NORTHWESTERN UNIVERSITY

The Experience and Expression of Emotion in Psychosis-risk

A DISSERTATION

SUBMITTED TO THE GRADUATE SCHOOL
IN FULFILLMENT OF REQUIREMENTS

for the degree of

DOCTOR OF PHILOSOPHY

Field of Psychology

By

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EVANSTON, ILLINOIS

June, 2022

Abstract

Emotional processing deficits are characteristic of psychotic disorders such as schizophrenia. However, impairments as such are not well understood prior to the onset of psychosis among individuals at clinical high-risk (CHR). This dissertation draws from prominent theories of emotion in schizophrenia and seeks to investigate the experience and expression of emotion in psychosis-risk.

The first aim of the study (Aim 1) addressed whether individuals with a CHR syndrome exhibit abnormalities in the experience of positive and negative emotions. To achieve this aim, an evocative film task was employed to a sample of CHR individuals and healthy controls. In this task, participants were asked to provide reports of the experience of positive (i.e., excitement, amusement) and negative (i.e., fear and sadness) emotions in response to video clips intended to evoke these noted emotions.

The second aim of the study (Aim 2) was to answer the question do CHR individuals show impairments in positive and negative facial expressions? To answer this question, a multi-modal approach was employed to a sample of CHR individuals and healthy controls in order to assess subtle and micro facial expressions. In this portion of the dissertation, segments of video recorded clinical interviews were submitted into an automated facial analysis software in order to investigate the presence of subtle facial expressions of emotions (e.g., joy, sadness), when compared to controls. To assess micro facial expressivity in this group, facial electromyography (EMG) (i.e., zygomaticus activity which is affiliated with smiling, and corrugator activity which is associated with negative displays of facial expressivity) was collected while individuals viewed the evocative film clip task discussed in Aim 1. Furthermore, facial expressivity during

the peak period of emotion (i.e., the period in which there were the highest levels of emotion shown in the video) was assessed in the video clips as well as changes in facial expressivity throughout.

An additional aim of this dissertation was to examine associations between facial expressivity and neurocircuitry in order to provide clues regarding the potential causes behind abnormalities in facial expressivity in those at CHR for psychosis (Aim 3). In this portion of the dissertation, associated neural circuitry was assessed using a region-of-interest to region-of-interest resting-state connectivity analysis approach. Here, the goal was to investigate whether emotion circuitry, motor circuitry, or both were related to facial expression abnormalities in this risk group. Aim 4 sought to examine if impairments in the experience (i.e., Aim 1 reports of experience of positive and negative emotions from the evocative film clip task) and expression of emotion (i.e., Aim 2, facial expressivity derived from automated facial analysis and facial electromyography) were associated with social functioning impairments and risk for ultimate conversion to psychosis based off of risk calculator scores.

The findings from this dissertation revealed that, when compared to controls, those with a CHR syndrome reported lower levels of excitement in response to the film clip intended to evoke excitement (Aim 1). Furthermore, it was found that those with a CHR syndrome showed alterations in subtle facial expressions during a neutral segment of a clinical interview including blunted joy, but also increased anger expressions derived from automated facial analysis (Aim 2). Similarly, when viewing a film clip intended to evoke excitement, those with a CHR syndrome displayed reduced zygomaticus facial activity. Relatedly, blunting was particularly pronounced during the peak period of the excitement clip (i.e., the most evocative section), but not prior to this peak. Furthermore the CHR group showed less changes in facial expressivity

during the excitement film clip as well. In terms of associated neural circuitry (Aim 3), it was found that greater connectivity patterns between motor-motor regions and motor-emotion regions were associated with blunted joy facial expressions in those with a CHR syndrome and this may be particularly the case in those with higher levels of facial expressivity. Finally, alterations in facial expressions derived from automated facial analysis in particular were related to both social functioning impairments and likelihood of conversion (based off of risk calculated scores), while less variability in zygomaticus facial activity when viewing the excitement clip during the evocative film clip task was related to higher risk scores.

Taken together, these data provide novel insights into the experience and expression of emotion in those with a CHR syndrome. Findings from this dissertation provide a foundation for further research that is needed in this area. Additionally, results inform early identification efforts, biobehavioral risk markers of psychosis, and shed light on targets for treatments and prevention strategies.

Acknowledgements

There are several people I would like to acknowledge that have been instrumental in my academic journey. To begin with, I would like to thank Dr. Vijay Mittal, who has mentored me for the last decade. I appreciate the commitment and dedication Dr. Mittal has shown to mentorship, providing endless opportunities and support throughout these ten years of training. With Dr. Mittal's mentorship, I begin my next chapter of my academic career with tools, skills, and knowledge that I believe will take me very far.

I would also like to thank my committee members Dr. Claudia Haase and Dr. Richard Zinbarg. Dr. Haase has been instrumental in my training in emotion. I have so much gratitude for all the patience and guidance along the way as I learned to integrate emotion theories and concepts into my work with those at risk for psychosis. Dr. Zinbarg has been incredibly supportive throughout these past six years of graduate training as well. Dr. Zinbarg's feedback and insights have been invaluable. I am also grateful for his leadership in our academic community – it is truly inspiring. Additionally, I have immense gratitude for the Northwestern University Psychology Department as a whole and appreciate all the additional training and efforts by Dr. Jennifer Tackett, Dr. Robin Nusslock, Dr. Michael Bailey, Dr. Bill Revelle, and Dr. Dan McAdams. This department is a remarkable training environment and it has been an honor to be a part of it.

I would also like to acknowledge my labmates and friends. First, Ivanka Ristanovic, Teresa Vargas, and Juston Osborne (and many more at ADAPT!) – I am unable to put into words how lucky I feel to have been on this graduate school journey with you. I want to especially thank Katherine Damme for her patience and guidance in the imaging portion of the dissertation and other collaborations. Additionally, I would also like to thank my Northwestern peers and

friends – Hollen Reischer, Henry Raffles Cowan, James Glazer, Natalie Gallagher, Amanda Kramer, and Kat Herzhoff – my time at Northwestern University would have not been the same without you. I would also like to express my gratitude for Jordyn Ricard, who was an incredibly committed research assistant involved in many of my research projects.

Additionally, I am grateful to have had the opportunity to cross paths with ADAPT lab members, collaborators, and friends from my time at the University of Colorado Boulder - Andrea Pelletier-Baldelli, Emily Carol, Derek Dean, Jessica Bernard, and Joseph Orr. Importantly, I would also like to acknowledge two previous graduate students at University of Colorado Boulder that introduced me to the study of psychology during my undergraduate training – Dr. Roselinde Kaiser Henderson and Dr. Blair Kleiber.

Finally, I want to express my deepest gratitude and acknowledgements to the many families and individuals that participated in these research projects whose lives have been impacted and touched by mental health challenges.

Preface

This dissertation is organized into eight chapters. The first chapter, Chapter I, is an introduction that provides background on symptoms of schizophrenia, characterizes clinical high-risk (CHR) for psychosis, and describes emotion and negative symptoms, integrating a brief review of possible mechanisms at play. In Chapter II, I describe the 4 aims and hypotheses for the study. Chapter III reviews materials and methods used to address each aim. Chapter IV describes data analyses employed to achieve each aim. Chapter V reviews the results of this dissertation while Chapter VI focuses on possible theories and interpretations of findings, integrates findings in the context of a broader model, and reviews clinical implications. Chapter VII reviews limitations and future directions while the last chapter, Chapter VIII is a brief conclusion.

Dedication

To my family

To my parents, who taught me to keep going no matter what challenge or adversity may arise. To my sister, who has been the constant in my life and always believes in me. To my husband, who supported me every step of the way in this journey and always reminds me of the bigger picture.

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CHAPTER I.

INTRODUCTON

Overview

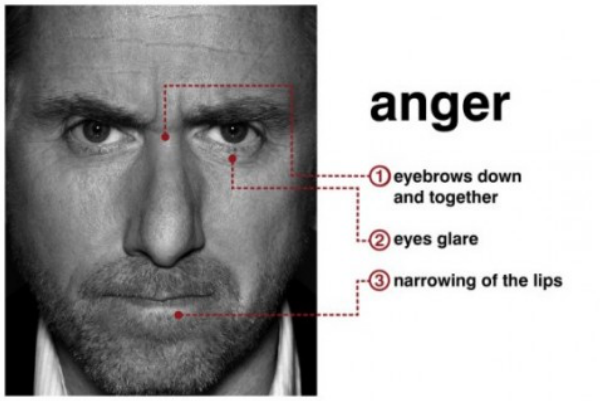
Emotion related impairments have long been held to play a central role in reflecting the etiology of psychosis and deficits are observed across the psychosis spectrum (Andreasen, 1997; Blanchard et al., 1998a; Kring et al., 1993; Kring & Bachorowski, 1999; Kring & Elis, 2013a; Zec, 1995). Eugen Bleuler, a Swiss psychiatrist known for his contributions to our understanding of mental disorders, introduced the term *schizophrenia* in the beginning of the 20th century. In his works, Bleuler suggested that affectivity in particular may be a core feature of the conceptions of the disorder, reporting that those with schizophrenia are "disturbed in a number of ways, but above all in the breakdown of emotion" (Bleuler, 1950). As Bleuler hinted, emotion related deficits occur across several domains, and are particularly represented in negative symptoms. Negative symptoms are characteristic symptoms of schizophrenia and are considered reductions in normal functions such as emotions, behaviors, and motivation (e.g., anhedonia, avolition) (Andreasen, 1997; Burbridge & Barch, 2007; Carpenter & Kirkpatrick, 1988; Fervaha et al., 2014a; Foussias et al., 2014; Kring & Elis, 2013a).

To date, behavioral studies indicate that individuals diagnosed with schizophrenia can exhibit a negative symptom called *anhedonia* (i.e., lack of capacity for pleasure) (Blanchard et al., 1998a; Burbridge & Barch, 2007; Foussias et al., 2014; Millan et al., 2014) reflected through decreased positive emotional experience ratings on a range of self-report measures and clinical interviews (Cohen & Minor, 2010a). However, the notion of positive emotional experiences is

complex especially given evidence from laboratory studies using evocative film clips (e.g., film clips intended to evoke specific emotions such as happiness, sadness etc). These studies find patients, on average, do not report reduced levels of positive emotional experiences as once expected, but instead report ratings similar to controls (Kring & Bachorowski, 1999; Kring & Elis, 2013a). Additionally, while accumulating evidence has found inconsistencies in reports of the experience of positive emotions, there are more consistent findings suggesting patients report increased negative emotions (e.g., fear, sadness) (Cohen & Minor, 2010a; Trémeau, 2006). Importantly, alterations in experience ratings have been found to relate to impairments in functioning (Cohen et al., 2013; Hooker & Park, 2002; Kee et al., 2003; Pogue-Geile & Harrow, 1985)

While phenomenology is one component of emotion research, there is also a critical, related construct that is not well-understood. Along these lines, one question that is often raised in the study of emotion in schizophrenia is if an individual is reporting, for example, increased levels of anger, is the report of emotional experience in line with what we would expect to see on the face? In this scenario, one would expect facial muscle movements in line with what is depicted in Figure 1 (e.g., eyebrows down and together). The study of facial expressions, independent of and in conjunction with phenomenology has been of interest across several disciplines since the development of the Facial Action Coding System by Paul Ekman and Wallace Friesen (Ekman & Friesen, 1978).

Figure 1. An example of facial expression movements in response to anger



Facial expressions of emotions have high social visibility (Hager & Ekman, 1979). They play a central role in signaling emotional states to others (Darwin & Prodger, 1998; Schmidt & Cohen, 2001) and in creating and maintaining social relationships (Keltner & Kring, 1998), reinforce desired behaviors (Martin et al., 1997), and help to cope with stress (Bonanno & Keltner, 1997). For example, happy facial expressions (e.g., upturned mouth; crowfeet around the eyes) can communicate enjoyment (Ekman & Friesen, 1982). In individuals with schizophrenia, evidence suggest these groups can exhibit *blunted* facial expressivity, another negative symptom characteristic of this group. However, findings vary depending on the type of methodology employed to assess *subtle* (e.g., more observable) and *micro* (not as commonly observable, fleeting) facial expressions. For example, there is evidence from clinical interviews in which assessors assign ratings reflecting the degree in which an individual displays facial expression blunting during the assessment; from this work, it is established that these individuals, on average, do exhibit blunted facial expression during clinical interviews (Kirkpatrick et al., 2006; Mandal et al., 1998). However, with the development of sensitive and precise tools such as automated facial analysis tools and facial electromyography (EMG) (Dimberg, 1982), there are studies that have found individuals with schizophrenia do exhibit expressions in line with the valence of stimuli (e.g., positive, negative), which may be undetected with the human eye (Kring & Elis, 2013a). Additionally, similar to ratings of the experience of emotion, facial expression deficits have been found to relate to impairments in functioning (Cohen et al., 2013; Hooker & Park, 2002; Kee et al., 2003; Pogue-Geile & Harrow, 1985).

In summary, our understanding of these noted impairments (i.e., alterations in the experience and expression of emotion) in schizophrenia is more established with the increasing evidence suggesting alterations in reports of the experience and expression of emotions

evidenced by clinical interviews, automated tools, and facial EMG (Foussias et al., 2014; Gur et al., 2006; Kring & Elis, 2013a; Messinger et al., 2011; Strauss et al., 2014). However, our understanding of the experience and the expression of emotion *prior* to the onset of psychotic disorders is not well-understood. This is a concern given that schizophrenia is a disorder characterized by several impairing symptoms that interfere with an individual's everyday functions (Addington et al., 2001; Andreasen, 1997; Carpenter & Kirkpatrick, 1988; Mackay & Crow, 1980). If individuals at risk for psychosis are accurately detected and identified, it may be possible to slow down or perhaps prevent the onset of psychotic disorders as well as aspects of functioning that take a "hit" once psychosis emerges. Additionally, understanding symptoms and behaviors prior to the onset of psychosis may be an opportunity to establish and implement treatments to those considered at risk before individuals experience rapid cognitive decline and motivational issues. In the last two decades, there is overarching consensus that there exists a group of individuals considered at clinical high-risk (CHR) for developing a psychotic disorder (Fusar-Poli et al., 2012). These individuals exhibit signs and symptoms such as seeing shadows in their corner of one's eye or believing in conspiracy theories that begin to be impactful on daily life and distressing.

Of relevance, this risk group is also suggested to exhibit a range of emotion related abnormalities (Fervaha et al., 2014a; Foussias et al., 2014; Millan et al., 2014; Remington et al., 2016). For example, there is evidence from laboratory paradigms of reduced levels of reports of positive and negative emotion in this group from one study in which participants rated emotional experiences in response to emotionally valence photos presented (Gruber et al., 2018). Furthermore, there is evidence of reduced emotional experiences from studies using clinical interviews which generally ask about broad emotional experience such as *"Do your emotions*

feel less strong than usual?" (Miller et al., 1993) and self-report measures (e.g., Yee et al., 2019). In terms of facial expressions, research suggests this group exhibits *blunted* facial expressions, but these data are largely drawn from studies utilizing clinical interviews in which raters assign a single rating to represent the degree in which there are facial abnormalities present (PINS citation; Pelletier-Baldelli, 2015; Strauss et al., 2020). However, with the current approaches for measuring the experience and expression of emotion, there are pieces of information we are missing.

First, it is not clear whether individuals with a CHR syndrome exhibit alterations in emotional experience ratings in response to evocative film clip tasks. This is important information because it can shed light as to whether abnormalities vary as a function of context. For example, do we see reduction in positive emotion ratings in response to a highly positive film clip but not in response to a film clip depicting a negative event? The answer to this question has the ability to provide insights regarding this risk group broadly and also inform models of emotion related impairments and interventions.

Second, as mentioned, no study has assessed *specific* facial expressions in the CHR group, but instead have focused on single observer ratings from clinical interviews. This is a gap in the literature and addressing this that has the ability to inform our understanding of facial expressivity (i.e., whether these abnormalities occur earlier in the disease process), reflecting vulnerability and mechanistic targets relevant for identification, biomarker, and treatment development. Along these lines, it is unclear whether individuals in this group exhibit subtle and micro facial abnormalities across different emotional expressions.

Third, it is unclear what the associated neural mechanisms are of facial expression abnormalities in CHR groups. To date, much of the research has focused on emotion related

brain regions (e.g., amygdala, emotion related regions in the cerebellum) (Aleman & Kahn, 2005; Anticevic et al., 2012; Baumann & Mattingley, 2012; Ferrucci et al., 2012; King et al., 2019). Furthermore, when considering the action of facial expressions, it is clear that producing facial expressions is in fact a motor movement (Gothard, 2014; Morecraft et al., 2004). With this, it not known whether emotion related pathways, motor pathways, or both are implicated in facial expression production abnormalities prior to the onset of psychosis. This is particularly critical given that aberrant emotion and motor abnormalities are characteristic of those with a CHR syndrome (Damme et al., 2020; Dean et al., 2018b; Mittal et al., 2007, 2008; Schiffman, 2017; Walker et al., 1999).

Fourth, if there are abnormalities in reports of positive and negative experience and expressions of emotion, it is not clear how these abnormalities relate to likelihood of conversion to psychosis (based off of risk calculator scores) (Cannon et al., 2016; Zhang et al., 2018) and social functioning (Cornblatt et al., 2007). Given that negative symptoms such as anhedonia and blunted affect are suggested to occur even prior to the emergence of positive symptoms and are predictive of converting to a psychotic disorder (Piskulic et al., 2015), it is valuable to understand relationships between experience and expression abnormalities and conversion risk scores. Additionally, emotional experiences and expressions are critical for effective social interactions. For example, the ability to experience positive emotions in social situations can help to foster a sense of belonging and community. Relatedly, displaying facial expressions that are positive and negative are adaptive (e.g., showing positive emotions can help an individual develop and maintain social relationships; negative emotions can signal danger) (Ekman, 1993).

As such, the current study seeks to assess emotional experience and expression in a CHR sample when compared to controls. Specifically, this study aims to answer four fundamental

questions: When compared to controls, (1) do CHR individuals exhibit abnormalities in the experience of positive and negative emotions? (2) Do CHR samples show impairments in positive and negative facial expressions? (3) Can looking at associated neurocircuitry provide any clues about the possible causes behind abnormalities in facial expressivity? (4) Are impairments in the experience and expression of emotion predictive of functioning and risk for ultimate conversion to psychosis?

In this current introduction section, I begin by discussing schizophrenia and CHR groups, highlighting how these disorders are phenotypically expressed. Then, in order to provide context of study aims and hypotheses, I review negative symptoms, focusing on anhedonia and blunted facial affect in particular, integrating findings from the emotion literature as well. Furthermore, I review neuroimaging studies in the study of facial expressivity drawing from studies conducted in healthy populations. After, the rest of the dissertation will be on study aims, hypotheses, results, discussion of results, limitations, and future directions.

Schizophrenia

Schizophrenia is characterized by a wide array of symptoms, often with distinct presentations (Carpenter & Kirkpatrick, 1988). Central symptoms of the disorder include positive symptoms which are symptoms "added on" to one's normative experience such as auditory and visual hallucinations. According to the DSM-5, a diagnosis of schizophrenia involves two or more of the following positive symptoms: (1) delusions (e.g., being spied on), and/or hallucinations (e.g., seeing fully formed figures). Other symptoms include negative symptoms which are reductions in normal functions (e.g., reduced experience of positive emotion, reductions in facial expressivity), as well as disorganized speech and disorganized or catatonic behavior. First (e.g., haloperidol) and second (e.g., risperidone) generation

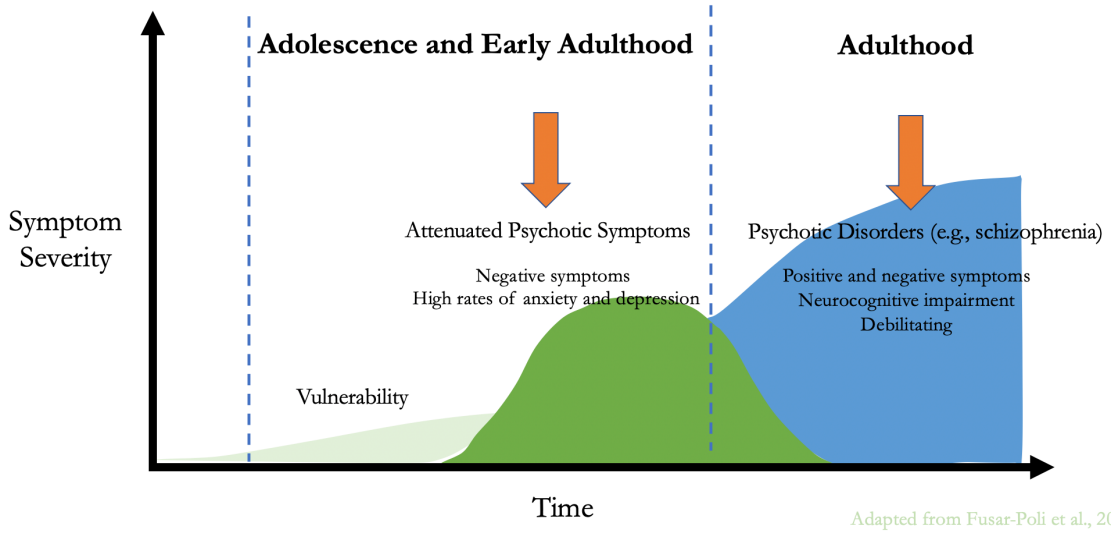
antipsychotic medications have been found to alleviate positive symptoms, but these tend to be ineffective for improving negative symptoms (Lally & MacCabe, 2015). Individuals with schizophrenia also take a "hit" in neurocognitive function, exhibiting abnormalities in processes such as working memory and executive function (Addington et al., 1991; Green et al., 2000; Green & Nuechterlein, 1999).

Clinical High-Risk

Individuals with clinical high-risk syndrome can exhibit recently emerging attenuated positive symptoms (e.g., seeing shadows, hearing whispers, reading into coincidences, feeling confused about what is real) that are frequent, distressing, and beginning to interfere with school/work and social relationships. Many of these individuals make contact with treatment providers as a result of experiences with comorbid diagnoses including anxiety and depression (Addington et al., 2017b, 2020; McAusland et al., 2017a). Additionally, negative symptoms (e.g., reductions in emotion, motivated behaviors, affect, social isolation) tend to emerge prior to positive symptoms and may be a central reason for an individual with risk symptoms to seek treatment to begin with. Other symptoms endorsed by this group can include impairments in neurocognitive functions, motor abnormalities, and poor social and role functioning. Individuals identified as falling in this risk category can exhibit different presentations (also termed states or syndromes) including predominantly (1) attenuated positive symptom syndrome (APSS) (e.g., seeing shadows, hearing whispers) (attenuated positive symptom syndrome), (2) very brief and intermittent psychotic experiences (Brief Intermittent Psychotic Syndrome; BIPS), and/or (3) genetic risk or schizotypal personality disorder with a decrease in functioning (Genetic Risk and Deterioration Syndrome; GRD). These subgroups exhibit different rates of transition to formal psychosis, with evidence pointing towards substantial numbers, particularly in those with brief

and intermittent symptoms (Fusar-Poli et al., 2016a). Though, a majority of the research focuses on the first category, APSS. See Figure 2 for a visual conceptualization of the etiology of psychosis.

Figure 2. A visual conceptualization of the pathogenesis of psychosis



This figure adapted from Fusar-Poli et al., 2013 depicts different stages of the psychosis spectrum. On the x-axis is time and on the y-axis is symptom severity. Individuals that exhibit attenuated psychotic symptoms often fall in the adolescent and early adulthood period and are considered meeting for a clinical high-risk (CHR) syndrome. Those with a CHR syndrome endorse positive symptoms such as hearing whispers, unusual thoughts, negative symptoms, and often endorse comorbid diagnoses. These individuals are a heightened risk of developing a psychotic disorder within a short window of time. Psychotic disorders are debilitating and typically emerge in adulthood and are characterized by positive and negative symptoms as well as neurocognitive deficits.

Diagnosing a CHR syndrome is multifaceted and there are different ways this is being done across the world (Addington and Heinssen, 2012). For example, McGorry and Yung, a group in Melbourne, Australia, pioneered the psychosis-risk construct and developed a measure called The Comprehensive Assessment of At-Risk Mental States (CAARMS) (Yung et al., 1996). In the United States, McGlashan and Miller developed the Structured Interview of Prodromal Symptoms (McGlashan et al., 2001; Miller, McGlashan, Rosen, Cadenhead, Ventura, et al., 2003) (SIPS; now called the Structured Interview for Psychosis-Risk Syndromes). The

goal of these interviews is to measure key symptoms of psychosis-risk syndromes (e.g., clinical high-risk) such as attenuated positive symptoms (e.g., hearing whispers, seeing shadows), negative symptoms (e.g., anhedonia, reductions in the experience and expression of emotion), disorganization (e.g., odd, eccentric, and/or bizarre behavior), and general symptoms (e.g., sleep disturbances). Another related yet alternative way of identifying and assessing risk syndromes stems from the basic symptom literature, which focus on individuals endorsing subclinical experiences such as difficulties with cognition and perceptions (Schultze-Lutter, 2009). With the increasing research on in this area, the DSM-5 introduced the diagnosis of Attenuated Psychosis Syndrome (DSM-5-APS) (American Psychiatric Association, 2013) in the Conditions for Further Study section of the manual (see criteria in Figure 3).

Figure 3. DSM-5 criteria of Attenuated Psychosis Syndrome

Proposed Criteria

- A. At least one of the following symptoms is present in attenuated form, with relatively intact reality testing, and is of sufficient severity or frequency to warrant clinical attention.
    1. Delusions.
    2. Hallucinations.
    3. Disorganized speech.
- B. Symptom(s) must have been present at least once per week for the past month.
- C. Symptom(s) must have begun or worsen in the past year.
- D. Symptom(s) is sufficiently distressing and disabling to the individual to warrant clinical attention.
- E. Symptom(s) is not better explained by another mental disorder, including a depressive or bipolar disorder with psychotic features, and is not attributable to the physiological effects of a substance or another medical condition.
- F. Criteria for any psychotic disorder have never been met.

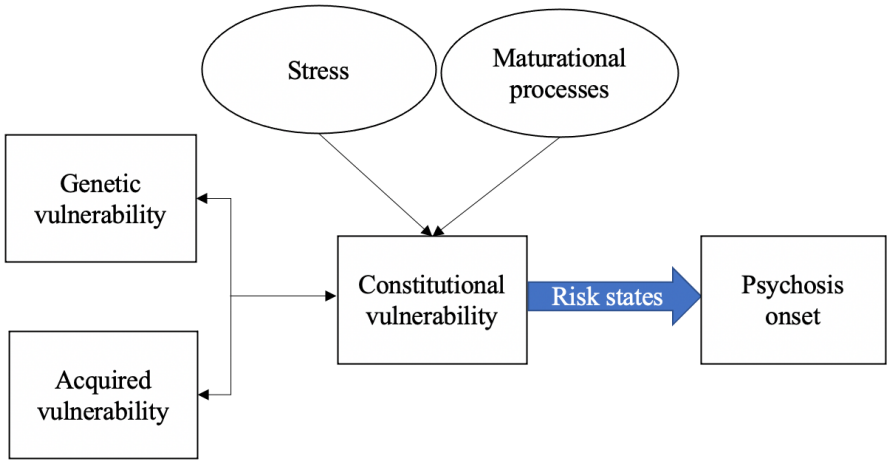
Although there is research on methodologies for diagnosing CHR syndromes, there is still important work to be done including considerations of race, ethnicity, and culture in the presentation of symptoms worldwide (Schiffman et al., 2019).

From an equifinality model framework, there are many different pathways to psychotic disorders, and this is evidenced in the trajectory of many with a CHR syndrome (Addington et al., 2011b; Fusar-Poli et al., 2012a, 2016a). For example, some individuals with CHR syndromes experience symptoms that rapidly progress (and this group tends to be at heightened risk for conversion), some exhibit symptoms that remain the same (possibly reflective of a trait-like vulnerability), and others may improve and even remit. Broadly, individuals in this group show early symptoms suggestive of risk for converting to a psychotic disorder, yet they have not fully transitioned. This risk window offers a unique opportunity for researchers in that we can identify vulnerability markers, such as emotion related deficits, contributing to the onset of psychosis individuals, which in turn can improve early detection, prevention efforts, and treatment development. While much of the focus in risk research to date has been on investigating questions surrounding risk markers contributing to the onset of both schizophrenia-spectrum disorders and mood disorders (e.g., depression, bipolar disorder) with psychosis, there are not the only outcomes. For example, two individuals can have the same genetic and environmental susceptibilities but have very different psychopathological outcomes. Some individuals may be at heightened risk for psychosis (e.g., hearing whispers, reading into coincidences), but develop other types of psychological disorders over time such as anxiety or depression. Others may show reductions in symptoms over time or even remit.

Diathesis-Stress Model

As adolescence is a time of dynamic change, increasing responsibilities, and escalating challenges and conflicts, individuals that fall within this period are susceptible to influences such as environmental stressors that may alter their developmental course. It is therefore not surprising that a range of psychopathology emerges during this critical developmental period. In the case of psychosis, a diathesis-stress conceptualization (see simplified version in Figure 4) posits that an early vulnerability (carried from birth through the confluence of heritable contributions such as psychosis-risk genes and prenatal insults) (Brown et al., 2000; Buka et al., 2008; Davies et al., 2020; Mittal et al., 2008) accumulates through childhood adversities and stressors (Fusar-Poli et al., 2013; Mayo et al., 2017), and later interacts with stressors and neurodevelopmental factors in adolescence. Through epigenetic changes, as well as gene/susceptibility by environment by normative development interactions, this can also lead to pathological development, which further predisposes youth to risk (Addington, Farris, et al., 2019; Corcoran et al., 2003; Gibson et al., 2016; Ristanovic et al., 2020; Vargas et al., 2020; Walker et al., 2008; Yee, Strauss, et al., 2019a). During these changes, new risk factors and signs emerge. Eventually, subsequent brain, social, and emotional dysfunctions cascade, driving the onset of psychosis.

Figure 4. A simplified diathesis stress model.

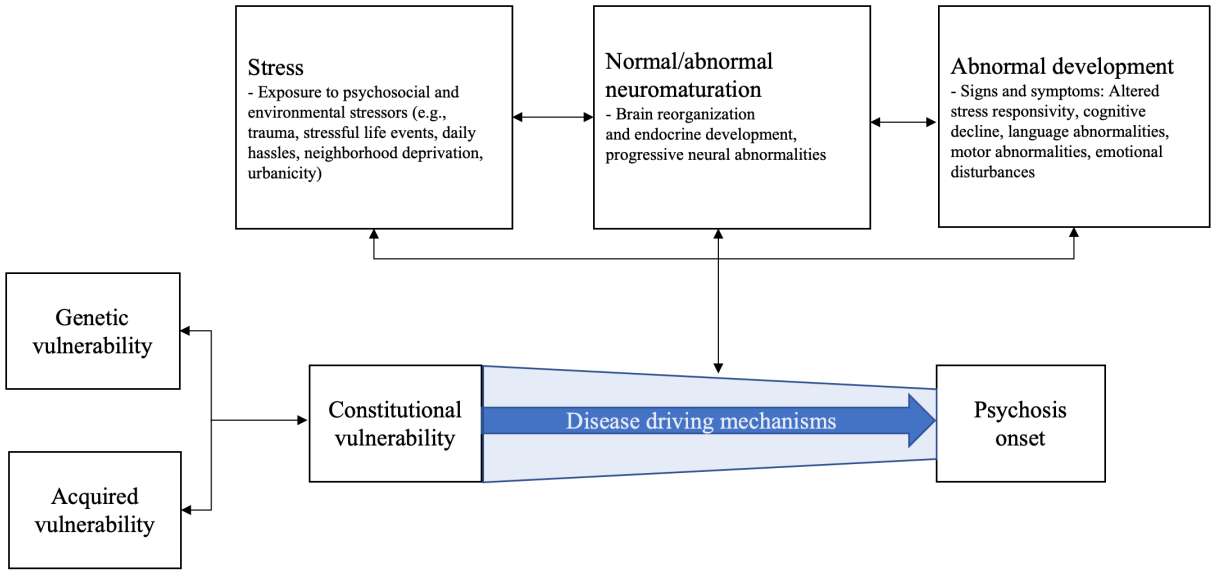


Note. In the diathesis-stress framework, psychosis onset is preceded by an early vulnerability that is carried from birth through the confluence of genetic vulnerability (e.g., familial risk), and acquired vulnerability (e.g., prenatal insults). This interaction is accumulated into a constitutional vulnerability, the diathesis, that interacts with additional stressors (e.g., childhood adversity) and maturational processes (e.g., neural development). Together, these interactions contribute to the emergence of risk states and ultimate onset of a first-episode of psychosis. It is important to note that individuals can carry an early vulnerability that is not expressed later in development due to several reasons such as protective factors and resiliency. Taken from Gupta et al., 2021

As discussed, several processes shape normative development during the adolescent period, allowing young individuals to develop skills and tools to navigate the world and gain a sense of autonomy. From earlier diathesis-stress models as shown in Figure 4, genetic vulnerability can interact with environmental influences that can alter an individual's developmental course (Corcoran et al., 2003; Mittal et al., 2008; Tessner et al., 2011). However, there is a need to consider and understand signs, symptoms, and risk factors that reflect pathogenesis during this period which holds significant potential for refining early identification and prediction efforts, as well as for informing novel targeted treatment development for psychosis-risk groups (see Figure 5 for more modern conceptualizations of the diathesis stress model that incorporate abnormal development; this highlights the need to better characterize signs and symptoms in those at risk for psychosis). In this dissertation, the focus is on emotion

related signs and symptoms, with an eye towards better understanding the nature of emotion related disturbances prior to the onset of psychosis.

Figure 5. A modern conceptualization of the diathesis stress model



Note. In this modern conceptualization of the diathesis stress model, constitutional vulnerabilities can carry across adolescence, interacting with exposure to psychosocial and environmental stressors as well as abnormal neuromaturational processes. What is needed is an understanding of signs and symptoms characteristic of abnormal development in this group. Critically, the nature of interactions between factors and the expression of signs and symptoms is heterogeneous. Adapted from Gupta et al., 2021

Emotion and Negative Symptoms in Schizophrenia

Emotions are suggested to include multiple components comprising of experience, expression, and physiology (Kring & Elis, 2013). Emotions and the way we manage and change them provide important functions in everyday life (Keltner & Gross, 1999a; Levenson, 1999a). Most emotions have developed from an evolutionary history and have allowed the ability to engage with one's environment and communities in efforts to combat or adapt to events and challenges that may arise (Keltner & Gross, 1999a; Kring & Bachorowski, 1999). Furthermore, emotions can coordinate with our systems such as response activation systems. For example, when in the presence of a dangerous situation, the emotion of fear can signal to ourselves and others to take action such as fighting or running away (Keltner & Gross, 1999b; Zinbarg, 1998).

Deficits in emotional processing can contribute to impairments in social functioning and challenges pursuing and persisting in goal directed behaviors (e.g., motivational and reward-based influences) (Blanchard et al., 1998b; Hooker & Park, 2002; Kee et al., 2003). Emotion related deficits are largely tied to negative symptoms in schizophrenia and CHR groups. As mentioned, interest in negative symptoms and emotional processing in schizophrenia have been prevalent since writings from historical figures such as Bleuler (Andreasen, 1997; Zec, 1995). In the 1950s, there was a focus primarily on positive symptoms, given the introduction of the antipsychotic medication (Andreasen, 1982; Foussias et al., 2014). However, it was not until the mid 1970s - early 1980s, that a broader understanding of the negative symptom dimension emerged and have continued on throughout the years (Fervaha et al., 2014a; Foussias et al., 2014; Millan et al., 2014).

Recent negative symptom conceptualizations draw from the National Institute of Mental Health consensus conference on *Negative Symptoms in Schizophrenia* that took place in 2005 (Kirkpatrick et al., 2006). Stemming from these discussions, negative symptoms are now understood as anhedonia (lack of capacity for pleasure), asociality (lack of pleasure in social interactions), avolition (lack of motivation towards goal-directed behaviors), alogia (reduced quantity of speech), and blunted affect (reductions in facial expressions, body movements, and voice tone). Early factor analytic studies in schizophrenia have supported a two-factor negative symptom structure consisting of diminished expression and motivation and pleasure (Blanchard & Cohen, 2006; Horan et al., 2011; Strauss et al., 2012). However, more recent confirmatory factor analytic and network analysis studies that directly tested the latent structure of the construct suggest that the two-factor model offers a poor fit; rather a five-factor model, corresponding to the five domains identified in the NIMH consensus conference on negative

symptoms (Kirkpatrick et al., 2006) provides an excellent fit. These findings have been replicated across contemporary negative symptom scales (e.g., Brief Negative Symptom Scale), across multiple cultures, using multiple mathematical techniques (confirmatory factor analysis, network analysis), and across chronic, first episode, CHR phases of illness (Azis et al., 2019; Chang et al., in press; Mucci et al., 2019). Furthermore, this evidence provides a basis for not only investigating negative symptoms as a whole but looking at individual constructs (i.e., anhedonia, blunted facial affect)

Emotion and Negative Symptoms in CHR groups

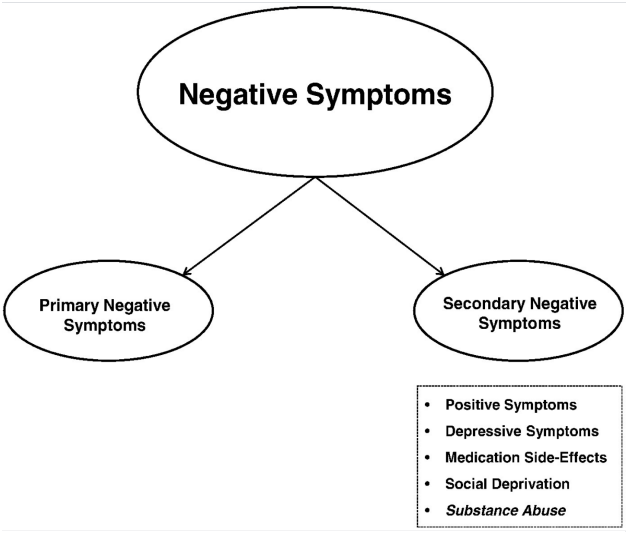
Emotion deficits in studies utilizing CHR samples are just beginning to be understood (Azis et al., 2019; Cannon et al., 2008; Fusar-Poli et al., 2012b; Gupta et al., 2019; Pelletier-Baldelli et al., 2017). Much of the work investigating negative symptoms stem from studies using the SIPS (Miller, McGlashan, Rosen, Cadenhead, Cannon, et al., 2003) that define negative symptoms as social anhedonia, avolition, reductions in the experience of emotion, reductions in the expression of emotions and self, ideational richness, and deficits in occupational functioning. Negative symptoms in this group have been found to relate to impairments in social functioning and may emerge prior to positive symptoms, and contribute to psychosis onset (Piskulic et al., 2012). Furthermore, work has sought to refine negative symptom measurement tools, with potential avenues including teasing apart what an individual is actually doing (e.g., behavior) vs. internal experience (e.g., how motivated were they got make time for hobbies?) given increasing evidence in schizophrenia suggesting differences between the two constructs (Barch & Dowd, 2010; Strauss, Visser, et al., 2017; Strauss et al., 2018). Specifically, findings in this area suggest, in schizophrenia, there may actually be the *capacity* for experiencing pleasure (e.g., reporting feeling the same amount of pleasure when engaging in

activities as those without schizophrenia), but individuals may vary in the frequency in which they are engaging in pleasurable activities (Berenbaum & Oltmanns, 2012; Kaiser et al., 2017; Oorschot et al., 2013; Strauss, Whearty, et al., 2017). However, this is not well-understood among CHR individuals.

Secondary Sources of Negative Symptoms in Schizophrenia

Negative symptoms can result from primary (i.e., idiopathic) or secondary (i.e., due to depression, anxiety, hallucinations, delusions, disorganization, medication effects) sources of influences (Carpenter & Kirkpatrick, 1988; Kirschner et al., 2017; Peralta et al., 2000) and this notion can complicate conceptualization and treatment. Essentially, two individuals can have the exact same score on a negative symptom rating scale, but due to very different reasons (i.e., equifinality). Historically, primary and secondary negative symptoms have been distinguished in schizophrenia clinically using the Schedule for the Deficit Syndrome (Kirkpatrick et al., 1989), which requires clinicians to judge whether negative symptoms are of clinically significant severity and whether they likely result from secondary sources (e.g., anxiety, depression, positive symptoms, extrapyramidal symptoms); in the absence of clear secondary sources deemed to drive negative symptoms, they are rated as primary. Cases where negative symptoms are considered of sufficient severity and to be driven by primary (rather than secondary) factors and to be persistent (rather than transient)

Figure 6. A visual representation of primary and secondary negative symptoms taken



Note. Taken from Kirschner et al., 2017

are deemed to meet criteria for the "deficit syndrome" (a putative schizophrenia subgroup with distinct etiological factors). Of note, secondary effects of positive symptoms on negative symptoms have been identified in the literature (Carpenter & Kirkpatrick, 1988; Kirschner et al., 2017). For example, an individual may experience persecutory thoughts, and withdraw from social interactions (Carpenter & Kirkpatrick, 1988). Other work has found influences of extrapyramidal side effects on blunted affect (Kelley, 1999) and relationships between negative symptoms and depression and anxiety (Kulhara et al., 1989; Lysaker & Salyers, 2007). Additionally, disorganization is associated with blunted affect (e.g., facial and vocal flattening due to limited available cognitive resources) (Cohen et al., 2014) as well as motivational impairments (Strauss et al., 2013).

Secondary Sources of Negative Symptoms in CHR

The extent to which negative symptoms are driven by secondary sources in this group is unclear. Currently, there are measures such as the Negative Symptom Inventory-Psychosis Risk (NSI-PR) (Pelletier-Baldelli et al., 2017; Strauss et al., 2020) that are geared towards isolating primary negative symptoms. However, distinct categorizations (i.e., non-deficit and deficit syndrome) observed in schizophrenia are not yet possible to implement in CHR groups. One might expect secondary sources to account for some of the proportion of variance in negative symptom scores given elevated rates of comorbid mood and disorders under the umbrella of anxieties, high rates of psychotropic medications prescription, and the presence of attenuated positive and disorganized symptoms (Addington et al., 2017b; Fusar-Poli et al., 2014). For example, one study reported from a sample of 245 help-seeking CHR individuals, half endorsed life-time depression. Furthermore, 34% met criteria for a current depressive disorder and 39% endorsed a current anxiety disorder. (Salokangas et al., 2012). Similarly, another study by

Addington and colleagues (2017) also reported in a sample of 744 CHR, the most common diagnosis was depression. Moreover, in recent work, it was found that anxiety (assessed with the Beck Anxiety Inventory and State-Trait Anxiety Inventory) contributed a large portion of variance in explaining blunted affect (reductions in gesturing, voice prosody, and facial expressivity) in a sample of individuals with a CHR syndrome (Gupta et al., 2021). Thus, it is imperative to consider possible secondary source (e.g., anxiety, depression) when investigating negative symptoms, particularly anhedonia and blunted facial expressions, in CHR groups.

Alterations in Emotional Experience in Schizophrenia

As mentioned, one of the cardinal negative symptoms is anhedonia which is the inability to experience pleasant emotions (Strauss et al., 2018). Despite theories positing individuals with schizophrenia have a lack of the *capacity* for pleasure (in contrast to anhedonia in depression which is a *loss* of pleasure, motivation, and interest), research findings in this area remain inconsistent. For example, studies report in the moment experiences of positive emotions comparable to controls (although there is some evidence of increases) according to laboratory-based paradigms while other research drawing from self-report measures report reductions in positive emotional experiences. As such, there appears to be an "emotion paradox" in the field (Gold & Strauss, 2012). One explanation for this paradox may be due to differences in methodologies. For example, some studies reveal individuals report normative levels of positive emotion when interacting with various emotional stimuli in laboratory tasks asking about in the moment experiences (Cohen & Minor, 2010b; Kring & Elis, 2013a; Kring & Moran, 2008a). Similarly, experience sampling methods have also found that individuals report, on average, pleasant emotional experiences in line with those without the disorder (Cho et al., 2017; Oorschot et al., 2013; Painter & Kring, 2016). However, self-report measures involving

recollection of past, general, or future experiences of positive emotion are impaired in this group, with reports of reduced emotional experiences reflective of anhedonia (Strauss et al., 2014). Furthermore, there is some evidence that self-report measures capture not the experience of a positive event (as with laboratory paradigms) but instead, the *frequency* in which individuals with schizophrenia engage in pleasurable activities, as mentioned. In other words, perhaps reductions are observed in self-reported anhedonia measures due to the fact that they are asking about the frequency of pleasurable experiences rather than the actual experience itself (Pelizza et al., 2020). Please note in this dissertation, the focus is on "in the moment" experiences of emotion using a laboratory paradigm. Different methodological approaches in assessing emotional experience are discussed in this dissertation to provide sufficient background.

While many laboratory-based studies suggest those with schizophrenia report comparable levels of positive emotions to those without the disorder (Kring & Elis, 2013a), increasing evidence in schizophrenia assessing reports of negative experiences of emotions indicate that negative, rather than positive, experiences of emotions are central to the disorder (Gold & Strauss, 2012). In terms of the experience of negative emotions, there is evidence indicating increased reports of negative emotions from self-report measures and clinical interviews among individuals with schizophrenia (Blanchard et al., 1998b; Horan et al., 2008; Kring et al., 1993; Kring & Elis, 2013a). For example, when individuals are asked in general how much they feel a specific emotion (e.g., *In general, how sad do you feel?*), schizophrenia patients, on average, report more negation emotions (Blanchard et al., 1998b; Horan et al., 2008; Kring et al., 1993; Kring & Elis, 2013a). Similarly, meta-analytic evidence from naturalist studies reveal schizophrenia patients report higher levels of negative emotions as well (Cho et al., 2017). Additionally, there are findings indicating in response to laboratory tasks displaying unpleasant

emotional stimuli such as film clips, pictures, odors, and foods, patients with schizophrenia report negative emotions (Kring & Moran, 2008a). However, in some work, there is evidence of more negative emotion reports compared to controls in response to positive and neutral stimuli as well (Cohen & Minor, 2010b) (i.e., inconsistent with the valence, nontarget emotions). Along these lines, in one experimental study, a subgroup of participants with schizophrenia endorsed higher negative emotion reports in response to both positive and negative picture presentations compared to individuals without schizophrenia, and this group also reported poor functional outcomes (Strauss & Herbener, 2011). These data indicate there may be heterogeneity in the experience of emotions.

Alterations in Emotional Experience in CHR groups

Emotional experience in those with a CHR syndrome have been investigated broadly, with few studies investigating specific "target" (e.g., presenting an image intending to evoke joy and asking how much joy an individual feels) and "nontarget" (e.g., presenting an image intended to evoke joy and asking how much fear they feel) emotions. Findings from studies in this area report attenuated levels of positive emotions and increased negative emotions. For example, our group administered a commonly employed self-report measure called the Differential Emotions Scale to assess for positive emotions and found CHR individuals exhibited reductions in positive emotions but increased negative emotions when compared to controls (Yee et al., 2019). However, studies assessing the nature of positive emotions in a laboratory setting which may capture in the moment, naturalistic, day-to-day experiences are limited.

In one study (Yee et al., 2010), chronic and first-episode schizophrenia groups and CHR individuals viewed static images conveying threat, mutilation, illness, food, and nature. After each image, participants were asked what they felt when viewing each image. In this study, CHR

individuals exhibited less positive emotion to pleasant and neutral images, and less negative emotions to unpleasant images when compared to chronic and first-episode schizophrenia groups (Yee et al., 2010), displaying a pattern of reduced emotional reactivity. These findings are consistent with a more recent study conducted by Gruber and colleagues (2018) in which CHR individuals and controls were presented with a series of photos taken from the International Affective Picture System (IAPS) (Gruber et al., 2018a). Participants were instructed to indicate how positive and negative they felt while viewing each photo. Findings indicated, similar to Yee and colleagues (2010), the CHR group reported reduced positive emotion ratings to pleasant stimuli and decreased negative emotion ratings to unpleasant stimuli. These findings of reduced positive and negative emotional reactivity may be counterintuitive when considering evidence in schizophrenia suggesting largely normative levels of positive emotion reports in laboratory settings. However, Gruber and colleagues (2018) also showed that lower reports of positive emotional experience were associated with greater severity in depression and anxiety in this CHR sample. These findings are consistent with evidence suggest CHR groups tend to endorse high rates of comorbid diagnoses such as disorders of anxieties (e.g. generalized anxiety disorder, social anxiety) and depressive disorders (e.g., major depression) (Addington et al., 2017a; McAusland et al., 2017b). Relationships with comorbid diagnoses hint towards the possibility that emotional impairments may be a result of these disorders rather than inherent to the CHR syndrome, however more studies are needed to further probe this question.

Alterations in Facial Expressivity in Schizophrenia

In addition to anhedonia, another negative symptom is blunted affect. Blunted affect in schizophrenia refers to reductions in facial expressions, gesturing, and voice prosody (Foussias et al., 2014). One of the most prominent impairments of blunted affect in schizophrenia is *blunted*

facial expressivity which will be the main focus of this dissertation. Specifically, there is established evidence indicating that patients with schizophrenia exhibit blunting in facial expressivity (Kring et al., 1993; Kring & Bachorowski, 1999; Kring & Elis, 2013a) and this has been linked with functional outcome (Fervaha et al., 2014a). Furthermore, these findings are observed when using different types of methodologies (e.g., behavioral observation, automated approaches, electromyography) and across different settings such as laboratory and naturalistic contexts (Kring et al., 1993; Kring & Elis, 2013a; Kring & Moran, 2008a). An important component of the study of facial expressions, drawing from the study of emotion more broadly, is that emotional processes such as facial expressivity can vary depending on one's environment and the significance of a situation (i.e., functionalist perspective of emotion generation) (Keltner & Gross, 1999b; Keltner & Kring, n.d.; Levenson, 1999b). In other words, the context in which facial expressions occur matters. Using methods such as clinical interviews, which has been historically the standard approach for assessing facial expressivity, does not allow for the ability to retrieve information related to contextual questions (e.g., when an individual is viewing something joyful, are they also smiling?). Furthermore, clinical interviews often require time intensive training, may be subject to rater biases, do not capture a full range of specific facial expressions of emotions, and are difficult to capture in real-time. The need for sensitive and objective tools to assess for facial expressions (i.e., automated facial analysis, facial electromyography) can more comprehensively inform emotion related conceptualizations across psychological disorders.

Automated facial analysis. It is now possible to use automated, computerized analyses of facial expressivity to remove the ambiguity and subjectivity associated with findings obtained from clinical rating scales. Historically, as mentioned, a common approach to assess for facial

expressions involved coding videos using approaches such as FACS (Ekman & Friesen, 1978). The FACS coding technique uses Action Units (AUs) that correspond with muscle movements observed from images or videos; collections of action units formulate specific emotions such as joy or sadness. Together, FACS coding has been proven to be effective in assessing for blunting in facial expressivity in adults with schizophrenia (Gaebel & Wölwer, 2004; Trémeau et al., 2005). One of the strengths of video coding techniques is that facial expressions can be captured in naturalistic settings. Specialized software now makes it possible to automate the extensive features previously done manually by coding systems like FACS. Early approaches of automated facial expression analysis used static images to classify facial expressions into broad emotion categories such as joy, fear, and disgust (Pantic & Rothkrantz, 2004). One downfall of coding static images is the temporal content is lost. As such, more recent work has begun to utilize automated analysis of continuous video output which produces ratings of facial action units (Cohen, Morrison, & Callaway, 2013; Hamm, Kohler, Gur, & Verma, 2011). There is evidence for the efficacy of automated facial expression analysis in populations such as schizophrenia (Hamm, et al., 2011), individuals with schizotypal traits (Cohen, et al., 2013), autism (Owada et al., 2018), and Parkinson's disease (Bandini et al., 2017). It is possible that this approach is a sensitive measure than can provide additional, unbiased information.

Facial electromyography. Additionally, in schizophrenia, facial expressivity research advanced with the use of facial electromyography (EMG) to detect micro facial expressions, often unobservable. Facial EMG is also an objective and sensitive tool that can measure muscle activity through sensors placed on the face and are less influenced by rater and/or software related confounds. In an early, groundbreaking facial EMG study with healthy individuals (Dimberg et al., 1982), the sample showed more zygomaticus activity (i.e., smiling) when

individuals were presented with positive emotional stimuli and more corrugator activity (i.e., frowning) when given negative emotional stimuli. Since this study, facial EMG has been used to measure micro facial expressions across psychological disorders. For example, this methodology was central to findings suggesting those with schizophrenia display blunted facial expressions, but do in fact show micro facial expressions including contraction of the zygomaticus major in response to positive stimuli and corrugator supercilii in response to negative stimuli (Kring et al., 1999; Kring & Elis, 2013a). It is likely that these contractions that were observed in line with the valence of stimuli may not have been detected without sensitive tools such as facial EMG.

One critical benefit of facial EMG is that data can be collected in real-time. With this, there are possibilities to answer questions regarding the temporal course of facial expressivity. For example, one question of interest might be what portion of time is there the most blunting? Investigating the temporal course of facial expressivity is a growing area. For example, one study in schizophrenia found reduced zygomaticus and corrugator activity during the first 500 ms of presenting a photo that was positively valenced in a study of facial mimicry (Varcin et al., 2010). Additionally, there is evidence using startle paradigms suggesting difficulties maintaining emotional experience states throughout time (Curtis et al., 1999; Kring et al., 2011; Volz et al., 2003). Together, assessing timing of facial expressions may provide novel insights and more precisely inform research on facial expressions and psychopathology.

Furthermore, using facial EMG methods provides the ability to look at changes in facial expressivity. This is an area of research that is not well documented. While not commonly studied in individuals with a CHR syndrome, rate of change in learning has been utilized in studies of motor function with groups along the psychosis continuum (Dean et al., 2014; Gupta, Dean, et al., 2018). The assessment of rate of change thus provides a metric that can capture the

degree to which there is variability in facial expressivity, which may serve as a useful clinical marker.

Challenges in studying facial expressivity. Critically, it is important to note that assessing facial expressions in psychopathology can be challenging for several reason. First, it is imperative to consider that blunted facial expressions are observed across other types of psychopathology such as in Parkinson's disease, depression, and autism (Bandini et al., 2017; Cohen et al., 2013; Owada et al., 2018). What makes blunted facial expressions in schizophrenia spectrum disorders particularly distinguishable from others is the persistent nature of abnormalities, although even so, disentangling comorbidity when it comes to this specific symptom can be very challenging. Additionally, there is some evidence in studies assessing blunted facial expressivity in both schizophrenia and depression samples suggesting patients with schizophrenia exhibit more pronounced expressive impairments when compared to patients with depression and parkinsonism (Borod & Clair, 1990; Yecker et al., 1999). Similarly, blunted facial expressivity is different from flat affect, where flat affect typically presents as extreme abnormalities in facial expressivity (Kirkpatrick & Fischer, 2006). Second, another aspect of studying blunting in facial expressions that can be difficult is assessing antipsychotic medication effects (Kirkpatrick & Fischer, 2006). Antipsychotic medications have been found to cause facial blunting, although this is more true for first rather than second-generation medications (Buckley & Stahl, 2007). Despite this, interestingly, converging evidence indicate that patients with schizophrenia exhibit fewer positive and negative facial expressions that are not actually due to antipsychotic medication effects (Kring & Bachorowski, 1999; Kring & Earnst, 1999; Kring & Elis, 2013a).

Alterations in Facial Expressivity in CHR groups

One of the benefits of assessing alterations in facial expressions in CHR groups is that these individuals tend to be free of confounds such as antipsychotic medications that can convolute research designs (e.g., as mentioned, antipsychotic medications may induce facial expression abnormalities) (Fusar-Poli et al., 2012b). To date, the widely used measure to assess for central symptoms in CHR is the SIPS (Miller, McGlashan, Rosen, Cadenhead, Cannon, et al., 2003). In this measure, there is one question that is intended to assess for the expression of emotion, which is "*Has anyone pointed out to you that you are less emotional or connected to people than you used to be?"* Within this context, this question conflates the experience and expression of emotion and also does not offer the possibility to assess differences in discrete emotions such as joy, anger, surprise, sadness etc. Thus, while clinical rating scales provide vital information that there is an abnormality in CHR groups, their lack of precision make it impossible to isolate the nature of this deficit or trust that the deficit truly reflects facial expressivity and not some other psychological process.

To date, only one study has assessed facial expressions among individuals considered at risk and did so in a prospective study design. Specifically, Walker and colleagues (1993) coded video home-movies taken at birthdays and family/holiday gatherings of patients that later developed schizophrenia (Walker et al., 1993). This group found by coding these videos that some individuals who developed schizophrenia showed blunting in facial expressions particularly in the expression of joy as a child up until adolescence compared to siblings without a diagnosis. Although hand-coding videos (as used in Walker et al., 1993) shows promise, this technique requires extensive training time and can be subject to rater bias. As such, there is increasing interest in using more precise methods (e.g., automated facial analysis and facial electromyography; EMG) (Kring & Elis, 2013a) in the assessment of facial expressions.

Neural Mechanisms of Facial Expressivity in Healthy Populations

The production of facial expressions in studies of healthy populations suggest that facial expressivity is a result of integrated activity of network structures of emotion and motor brain circuitry (Car et al., 2003; Dworkin et al., 1996; Gothard, 2014; Hennenlotter et al., 2005; Leslie et al., 2004; Livneh et al., 2012;). While the focus of this section is to provide an overview of the relevant brain regions likely involved in facial expression production, it is important to highlight the brain is comprised of integrated and interacting networks, where no one brain region is solely in charge of a single behavior.

In terms of implicated emotion network brain regions in the production of facial expressions, there is evidence of the involvement of the amygdala, anterior cingulate, orbitofrontal cortex, and cerebellar crus I/II in emotional processes (Davidson, 2002; Etkin et al., 2015; King et al., 2019; Phillips et al., 2003).

In terms of the *amygdala*, this region is particularly vital in selecting facial expressions that may be relevant for a particular context (e.g., different social situations) (Gothard et al., 2014). Furthermore, the amygdala is also responsible for processing fearful and aversive stimuli (e.g., Davis, 1992). Other emotion related regions such as the *anterior cingulate* may play a role in several aspects of emotion such as regulation, generating positive emotion, and suppressing positive emotion (e.g., Dowd & Barch, 2010; Rolls et al., 2003) and *the orbitofrontal cortex* in reward processes such as hedonics and value representations (e.g., Dowd & Barch, 2010). Lastly, crus I/II is a brain region within the cerebellum. There is increasing evidence suggesting that the cerebellum, which has been historically known for its contributions to motor related behaviors, is also involved in emotion (King et al., 2019). In particular, crus I/II may play a role in social cognitive processes such as mentalizing (e.g., being able to understand another person's

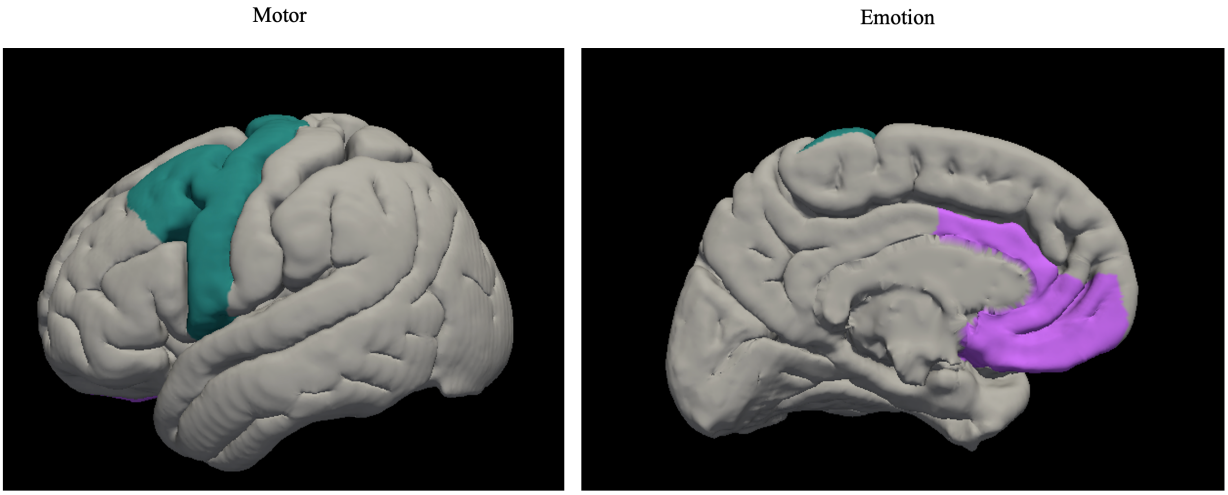
emotional states) (Van Overwalle et al., 2014; Van Overwalle et al., 2020; King et al., 2019). Given that individuals tend to react with facial expressions that are similar to what is observed on another's face (i.e., smiling when seeing another person smile or, in other words, imitating another's behavior) (Dimberg, 1982), it is very possible that mentalizing (and inherently, brain circuitry involved in these processes such as crus I/II) may play a role in facial expression production as well.

In terms of motor structures, it has been suggested that certain brain regions such as the caudate, putamen, thalamus, primary and secondary motor area, caudal middle frontal gyrus, bilateral lobules IV (also known as the anterior cerebellum and represent the combined lobules I-IV of the cerebellum), and cerebellar vermis IIIa/IIIb are implicated.

To begin with, the caudate and putamen are brain regions involved in the basal ganglia cluster, which is an area heavily involved in motor processes. The basal ganglia relies on a normal functioning dopaminergic system and aberrations may result in movement abnormalities as observed in diseases such as Parkinson's and Huntington's (Obeso et al., 2000). The thalamus is a central "relay station" that transmits sensory and motor signals to different pathways in the brain. The primary and secondary motor areas are suggested to be involved in motor processes as well and there is some evidence to even suggest their role in facial expressivity. For example, as discussed in Gothard and colleagues (2014), the primary motor cortex controls facial muscle movements in the lower portion of the face. Contrastingly, the secondary motor area is suggested to control facial muscle movements of the upper facial area. The caudal middle frontal gyrus is responsible for scanning information within the visual field and speech production (speech production and motor movements share similar circuitry) (Kircher et al., 2009). Finally, as discussed, both historical and present-day conceptions of the cerebellum suggest regions such as

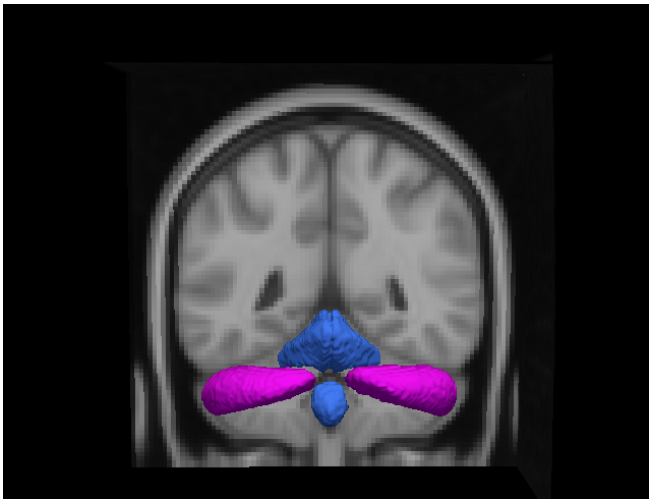
bilateral lobules IV are involved in process including motor control while vermis IIIa/IIIb constitutes the secondary motor representation in the cerebellum (King et al., 2019). See Figure 7 and 8.

Figure 7. Brain regions suggested to be involved in motor and emotional processes



Note. Motor (green; caudate, putamen, thalamus, primary and secondary motor area, caudal middle frontal gyrus) and emotion regions (purple; amygdala, anterior cingulate, orbitofrontal cortex) potentially implicated in facial expressivity

Figure 8. Example of cerebellar regions that may be implicated in facial expressions



Note. Motor cerebellum (blue; bilateral lobules I-IV, vermis lobules IIIa, IIIb), posterior/social (purple; crus I/II)

Neural Mechanisms of Facial Expressivity in Schizophrenia and CHR groups

There are few studies investigating the underlying neural circuitry of alterations in facial expressivity in individuals diagnosed with schizophrenia. Much of the work that currently exists stems from studies of facial expression perception (e.g., perceiving other's facial expressions) using tasked-based functional magnetic resonance imaging (fMRI) paradigms. For example, one study, using a task-based fMRI approach (i.e., completing a task in the scanner) found that more severe blunted affect was associated with increased amygdala activation in response to fearful faces presented (Gur et al., 2007). Another study found using a task-based fMRI paradigm in which sad, happy, and neutral faces were presented in the scanner, that the severity of blunted affect (obtained during clinical interviews) in patients with schizophrenia was related to reduced activation of regions including the amygdala (Lepage et al., 2011). It is well established that the amygdala, amongst the author noted regions, play a role in emotional processing (Aleman & Kahn, 2005).

What is not as well understood is the role of motor circuitry in emotional processing. Perhaps it is the case that brain regions of emotion are solely underlying facial expression abnormalities, but what if motor circuitry is involved as well? The current research on emotional processing is dominated by investigations focusing on brain regions such as the amygdala, as mentioned. However, this may not be the full picture. While there is more evidence of the involvement of motor networks in studies of facial expressions in healthy populations (e.g., Gothard, 2014), research on motor circuitry underlying facial expressions in schizophrenia and in studies of those with a CHR syndrome are limited. However, there is a large body of evidence to suggest that those diagnosed with schizophrenia as well as those with a CHR syndrome are characterized by motor abnormalities (Bernard et al., 2015; Dean et al., 2018b; Mittal et al., 2008; Mittal & Walker, 2007; Schiffman, 2017; Walker et al., 1999; Walther & Mittal, 2017). This includes work suggesting cerebellar pathways comprising of regions such as bilateral lobules IV may play a fundamental role in motor dysfunction in schizophrenia (Andreasen et al., 1998). Additionally, there is evidence of abnormalities in the use of hand gestures which is another component of blunted affect (Walther & Mittal, 2016). When considering blunted facial expressivity under the same umbrella as blunted affect, broadly, it is especially likely that motor circuitry, to an extent, may be underlying disruptions in facial expressivity as well. Relatedly, there is also increasing evidence to indicate that language processing, which involves motor movements of the face, are disrupted in these groups as well (Corcoran et al., 2020; Gupta, Hespos, et al., 2018; Hitczenko et al., 2021).

Importantly, neuroanatomical models of motor dysfunction provide additional support for the role of motor circuitry in facial expressions. For example, motor abnormalities can occur early on in the motor system through transmission of information from the cerebellum to the

thalamus via the cerebellothalamocortical system which projects signals for motor sensory areas such as the primary motor cortex. Once motor signals are projected onto these motor and sensory areas, it is possible to either go through the facial pathway (e.g., facial expressions, language) or through pathways involved in other bodily motor movements. As such, it is possible motor disruptions are present even prior to impulses reaching the facial pathway.

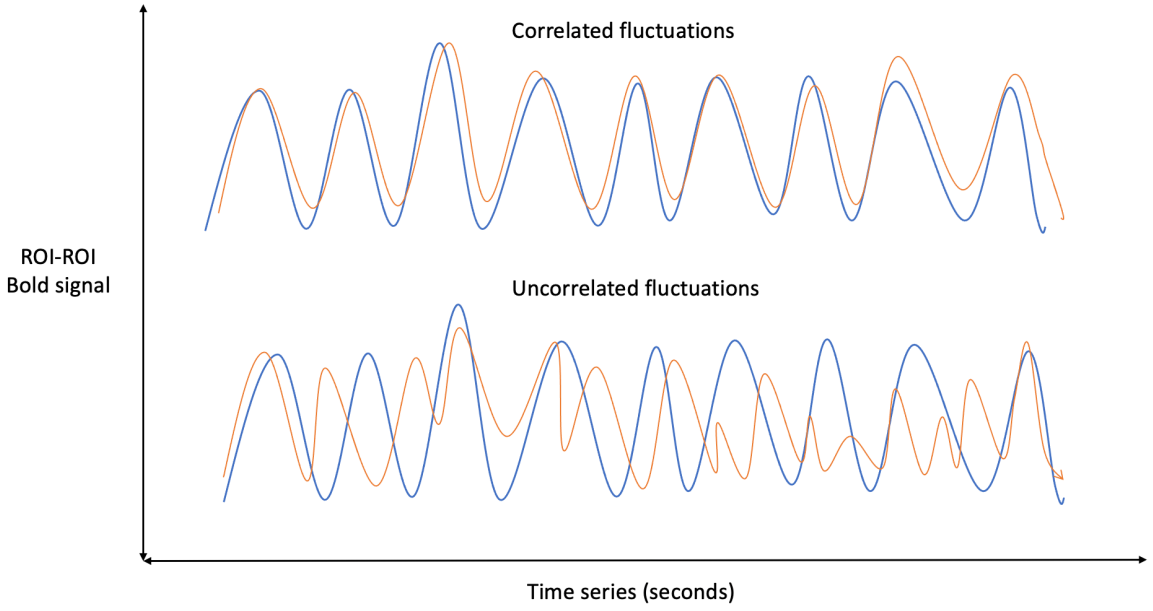
Resting-State Functional Connectivity

The neuroimaging approach called fMRI is a technique that is non-invasive and is used to investigate changes in blood oxygen level-dependent (BOLD) signal in order to pinpoint directions of neural activity (Logothetis et al., 2003; Raichle & Mintun, 2006). A common fMRI approach is using tasks (i.e., task-based fMRI) in the scanner in order to better understand neural mechanisms through BOLD signal activation. A challenge with task-based fMRI paradigm is that the translational, clinical application of findings can be difficult; the scanner is one context and a unique environment in which performance on tasks may not translate to real-world settings. Examining the BOLD signal and spontaneous modulation at rest have been suggested to address the difficulties with clinical adaptability. This approach called resting-state, involves asking participants in the MRI scanner to lie quietly either with eyes closed or stare at a fixation cross. This approach builds off of the notion that the brain is a network comprised of regions that have various functions that are engaged even at rest (i.e., when asked to stay still). Furthermore, this approach can shed light on how information is shared and exchanged intrinsically between regions given that regions are spatially distributed but functionally linked, without the confounds of task-based approaches (e.g., movement, task fatigue). Using resting-state connectivity analyses may reflect stable neural disruptions, instead of brain abnormalities that emerge as a result of task manipulation (Logothetis, 2003; Raichle & Mintun, 2006). This technique maps

closely to the dysconnecitvity hypothesis of schizophrenia (Friston et al., 2016). This theory, developed in the 1990s, posits that symptoms of schizophrenia may result in aberrant connections and integration between unique brain regions. To date, many of the emotion related studies have employed task-based fMRI studies in schizophrenia (Anticevic et al., 2012; Gur et al., 2002; Harrison et al., 2007; Taylor et al., 2012). However, here, I take a different approach and apply resting-state functional connecitivty analyses, which has also been adopted in the study of schizophrenia and CHR symptomatology (Bernard et al., 2017; Fox & Greicius, 2010; Gupta et al., 2016; Mamah et al., 2013; Pelletier-Baldelli et al., 2015, 2018; Shim et al., 2010). With this, I can investigate connectivity patterns that may be related to behavioral data collected outside the scanner and provide insights regarding more stable fluctations (vs. fluctuations in response to task manipulation, as mentioned). However, it is important to note that with resting-state, these analyses are correlational and thus cannot infer causation or provide exact conclusions regarding direction.

To further expand on resting-state connectivity, in Figure 9 below, two brain regions (blue and orange line) signal responses for one participant are depicted in a period of time. The top panel depicts correlated fluctations between two regions while the bottom panel depicts fluctations are not correlated between two regions. Correlated fluctations suggest there is similar signal bold signal activity between two regions. Uncorrelated fluctations as shown in the bottom panel shows two regions that do not have overlapping signals (and thus, are not statistically associated). Another benefit of resting-state analyses is that one can extract neural association values and conduct correlations to better understand relationships between brain activity between regions and behavioral data collected outside of the scanner (which is done in the current study, see Aim 3).

Figure 9. Example of correlated and uncorrelated fluctuations from resting-state connectivity analyses



Note. The top panel represents connectivity between two regions from the same person that are correlated while the bottom panel represents two connectivity patterns that are not correlated; Region of interest (ROI)

Summary

Taken together, there are several questions that remain unanswered in the study of the experience and expression of emotion in those with a CHR syndrome. First, those with schizophrenia report experiencing positive emotions similar to those without the disorder as well as increased negative emotions in response to both pleasant and unpleasant stimuli (Cohen & Minor, 2010b; Kring & Elis, 2013; Strauss et al., 2018). However, it is not well documented as to whether individuals with a CHR syndrome show reductions in reports of positive and negative emotions when viewing video clip stimuli. Secondly, individuals diagnosed with schizophrenia show blunted facial expressivity in studies employing clinical interviews and facial EMG. However, its unknown whether those with a CHR syndrome show subtle and/or micro blunted

facial expressions when using automated facial analysis and facial EMG, respectively. Third, there is limited work investigating associated neural circuitry of blunted facial expressivity in clinical populations and as a result, there is a need to tease apart mechanistic questions related to blunted facial expressivity. One way to do this is to examine associations between alterations in facial expressivity with neural connections between motor and emotion regions. Fourth, given the impacts negative symptoms have on psychosis conversation status and functional outcomes in schizophrenia (Fervaha et al., 2014), it is critical to better characterize these relationships in the context of the experience and expression of emotion in CHR groups.

THE PRESENT STUDY

The current study draws from prominent theories of negative symptoms and emotion in efforts to assess the experience and expression of emotion in CHR individuals. To achieve this broader goal, I first sought to assess differences between CHR individuals and healthy controls in experience ratings (i.e., excitement, amusement, sadness, and fear) from an evocative film clip task. Specifically, participants viewed video clips intended to evoke specific emotions and were asked to provide a rating indicating the intensity in which they felt a range of emotions. Second, alterations in subtle facial expressivity (i.e., joy, anger, surprise, fear, contempt, disgust, sadness) were investigated by processing segments of video recorded clinical interviews into automated facial analysis. To further probe at facial expressivity and understand micro facial expressions, facial EMG data (zygomaticus and corrugator activity) were collected during the noted evocative film clip task. Using facial EMG data, timing was examined between groups in the most evocative sections of the film clips as well as the period before. Furthermore, analyses assessing rate of change in facial expressivity was also investigated. Third, this study assessed associated neural circuitry of facial expressivity alterations using resting-state connectivity analyses and emotion (e.g., amygdala) and motor brain regions (e.g., primary and secondary motor areas). Fourth, relationships between alterations in the experience and expression of emotion (i.e., reports of the experience of emotion and facial expressivity derived from automated analysis and facial EMG) and psychosis risk calculator scores and social functioning were also investigated. More specific aims are noted below in the next chapter.

CHAPTER II.

AIMS AND HYPOTHESES

Aim 1. To investigate if CHR individuals exhibit abnormalities in the experience of positive and negative emotions when compared to controls.

Hypothesis 1. As mentioned, studies assessing reports of positive and negative emotions are limited in CHR groups. To date, findings utilizing laboratory, experimental paradigms suggest reduced reports of positive and negative emotion ratings in response to viewing both positive and negative picture presentations in CHR groups (Gruber et al., 2018a). I sought to assess reports of positive and negative emotions by employing an evocative film clip task which presents video clips (e.g., excerpts showing an exciting situation) intended to evoke positive (i.e., excitement and amusement) and negative (i.e., fear and sadness) emotions. This emotion task has the potential to provide an understanding of potential "real-world" experiences of emotion that may translate outside the laboratory-based context. Given evidence in studies utilizing CHR samples suggesting reduced reports of positive emotions when presented with pleasant stimuli and lower reports of negative emotions when presented with unpleasant stimuli (Gruber et al., 2018), I predicted that CHR individuals would exhibit reductions in reports of positive emotional experience after viewing the positively valenced clips, and reductions in negative emotion ratings after viewing the negatively valenced clips.

Exploratory Aim 1.1. Differences in nontarget emotions. There is also evidence in studies of schizophrenia that patients report increased negative emotion ratings in response to pleasant stimuli in some laboratory paradigms (e.g., Cohen & Minor, 2010). Given this, I also assessed nontarget emotion ratings in an exploratory fashion (i.e., self-reported negative emotions after

viewing the positive emotional clips and self-reported positive emotions after viewing the negative emotional clips) in CHR individuals when compared to controls.

Exploratory Aim 1.2 Relationships with anxiety and depression. As noted, secondary negative symptom sources may also play a role in reports of the experiences of emotion among CHR samples (Addington et al., 2011a; Gruber et al., 2018a; Gupta, Strauss, et al., 2021). Similarly, one study found a relationship between reduced reports of positive emotional experience after viewing pleasant stimuli and increased anxiety and depression (Gruber et al., 2018). As a result, I sought to investigate relationships between reports of positive emotion to pleasant stimuli and negative emotions to negative stimuli and relationships with anxiety (i.e., panic, arousal symptoms) and depression.

Aim 2. To determine whether CHR individuals exhibit alterations in facial expressions when compared to controls.

Hypothesis 2a. Given increasing evidence indicating that alterations in facial expressions are present in studies with schizophrenia samples (Kring & Elis, 2013) and research to suggest these impairments are present among CHR individuals (Walker et al., 1993), I sought to assess whether alterations in facial expressivity are present in sample of CHR individuals using a multi-dimensional approach. To assess subtle facial expressions, video-recorded segments were submitted into an automated facial analysis (iMotions) software to assess seven basic facial expressions (joy, anger, surprise, fear, contempt, disgust, sadness) when compared to controls. In order to stay update to date with research and given the noted iMotions program is no longer available for public use, an additional automated facial analysis program was used for Aim 4 (see below). I predicted that CHR individuals would exhibit blunting in all facial expressions when compared to controls.

Hypothesis 2b. Facial EMG was used to determine if subtle alterations in expressivity were present with a focus on two facial muscles that are centrally implicated in emotional responding, the zygomaticus and corrugator muscle (Dimberg, 1982). The use of dynamic, multimodal film clip stimuli specifically designed to elicit positive, negative, and neutral emotions allowed the ability to determine information regarding (a) context and (b) timing in facial expressivity. Specifically, established film clips designed to elicit positive (i.e., excitement, amusement), negative (i.e., fear, sadness), and neutral emotions were used. To determine whether facial expressions might differ depending on the timing of the clip, for those film clips where alterations in facial expressivity were observed, facial expressions during the most emotionally evocative peak period and prior to this peak period (Johnson et al., 2017) were examined. Given findings from schizophrenia of blunted facial expressivity (e.g., Kring & Elis, 2013), it was predicted that CHR individuals would show blunted zygomaticus activity in response to positive and negative film clips. To further determine examine timing, I also assessed whether the CHR group would show increased corrugator activity in response to positive and negative clips film clips based on current findings suggesting those with a CHR report increased negative facial expressions (Gupta et al., 2020, 2021). Additionally, for those film clips where alterations in facial expressions were detected in the CHR group, facial expressions were examined during the most emotionally evocative positive film clip period in which I predicted I would see differences during the peak period of emotion in the video clip (but not prior to that peak). Finally, the rate of change in facial expressivity was assessed and I predicted that the CHR group would show reduced rates of change in facial expressions when compared to controls.

Exploratory Aim 2.1 Differences in nontarget expressions. I sought to assess nontarget facial expressivity: zygomaticus facial muscle activity in response to the negative emotion film clips and corrugator facial muscle activity in response to the positive emotion film clips.

Exploratory Aim 2.2 Relationships with anxiety and depression. To examine the relationships between facial expressions derived from automated analysis and facial EMG (zygomaticus activity in response to pleasant clips and corrugator activity in response to unpleasant clips) and secondary sources of negative symptoms (e.g., anxiety and depression).

Aim 3. To examine whether alterations in facial expressions are related to dysconnectivity between (a) emotion regions (e.g., amygdala, crus I/II), motor regions (e.g., bilateral lobules IV, primary and secondary motor areas), or both emotion and motor regions.

Hypothesis 3. Resting-state functional connectivity analyses were employed to investigate relationships between alterations in facial expressions (if observed) derived from FaceReader (a more up to date facial analysis software program that is currently available) and emotion (left/right caudal anterior cingulate, left/right medial orbitofrontal cortex, left/right rostral anterior cingulate, left/right amygdala, crus I/II) and motor regions (left/right caudate, left/right putamen, left/right thalamus, left/right primary motor area, left/right secondary motor area, left/right caudal middle frontal gyrus, bilateral lobules IV, Vermis IIIa, Vermis IIIb). Hypotheses for this aim were competing (Platt, 1964). Specifically, it was predicted if connectivity between emotion-emotion regions are found to relate to alterations in facial expressions within the CHR group, then one could speculate that the underlying mechanisms of alterations in facial expressivity could be driven by disruptions in emotion circuitry primarily. If motor-motor connectivity is found, then it may be that more motor-based abnormalities underlie facial expression alterations. If emotion-motor regions are found to relate to alterations in facial

expressions in the CHR group, it is possible that both emotion and motor regional connections may be contributors.

Exploratory Aim 3.1 Comparisons with control connectivity patterns. Once atypical patterns of connectivity are identified in the CHR group, an exploratory aim is to compare these relationships with connectivity patterns within a control group.

Exploratory Aim 3.2 Analyses with high and low facial expressivity. Exploratory analyses were also employed to investigate whether these results are driven by those with lower or higher levels of facial expressivity within the CHR group.

Aim 4. To assess whether abnormalities in the (a) experience ratings from the evocative film clip task and (b) facial expressivity alterations derived from automated facial analysis and facial EMG are related to social functioning and likelihood of conversion to psychosis (based off of risk calculator scores).

Hypothesis 4a. Given studies indicating that abnormalities in the experience of emotion are related to functional outcomes and conversion to psychosis in CHR samples (Piskulic et al., 2012), I predict that reduced self-reported positive and negative emotional experience will be related to impairments in social functioning and likelihood of conversion to psychosis based off of risk calculator scores.

Hypothesis 4b. There is also recent evidence from our group to indicate that blunted facial expressions of joy (and not increased anger facial expressions) are related to deficits in social functioning and increased likelihood of conversion to a psychotic disorder in a sample of CHR individuals. As such, I predict that reduced positive zygomaticus activity in response to positively valenced video clips and reduced corrugator activity in response to negative film clips will be related to impairments in social functioning and conversion to psychosis scores.

CHAPTER III.

METHODS

Participants

Sample 1 is from the ADAPT lab at Northwestern University consisting of 83 participants (43 CHR and 40 controls), aged 15-28 (M = 20.73, SD = 2.43). Sample 1 is used to address Aim 1 (experience ratings during an evocative film clip task), and a subsample (CHR = 34, control = 32) with available data is used for the facial EMG portion of Aim 2 (*Hypothesis 2b)*, aged 15-28 (M = 20.56, SD = 2.37). Sample 2 consists of a total of 84 participants (42 CHR and 42 control individuals), aged 12-21 recruited through ADAPT at University of Colorado (M = 18.51, SD = 2.31). These participants have been used to assess for *subtle* facial expressions derived from segments of video-recorded clinical interviews submitted into an automated facial analysis software that was available at that time called iMotions (*Aim 2, Hypothesis 2a*). A subsample of Sample 2 included a total of 37 CHR individuals and 33 controls that had available neuroimaging data and this sample is used for Aim 3, aged 16-21 (ages 16 and above as used only given neurodevelopmental differences throughout adolescence), (M = 19.11, SD = 1.43). All participants were recruited using Craigslist, e-mail announcements, newspaper advertisements, flyers, and community health referrals. Exclusion criteria consisted of head injury, the presence of a neurological disorder, IQ < 70, and lifetime substance dependence. The presence of DSM-5 schizophrenia spectrum disorder (e.g., schizophrenia, schizoaffective disorder, schizophreniform) was an exclusion criterion for CHR participants. The presence of a psychotic disorder or a psychotic disorder in a first-degree relative was an exclusion criterion for control participants.

The SIPS (Miller, McGlashan, Rosen, Cadenhead, Cannon, et al., 2003) was administered to diagnose a CHR syndrome (see Appendix A, B for structure and rating scale). CHR participants in the present study met SIPS criteria defined by moderate-to-severe but not psychotic levels of positive symptoms (rated from 3 to 5 on a 6-point scale) or a decline in global functioning accompanying the presence of schizotypal personality disorder or a family history of psychosis. The SIPS gauges several distinct categories of prodromal symptom domains, including positive, negative, and disorganized dimensions. A mean score for each category is used as an indicator of the respective dimensions of symptomatology.

The Structured Clinical Interview for the DSM-5 (First et al., 1997) is administered to rule out a psychotic disorder diagnosis. Training of advanced doctoral student interviewers was conducted during a 2-month period for both clinical interviews, and interrater reliabilities exceeded the minimum study criterion ($\kappa \geq 80$). All participants included in this study consented to being video-recorded during clinical interviews.

Anxiety and Depression

Anxiety (panic/arousal symptoms) were investigated using sum scores from the Beck Anxiety Inventory (Beck et al., 1988). While there are several other types of anxieties, for the purposes of the current proposal aims, we limit analyses to panic/arousal symptoms. To ease readability, panic-symptoms measured by the BAI is referred to as “anxiety” throughout. Depression sum scores from the Beck Depression Inventory (Beck et al., 1996) were also used.

Social Functioning

The Global Functioning Scale: Social (GFS-S) (Cornblatt et al., 2007b) was used to assess for social functioning in an interview format. A score is given on a 1-10 scale, with 1 indicating low levels of social functioning and 10 indicating very high levels of social functioning (e.g.,

number of friends, how often the individuals engaged in social activity, any reports of arguments or falling outs).

Risk Calculator Scores

Two risk calculators were used in the present study given available data. For data derived from the University of Colorado sample (Sample 2) - each CHR participant was assessed for risk of conversion to psychosis. A freely available online calculator was used – http://riskcalc.org:3838/napls/ developed by a large prodromal consortium called the North American Prodrome Longitudinal Study (NAPLS). In the initial development, this calculator seemed to have good predictive ability to classify CHR who did or did not convert to psychosis (Cannon et al., 2016). The measures used to create the risk calculator score will be the same materials as described in initial development of the calculator, collected during the clinical interviews. This includes cognitive measures (Nuechterlein et al., 2008), family history of psychosis collected during the clinical interview, SIPS unusual thoughts and suspiciousness (Miller et al., 2003) scores, age, the number of negative life events, and the number of traumas reported from the SCID interview. We have used this calculator in previous studies published by our group (Dean et al., 2018b; Gupta et al., 2019).

A newer risk calculator available for the Northwestern Sample (Sample 1) was used. This calculator was developed by the Shanghai At-Risk for Psychosis (SHARP) program at the Shanghai Mental Health Center (Zhang et al., 2018) was used. The noted risk calculator provides a probability estimate that an individual with a CHR syndrome will develop psychosis and was developed in order to provide a practical and individualized risk score for psychosis conversion that can be used by both clinicians and researchers (Osborne & Mittal, 2019, 2021; Zhang et al., 2020).

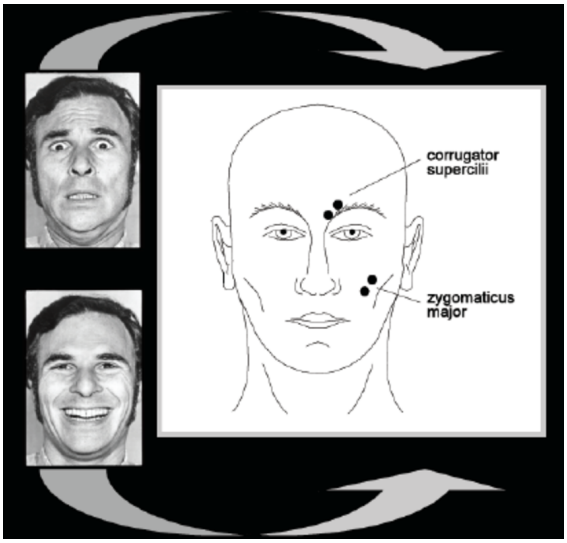
Film Clip Stimuli

An evocative film clip task was used for Aim 1 and Aim 2 in which experience ratings and facial electromyography data, respectively, (described below) was collected. This film clip task is designed to elicit positive, negative, and neutral emotional states and has been used extensively in prior work with healthy and clinical populations (Gross & Levenson, 1995; Kreibig, 2010). Specifically, participants watched film clips eliciting (a) excitement (a short film clip showing Sarah Hughes winning the Olympic Gold medal and celebrating her success) (Gruber et al., 2008; Johnson et al., 2017), (b) amusement (a short clip from "I love Lucy" showing Lucy and her friend manage wrapping chocolate pieces on a conveyor belt that continues to increase in speed) (Chen, Lwi, et al., 2017; Chen, Wells, et al., 2017); (c) fear (a short film clip from "The Silence of the Lambs" showing a detective encountering a criminal they have been searching for) (Gross & Levenson, 1995), (d) sadness (a short clip from "21 Grams" showing a mother who learns that her two young daughters died in a car accident) (Rompilla Jr. et al., 2021; Shiota & Levenson, 2009); and (e) a neutral video clip (a short film clip showing geometric shape moving along the screen; Johnson et al., 2017). Additionally, baseline ratings and facial electromyography was collected prior to each video clip in which participants were asked to view a "X" for 1 minute. Participants received a 30 second break between each video. See Appendix C and D for example of film clip task structure and scale presented for experience ratings. See Appendix E for example of pre-condition experience rating scale.

Facial Expression Measures

Facial Electromyography

Figure 10. Image depicting corrugator supercilli and zygomaticus major



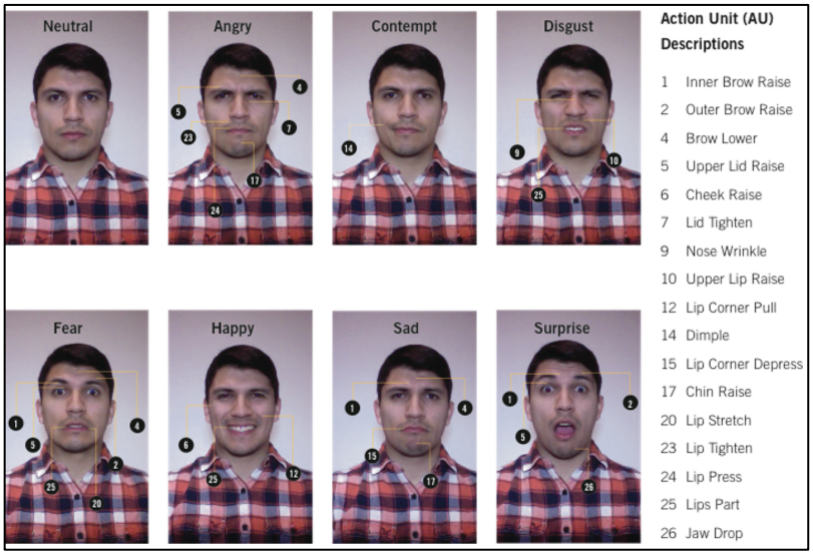
Note. Corrugator supercilli activity is commonly associated with negative facial expressions while zygomaticus major is affiliated with positive emotions such as smiling

Facial electromyography data (zygomaticus and corrugator) was collected continuously using Biopac Systems. Specifically, following established guidelines detailed in Fridlund and Cacioppo (1986), Ag/AgCL electrodes (4 mm) were placed over the left zygomaticus major and left corrugator supercilii muscle (Figure 10). A ground electrode was placed on the forehead right below the hairline. EMG data were collected with a sampling rate of 2000 Hz, with an online bandpass filter between 10 and 5000 Hz for zygomaticus activity and between 1 and 500 Hz for corrugator activity. Offline, zygomaticus signals were run through a 50-500 Hz bandpass filter, while corrugator raw signals were run through a high pass filter of 50 Hz, to achieve a final bandpass filter between 50 and 500 Hz for both muscles. Collected EMG data was visually inspected for usability and artifacts and preprocessed using Biopac Systems Acknowledge Software, version 4.4. Following, filtered data was integrated (root square mean). Zygomaticus, and corrugator values (microvolts) deduced from each video clip were averaged.

Automated Facial Analysis Software

Two computerized facial analysis programs are used in this dissertation. Aim 2 uses iMotions (2016), which is a computerized software that uses a commercial automated facial coding tool (Emotient FACET) (https://imotions.com) which grew out of The Computer Expression Recognition Toolbox (CERT) (Littlewort et al., 2011) and computes evidence scores indicating the likelihood a specific expression (based off of AUs) (Figure 11) is present based off of matching predetermined algorithms and provides a percent score (*Hypothesis 2a)*. Second, these same segments were submitted into an additional automated analysis tool called FaceReader (version 7.1) developed by Noldus Information Technology given that iMotions was no longer available at the time of Aim 3 (aim at investigating underlying neurocorrelates of alterations in facial expressivity). This program also uses predetermined algorithms and provides a value indicating how much facial activity resembles expressions (e.g., joy). FaceReader values were averaged for each participant and a percent was computed indicating the likelihood the expression matches a predetermined algorithm (i.e., likelihood an expression is present). Both programs were developed based on FACS. FACS is an anatomically-based coding system and quantifies facial muscle movements using AUs (e.g., AUs; AU 6 indicates movement of the

Figure 11. Action units for facial expressions derived from iMotions



orbicularis oculi, pars orbitalis, which raises the cheeks; AU 12 indicates movement of the zygomaticus major, which pulls the lip corners upwards). Specific configurations of AU are then used to identify specific emotion prototypes (e.g., presence of both AU 6 and 12 identify joy).

It is important to note that there were no group differences in the ability to register the video recording of participant faces in either program (iMotions: $t(82) = .83$, $p = 0.41$; FaceReader: $t(82) = 0.23$, $p = 0.82$). Over 91% of video frames were recognized by the iMotions (2016) software and over 85% were recognized by FaceReader in this sample at a sampling rate of 30 Hz suggesting that these videos, on average, were suitable for facial analysis.

Resting-State Functional Connectivity

As mentioned, a subsample of participants from the University of Colorado Sample (Sample 2; see results section for more details) completed a brain imaging session that included structural and imaging data. Here, the focus was on the resting-state function connectivity data. Scans were acquired using a 3-Tesla Siemens Tim Trio MRI scanner (Siemens AG, Munich, Germany) with a standard 12-channel head coil. Structural images were acquired with a T1-weighted 3D magnetization prepared rapid gradient multi-echo sequence (MPRAGE; sagittal plane; repetition time [TR] = 2530 ms; echo times [TE] = 1.64 ms, 3.5 ms, 5.36 ms, 7.22 ms, 9.08 ms; GRAPPA parallel imaging factor of 2; 1 mm^3 isomorphic voxels, 192 interleaved slices; FOV = 256 mm; flip angle = 7°; time = 6:03 min). A 5-minute 34 s resting-state blood-oxygen-level dependent (BOLD) scan was acquired with a T2-weighted echo-planar functional protocol (number of volumes = 165; TR = 2000 ms; TE = 29 ms; matrix size = 64 × 64 × 33; FA = 75°; 3.8 × 3.8 × 3.5 mm^3 voxels; 33 slices; FOV = 240 mm). A 5-minute resting-state scan has been shown to have the same robust correlations as longer scans (Van Dijk et al., 2010). Participants were instructed to relax with their eyes closed during this

time. A turbo spin echo proton density (PD)/T2-weighted acquisition (TSE; axial oblique aligned with anterior commissure-posterior commissure line; TR = 3720 ms; TE = 89 ms; GRAPPA parallel imaging factor of 2; FOV = 240 mm; flip angle: 120°; 0.9 × 0.9 mm^2 voxels; 77 interleaved 1.5 mm slices; time = 5:14 min) was generated to investigate incidental pathology.

FSL (v. 5; http://fsl.fmrib.ox.ac.uk/fsl) was used for data processing. Data processing included motion correction, brain extraction, high-pass filtering (100 s), and spatial smoothing (6mm FWHM). Next, functional images were aligned to the MNI 2-mm brain template with a two-step procedure. First, the resting state scan was aligned to the high-resolution MPRAGE using a linear boundary-based registration method, which relies on white matter boundaries (Greve & Fischl, 2009; Jenkinson et al., 2002; Jenkinson & Smith, 2001). Second, the MPRAGE was nonlinearly aligned to the template, and the two registrations were then combined to align the functional resting state scan to the template. We used the Artifact Rejection Toolbox (ART; http://www.nitrc.org/projects/artifact_detect/) to create confound regressors for motion parameters (3 translation and 3 rotation parameters), and additional confound regressors for specific image frames with outliers based on brain activation and head movement. In order to identify outliers in brain activation, the mean global brain activity (i.e., the mean signal across all voxels) was calculated as a function of time and was then Z normalized. Activation outliers were defined as any frames where the global mean signal exceeded 3 SD. Similarly, frame-wise measures of motion (a composite measure of total motion across translation and rotation) were used to identify any motion outliers (i.e., motion spikes). Motion outliers were defined as any frame where the motion exceeded 1 mm.

Anatomical images were segmented into gray matter, white matter, and CSF with SPM8 in order to create masks for signal extraction. The CONN toolbox (Whitfield-Gabrieli & Ford,

2012) uses principal component analysis (PCA) to extract 5 temporal components from the segmented CSF and white matter, which were entered as confound regressors in the subject-level GLM. This approach corrects for confounds of motion and physiological noise without regressing out global signal, which has been shown to introduce spurious anticorrelations (Chai et al., 2011; Murphy et al., 2009). Motion from the ART toolbox was included as a confound regressor. From the motion translation parameters, the ART toolbox calculates mean displacement, and we used this measure as well as the number of motion and mean signal outliers in order to compare the degree of head movement between the groups. Further preprocessing included a band-pass filter (0.008 to 0.09 Hz), detrending, and despiking, in accordance with procedures used to target resting-state data. The mean time-series averaged across all voxels within each seed was used as a regression parameter and correlated with all other voxels in the brain in seed-to-voxel connectivity analyses.

The CONN toolbox version 20.b (Whitfield-Gabrieli & Ford, 2012), with SPM12 (Wellcome Department of Imaging Neuroscience, London, UK; www.fil.ion.ucl.ac.uk/spm) was used using a voxel-level cluster forming threshold of $p_{uncorrected} < 0.001$ and then corrected at the cluster-level using a false-discovery rate (FDR) of $p < 0.05$ (Chumbley & Friston, 2009). Additional denoising regressors corresponding to gray matter, white matter, cerebrospinal fluid, and mean global signal were included to remove variance. Anatomical images were segmented into gray matter, white matter, and CSF with SPM12 to create masks for signal extraction. The CONN toolbox extracts five temporal components from the segmented. CSF and white matter, which were entered as confound regressors in the subject-level GLM. Seeds were defined in MNI template space and generated using the Desikan atlas (Desikan et al., 2006) for all cortical and subcortical regions and a spatially unbiased atlas template (SUIT) for cerebellar regions

(Diedrichsen, 2006). The final connectivity measures were corrected for nuisance variables (e.g., motion, artifacts) but not model corrected.

CHAPTER IV.

DATA ANALYSIS

Independent t-tests and chi-square tests were employed to examine differences between groups in continuous and categorical demographic variables. If group differences were observed in demographics, further analyses were applied (see results for more detail).

Aim 1. To investigate if CHR individuals exhibit abnormalities in the experience of positive and negative emotions when compared to controls.

Data was not highly skewed for reports of positive and negative emotional experiences from the evocative film clip task. Group differences in pre-condition ratings were investigated first by employing independent t-tests. Then, group differences in experience ratings were investigated by employing analysis of covariance (ANCOVA). Independent t-tests were used to assess for nontarget emotion group differences (exploratory aim). Partial correlations, adjusting for pre-condition ratings, were applied to investigate associations between experience reports (in which group differences were observed to limit the number of comparisons) and anxiety/depression scores (exploratory aim).

Aim 2. To determine whether CHR individuals exhibit alterations in facial expressions when compared to controls.

For automated computerized facial analysis (Aim 2), tests of normality revealed data was not highly skewed. Independent t-tests were used to look at group differences in facial expression measures between CHR and control groups. Additionally, exploratory analyses were conducted to determine the influence of sex on facial expression using two-way ANOVA with sex (male/female) and group (CHR/control) given demographic differences in the distribution of

sex (see results for further detail) in this specific sample. Bivariate correlations were applied to investigate associations between automated facial expression variables (in which group differences were observed to limit the number of comparisons) and anxiety/depression scores (also exploratory).

For analysis with facial EMG, tests of normality were employed and results indicated data was not highly skewed except for zygomaticus activity during the amusement and sadness videos. Analyses with log transformed variables did not change the magnitude or direction of findings. As a result, untransformed data was employed in analyses to ease interpretation of results. Independent t-tests were employed to investigate group differences in pre-condition facial expressivity values (microvolts). ANCOVA, adjusting for pre-condition values, were used to assess for group differences in facial expressivity during each video clip, time segments, and rate of change. In terms of timing, this was investigated by taking facial activity from the film clips in which there were group differences in facial expressivity. For this, the peak segment depicts the peak level of emotions (i.e., high levels) intended to be evoked and the pre-peak period is prior to that period. With this in mind, a group (CHR/control) by facial expressivity (during pre-peak and peak period) was employed. Then, post-hoc ANCOVAs investigating group differences (CHR/control), adjusting for pre-condition facial expressivity, were examined. Partial correlations, adjusting for pre-condition facial expressivity were applied to investigate correlations between facial EMG variables (in which group differences were observed to limit the number of comparisons) and anxiety/depression scores. Group differences in nontarget facial expressivity was also examined in an exploratory fashion using independent t-tests.

Aim 3. To examine whether alterations in facial expressions are related to dysconnectivity between (a) emotion regions (e.g., amygdala, crus I/II), motor regions (e.g., bilateral lobules IV, primary and secondary motor areas), or both emotion and motor regions.

For resting-state connectivity analyses, independent t-tests were employed to assess for group differences in behavioral facial expression data to establish if there were, in fact, group differences in facial expressions derived from FaceReader. Given evidence from my previous work suggesting that it is is possible to detect facial expression abnormalities in 1-minute slices of time (Gupta et al., 2020), 1-minute segments of clinical interview data were submitted into FaceReader (an available facial analysis program) and independent t-tests were employed assessing for group differences. Resting-state connectivity analyses were employed only for facial expressions in which group differences were observed. Additionally, resting-state connectivity analyses were employed within the CHR group (interaction analyses were not employed given limited power) using a priori seeds for emotion and motor regions defined based on previous studies suggesting both emotion and motor regions may be implicated in facial expression related abnormalities (e.g., Gothard et al., 2014). Facial expression variables used within group (CHR, healthy control) analyses were mean centered by that group, and in whole group analyses the variable was mean centered by whole study sample. ROI-ROI analyses within the CHR group only were first employed, given that this group has been suggested show facial expression abnormalities when compared to controls (Gupta et al., 2019; Gupta et al., 2021). Seeds entered into analyses included emotion (left/right caudal anterior cingulate, left/right medial orbitofrontal cortex, left/right rostral anterior cingulate, left/right amygdala, crus I/II) and motor regions (left/right caudate, left/right putamen, left/right thalamus, left/right primary motor area, left/right secondary motor area, left/right caudal middle frontal gyrus, bilateral lobules I-IV

termed bilateral lobules IV throughout, Vermis IIIa, Vermis IIIb). To appropriately account for false positives in multiple comparisons, results are thresholded at the voxel-level at $p_{uncorrected}$ <0.001 for cluster formation and then corrected at the cluster-level using a false-discovery rate (FDR) of $p < 0.05$ (Gupta et al., 2020). Once atypical connectivity patterns were identified in the CHR group, follow up correlational analyses (using extracted Fischer's transformed bivariate coefficients) within the CHR group and a matched control group were employed. To further interpret correlational findings, group differences in the associations between facial expressivity and connectivity were employed. Then, group differences in connectivity patterns in CHR and control groups showing lower and higher levels of facial expressivity were assessed in an exploratory fashion by taking the median split of facial expressivity values (high/low facial expressivity).

Aim 4. To assess whether abnormalities in the (a) experience ratings from the evocative film clip task and (b) facial expressivity alterations derived from automated facial analysis and facial EMG are related to social functioning and likelihood of conversion to psychosis (based off of risk calculator scores).

Finally, bivarte correlations were employed to investigate relationships between facial expressions derived from iMotions and psychosis risk conversion scores and social functioning. To assess for links between facial expressions derived from facial EMG and psychosis-risk conversion scores and social functioning, partial correlations were employed adjusting for pre-condition activity. These analyses were only applied to findings in which there were group differences in facial expressions.

Please note, group differences in experience ratings and facial EMG values were employed using difference scores as well – see Appendix F.

CHAPTER V.

RESULTS

Sample Characteristics and Details

Sample 1. Northwestern Sample

As noted, one sample consisted of individuals recruited at Northwestern University. The sample was a total of 83 participants (CHR = 43, control = 40) with experience reports (Aim 1) from the evocative film clip task. These individuals did not exhibit any significant differences in age, biological sex, or parental education. As expected, the CHR group endorsed more positive, negative, depressive, and anxious symptoms compared to controls. Furthermore, the CHR group endorsed more social impairments compared to the control group. Only one individual was taking an antipsychotic medication. See Table 1 for demographic details.

Table 1. Means and standard deviations for demographics and symptom details in a sample with a CHR syndrome and controls collected at Northwestern that completed experience portion of the evocative film clip task (Sample 1)

	CHR	Control	Total	Statistic
Demographics				
Number of participants	43	40	83	
Age	21.13 (2.60)	20.30 (2.20)	20.73 (2.43)	$t(81) = 1.58$, $p = 0.12$
Number of Females	24	28	52	$x^2(1) = 2.25$, $p = 0.13$
Parent Education	16.70 (2.05)	15.38 (3.14)	16.11 (2.67)	$t(78) = -1.34$, $p = 0.89$
Race and Ethnicity				
First Nations	0	1	1	
Asian/Middle Eastern	6	10	16	
Black	12	5	17	
Central/South American	2	0	2	
White	19	19	38	
Interracial	3	5	8	
Unknown	1	0	1	
Symptoms				
Positive	10.60 (4.62)	0.84 (1.65)	6.38 (6.07)	$t(72) = 12.67$, $p < 0.001$
Negative	7.00 (5.54)	1.16 (1.89)	4.44 (5.20)	$t(71) = 6.31$, $p < 0.001$
Depression	15.45 (12.68)	4.39 (4.75)	10.06 (11.10)	$t(76) = 5.15$, $p < 0.001$
Anxiety	18.22 (14.04)	7.43 (7.36)	13.25 (12.61)	$t(74) = 4.28$, $p < 0.001$

Social Functioning	7.38 (1.44)	9.00 (0.91)	8.07 (1.47)	$t(57) = -4.94$, $p < 0.001$

Note. Parental education is the average of mother and father education in years; Positive and negative symptoms are sum scores taken from the Structured Interview for Psychosis-Risk Syndromes (SIPS); Biological sex and race and ethnicity are recorded as counts; Depression and anxiety are sum scores deduced from the Beck Depression Inventory and Beck Anxiety Inventory, respectively; Social functioning scores fall on a 1 (very impaired social functioning) to 10 (no impairments) scale

A subsample of the noted participants had usable and available facial EMG data (Aim 2). Sample characteristics and details for this subsample are depicted in Table 2. There were no significant differences in age, biological sex, and parental education. As expected, and in line with the larger sample, there were group differences in positive, negative, depressive, and anxious symptoms as well as social functioning.

Table 2. Means and standard deviations for demographics and symptom details in a sample with a CHR syndrome and controls of individuals with EMG data available from the Northwestern sample (Sample 1)

	CHR	Control	Total	Statistic
Demographics				
Number of participants	34	32	66	
Age	20.94 (2.41)	20.16 (2.29)	20.56 (2.37)	$t(64) = 1.36$, $p = 0.18$
Number of Females	20	22	42	$x^2(1) = 1.05$, $p = 0.31$
Parent Education	16.39 (2.40)	16.42 (2.92)	16.41 (2.64)	$t(62) = -0.04$, $p = 0.97$
Race and Ethnicity				
First Nations	0	0	0	
Asian/Middle Eastern	5	10	15	
Black	8	5	13	
Central/South American	2	0	2	
White	15	15	30	
Interracial	3	2	5	
Unknown	1	0	1	
Symptoms				
Positive	10.91 (4.34)	0.92 (1.71)	6.68 (6.06)	$t(57) = 12.19$, $p < 0.001$
Negative	7.48 (5.62)	1.24 (2.01)	4.79 (5.40)	$t(56) = 5.91$, $p < 0.001$
Depression	16.61 (12.32)	4.20 (4.52)	10.70 (11.27)	$t(61) = 5.40$, $p < 0.001$
Anxiety	18.97 (14.50)	7.21 (7.36)	13.48 (13.04)	$t(58) = 4.03$, $p < 0.001$
Social Functioning	7.41 (1.45)	9.10 (0.83)	8.12 (1.48)	$t(48) = -4.76$, $p < 0.001$

Note. Parental education is the average of mother and father education in years; Positive and negative symptoms are sum scores taken from the Structured Interview for Psychosis-Risk Syndromes (SIPS); Biological sex and race and ethnicity are recorded as counts; Depression and anxiety are sum scores deduced from the Beck Depression Inventory and Beck Anxiety Inventory, respectively; Social functioning scores fall on a 1 (very impaired social functioning) to 10 (no impairments) scale.

Sample 2: University of Colorado Sample

Sample 2 consisted of individuals recruited at the University of Colorado Boulder (N = 84; CHR = 42, controls = 42) (Aim 2; automated analysis). In terms of sample 2 characteristics and details, CHR individuals did not differ from controls in age or parental education but did significantly differ in biological sex. As such, the influence of sex on facial emotion variables was evaluated in exploratory analyses noted below. As expected, the CHR group endorsed more positive, negative, anxious, and depressive symptoms. Furthermore, the CHR group had lower social functioning scores (suggestive of more impairments) compared to controls). See Table 3 for means and standard deviations.

Table 3. Means and standard deviations for demographics and symptom details in a sample with a CHR syndrome and controls from University of Colorado Boulder that had facial expression (iMotions), automated analysis data of clinical interviews (Sample 2)

	CHR	Control	Total	Statistic
Demographics				
Number of participants	42	42	84	
Age	18.90 (1.91)	18.12 (2.61)	18.51 (2.30)	$t(82) = 1.58$, $p = 0.12$
Number of Females	19	30	49	$\chi2(1) = 5.93$, $p = 0.02$
Parent Education	15.51 (2.10)	15.76 (2.89)	15.63 (2.49)	$t(74) = -0.44$, $p = 0.66$
Race and Ethnicity				
First Nations	1	0	1	
Asian/Middle Eastern	2	3	5	
Black	1	2	3	
Central/South American	5	14	19	
White	33	23	56	
Hispanic	6	14	20	
Symptoms				
Positive	11.69 (4.70)	0.45 (1.19)	6.07 (6.60)	$t(82) = 15$, $p \leq .001$
Negative	9.55 (6.84)	0.43 (0.83)	4.98 (6.67)	$t(82) = 8.58$, $p \leq .001$
Depression	18.37 (11.98)	4.81 (5.54)	11.50 (11.43)	$t(75) = 6.39$, $p \leq .001$
Anxiety	23.39 (13.59)	6.36 (6.93)	14.77 (13.69)	$t(58) = 1.67$, $p < 0.001$
Social Functioning	6.83 (1.41)	8.60 (0.59)	7.71 (1.39)	$t(82) = -7.46$, $p \leq .001$

Note. Parental education is the average of mother and father education in years; Positive and negative symptoms are sum scores taken from the Structured Interview for Psychosis-Risk Syndromes (SIPS); Biological sex and race and ethnicity are recorded as counts; Depression and anxiety are sum scores deduced from the Beck Depression Inventory and Beck Anxiety Inventory, respectively; Social functioning scores fall on a 1 (very impaired social functioning) to 10 (no impairments) scale.

Additionally, a subsample of participants had imaging data available (Table 4) (Aim 3). Of this sample, there were no group differences in age or parental education. However, there was a significant difference in the distribution of biological sex. Additional analyses revealed sex was not associated with connectivity patterns but was associated with joy facial expressions when looking at whole group analyses, $r = 0.27$, $p = 0.02$. Within the control and CHR groups, sex was not significantly associated with the noted variables. However, sex was included as a covariate in all analyses. The CHR group, as expected, endorsed more symptoms compared to the control group. A total of 3 individuals in the sample were taking antipsychotic medications. Analyses without individuals taking antipsychotic medications did not change the magnitude or direction of findings; to ensure generalizability of findings, those taking these medications were included in final analyses. There were no significant group differences in the quality of resting-state data in either number of volumes removed for signal outliers, $t(68) = -0.95$, $p = 0.35$ and motion outliers, $t(68) = -1.10$ $p = 0.28$. In terms of behavioral facial expression data, there were group differences in facial expressions of joy only (in line with Aim 2) derived from automated analysis 1-minute clips in that the CHR group displayed more blunted facial expressions of joy compared to controls, $t(68) = -2.93$, $p = 0.005$ (CHR $M = 0.13$, $SD = 0.11$; control $M = 0.24$, $SD = 0.03$).

Table 4. Means and standard deviations for demographics and symptom details in a sample with a CHR syndrome and controls with neuroimaging data collected at University of Colorado (Sample 2)

	CHR	Control	Total	Statistic
Demographics				
Number of participants	37	33	70	
Age	19.24 (1.23)	18.97 (1.63)	19.11 (1.43)	$t(68) = 0.80$, $p = 0.43$
Number of Females	16	24	40	$x^2(1) = 6.19$, $p = 0.01$
Parent Education	15.29 (2.02)	15.28 (2.90)	15.29 (2.46)	$t(62) = -0.04$, $p = 0.97$
Race and Ethnicity				
First Nations	1	0	1	
Asian/Middle Eastern	2	3	5	
Black	1	1	2	

Central/South American	6	12	17	
White	28	17	45	
Symptoms				
Positive	10.91 (4.34)	0.92 (1.71)	6.68 (6.06)	$t(68) = 14.06$, $p < 0.001$
Negative	7.48 (5.62)	1.24 (2.01)	4.79 (5.40)	$t(68) = 7.65$, $p < 0.001$
Depression	19.38 (11.40)	4.18 (4.49)	12.11 (11.61)	$t(67) = 7.39$, $p < 0.001$
Anxiety	19.50 (10.92)	3.97(4.73)	12.07 (11.53)	$t(67) = 7.77$, $p < 0.001$
Social Functioning	6.86 (1.53)	8.58 (0.61)	7.67 (1.46)	$t(68) = -6.02$, $p < 0.001$

Note. Parental education is the average of mother and father education in years; Positive and negative symptoms are sum scores taken from the Structured Interview for Psychosis-Risk Syndromes (SIPS); Biological sex and race and ethnicity are recorded as counts; Depression and anxiety are sum scores deduced from the Beck Depression Inventory and Beck Anxiety Inventory, respectively; Social functioning scores fall on a 1 (very impaired social functioning) to 10 (no impairments) scale.

Aim 1. To investigate if CHR individuals exhibit abnormalities in the experience of positive and negative emotions when compared to controls.

Group differences in target emotional ratings from the evocative film clip task

There were group differences in pre-condition rating in that the CHR group had higher pre-condition amusement ratings compared to controls, $t(81) = 2.05$, $p = 0.04$, $d = 1.90$, as well as more pre-condition sadness ratings compared to controls, $t(81) = 2.10$, $p = 0.04$, $d = 1.77$. There were no other group differences, $p > 0.71$. See Table 5.

Table 5. Group differences in pre-condition ratings

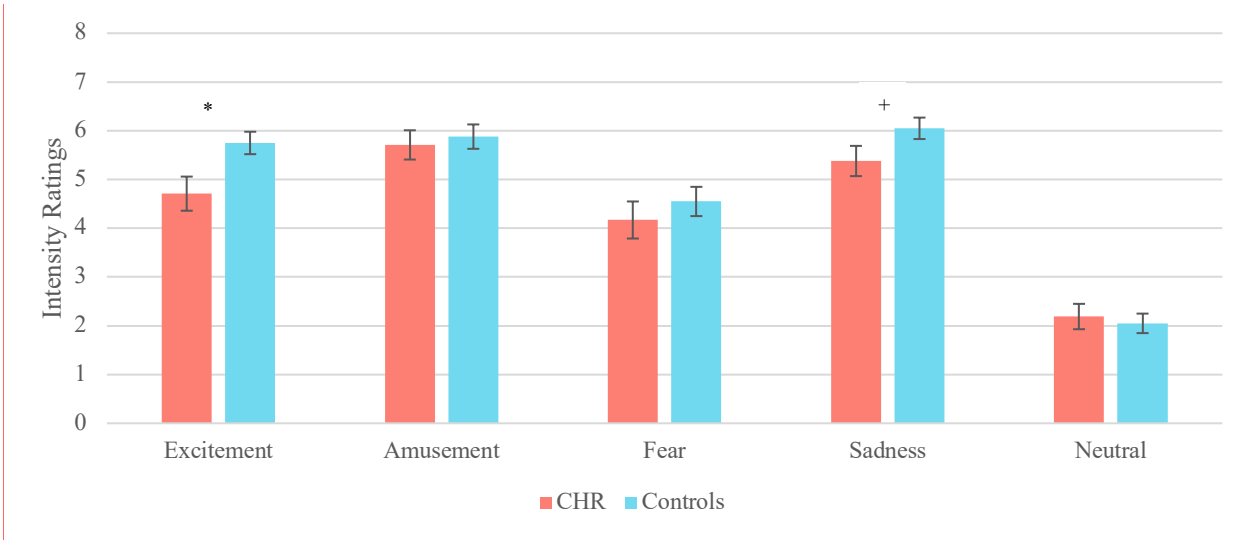
	Pre-excitement	Pre-amusement*	Pre-fear	Pre-sad*	Pre-neutral
CHR	3.88 (2.07)	4.30 (2.03)	1.86 (1.77)	5.40 (2.01)	4.32 (1.43)
Control	3.73 (1.69)	3.45 (1.74)	1.76 (1.37)	6.05 (1.34)	3.65 (1.61)

Note. Pre-condition ratings are on a scale of 1 (no emotion at all) to 8 (strongest emotions felt) – these ratings are collected prior to participants watching evocative film clips in order to get a rating of emotional experience at baseline; *group differences in pre-condition ratings, $p < 0.05$.

To be thorough and ease interpretation of findings, pre-condition ratings were controlled for in all analyses. In the stated analyses, ANCOVAs were employed to investigate group differences in target emotions (e.g., group differences in excitement ratings when viewing the excitement video) when adjusting for pre-condition ratings. In terms of positive emotion ratings

when viewing film clips intended to elicit positive emotions, the CHR group reported significantly lower excitement ratings when viewing the excitement video compared to controls, $F(80) = 5.92$, $p = 0.017$, $\eta_p^2 = 0.07$ (Figure 12). However, there were no significant group differences in amusement ratings, $F(80) = 0.92$, $p = 0.34$. For negative emotion ratings when viewing film clips intended to evoke negative emotions, the CHR group reported reduced ratings of sadness when viewing the sadness film clip at trend level, $F(80) = 3.36$, $p = 0.07$, $\eta_p^2 = 0.04$. However, there were no group differences in fear ratings in response to the fear clip, $F(80) = 0.81$, $p = 0.37$. Additionally, there were no significant group differences in the general emotion ratings in response to the neutral film clip, $F(79) = 0.12$, $p = 0.73$.

Figure 12. Group differences in experience ratings for target emotions derived from an evocative film clip task

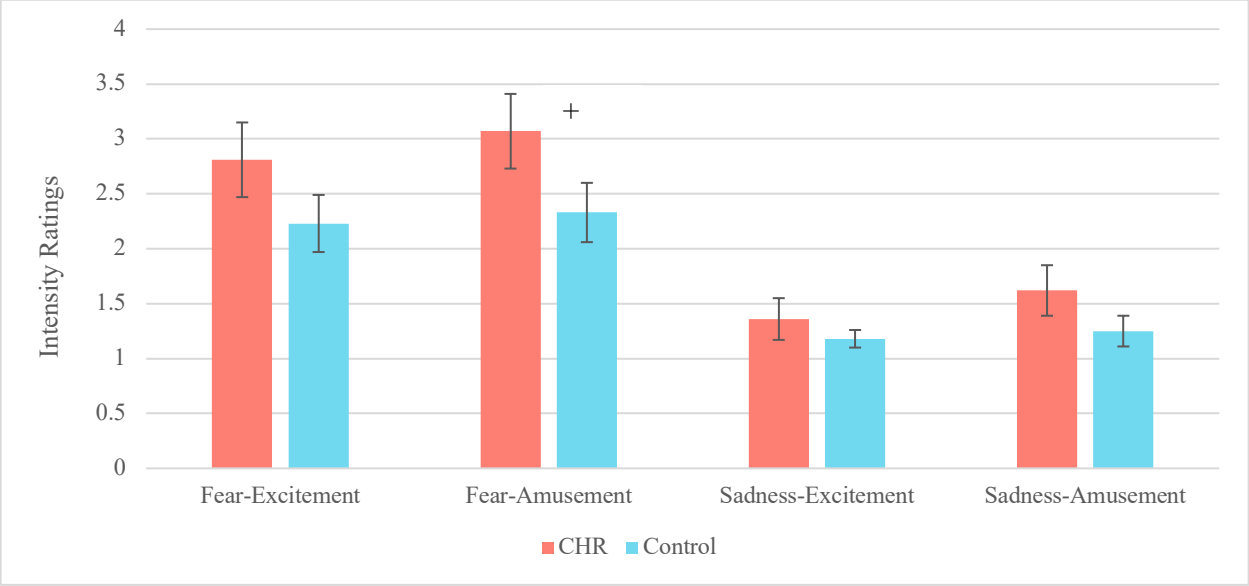


Note. The sample was collected from Northwestern University and include 43 CHR individuals and 40 controls; this image depicts target emotions which are emotions intended to be elicited in the evocative film clip task (e.g., excitement ratings when viewing the excitement film clip, amusement ratings when viewing the amusement film clip, fear ratings when viewing the fear film clip, sadness ratings when viewing the sadness clip); general emotion ratings are depicted when viewing the neutral video clip; intensity ratings reflect scores that fall on a 0 (no emotion at all) to 8 (strongest emotion ever felt) scale and reflect emotional experience; Error bars are standard error; $*p < 0.05$, $+ p < 0.10$

Exploratory analyses of group differences in nontarget emotions from the evocative film clip task

Exploratory analyses were conducted to assess nontarget emotions. Specifically, statistical analyses (i.e., independent t-tests) were employed to examine group differences in (1) ratings of excitement and amusement in response to viewing negative emotional film clips (i.e., clips evoking fear and sadness), and (2) ratings of fear and sadness in response to viewing amusement and excitement clips. In terms of (1) amusement and excitement ratings when viewing negative emotional film clips, there was a trend suggesting higher amusement ratings in response to the fear film clip, $t(80) = 1.72$, $p = 0.09$, $d = 0.37$. There were no other significant level differences (excitement when viewing the fear film clip, $t(80) = 1.37$, $p = 0.17$; amusement, $t(80) = 1.38$, $p = 0.17$, and excitement when viewing the sadness clip, $t(80) = 0.89$, $p = 0.38$. See Figure 13.

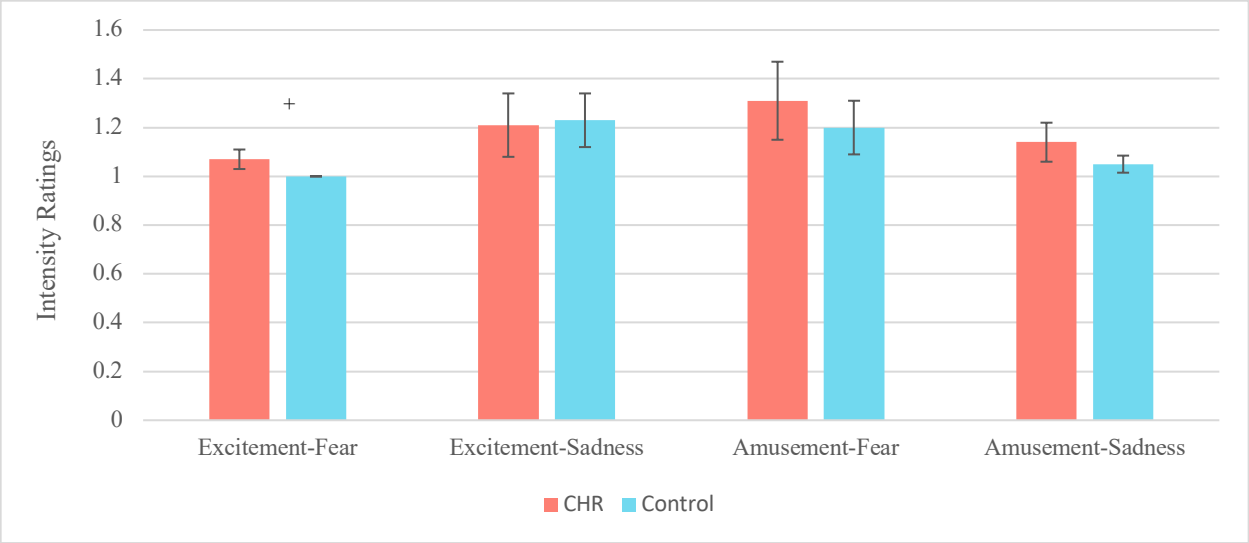
Figure 13. Group differences in excitement and amusement ratings in response to negative film clips



Note. The sample was collected from Northwestern University and include 43 CHR individuals and 40 controls; Video-Rating Category (e.g., Fear-Excitement, fear is the video, and excitement indicates excitement ratings in response to the fear video); Intensity ratings reflect scores that fall on a 0 (no emotion at all) to 8 (strongest emotion ever felt) scale; Error bars are standard error; + $p < 0.10$.

When assessing group differences in fear and sadness ratings when viewing positive emotional film clips (amusement and excitement), findings revealed that the CHR group showed a trend suggesting higher fear ratings when viewing the excitement film clip at a marginally significant level, $t(80) = 1.78$, $p = 0.08$, $d = 0.38$. There were no other differences (fear, $t(80) = 0.56$, $p = 0.58$ and sadness, $t(80) = 1.06$, $p = 0.29$, ratings when viewing the amusement film clip; sadness ratings when viewing the excitement clip, $t(80) = -0.061$, $p = 0.95$). See Figure 14.

Figure 14. Group differences in fear and sadness ratings in repsonse to positive film clips



Note. The sample was collected from Northwestern University and include 43 CHR individuals and 40 controls Video-Rating Category (e.g., Fear-Excitement, fear is the video, and excitement indicates excitement ratings in response to the fear video); Intensity ratings reflect scores that fall on a 0 (no emotion at all) to 8 (strongest emotion ever felt) scale; Error bars are standard error; + $p < 0.10$.

Exploratory analyses examining relationships between target emotion ratings and depression and anxiety

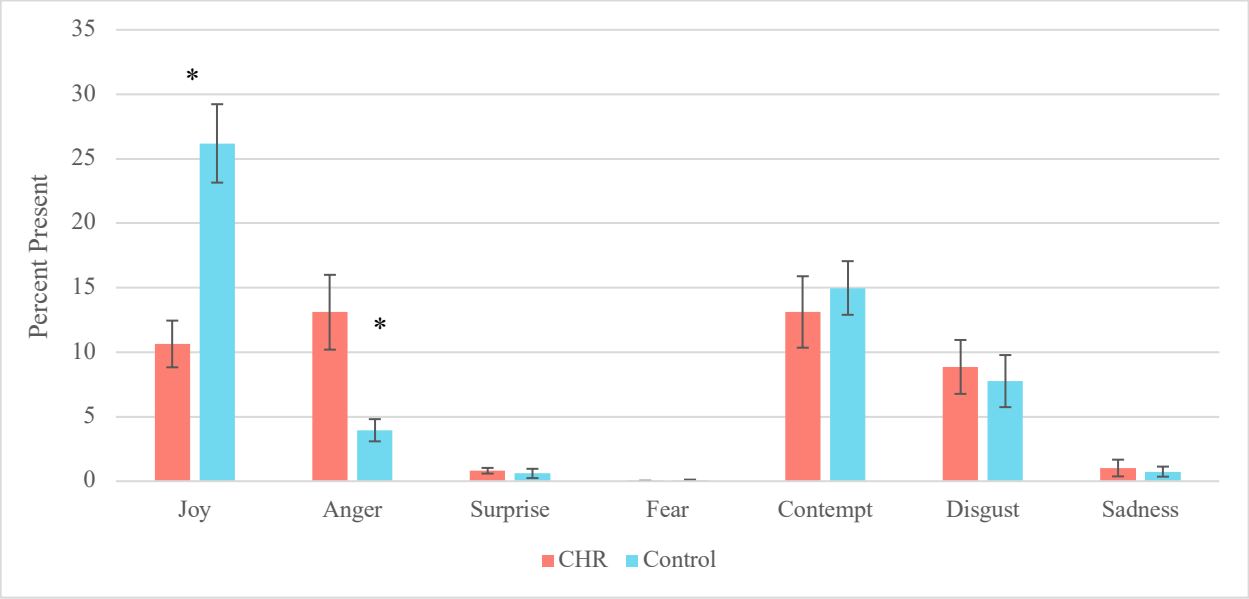
As mentioned, significant group differences in excitement ratings when viewing the excitement film were observed. Partial correlations were employed within the CHR group to investigate relationships between lower excitement ratings and depression and anxiety scores. Findings revealed no significant associations between excitement ratings and depression, $r = 0.13$, $p = 0.45$, and anxiety, $r = -0.02$, $p = 0.92$.

Aim 2. To determine whether CHR individuals exhibit alterations in facial expressions when compared to controls.

Automated facial analysis

When examining group differences in the automated variables to assess for subtle facial expressions, the CHR group showed lower levels of joy expressions, $t(82) = -4.39$, $p < .001$, $\eta_p^2 = 0.12$, and increased anger expressions, $t(82) = 3.02$, $p = .004$, $\eta_p^2 = .09$, compared to the control group. There were no differences between CHR and controls in expressions of surprise, $t(82) = 0.51$, $p = 0.61$, fear $t(82) = -0.82$, $p = .41$, contempt $t(74) = .002$, $p = .96$, disgust $t(82) = 0.38$, $p = 0.71$, or sadness $t(82) = 0.38$, $p = 0.71$. See Figure 15.

Figure 15. Group differences in facial expressions derived from automated analysis



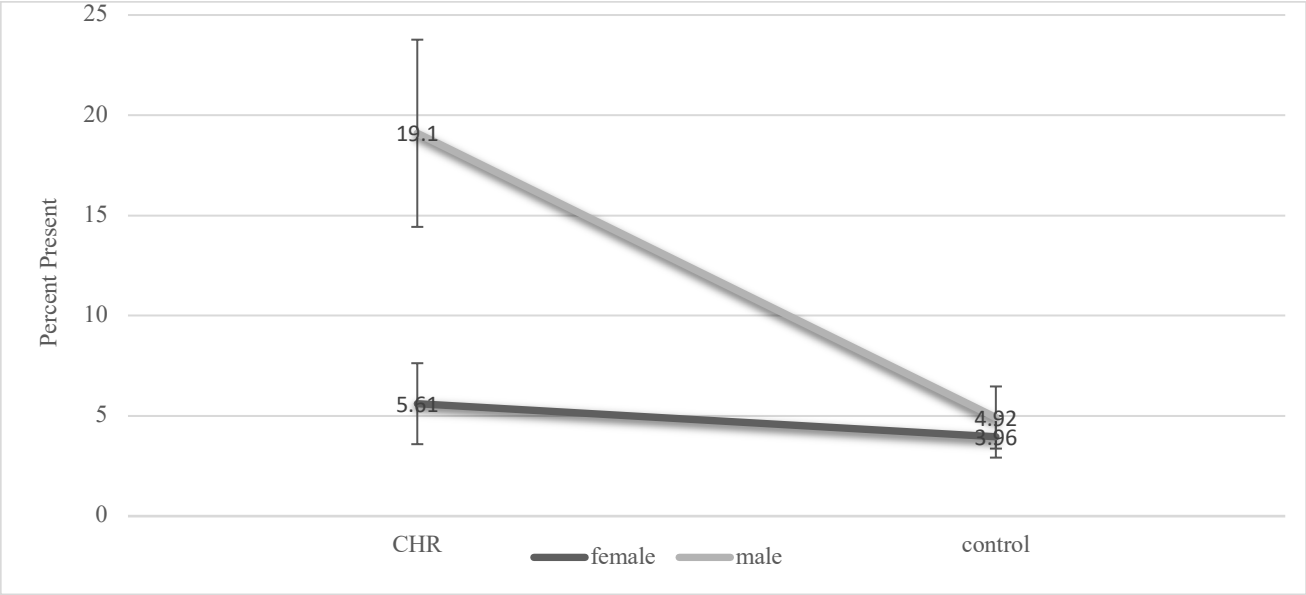
Note. The sample depicted here is from University of Colorado Boulder and include 42 CHR and 42 controls; Scores are based off of the percent likelihood an expression is present (%). *$p \leq .05$. Error bars represent standard error.

Follow-up analyses investigating facial expressions and biological sex

Given demographic details suggesting significant differences in the distribution of biological sex, the influence of sex on facial expression measures derived from automated analysis were examined in exploratory analyses. While there were no findings for the majority of

facial expressions, there was a significant interaction for one expression. Specifically, there was a significant interaction between group (CHR/control) and sex (male/female) predicting increased anger facial expressions derived from the automated analysis, $F(72) = 6.14$, $p = 0.02$, $\eta_p^2 = 0.08$. Post hoc analyses revealed that there were significant differences in anger expressions between CHR and control males in that the CHR males had greater anger expressions compared to the control males, $F(29) = 8.80$, $p = 0.006$, $\eta_p^2 = 0.23$. There were no significant differences between CHR and control females, $F(42) = 0.20$, $p = 0.66$. See Figure 16.

Figure 16. Significant group (CHR, control) x sex (male, female) interaction predicting alterations in facial expressions of anger



Note. The sample depicted here is from University of Colorado Boulder and include 42 CHR and 42 controls; Scores are based off of the percent likelihood an expression is present (%). Error bars represent standard error.

Facial Electromyography

There were no significant group differences in pre-condition facial expressions (i.e., facial expressions collected as individuals watched an "X prior to each video clip); zygomaticus activity, $p > 0.26$, corrugator activity, $p > 0.54$. See Table 6.

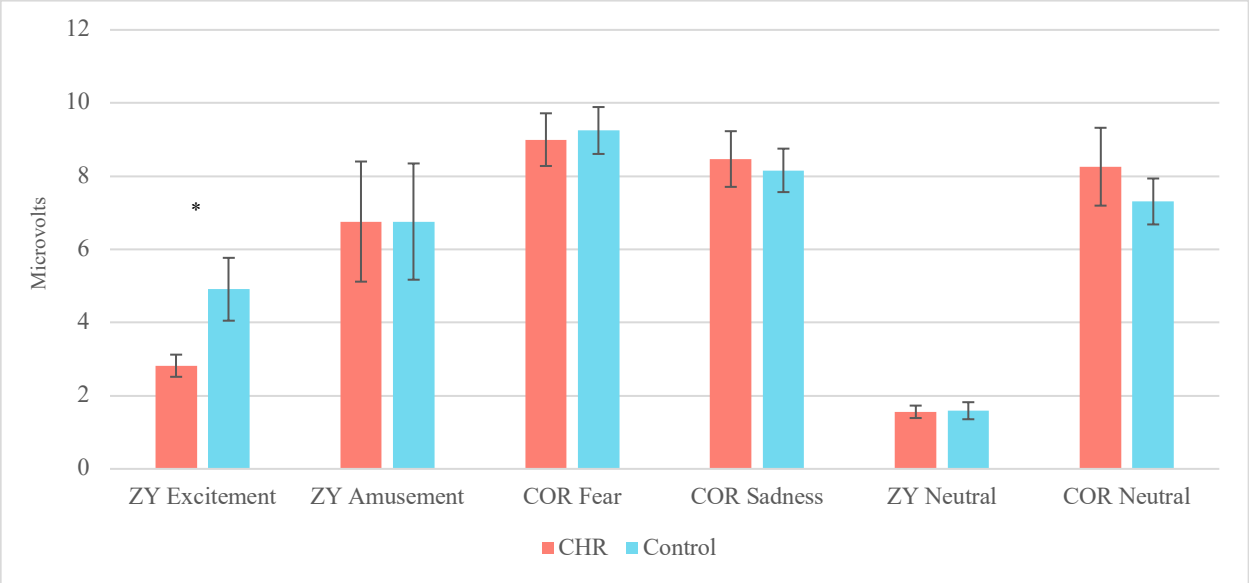
Table 6. Group differences in pre-condition zygomaticus and corrugator activity

	Pre-excitement Zy	Pre-amusement Zy	Pre-sad Cor	Pre-fear Cor	Pre-neutral Zy	Pre-neutral Cor
CHR	1.85 (1.64)	1.76 (1.16)	8.26 (5.72)	7.15 (3.39)	4.32 (1.43)	8.44 (7.33)
Control	2.44 (2.50)	2.44 (4.25)	7.81 (3.95)	7.72 (3.30)	3.65 (1.61)	7.59 (5.00)

Note. Zygomaticus activity (Zy), Corrugator activity (Cor); Pre-condition facial expressivity was collected and involved participants viewing an X right before a specific evocative film clip; Zygomaticus activity is affiliated with smiling and was assess with excitement and amusement videos; corrugator activity is associated with negative emotion and was assessed with fear and sadness videos; Both zygomaticus and corrugator values were deduced for the neutral film clip; values are in microvolts

Despite this and in efforts to be through and ease interpretation of findings, analyses were conducted adjusting for pre-condition ratings. Findings from a series of ANCOVAs revealed, compared to healthy controls, the CHR group displayed reduced zygomaticus activity in response to the excitement film clip, $F(63) = 4.40$, $p = 0.04$, $\eta_p^2 = 0.07$ (Figure 17), but not in response to the amusement, $F(63) = 0.04$, $p = .84$, sadness, $F(63) = 2.49$, $p = 0.12$, and fear, $F(63) = 0.63$, $p = 0.43$ film clips. No significant group differences were found for corrugator activity across film clips, $p > 0.45$. There were also no significant differences in facial activity when watching the neutral film clips, $p > 0.59$.

Figure 17. Group differences in facial activity for target emotions derived from an evocative film clip task



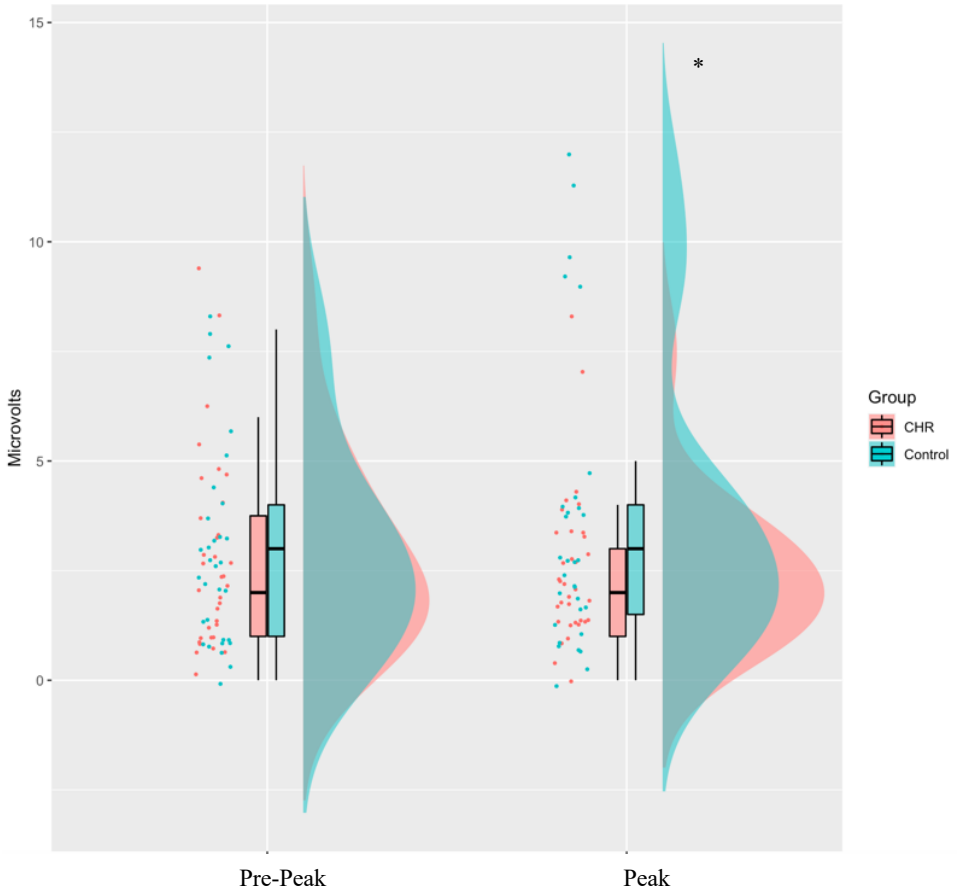
Note. The sample shown here is from Northwestern University and include 34 CHR individuals and 32 controls with available EMG data; Target emotions are emotions intended to be elicited in the evocative film clip task (e.g., zygomaticus activity from viewing excitement and amusement film clips; corrugator activity from viewing fear and sadness film clips); Error bars are standard error; *$p < 0.05$.

Unpacking reduced zygomaticus activity during the excitement clip

To further understand the nature of group differences in zygomaticus activity when viewing the excitement film clip, I analyzed the segment of peak emotional responding – for the excitement film clip as in prior work (Johnson et al., 2017) and lasting about one minute – and compared it to the segment before this peak (i.e., 80 second segment from the start of the film clip up until the start of the peak) termed pre-peak.

Interaction analyses for the excitement film clip, adjusting for baseline zygomaticus activity, revealed a significant group (CHR, control) x facial expressivity (pre-peak, peak) interaction, $F(58) = 7.79$, $p = 0.008$, $\eta_p^2 = 0.12$. Follow up analyses revealed no significant group differences in the pre-peak segment, $F(62) = 0.22$, $p = 0.64$, but did show a significant difference during the peak in that the CHR group exhibited less zygomaticus activity during the peak segment of the clip compared to controls, $F(59) = 4.79$, $p = 0.03$, $\eta_p^2 = 0.08$. See Figure 18.

Figure 18. Facial expressivity across time segments



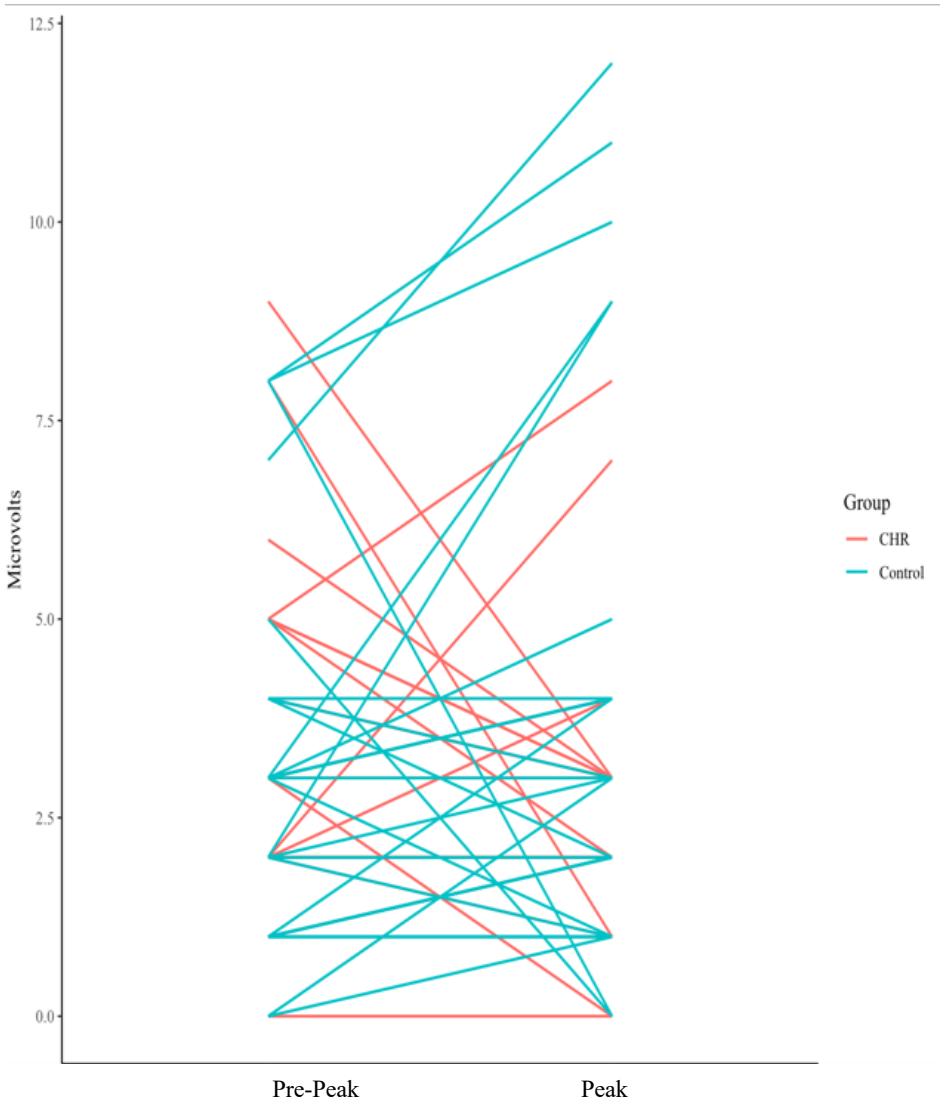
Note. Raincloud plot depicting distribution of zygomaticus activity (in microvolts) when viewing the excitement clip across time segments; Second segment is termed peak (i.e., the clip depicting very high levels of emotion); Pre-peak is the period before; *$p < 0.05$

Rate of Change in Facial Expressivity

Additional analyses were conducted in efforts to deduce the average rate of change in zygomaticus activity between the CHR group and healthy controls across the two excitement film clip segments (pre-peak subtracted from peak). As expected, there were significant group differences in the rate of change in that the CHR group had a lower amount of rate of change

during the excitement video, $F(58) = 7.79$, $p = 0.007$, $\eta_p^2 = 0.12$, CHR $M = 0.05$, SD = 2.00, Control $M = 1.00$, $SD = 2.00$. See Figure 19.

Figure 19. Rates of change for each individual in the facial activity



Note. Rate of change was computed by taking the difference between pre-peak and peak; each individual participant pre-peak and peak data is plotted here.

Exploratory analyses of group differences in nontarget emotions from the evocative film clip task

There were no group differences in corrugator activity (when adjusting for pre-condition corrugator activity) when viewing the excitement film clip, $F(64) = 0.33$, $p = 0.57$, and amusement clip, $F(64) = 0.08$, $p = 0.78$. Additionally, there were no group differences in zygomaticus activity (when adjusting for pre-condition zygomaticus activity) when viewing the fear film clip, $F(64) = 0.64$, $p = 0.43$, sadness clip, $F(64) = 2.74$, $p = 0.10$, and disgust film clip $F(64) = 0.95$, $p = 0.34$.

Exploratory analyses investigating alterations in facial expressions and depression and anxiety

Bivariate correlations investigating relationships between reduced joy expressions and depression and anxiety from automated analyses revealed no significant associations, $r = -0.30$, $p = 0.07$, $r = -0.15$, $p = 0.35$, respectively. There were also no significant group differences between increased anger expressions derived from automated analysis and depression, $r = -0.09$, $p = 0.59$ and anxiety, $r = 0.11$, $p = 0.50$. Similarly, partial correlations assessing relationships between zygomaticus activity when viewing the excitement video clip (adjusting for baseline facial activity) and depression, $r = -0.14$, $p = 0.47$, and anxiety, 0.06, $p = 0.76$, revealed no significant results.

Aim 3. To examine whether alterations in facial expressions are related to dysconnectivity between (a) emotion regions (e.g., amygdala, crus I/II), motor regions (e.g., cerebellar bilateral lobules IV, primary and secondary motor areas), or both emotion and motor regions.

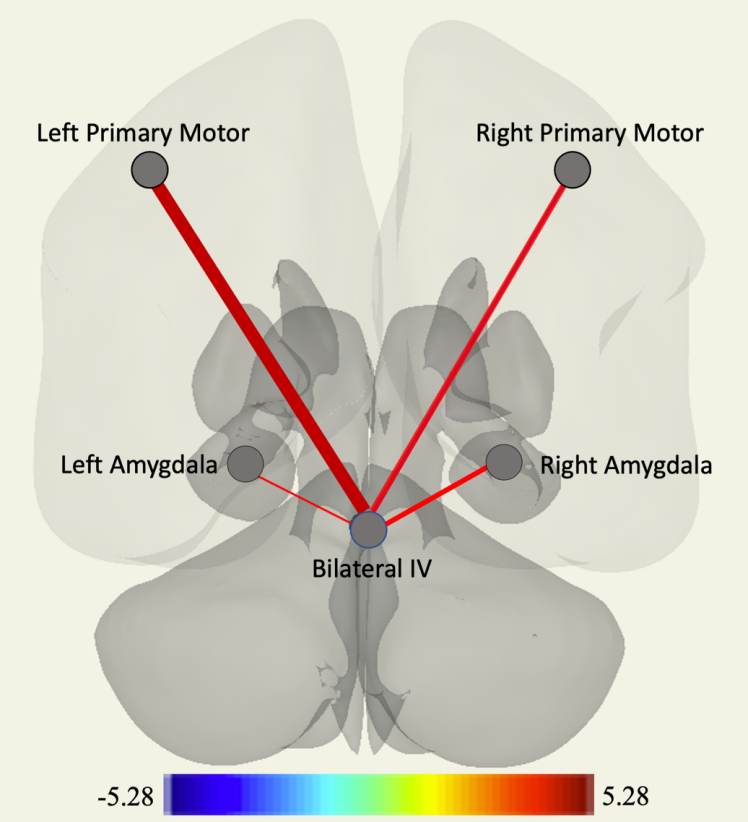
ROI-ROI Analyses

As mentioned, there were group differences in joy facial expressions derived from FaceReader's automated analysis software and as such, ROI-ROI analyses were employed with

joy facial expressions specifically. Findings from ROI-ROI analyses (within the CHR group only) revealed there was an positive association between facial expressions of joy and connectivity between motor-motor regions including cerebellar bilateral lobules IV to left primary motor area, $t(34) = 5.28$, $p_{FDR} = 0.002$, $r = .67$, $p < 0.001$ and cerebellar bilateral lobules IV and right primary motor area, $t(34) = 4.12$, $p_{FDR} = 0.03$, $r = 0.58$, $p < 0.001$. There were also significant positive associations between joy facial expressions and connectivity between motor-emotion areas; cerebellar bilateral lobules IV - left amygdala, $t(34) = 3.84$, $p_{FDR} = 0.03$, $r = 0.56$, $p < 0.001$, and cerebellar bilateral lobules IV - right amygdala, $t(34) = 3.92$, $p_{FDR} = 0.03$, $r = 0.55$, $p = 0.001$. See Figure 20.

Figure 20. Significant associations between facial expressions of joy and connectivity between regions in a CHR sample

B. Results



A. Regions entered into model

Motor
L/R caudate
L/R putamen
L/R thalamus
L/R primary motor area
L/R secondary motor area
L/R caudal middle frontal gyrus
Cerebellar bilateral lobules IV
Cerebellar vermis IIIa/vermis IIIb
Emotion
L/R caudal anterior cingulate
L/R medial orbitofrontal cortex
L/R rostral anterior cingulate
L/R amygdala
Cerebellar crus I/II

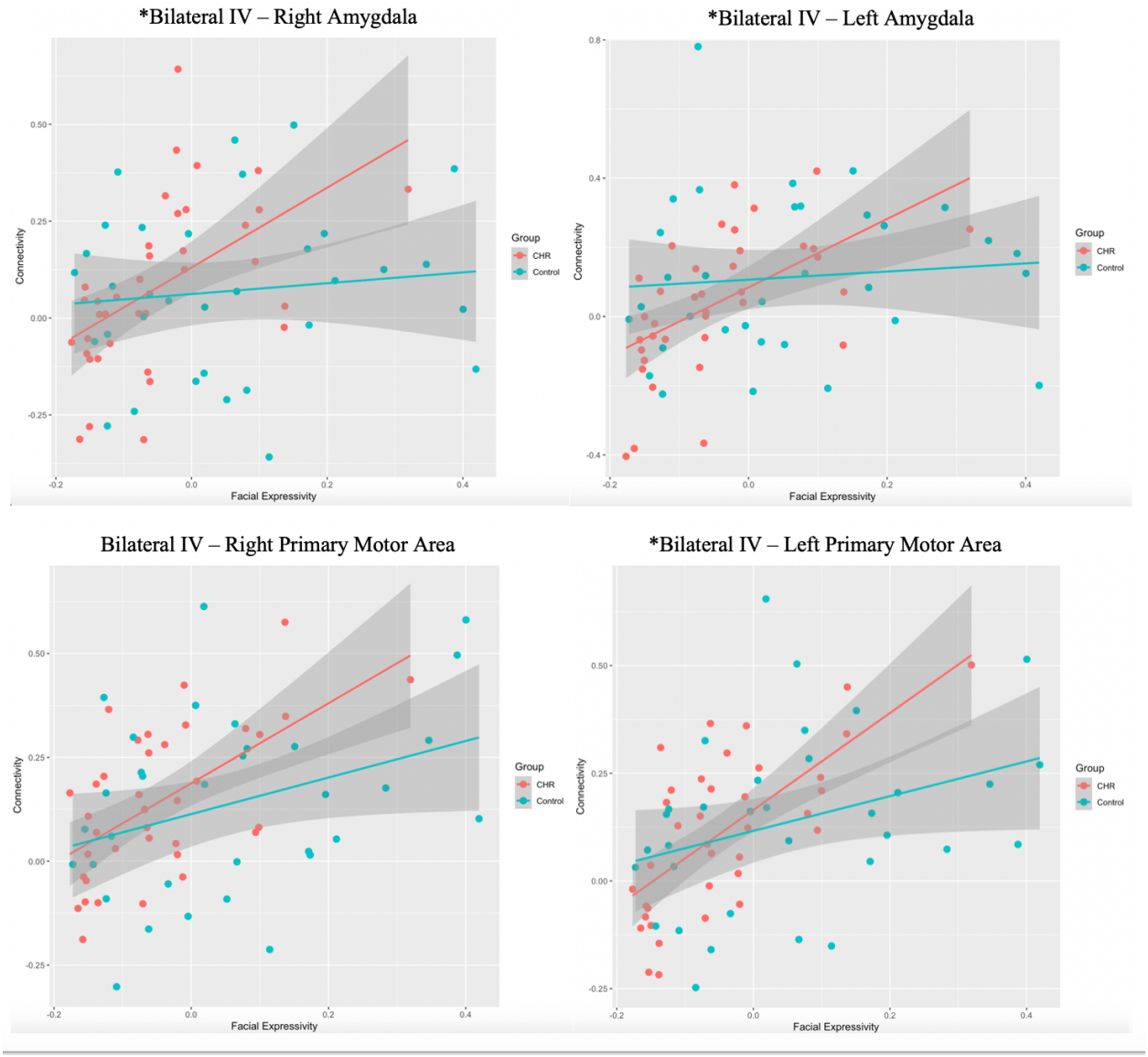
Note. Facial expressions of joy are derived from FaceReader; L = left, R = right; A. Regions entered into the model, B. Color bar indicates t-values; Line thickness reflects magnitude (thicker lines indicate larger effects); Results are

thresholded at the voxel-level at $p_{uncorrected} < 0.001$ for cluster formation, and then corrected at the cluster-level using a false-discovery rate (FDR) of $p < 0.05$.

Group comparisons of facial expressions and connectivity

To unpack ROI-ROI results, within group correlations were employed to investigate associations between facial expressivity and connectivity patterns in the CHR group, but also a comparison control group. Within the CHR group, as defined in exploratory analyses, there were positive associations between facial expressivity and connectivity between cerebellar bilateral lobules IV - left primary motor areas, $r = .67$, $p < 0.001$ and right primary motor area, $r = 0.58$, $p < 0.001$ while in the control group, these results were nonsignificant, left: $r = 0.30$, $p = 0.10$, right: $r = 0.32$, $p = 0.07$, respectively. When investigating group differences in associations between facial expressions and connectivity between cerebellar bilateral lobules IV to right/left primary motor area, there was no significant group difference between facial expressivity and connectivity between cerebellar bilateral lobules IV and right primary motor, $z = 1.32$, $p = 0.09$, but there was for the correlation between facial expressivity and connectivity between cerebellar bilateral lobules IV and left primary motor area, $z = 2.00$, $p = 0.02$. Furthermore, within the CHR group, facial expressivity was related to reduced cerebellar bilateral lobules IV - left amygdala connectivity, $r = 0.55$, $p = 0.001$, and cerebellar bilateral lobules IV - right amygdala, $r = 0.56$, $p < 0.001$, while these associations were nonsignificant in the control group, $r = 0.18$, $p = 0.31$, $r = 0.09$, $p = 0.62$, respectively. There was a group difference in the relationship between facial expressivity and cerebellar bilateral lobules IV- left amygdala connectivity, $z = 1.74$, $p = 0.04$ as well as cerebellar bilateral lobules IV - right amygdala connectivity, $z = 2.17$, $p = 0.015$. See Figure 21 for group associations.

Figure 21. Group differences in associations between facial expressions and connectivity patterns



Note. Specific region-region connectivity patterns are shown from analyses within the clinical high-risk (CHR) group; Connectivity beta values are reflected on y-axis; Facial expressivity deduced using an automated facial analysis tool are depicted on x-axis and values fall on a 0-1 scale with higher values indicating more facial expressivity; * reflect significant differences between groups in associations between facial expressions and connectivity, $p < 0.05$.

Follow-up analyses assessing group difference in those with low and high facial expressivity

Furthermore, exploratory analyses were conducted to examine if these effects were driven by emotional blunting (low expressivity in CHR) or high levels of facial expressivity. In the group with higher levels of facial expressivity (CHR $N = 14$, control $N = 21$), individuals with a CHR syndrome showed significantly less facial expressivity compared to the control group, $F(32) = 4.33$, $p = 0.046$, $\eta_p^2 = 0.12$ while there were no significant differences in the low facial expressivity group between CHR (N = 23) and controls (N = 12), $F(32) = 0.17$, $p = 0.69$.

When assessing differences in connectivity values between CHR individuals with high facial expressivity and controls with high facial expressivity, there was a significant difference, $F(32) = 4.70$, $p = 0.038$, $\eta_p^2 = 0.13$ in that the CHR group had higher connectivity values between cerebellar bilateral lobules IV and left amygdala compared to controls. There were no other significant differences in connectivity levels in the high and low facial expressivity groups, $p < 0.22$. See Table 7 for effect sizes of mean group differences in connectivity.

Table 7. Cohen's *d* Effect size of mean difference in connectivity between CHR and control groups

	Bilateral lobules IV – L Amygdala	Bilateral lobules IV – L Primary Motor	Bilateral lobules IV – R Amygdala	Bilateral lobules IV – R Primary Motor
Low	0.17, CI [-1.2, 0.21]	0.17, CI [-0.6, 0.8]	0.21, CI [-1.55, -0.1]	0.17, CI [-0.6, 0.8]
High	*0.21, CI [0.17, 1.59]	0.19, [-0.06, 0.75]	0.18, CI [-0.24, 1.14]	0.21, CI [-0.42, 0.94]

Note. A median split in behavioral facial expressivity data was applied in order to categorize individuals (CHR, control) into low and high facial expressivity groups; "Low" represents comparisons between CHR individuals with low facial expressivity and controls with low facial expressivity; "High" represents comparisons between CHR and controls with high facial expressivity; L = left, R = right; CI = confidence intervals; *$p < 0.05$.

Aim 4. To assess whether abnormalities in the (a) experience ratings from the evocative film clip task and (b) facial expressivity alterations derived from automated facial analysis and facial EMG are related to social functioning and likelihood of conversion to psychosis (based off of risk calculator scores).

Associations with experience ratings

As noted, the CHR group reported less excitement ratings when viewing the excitement video clip when compared to controls. Partial correlations were employed within the CHR group, when adjusting for baseline ratings, to investigate relationships between alterations in emotional experiences of excitement and social functioning and psychosis risk conversion scores. Findings indicated no significant relationships between excitement ratings when viewing the excitement film clip and social functioning, $r = -0.16$, $p = 0.40$, and risk scores, $r = -0.21$, $p = 0.50$.

Associations with automated facial analysis variables

Reduced joy expressions derived from automated facial analysis were significantly related to social functioning, $r = 0.37$, $p = 0.02$, and increased likelihood of conversion based off of psychosis risk conversion scores, $r = -0.37$, $p = 0.02$. Increased anger expressions were not significantly related to social functioning, $r = 0.02$, $p = 0.90$, and psychosis risk scores, $r = 0.11$, $p = 0.51$. See Figure 22 and 23.

Figure 22. Facial expressivity derived from iMotions and psychosis-risk conversion scores within the CHR sample

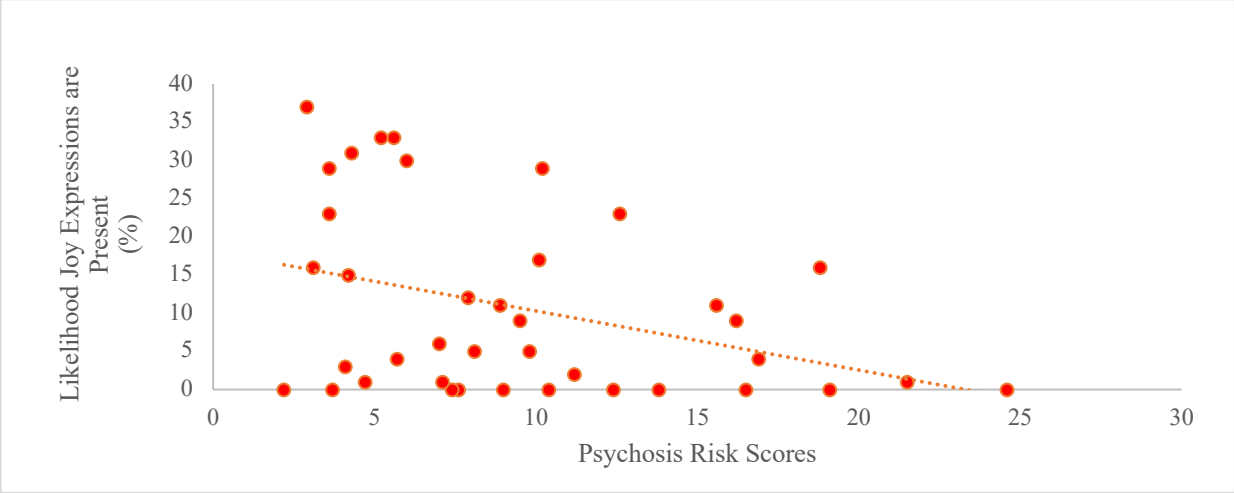
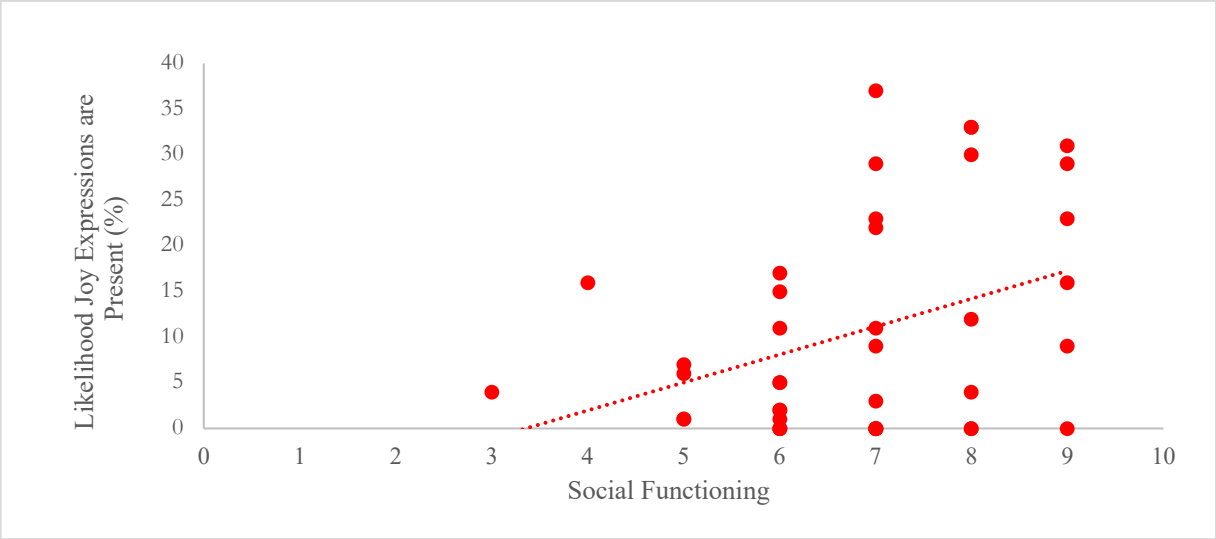


Figure 23. Facial expressivity derived from iMotions and social functioning scores within the CHR sample



Associations with facial EMG

When adjusting for baseline zygomaticus activity, within the CHR group, findings indicated no significant relationships between zygomaticus activity during the excitement clip and psychosis risk conversion scores, $r = 0.09$, $p = 0.64$ and social functioning scores, $r = 0.05$, $p = 0.81$. There were no significant associations observed between zygomaticus activity during the peak segment of the excitement clip and psychosis risk conversion scores, $r = -0.17$, $p = 0.39$ or social functioning scores, $r = -0.12$, $p = 0.57$. However, lower rate of change during the excitement clip was related to greater likelihood of conversion to psychosis, $r = -0.58$, $p = 0.002$, but not social functioning, $r = -0.07$, $p = 0.74$.

CHAPTER VI.

DISCUSSION

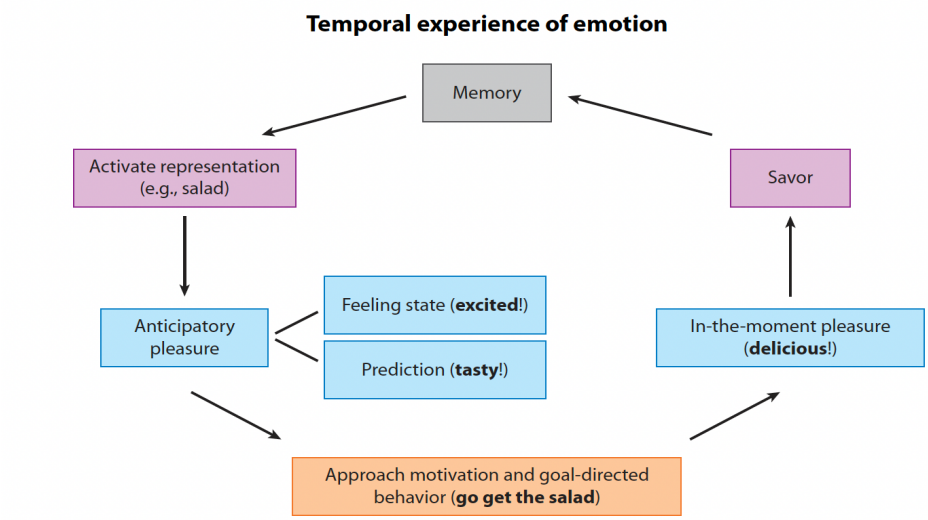
Overview

The current dissertation sought to unravel unknown questions related to emotional processes in those with a CHR syndrome when compared to controls. Results revealed that the individuals at CHR for psychosis reported lower excitement ratings when viewing a film clip intended to evoke excitement. Furthermore, the assessment of subtle and micro facial expressions revealed those with a CHR syndrome showed blunted facial expressions of joy (note: increased anger was also observed from automated facial analysis findings) and excitement (deduced from zygomaticus activity when viewing an exciting film clip), respectively. Furthermore, assessment of micro facial expressivity allowed for the ability to also look at timing, in which it was found that blunting was occurring specifically during the most evocative section of the film clip, and not the period prior. Additionally, it appeared those with a CHR syndrome were not showing variability in facial expressivity when viewing the excitement film clip. Analyses disentangling associated neural circuitry suggested that both motor and emotion regions may be implicated in alterations in facial expressions and this may be particularly driven by CHR individuals with higher levels of facial expressivity. Finally, reduced joy facial expressions derived from automated facial analysis were found to relate to psychosis risk conversion scores and social functioning impairments. Together, these dissertation aims and results are novel and suggest that alterations in the experience and expression of emotion occur prior to the onset of psychosis. Additionally, these results inform emotion related conceptualizations, the pathogenesis of psychosis, and shed light on biomarker research in this area as well as the development of intervention and prevention strategies for this group.

Evidence of lower excitement ratings when viewing the video clip intended to evoke excitement in the CHR group compared to controls (Findings from Aim 1)

Experience ratings. Findings suggested CHR individuals reported significantly lower excitement ratings in response to the excitement video clip, but this was not observed for other facial expressions of emotions. These data are in line with evidence of previous studies suggesting individuals with a CHR syndrome endorse reductions in reports of positive emotional experience. For example, studies have found reductions in self-report ratings of empathy (Montag et al. 2020), ratings of trait level positive affect (Yee et al., 2019), and positive reactivity in response to positively valences photos (Gruber et al., 2018). Furthermore, the findings with excitement suggests that emotional abnormalities may be unique to excitement. Importantly, these data provide insights on both valence and arousal, with the possibility that those with a CHR syndrome may exhibiting a dampening in arousal during the excitement film clip. One additional possible interpretation of reduction in excitement ratings when presented with the excitement video stems from theories of anhedonia in schizophrenia that suggest difficulties with reward signaling may be at the core of reductions in positive emotional experiences as seen in experimental paradigms (Schlosser et al., 2014). As depicted in Figure 24,

Figure 24. An example of the temporal experience of emotion conceptualizations



Note. Taken from Kring & Elis, 2013

on the simplest level, the experience of a positive event such as eating something delicious is savored, stored in memory, and the active representation is used to anticipate future events related to eating, which guides approach motivation and goal-directed behavior (Kring & Elis, 2013). It is possible that this cycle is disrupted in those with a CHR syndrome. If perhaps this theory is on track in explaining reduced positive emotional experiences, the exact point in which the breakdown occurs in this cycle is unclear and future studies should examine this. For speculation purposes, one hypothesis is that the ability to savor the positive experience is

disrupted, and thus the experience of eating is not stored in memory and as a result, not recollected. An important future direction of this work will be to understand whether disruptions are observed in the anticipation of positive experiences as well.

However, findings of lower positive emotional experience in those with a CHR syndrome are largely in contrast to what is observed in schizophrenia. Studies of schizophrenia find similar levels of positive emotion ratings in response to photo or video stimuli when compared to those without the disorder, on average (Kring & Elis, 2013). These findings from the literature are challenging to interpret, raising the question of why do we see lower ratings of excitement in CHR groups but not in studies of schizophrenia? This question is largely understudied in the CHR and schizophrenia literatures and one in which further research is warranted. Of the limited work that does exist, there continues to be emerging results of reductions in positive emotional experiences (Strauss et al., 2017; Jhung et al., 2016) in which the phrase "schizophrenia spectrum anhedonia paradox" has been coined (Pelizza et al., 2020). This paradox suggests less severe pathology (i.e., CHR) show impairments in experiences of positive emotions where more severe forms (i.e., schizophrenia) do not. There are several additional possible interpretations of these findings discussed below. While the discussion below focuses on theoretical differences that may explain this "schizophrenia spectrum anhedonia paradox," it is also possible that methodological differences may be at play as well. Additionally, it is important to note it was not possible to test differences in emotion related variables between CHR and schizophrenia samples in this dissertation but this will be important future direction of this work.

Comorbidity. One factor that is discussed in the literature within the context of emotion in CHR is the role of comorbidity. For the purposes of speculation, it is tempting to explain inconsistent findings (i.e., reports of lower positive emotion ratings observed in the CHR

literature, normative levels of positive emotion in schizophrenia) (Gruber et al., 2018b; Kring & Elis, 2013a; Yee et al., 2019; Yee et al., 2010) as a product of comorbid diagnose such as anxiety and depression commonly observed in CHR groups. While this may be the case, correlational analyses in the current study with anxiety and depression did not support this potential theory (note: this could be due to power). However, it is important to note that only panic type symptoms of anxiety were assessed with the BAI and depression was only assessed with the BDI. As such, it is possible there may be other contributing comorbid related factors that were not fully addressed in the current study (e.g., generalized anxiety disorder, social anxiety disorder, post-traumatic stress disorder, persistent depressive disorder, bipolar disorder) in which it will be critical to further studies to examine in the future. Aside from the noted limitation, the findings suggesting no links between anxiety and depression and emotional experience are not in line with Gruber and colleagues (2018) in which relationships between lower reports of positive emotions in response to pleasant, static stimuli were related to scores of anxiety (BAI) and depression (BDI). Gruber and colleagues (2018) conducted their study with our ADAPT University of Colorado sample, whereas the current study was conducted using the Northwestern Sample. Along these lines, it may be that there are unique characteristics of the different samples that contribute to contrasting results as well as other methodological related issues, aside from potential theoretical explanations provided. For example, there may be differences due to geographic location (Boulder vs Chicago) and including individuals endorsing high levels of cannabis use (perhaps higher rates in Boulder given legalization occurred during the time of data collection). More research is needed with larger sample sizes to better understand these relationships.

Medications. Furthermore, individuals with a CHR syndrome are often not at the point where they are taking several medications, which is one reason studying emotional experience, amongst other risk signs and symptoms, in those with a CHR syndrome can be valuable (i.e., findings are often not convoluted by medication effects). It is possible that medications as such have impacts on the mechanisms underlying the experience of emotion. However, only one individual was taking an antipsychotic medication in this sample. Other studies have also found lower positive emotion ratings in samples in which individuals with a CHR syndrome were neuroleptic-free (e.g., Gruber et al., 2018). It could be that reduced positive emotional experience in CHR groups, in which these individuals can be generally neuroleptic-free, are reflecting emotion abnormalities which becomes normalized in later stages of illness due to antipsychotic medications and therapeutic intervention (Strauss et al., 2018). More research, however, is needed before definitive conclusions can be made.

Trends in reports of sadness. Results suggesting trend level group differences in sadness experience ratings in response to the sadness clip are difficult to interpret. Given that effect sizes for this difference were small and findings were only marginally different, interpretations should be made with caution. In future studies with larger sample sizes, it may be noteworthy to discuss trends with sadness. In particular, in this study, the CHR group reported lower sadness ratings than the control group in response to viewing the sadness video clip. Similarly, the CHR group endorsed a pattern of lower ratings in amusement and fear (target emotional ratings. Future work is needed with larger sample sizes in order to tease apart if there is in fact a generalized emotion deficit (i.e., is there an overall reduction in emotions or is this truly unique to excitement?). This hypothesis would be in line with previous work in this area suggesting overall reduced emotional

reactivity in those with a CHR syndrome (from a study using picture presentations) (Gruber et al., 2018).

Trend with nontarget emotions. As mentioned, exploratory analyses investigating nontarget emotions (i.e., excitement ratings in response to the fear clip) revealed a trend level difference in that the CHR group reported higher amusement ratings when watching the fear clip compared to the control group. Interesting, similar with trend level findings noted above, there is a pattern in which overall, CHR individuals have higher ratings, descriptively speaking, compared to controls on positive ratings in response to the fear clip and the sadness clip. Similarly, there was a trend level difference for fear ratings in response to excitement video in that the CHR group is showing a pattern of more fear ratings in response to the excitement film clip than the control group. Although these findings are trends, there may be possible future directions that are warranted from these notable patterns. For example, it will be critical for future studies to continue to investigate nontarget emotions in CHR groups which are defined more generally as emotions that are incongruent with what we would expect one would feel in response to a situation (Berrios et al., 2015; Ong et al., 2018). To further elaborate, nontarget emotions in particular may be detrimental to overall wellbeing (e.g., feeling happiness when sadness would be expected). Target emotions, stemming from functional theories of emotion generation (Levenson et al., 1999) suggest that matching emotions that are expected in a situation involves the ability to process incoming information such as sensory cues and inputs from a situation, establish which information is most relevant of the information being processed, and match this association with relevant information to prototypes of emotions (Stephens et al., 2022). This information can then guide appropriate response tendencies (which

have been evolutionarily selected as responses that are effectively dealt with in a certain situation) (Levenson et al., 1999).

With nontarget emotions, there may be difficulties in the ability to tease apart relevant and irrelevant information. It will be imperative to decipher in future work if there are in fact group differences in nontarget emotions in CHR groups. If so, it will be beneficial for additional research to examine whether findings are better explained by difficulties in deciphering irrelevant vs. relevant information or if this is a normative response in which an individual is experiencing a wide array of emotions (which can aid perspective taking) and can be adaptive. For example, it could be perhaps the case that individuals with a CHR syndrome are experiencing more fear in response to the film clip (and that may explain reduced levels of excitement in this group when compared to controls) which is in line with studies in schizophrenia (Cohen & Minor, 2010). However, these interpretations are highly speculative and future data is needed with larger samples before conclusions can be drawn.

Taken together, it is possible that the evaluation of a positive experience may be particularly faulty prior to the onset of psychosis among those with a CHR syndrome. Given that studies of positive emotional experiences in schizophrenia find reports similar to controls (Kring & Moran, 2008), reduced reports of positive emotional experiences in CHR groups may be a unique feature that can be perhaps targeted with intervention strategies (e.g., behavioral activation, cognitive training). While the exact mechanism of reduced reports of positive emotional experience is not well-understood, it will be critical for future work to continue to unravel this mystery. It will also be beneficial to investigate the role of cognition in the experience of positive emotion such as memory (Barch, 2005). Additionally, further research is needed to tease apart whether other comorbid diagnoses explain findings of reduced reports of

positive emotional experiences and nontarget emotions (Addington et al., 2017a; Fusar-Poli et al., 2014).

Evidence of alterations in facial expressions in the CHR group when compared to controls derived from automated facial analysis and facial electromyography (Findings from Aim 2)

Automated facial analysis. Findings suggesting CHR individuals displayed alterations in facial expressions derived from automated facial analysis contribute to the growing literature investigating emotional processing and are consistent with evidence among schizophrenia populations (Kring & Elis, 2013; Kring, Gur, Blanchard, Horan, & Reise, 2013). Furthermore, these data provide additional evidence for the utility of automated analysis as a means to assess facial expressions among CHR groups. In particular, automated analysis revealed additional data that would not have been available with clinical interviews alone. Specifically, it was found that CHR individuals displayed blunted facial expressions of joy, but increased anger expressions compared to controls. It is important to note while the clinical interview section that was submitted for analysis consisted of a neutral segment (e.g., discussing background information), the automated analysis program did not offer the ability to assess the degree to which individuals showed neutral facial expressivity and this will be necessary to examine in the future. Despite this, reductions in joy facial expressions are in line with studies suggesting those with a CHR syndrome exhibit reduced joy expressions (Walker et al., 1993) and negative symptoms such as blunted affect, more broadly (Gupta et al., 2021; Kirkpatrick et al., 2006; Piskulic et al., 2012; Strauss et al., 2020). Some interpretations of these findings may be that individuals with a CHR syndrome are experiencing cognitive overload and may not have the cognitive resources to show facial expressions of joy (Strauss & Cohen, 2017). Alternatively, given the neutral nature of the video segment as discussed, those with a CHR syndrome may not in fact be experiencing a

cognitive load but may be experiencing anxiety or other types of emotional experiences that might explain reductions in joy facial expressions. Clinical visits can evoke anxiety and it is possible a new environment may be threat activating. It will be imperative for future studies to assess facial expressivity and the experience of emotions in conjunction to better understand the interplay between internal experience and the facial expression of emotion similar to the series of studies conducted by Kring and colleagues (1999), although the degree to which internal experience and expression co-occur has been recently up for debate (Barrett et al., 2019) . Altogether, these findings provide a nuanced perspective. Furthermore, these data highlight the potential for automated facial analysis to detect increased frequency of emotion in some domains, in contrast to clinical interviews, which emphasize symptom deficits.

Findings of increased anger facial expressions in the CHR group when compared to controls are in line with a previously noted prospective study in which boys and girls that went on to develop schizophrenia expressed more negative (e.g., anger, sadness) emotion compared to siblings. Furthermore, this finding is in line with a study in which individuals with a CHR syndrome reported increased self-reported experiences of negative emotions (Yee et al., 2019). Even so, results indicating increased anger among the CHR group are still difficult to interpret. Given work from Horan and colleagues (2006) suggesting that patients with schizophrenia have difficulties inhibiting the experience of negative emotion, it is possible that these interpretations may translate to the expression of negative emotion among those with a CHR syndrome, representing a larger emotion regulation deficit. Alternatively, increased anger expressions could be more representative of difficulties in concentration or regulating cognitive demand given that the muscle movements of anger and concentration overlap on an action unit level (i.e., specific facial muscle movements) (Ekman & Friesen, 1978). One critical point to make is that one can

speculate that perhaps reductions in joy expressions may just reflect an increase in anger expressions. However, descriptively speaking, the CHR group is showing almost equal amounts of joy and anger facial expressions. Future work is warranted to understand facial expressions in both positive and negative emotion among this group.

Automated facial analysis: exploratory analyses with biological sex. When exploratory analyses were conducted to determine sex differences in facial expression measures derived from automated analysis, findings indicated that CHR males showed greater anger expressions compared to control males. This is in support of the sensitivity of automated analysis of facial expressions in picking up these differences that could have been missed with clinical interviews. Replication and additional studies are needed to provide information regarding the impacts of biological sex on negative symptoms. It is possible that there are unique sex differences in facial expressivity; however given the small sample sizes and the exploratory nature of the findings, it will be important for future studies to continue to understand these differences before definitive conclusions can be made. For speculation purposes, in recent work (Gupta, Strauss, et al., 2021), it would found that depression explained a large portion of the variance in negative symptom domains including anhedonia while serotonin reuptake inhibitor use explained the largest variance in alogia (e.g., reduction in quantity of speech). While differences unique to facial expressions were not found (and this could be due to the use of the SIPS which has limitations in truly isolating facial expressions), it is possible that secondary sources may be playing a role in facial expressions in the context of biological sex. However, this was not directly tested and additional research in this area is warranted to better understand if there is in fact a true signal.

Facial electromyography. While findings of automated analysis suggested reduced joy facial expressions, this method focused on a relatively neutral conversation during the

background section of an interview and did not provide the ability to detect the ability to assess a wide range of evocative emotions. Furthermore, automated analysis provides information on subtle facial expressions but there may be additional information regarding facial expressions that are more micro and fleeting that could be missing. The use of facial EMG in addition to automated facial analysis provided the ability to assess facial expressivity when viewing videos intended to evoke a wide array of emotions. From these analyses, findings revealed that the CHR group showed reductions in zygomaticus facial activity when viewing a film clip intended to evoke excitement. These findings are consistent with previous work applying facial EMG to individuals with schizophrenia who showed reduced zygomaticus activity in response to the presentation of positive pictures (Wolf et al., 2006) and, more broadly, a long line of working showing blunted facial expressions in individuals with schizophrenia (e.g., Kring et al., 1999). At the same time, there was important findings of blunted zygomaticus activity in terms of context and timing. In terms of context, blunting in zygomaticus activity was observed uniquely in response to the excitement film clip but not in response to the amusement, neutral, or during the negative film clips. In terms of timing, blunting in zygomaticus activity was observed only during the most emotionally evocative peak segment of the excitement film clip (whereas no group differences were observed prior to the peak). These findings suggest that alterations in facial expressions in individuals with a CHR syndrome may only become apparent at sufficiently high levels of arousal, which underscores the value of using experimental stimuli that are able to elicit emotions at intense levels (Levenson, 2014).

Facial electromyography and timing. Findings revealed results regarding the timing of the clip, with group differences identified in zygomaticus activity at the end of the excitement video clip during the peak stage (but no differences were observed during the segment prior). As

previously mentioned, timing has been investigated in some studies using facial EMG in schizophrenia although this is quite limited (Varcin et al., 2010). While the underlying mechanism of this possible explanation of findings is unknown, it could be that abnormalities are a result of cognitive deficits such as attentional issues or limited cognitive resources (Strauss & Cohen, 2017). It is also perhaps possible that difficulties with motivation (i.e., challenges with drive to sustain a facial expression) may be contributing (e.g., not feeling the drive to maintain facial muscle movements). This is an especially plausible hypothesis given recent work suggesting motivation deficits may be central to symptoms endorsed by individuals with a CHR syndrome (Gupta, et al., 2021). This finding may suggest that CHR groups display facial expressivity, however, the deficit may lie in difficulties with holding, sustaining, or even coordinating facial expressions throughout time. Taken together, timing of alterations in facial expressions is a rich area of investigation for future work to consider building on given the importance of timing in social interactions (e.g., smiling when smiling might be appropriate). For example, one additional point of future inquiry is whether timing deficits occur in dyadic interactions as well (e.g., are individuals with a CHR syndrome more expressive at the beginning of an interaction but not at the end).

Facial electromyography and change in facial expressivity. These data also suggest that the CHR group are showing less changes in overall facial expressivity during the excitement clip evidenced by lower rate of change scores, which was significantly related to increased likelihood of conversion to psychosis (based off of risk calculator scores). In terms of rate of change, it is possible that less changes in facial expressivity could be representative of individuals being particularly threat activated or emotionally dysregulated during the excitement film clip (i.e., suppressing facial expressions of emotions). Additionally, it may be that these individuals have

an impaired ability on a motor system level to show varying degrees of zygomaticus facial muscle activity given the high rates of motor abnormalities observed in this group (Dean et al., 2018b; Mittal et al., 2014; Schiffman, 2017; Walker et al., 1999; Walther & Mittal, 2017). It is also possible that cognition might be playing a role in the lack of changes in facial expressivity and this could be particularly the case given those with a CHR syndrome endorse a wide array of cognitive deficits (Niendam et al., 2009). It is perhaps possible that there are cognitive processes such as difficulties processing incoming information.

Associations with anxiety and depression. As discussed, there were no links between zygomatic activity and common mediating or confounding factors, including depression and anxiety (Addington et al., 2017b). While small to moderate associations were found between expression and experience variables and symptom and functioning domains (discussed later in this discussion), none were observed with depression and anxiety. As such, it is likely that emotion abnormalities are not simply a byproduct of comorbid depression and anxiety (in this case, panic symptoms). Importantly, while null findings are not interpretable, this potential hypothesis is useful to consider in light of future research. Additional research is needed to better characterize the role of comorbidity in different aspects of facial expressions. This is particularly needed given that previous findings, as mentioned, from Gruber and colleagues (2018) found links between reports of emotional reactivity derived from viewing static photo presentations and anxiety and depression measured by the BDI and BAI, respectively. Along these lines, in prior work (Gupta et al., 2021), depression explained the largest proportion of variance in anhedonia in a sample of 192 individuals with a CHR syndrome, while anxiety was the most predictive of blunted affect (note: blunted affect included all aspects of affect including blunted facial expressivity but also reductions in gestures and vocal features like prosody). Importantly,

specific anxiety and depression were used in this study - the BDI and BAI. Along these lines, there are many anxieties (e.g., social anxiety) and other aspects of depression (e.g., persistent) that were not measured in this study that future work would benefit from investigating. There is also increasing evidence that reports of adverse childhood experience such as bullying might play a role in emotional processes (Ricard et al., 2021; Tognin et al., 2020). Altogether, additional research is needed to comprehensively assess the role of comorbidity in emotional processes prior to the onset of psychosis.

Anger/negative facial expression findings from automated analysis and facial electromyography. One area of that was not in line with expectations for this study was that with the automated analysis approach revealed more anger facial expressions, however, there were no findings observed in more negative (i.e., corrugator) facial expressions during the evocative film clip task. While these relationships were not empirically tested/compared, and should not be over-interpreted, it is possible that this area of divergence may be a result of differences in methodologies and different samples. However, it could be a critical point of future research to further example when increased anger in particular is observed in different contexts. For example, perhaps it is the case that those with a CHR syndrome show increased anger facial expressions in more neutral conversation (as observed from findings from automated analysis) but there may be differences depending on the context (i.e., when viewing video clips). Relatedly, those with a CHR syndrome (in a different sample) the also showed, descriptively speaking, increased corrugator activity in response to the neutral film clip from the evocative film clip task. This may reflect a pattern that could be interpretable especially with an increased sample size. Altogether, it will be critical to further investigate, with larger samples sizes, to examine these patterns and relationships.

Blunted joy facial expressions derived from iMotions, FaceReader, and facial EMG. Another critical pattern observed from findings was that blunted joy facial expressions were detected across methods and across samples. While direct comparisons were not made across methods and samples, interpretations are provided in efforts to detect patterns for future studies to consider (and should be interpreted with caution until more research is conducted comparing across methods and samples). For speculation purposes, findings of blunted joy expressions from iMotions and FaceReader were observed in the sample collected at University of Colorado Boulder and in 5-minute and 1-minute segments (note: in a previous study, 5-minute and 1-minute segments of clinical interviews submitted into iMotions and FaceReader, respectively in this sample were found to be positively correlated, $r = 0.83$, $p < 0.001$) (Gupta et al., 2020). Interestingly, with the Northwestern sample, there were also displays of blunted zygomaticus facial activity observed with facial EMG. Further research is needed to understand to what degree these findings generalize to other samples of CHR individuals in other cultures, geographic locations, racial and ethnic backgrounds and more. However, the convergence/replication of findings across samples suggesting blunted facial expressions of joy may provide some evidence that this finding is robust and perhaps generalizable. However, replication is needed with similar methodologies (e.g., different samples, same facial expression method such as automated analysis or facial EMG) before definitive conclusions can be drawn.

Evidence of altered emotion-motor and motor-motor circuitry underlying blunted facial expressions of joy within the CHR group (Findings from Aim 3)

Emotion and motor circuitry. It was found that there was a positive association between facial expressions and connectivity between cerebellar bilateral lobules IV (i.e., anterior cerebellum) and left and right primary cortex (motor-motor connectivity). Furthermore, there

was a relationship between facial expressions and connectivity between cerebellar bilateral lobules IV and the left and right amygdala (motor-emotion/emotion-motor). In efforts to unpack these results/understand the direction of findings, I compared these associations with relationships in the control group. Findings revealed there was a strong effect for differences in control and CHR associations between facial expressivity and connectivity between the cerebellar bilateral lobules IV and left primary motor area, right amygdala, and left amygdala. These findings hint towards the possibility that both emotion and motor regional network associations may be involved in alterations in facial expressivity in those with a CHR syndrome. Additionally, these findings add a nuanced perspective to the literature. Much of the current literature has focused on emotion regions in particular in the study of emotional processes (Aleman & Kahn, 2005; Anticevic et al., 2012; Bjorkquist et al., 2016). However, these data provide support that the inclusion of motor networks may provide important clues in our understanding of facial expressions. As discussed, the potential role of motor circuitry is in line with studies suggesting those with a CHR syndrome experience motor abnormalities (Damme et al., 2020; Mittal et al., 2008, 2014; Mittal & Walker, 2007). In this study, facial expressions derived from FaceReader were analyzed during a dyadic interaction (i.e., a clinical interview) and used in neuroimaging analyses.

When considering possible interpretations of findings, it may be that emotion-motor circuitry may be reflective of attempts at facial mimicry. There is evidence that when individuals observe other's facial expression movements (e.g., in conversation), motor-related brain areas are recruited (Philippot et al., 2003). Perhaps the recruitment of motor areas represents an individual's involuntary and rapid response at mimicking another's facial expression during an interaction (Dimberg et al., 1982; Dimberg et al., 2000). While this theory draws more directly

from studies of empathy (e.g., Varcin et al., 2010) and studies investigating the role of the mirror neuron system in facial emotion perception (e.g., van der Gaag et al., 2007), it may be relevant for facial expressions of joy as well. However, future studies, drawing from models of social cognition are needed to better understand this explanation. Additionally, while the amygdala may not be crucial in the actual action of facial expressions, it does have a role in facial expression selection (Gothard et al., 2014); another possible explanation of findings is that there may be difficulties in selecting appropriate facial expressions in different contexts. This explanation is supported by research in individuals with lesions to the amygdala, showing a tendency for these individuals to appear more restricted and display less affiliated displays of facial expressions (Adolphs et al., 2010; Bliss-Moreau et al., 2013; Gothard et al., 2014; Meunier et al., 1999).

High and low levels of facial expressivity. Importantly, it appeared that the difference may not be driven by those with lower facial expressivity but instead by those with higher facial expressivity (note: there were group differences in behavioral data in that the CHR high facial expressivity group had lower levels of facial expressivity compared to the control group with high facial expressivity). A possible interpretation of these findings is perhaps in efforts to match or display somewhat normative levels of facial expressivity (i.e., in line with controls), motor regions are thus overrecruited to help facilitate facial expression movements. This explanation is supported by the additional analyses conducted to explore group differences in CHR and controls with low facial expressivity and CHR and controls with high levels of facial expressivity. From these exploratory analyses, results suggested that CHR individuals with more facial expressivity (as opposed to blunting) had greater connectivity between cerebellar bilateral lobules IV and right amygdala compared to controls. These additional analyses were critical in the ability to extract possible explanations of these findings. Specifically, there were no differences in

associations between blunted (i.e., lower) facial expressivity and connectivity in both the CHR and control groups. However, as mentioned, there seems to be a pattern in which the difference is driven by those with higher levels of facial expressivity, in particular. While the sample sizes were very low for the high facial expressivity group (CHR = 12, control = 21), effect sizes derived from mean comparisons of connectivity patterns between high facial expressivity CHR and control groups were strongest for increased connectivity between cerebellar bilateral lobules IV and left amygdala. As such, it appeared that CHR individuals with higher facial expressivity also had greater connectivity between cerebellar bilateral lobules IV and left amygdala.

It is possible, as already briefly discussed, those with a CHR syndrome may not be displaying facial expressions as controls are (potentially due to difficulties selecting facial expressions as discussed above which may be one explanation of the amygdala's role in this connectivity pattern). Interestingly, behavioral data suggested the CHR group had lower facial expressions of joy even among those with higher levels with facial expressivity. Perhaps to match facial expressions of joy similar to the control group, the CHR group may be over-recruiting motor regions.

The cerebellum and facial expressions. Furthermore, these data provide insights on the role of the cerebellum in facial expressivity given the prevalence of cerebellar bilateral lobules IV, a cerebellar region, in connectivity findings. Of note, cerebellar pathways have been suggested to be relevant in symptomatology of both schizophrenia and CHR groups (Andreasen & Pierson, 2008; Bernard et al., 2015). Furthermore, there is a growing body of evidence that suggest that cerebellar circuitry is involved in emotional processes (King et al., 2019; Schmahmann & Caplan, 2006). This is in addition to the more established research suggesting the cerebellum implicated in motor processes such as the initiation and imitation of actions (King

et al., 2019). Of emotional processes, the cerebellum has been more commonly associated with affective processing. For example, there are studies of individuals with cerebellar lesions that report difficulties in patients accurately detecting facial expressions of emotion using facial recognition tasks (Adamaszek et al., 2014). Furthermore, there is evidence that transcranial direct current stimulation (tDCS) of the cerebellum can improve facial expression processing, particularly of negative facial expressions (Ferruci et al., 2012). However, the study of the cerebellar roles in facial expression production is very limited. These data provide a foundation for future research that is needed in this are area unraveling the role of the cerebellum in emotion related processes.

Relationships between experience reports and blunted facial expressivity derived from automated analysis and facial electromyography and links with (1) psychosis-risk scores and (2) social functioning (Findings from Aim 4)

Psychosis-risk conversion scores. As discussed, it is suggested that negative symptoms contribute to the transition to psychosis and these data add to the larger literature investigating vulnerability markers that contribute to the onset of psychosis (Cannon, et al., 2008; Fusar-Poli, et al., 2012). Here, findings that blunted facial expressivity derived from automated facial analysis was related to psychosis-risk conversion scores adds to the growing literature identifying risk markers of psychosis. Furthermore, findings from facial EMG suggest that reduced change in facial expressivity are related to psychosis-risk conversion scores. Together, these findings are in line with previous research that found blunted facial expressivity derived from clinical interviews as well as inappropriate affect were predictive of transition to psychosis (Mason et al., 2004). While longitudinal analysis was not possible given the available data, these results do suggest that blunted facial expressivity may contribute to risk for conversion to

psychosis within a two-year period. However, these analyses were correlational and cannot infer causation. As such, future studies are needed to better understand the predictive, causal nature of blunted facial expressivity and risk for psychosis. These associations inform vulnerability models and it may be useful to integrate alterations in facial expressivity in psychosis risk prediction calculators. It is also possible that targeting facial expression abnormalities with interventions may be useful in the prevention of psychosis

When considering the other direction of findings, it is possible that risk for conversion to psychosis may result in blunted facial expressivity. Interestingly, there is evidence to suggest that negative symptoms such as blunted facial expressivity emerge even prior to positive symptoms. As such, one might speculate that blunted facial expressivity may be a part of the pathogenesis of psychosis. This is particularly noteworthy given that those with schizophrenia also exhibit facial expressivity alterations (Kirkpatrick et al., 2006).

Social functioning. Several studies have reported that negative symptoms are impactful on social functioning in both schizophrenia and CHR populations (Corcoran, et al., 2011; Evensen, et al., 2012; Gur, et al., 2006; Meyer et al., 2014; Schlosser et al., 2015) which are consistent with our current findings indicating blunting in facial expressivity derived from automated analysis was related to decreases in social functioning. Additionally, social impairments have been found to be uniquely predictive of transition to psychosis (Cannon, et al., 2008). These data support findings from Schlosser and colleagues (2015) in which negative symptoms were predictive of social functioning in a sample of CHR individuals. However, the present study differs from the literature in that we found a specific negative symptom category (i.e., blunted joy derived from automated analysis) to be associated with decreased social functioning. Perhaps blunted facial expressions leads to difficulties developing relationships

(e.g., not showing joy when joy is expected for example may be problematic when developing and maintaining relationships). However, when considering the other direction of correlational findings, it is possible social functioning impairments cause blunted facial expressions. One might speculate that social functioning impairments may cause depressive symptoms which in turn could lead to blunted facial expressions. However, this is unlikely to be the case given findings suggest blunted facial expressions were not related to depressive symptoms (although this could very well be due to power). Further research is warranted to understand directional causal relationships, which were not possible in the current study given the correlational analyses employed. Furthermore, it will be critical for other studies to assess different components of social functioning. Here, I used a commonly employed social functioning scale (Cornblatt et al., 2007). However, there are other measures of social functioning that are useful to add in future studies. Using a battery of social functioning scales may provide a bit more information (e.g., What aspects of social functioning are impaired? Is it developing relationships? Maintaining relationships? Communication?).

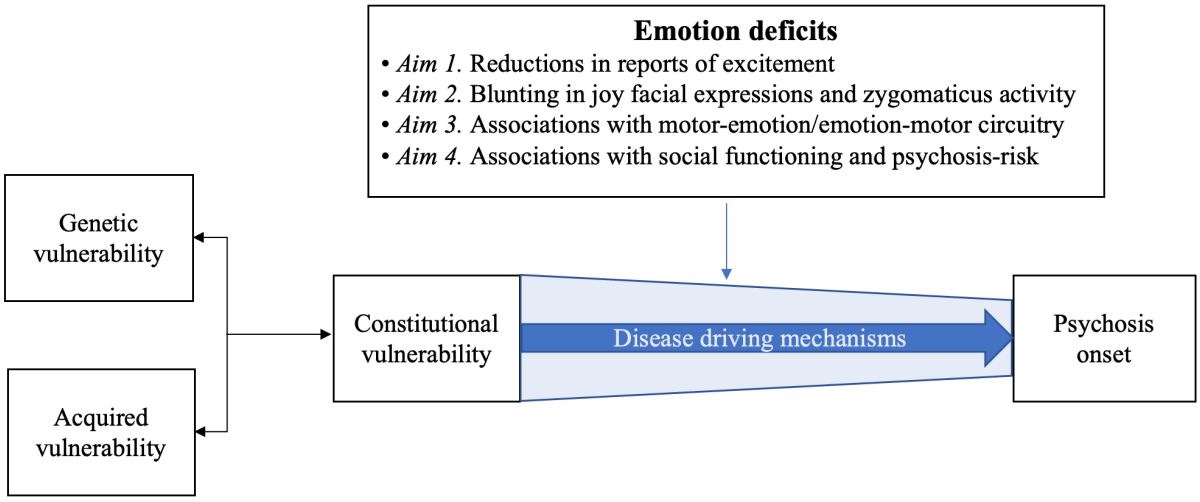
Emotion Deficits and the Diathesis Stress Model

Here, I come back to the diathesis stress model in order to provide further detail, based off of the findings of this dissertation, in how perhaps emotion deficits may be conceptualized in this framework. As discussed, adolescence is a time in which several changes are occurring, socially, emotionally, and neurodevelopmentally. For some, the interaction between genetics and acquired vulnerability (e.g., prenatal exposure to toxins), may form a constitutional vulnerability, the diathesis, that one may carry in time. This vulnerability can interact with stress, environmental factors, and aberrant neurodevelopment, that can make an individual at heightened risk for developing psychosis and even convert over time. There may be risk signs

and symptoms that can be expressed, as conceptualized in modern takes of the diathesis stress framework, that are emerging. One piece that has been missing in this model is the nature of the emotion deficits in at risk for psychosis. While further research is needed to understand several components of emotion in the context of this framework before definitive conclusions can be drawn, the findings from this dissertation do suggest that emotion deficits may be apart of the etiology of psychosis (see Figure 25).

In light of speculating how these dissertation findings fit into the larger diathesis stress model, it is possible that reductions in the experience of positive emotions as well as blunting in facial expressions of joy can lead to a cascade of negative events such as social challenges, difficulties pursing goals, and overall withdrawal. These behaviors can interact with stress and neurodevelopment which together, could contribute to unmasking one's vulnerability and ultimate emergence of psychosis. While these data do hint towards the potential for other factors at play as well such as abnormal neurodevelopment, motor process, cognition, and social functioning, a takeaway from this dissertation is that there appears to be a failure, based off of these data, in recruiting emotional experience and expressions (excitement and joy) in particular in those at risk for psychosis. With this, it will be useful for models of the pathogenesis of psychosis such as this diathesis framework to incorporate emotional deficits into our understanding of individuals with a CHR syndrome. With this information, we can further understand and test how emotional deficits interact with other key components of the diathesis stress model, such as stress which can inform prevention and intervention strategies.

Figure 25. Dissertation findings in the context of the diathesis stress model



Clinical Implications

Along these lines, these data have important clinical implications that inform prevention and intervention strategies. It may be useful to consider integrating emotion deficits into risk calculator algorithms. In the last few years, risk calculators such as the NAPLS risk calculator and the SHARP calculator have been developed (Cannon et al., 2016; Zhang et al., 2018). While these data are not conclusive regarding the nature of emotional impairments in those with a CHR syndrome, there is evidence within the literature more broadly that emotional impairments (e.g., expression, experience, perception, regulation) are present in this group.

Furthermore, there may be beneficial implications for clinical staging models which have grown in popularity in the last decade for the treatment of psychological disorders including psychosis. In these models, clinical staging refers to the idea that the development of mental health challenges has the potential to progress to formal illness at some point in an individual's life. These models take a developmental perspective and integrate ideas for treatment that begin as early as when a child is in utero (e.g., one suggestion is taking choline). The introduction of

clinical staging into psychiatry has been instrumental in setting up a blueprint for the development of prevention and intervention strategies among severe mental illness (McGorry et al., 2010). Critically, staging models are important in that they define risk markers and interventions can be offered on a broader level earlier in treatment, resulting in less harmful, expensive, and stigmatizing interventions initially and addressing concerns regarding predictive power. While these models touch on emotional impairments, it would be useful to integrate emotional impairments into these models more fully. However, in order to do this effectively, it will be critical for future work to tease apart the developmental trajectories of emotional impairments in this group including further understanding whether early emotional disturbances (e.g., early childhood) predict negative symptom emergence as well as psychosis onset later on. Perhaps if we can improve emotional disturbance identification and detection early on in one's life, from a clinical staging perspective, we can intervene before symptoms worsen over the course of development.

Importantly, with efforts intended to improve the accurate detection and identification of emotional disturbances in those with a CHR syndrome, it would be possible to intervene with various treatment approaches. As noted, common medications for the treatment of psychosis such as antipsychotics are proven to be ineffective for the treatment of negative symptoms, generally speaking. As such, clinical interventions such as cognitive behavioral therapy (and pulling techniques such as behavioral activation) could be useful for disturbances related to lower reports of positive emotional experience. One model developed by Grant, Beck, and colleagues (Beck et al., 2009; Grant & Beck, 2009) suggest experiences with repeated setbacks and social challenges can lead to negative beliefs and attitudes of oneself which can, over time, develop into negative symptoms and poor clinical outcomes in psychosis/psychosis-risk. Given

that cognitive behavior therapy involves challenging unhelpful thoughts, it may be useful especially useful when considering unhelpful thoughts may contribute to reductions in positive emotional experiences as well, which may impact behaviors. Furthermore, psychoeducation on the utility of emotional experiences including the use of facial expressions in social situations would be advantageous.

Additionally, third-wave behavioral approaches may be particularly beneficial for blunted facial expressions. In particular, for example, radically open dialectical behavioral therapy (RO DBT) is intended to improve social signaling such as using facial expressions effectively in those that endorse overcontrolled coping styles (Lynch et al., 2015). The underlying premise of RO DBT is individuals that are overcontrolled tend to actually be quite threat activated which contributes to social signals such as reductions in the use of hand gestures and facial expressions. It could be that some of the skills from RO DBT that involve activating one's social safety system (which is interconnected with reward circuitry) could be useful for improving facial expressions in those with a CHR syndrome. While this has not been tested yet, it could be a beneficial future, translational application of this work.

Lastly, there may be benefits of applying cerebellar transcranial direct current stimulation as a means to improve facial expressivity in this group. While much more research is needed first assessing the efficacy before application of a technique as such, there is evidence of cerebellar tDCS in particular to improve processes in those with psychiatric disorders including individuals falling on the psychosis continuum (Bose et al., 2014; Ferrucci et al., 2015; Gupta, Dean, et al., 2018; Kekic et al., 2016; Mondino et al., 2015).

CHAPTER VII.

Limitations and Future Directions

While several limitations are already discussed throughout the discussion section of the dissertation, there are further limitations and future directions to consider.

Sample. To begin with, it will be important for studies to examine these research questions with age and biological sex matched samples. In this dissertation, there were significant differences in one sample (Sample 2) in the distribution of sex. Additional studies would benefit from matching demographics more closely. Furthermore, it is evident that across all samples used in the study, their racial and ethnic composition was relatively homogenous. It will be critical to also recruit participants that represent a broad range of ethnic and minority backgrounds. This is particularly useful to consider in light of the use of automated facial analysis. While automated approaches can reduce biases in many ways, there is also recent research to suggest computational methods as such can potentially perpetuate health disparities. For example, there is evidence of higher error rates by automated approaches in detecting facial expressivity in darker-skinned women compared to 0.9% on lighter skinned men (Buolamwini & Gebru, 2018). Many of the machine learning algorithms have been trained on White individuals and as such, there may be biases towards this social group (Hitczenko et al., 2021). It will be useful for future research to continue to develop training models for computational approaches like automated facial analysis that include a wide range of social groups.

Additionally, even though the sample size is comparable to studies in schizophrenia populations (Kring & Elis, 2013), larger samples sizes could be useful in future work. Additionally, comparing different samples on the psychosis continuum/across stage (e.g.,

nonclinical psychosis, CHR, first-episode, schizophrenia) would be valuable in future work as well. It is important to note that in our study, we used correlational analysis so causation cannot be inferred. Furthermore, this dissertation used cross-sectional data; longitudinal analyses are needed to comprehensively understand these findings, particularly those related to conversion to psychosis.

Symptom assessments. While the SIPS is a commonly used instrument for diagnosing a CHR syndrome (Fusar-Poli et al., 2012; Miller et al., 1993), there are other assessments used around the world to diagnose those at risk for psychosis, discussed in the introduction section of the dissertation. With this in mind, a future direction will be to continue to improve assessment approaches and tools in diagnoses a CHR syndrome. This will be important as 2/3 of individuals identified as CHR often do not go on to develop psychosis but instead either remit or develop a different psychological disorder (Addington et al., 2011c). These efforts have the potential to improve our ability, as a field, to accurate detect and thus, intervene early in the prevention of psychosis.

Additionally, while the BAI and BDI were used given that these are widely adopted in CHR studies (e.g., Gruber et al., 2018), we did not utilize a wide range of assessments in our measures of anxieties and depression, which likely impact conclusions and interpretations of results. The BAI assesses more panic, arousal type anxiety symptoms. Additionally, the BDI measures depression in a two-week time frame (Beck et al., 1996). It could be useful for future research to more fully assess anxieties and depression. For example, it may be more accurate to consider DSM-V diagnoses. Moreover, it could be useful to include a battery of measures of anxieties and depression in additional research. Furthermore, as mentioned already, social functioning measures were limited and future studies should more comprehensively assess social

functioning using a battery perhaps in efforts to obtain a bit more specificity. It could also be useful to assess occupational functioning as well given evidence of impairments in roles in different contexts in this group (e.g., school performance, difficulties completing chores) (Cornblatt et al., 2007b).

Lastly, in this dissertation, I examined risk calculator scores using two risk calculators due to available data (NAPLS was used with the automated facial analysis aim and SHARP was used with the facial EMG aim given available data across both sites). Given that both these risk calculators are built on different algorithms, it will be important for future studies to test different domains of emotional processing and risk for psychosis scores using similar calculators.

Evocative film clip task. In Aim 1, while the use of reports of experiences of emotions were useful, it is important to note that these data were collected a few seconds after the presentation of the video clip. It would be interesting for future work to consider collecting real-time reports of experience of emotion throughout the video presentations. Additionally, it was not possible with the current methods to examine the intersections between the experience and expression of emotion. Additional research could benefit from utilizing methods that may couple experience reports and facial expressivity data in order to truly understand if one is not showing facial expressions on the face, if that also means they are not experiencing a specific emotion (or vice-versa). One line of research that may provide insights that could extend the current findings is using approaches such as ecological momentary assessment (EMA). EMA may be able to capture real-time reports of the experience of emotion more closely and in more of a naturalistic context than a laboratory task. While finding ways to assess facial expressivity using EMA methods is still underway, this could be a particularly unique and translational way to assess facial expressivity in conjunction with reports of the experience of emotion.

Automated facial analysis. In terms of automated facial analysis, additional limitations include the use of clinical interviews. While on the one hand, clinical interviews provide a snapshot of what behavior in a more naturalistic setting (as opposed to an experimental paradigm) may look like, these interviews do not produce a range of emotions (facially and in terms of experience). The use of facial EMG addressed this limitation to some degree. However, additional studies utilizing experimental paradigms as such are needed in this area. Furthermore, in the clinical interview, the first-five minutes were used. It is possible that when using the first-five minutes of the clinical interviews, a baseline read of facial expressions may be influenced by social relatedness, rapport, and comfort with the interviewer. Studies using other paradigms that are able to better control for these issues could be of value. Furthermore, it is possible that interviewer biases can inflate correlational analyses (this limitation highlights the potential value of automated analysis given the objective nature of this approach), which provides further support regarding the need for replication studies using experimental paradigms. Additional studies are required to better understand the validity of automated analysis. Furthermore, there may be differences in the production of facial expressions while talking vs viewing. Given we did not directly test this question, it will be critical for future studies to continue to understand whether facial expressions vary as a function of context such as in conversation, when viewing videos, and when imitating facial expressions of others. Furthermore, understanding facial expressivity in conjunction with other components of affect such as gesture and voice may provide novel insights in the study of emotion.

Facial EMG. While the use of facial EMG was able to address limitations presented from the use of automated facial analysis such as restricted range of emotions evoked, there are additional limitations to discuss related to this portion of the dissertation. For example, while

several different videos were used to evoke a range of facial expressions, not all emotions were assessed; there was no video for anger. It will be important for additional research to also consider assessing zygomatics and corrugator activity in response to stimuli intended to evoke anger. More research is also needed utilizing other physiological and behavioral measures accompanying EMG in order to understand the interplay between the experience and the expression of emotion in these groups. Finally, it will be important for additional studies to understand the effects of culture as well as race and ethnicity using larger samples sizes (Soto et al., 2005). It may be useful for future work to continue to disentangle the mechanisms underlying alterations in facial expressivity, particularly testing to see if these abnormalities are a result of difficulties identifying and discriminating between emotions, impairments in perspective-taking, or deficits in motor abnormalities.

Another related limitation that serves as an opportunity for future work is to understand different components of joy facial expressions. For example, in recent work by our group assessing genuine (e.g., thought to reflect genuine levels of positive emotion) and nongenuine smiles (e.g., thought to represent fake positive or mask negative emotions), it was found that those with a CHR syndrome displayed reduced genuine but not non-genuine smiles (Ricard et al., in press). One idea for future work could include assessing genuine and nongenuine smiles in the context of an evocative film clip as used in this dissertation. Furthermore, it would be informative to assess whether emotion and/or motor circuitry are uniquely related to these types of smiles. Additionally, it would be useful to investigate facial expressions as such transdiagnostically, across clinical disorders such as autism and depression.

Imaging. The imaging component of the dissertation provided the opportunity to dive into possible mechanistic questions underlying facial expressivity. It will be important for future

studies to also assess underlying mechanisms of the experience of emotion as well. It is also possible that as with other components of the study, the imaging portion in particular may have lacked power given the small sample size, particularly in exploratory analyses with those with high/low facial expressivity. This is a broader concern in the field of neuroscience which agree there is a need for imaging studies to utilize larger sample sizes in imaging studies (Button et al., 2013). Another future direction of this work involves investigating the role of motor abnormalities in facial expressivity. As discussed, motor functions have been more historically considered to play a role in blunted facial expressivity. However, current interpretations draw from the emotion literature and suggest facial expressivity may be a result of impairments in emotion and cognitive neural circuitry. There may be new insights by integrating motor processes in theories and conceptualizations of facial expressions. Future research is also needed to understand the role of other aspects of possible associated neural circuitry of facial expression abnormalities such as prefrontal regional connections (Kring & Elis, 2013). Additionally, while resting-state connectivity has many benefits, there are important limitations of this approach to discuss as well. For example, with resting-state, it is not possible to infer causation and the exact biological mechanisms at play. Furthermore, many brain regions are interconnected so while the current dissertation used the literature to pinpoint brain regions in emotion and motor areas that may be implicated, there may be other brain regions that may be relevant that were not included. Furthermore, the identified brain regions may overlap with other regions in the brain as well that have unique network properties so it is perhaps the case that other networks may be playing a role in facial expressivity that have yet to be identified. Importantly, resting-state does not provide information on activation during a task-based fMRI paradigm and it could be useful to also assess facial expressivity during tasks such as facial imitation tasks (Kring & Elis, 2013).

Furthermore, it is important to note that in the current study, only underlying neural circuitry of joy facial expressions derived using FaceReader were examined. Additional studies may benefit from investigating associated neural circuitry in other emotional facial expressions (e.g., anger) and using other facial expression tools such as facial EMG.

Other. Other future directions include assessing the role of cognition in emotional processes in those at risk for psychosis. It is possible that working memory deficits or other cognitive impairments as discussed may be playing a role in the experience and expression of emotion. This may be particularly the case given that those with a CHR syndrome begin to experience declines in cognitive functioning (Fusar-Poli et al., 2012). Relatedly, one idea for a future study is to test theories of cognitive demand in blunted facial expressivity (Strauss & Cohen, 2017). This might include presenting individuals with cognitively demanding tasks, as well as a neutral task, and assessing facial expressivity and reports of the experience of emotion throughout. Furthermore, it would be useful to test theories of emotion regulation in efforts to better understand the interplay with the experience of positive emotions and facial expressivity (Chapman et al., 2019). One aspect of the current dissertation that was not assessed was skin conductance. Given that skin conductance is a component of the way emotion is defined (Kring & Elis, 2013), it could be informative to assess relationships between skin conductance, experience of emotion reports, and facial expressivity. Furthermore, findings from the imaging aim of the dissertation hint towards the possibility that there may be unique subgroups of facial expressivity (and perhaps too when consider the experience of emotion). Given that those with a CHR syndrome are a largely heterogenous group (Fusar-Poli et al., 2016b), it is possible that some may experience impairments, while others do not. This is in line with a broader study of negative symptoms conducted in our group in which it was found that there were unique

negative symptom subgroups in those with a CHR syndrome (Gupta et al., 2020). While one subgroup included both reports of the experience and expression of emotion, the SIPS was used in this study to define negative symptom subgroups. Recently, more negative symptom measurements that more fully assess negative symptom items such as anhedonia and blunted facial affect have been developed (Pelletier-Baldelli et al., 2017; Strauss et al., 2020) and could be useful to examine using cluster analysis.

Furthermore, it will be useful to better understand differences in emotional disturbances across the psychosis spectrum. There is evidence of emotional disturbance occurring even earlier on in development in the premorbid period. Evidence suggests that these behaviors early on can include preferences for solitary play, higher levels of aggression, social isolation/withdrawal, and fewer expressions of joy (Amminger et al., 1997; Liu et al., 2015; Pauly et al., 2008; Walker et al., 1993). It will be critical for additional studies to better understand the emotional disturbances during the premorbid period and how it might relate to symptoms in the CHR period as well as after psychosis onset. Additionally, facial expressions are just one component of expression; it will be important for future work to investigate other components of expression such as gesture and voice prosody.

CHAPTER VIII.

CONCLUSION

Conclusion

Taken together, findings suggest reduced reports of the experience and expression of excitement/joy in particular may not solely be a part of the schizophrenia onset but instead, may be unique to the progression of symptoms over time, occurring prior to the onset of psychosis as well. Furthermore, these data shed light on the potential role of motor functions and processes in facial expressivity (possible theories are largely discussed in light of emotion-motor, motor-emotion neural findings. Additionally, these data provide insights regarding emotive dysfunction in CHR groups, perhaps shed light on the possibility this is a true biomarker, introduce techniques and methods for examining the experience and expression of emotion that go beyond clinical interviews, shed light on possible associated neural mechanisms, as well as relationships with functional outcomes and psychosis risk likelihood. The hope of this dissertation is that it serves as a leaping point for the additional research that is needed in this area seeking to understand the nature of emotional processes in those with a CHR syndrome.

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Appendix A. Structure of the SIPS Interview

Table of Contents

Appendix B. Example of SIPS Rating Scale

PtID _____Date:_____/_____/_____Interviewer Code:_____SIPS

P. 1. UNUSUAL THOUGHT CONTENT/DELUSIONAL IDEAS

From the preceding Inquiries, Qualifiers, and Description, choose the level that best describes the subject on the scale below, based equally on the type of unusual ideas and the tenacity of belief in the unusual ideas. That level will generally but not necessarily match the supporting descriptions of distress from the unusual ideas and/or interference with life or functioning by unusual ideas. Basis for ratings includes both interviewer observations and subject reports.

UNUSUAL THOUGHT CONTENT/DELUSIONAL IDEAS **Positive Symptom Severity Scale (circle one)**

0 Absent	1 Questionably Present	2 Mild	3 Moderate	4 Moderately Severe	5 Severe but Not Psychotic	6 Severe and Psychotic
No unusual thoughts.	Unusual thoughts such as déjà vu or other "mind tricks" that occur often in the general population.	Unusual thoughts such as over interested in fantasy life or unusually valued ideas/beliefs or superstitions. Beliefs beyond what might be expected by the average person but within cultural norms.	Unusual thoughts such as pathological ideas/experiences/ mental events that seem to come from within and to be imaginary.	Unusual thoughts with the sense that pathological ideas/experiences /mental events may be coming from outside oneself or may be real.	Unusual thoughts such as pathological ideas/experiences/ mental events that seem real and external to self.	Unusual thoughts such as pathological ideas /experiences/ mental events that feel completely real and distinct from the person's own experiences.
No tenacity of unusual thoughts.	Fleeting sense that something is different.	May defend beliefs.	Experiences seem meaningful because they recur and will not go away. Self-generates skepticism with little effort.	Able to self-generate skepticism with effort.	Skepticism can be induced, but only by the efforts of others.	Qualifies as delusional conviction: skepticism cannot be induced, at least intermittently.
No distress from unusual thoughts.	May be experienced as curious.	May be puzzling but not distressing. Ignorable.	May be unanticipated, puzzling and distressing. Unwilled, and not easily ignored.	May be distracting, bothersome.	Content may be familiar, anticipated. May cause significant distress.	May cause severe distress.
No interference by unusual thoughts.	Thinking, feeling, social relations and behavior not affected.	Thinking, feeling, or social relations may be altered but not impaired. Behavior not affected.	Thinking, feeling, or social relations may sometimes be affected. Behavior not affected.	Thinking, feeling, or social relations may often be affected. Behavior may sometimes be affected.	Thinking, feeling, or social relations may be affected daily. Behavior may often be affected.	May interfere persistently with thinking, feeling, or social relations and with behavior.

Rating rationale:_____

_____

_____

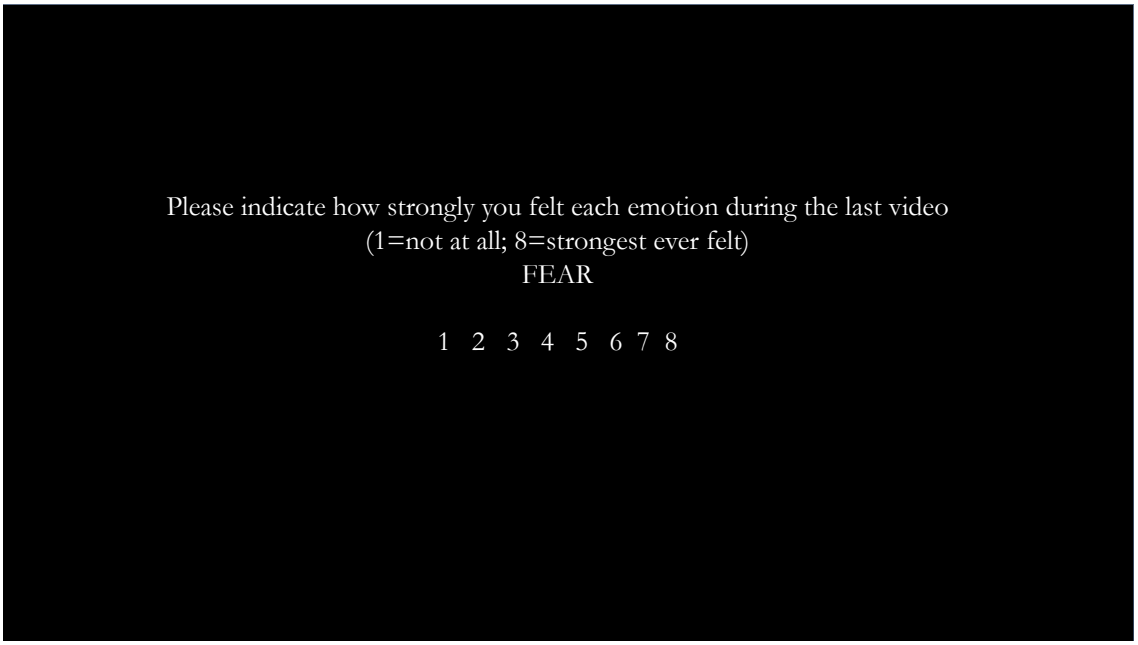
_____

_____

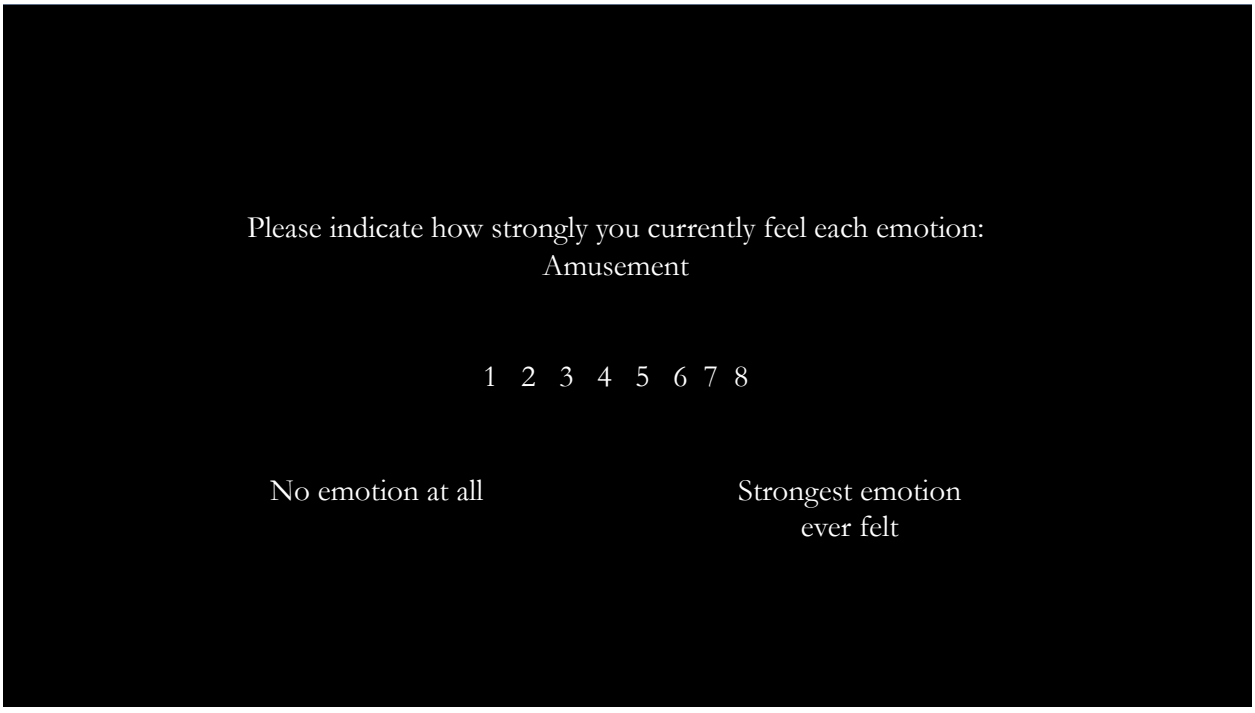
For Symptoms Rated at Level 3 or Higher			
Symptom Onset	**Symptom Worsening**	**Symptom Frequency**	**Better Explained**
Record date when a positive symptom first reached at least a 3: ☐ "Ever since I can recall" ☐ Date of onset ___/___ Month/Year	Record most recent date when a positive symptom currently rated 3-6 experienced an increase by at least one point: Date of worsening ___/___ Month/Year	Check all that apply: ☐ ≥ 1h/d, ≥ 4d/wk ☐ ≥ several minutes/d, ≥ 1x/mo ☐ ≥ 1x/wk ☐ none of above	Symptoms are better explained by another DSM disorder. Check one: ☐ Likely ☐ Not likely

Appendix C. Example Structure of Evocative Film Clip Task

Appendix D. Example of Task Scale



Appendix E. Example of Pre-condition Experience Rating Scale



Appendix F. Difference Scores in Experience Ratings and Facial EMG

Note. Difference scores were made for experience ratings and facial EMG by taking the difference between precondition ratings/expressivity and expression and facial expressivity during the evocative film clip task. No significant differences were observed; Excitement and amusement in the bottom graph depicting difference scores from facial EMG reflect zygomaticus activity while fear and sadness are corrugator activity (microvolts).