2018-05-22

Physiological and Affective Reactivity to Emotional Comments in Individuals at High Risk for Psychosis

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PHYSIOLOGICAL AND AFFECTIVE REACTIVITY TO EMOTIONAL COMMENTS IN INDIVIDUALS AT HIGH RISK FOR PSYCHOSIS

By

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A DISSERTATION

Submitted to the Faculty
of the University of Miami
in partial fulfillment of the requirements for
the degree of Doctorate of Philosophy

Coral Gables, Florida

August 2018

UNIVERSITY OF MIAMI

A dissertation submitted in partial fulfillment of
the requirements for the degree of
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WEINTRAUB, MARC JOSHUA (Ph.D., Psychology)
Physiological and Affective Reactivity (August 2018)
To Emotional Comments in Individuals
At High Risk for Psychosis.

Abstract of a dissertation at the University of Miami.

Dissertation supervised by Associate Professor Amy Weisman de Mamani.
No. of pages in text. (109)

Psychotic symptoms are distributed along a continuum, ranging from subclinical experiences to clinically-defined psychosis. Individuals who are on the clinically diagnosable end of the psychosis spectrum (e.g. schizophrenia) tend to have a heightened sensitivity to social stressors, like Expressed Emotion (EE). EE measures how critical or overly-involved a family member is towards an identified patient, and is positively associated with higher rates of relapse and greater symptomatic presentations following a patient's hospitalization. Additionally, the mere exposure to a family member who is high in EE leads to greater physiological arousal in patients with schizophrenia compared to healthy controls. It is unclear, however, whether individuals not yet diagnosed, but at high-risk for a psychotic disorder, also have a heightened sensitivity to the social stress of EE. This study recruited individuals who are at high-risk for a psychotic disorder based on either genetic ties linking them to a first-degree family member (i.e. a parent or sibling) with schizophrenia and moderately elevated prodromal symptoms *or* elevated prodromal symptoms. The primary study aims were to examine whether high-risk individuals demonstrate greater physiological and subjective affective changes compared to low-risk controls after hearing critical, praise, and neutral comments directed at them. Measures of cardiovascular arousal (heart rate and heart rate variability), skin

conductance, cortisol, affect and anxiety ratings were used to assess differential responses to EE in patients at high-risk for psychosis compared to low-risk controls. Data was analyzed using repeated measures ANOVAs on a total of 38 high-risk individuals and 38 low-risk controls. Contrary to hypotheses, high-risk individuals did not show differences in reactivity to critical comments compared to controls. However, following critical comments, high-risk individuals did have slower heart rate recovery to baseline compared to controls. Further, high-risk individuals showed significant responses to praise comments. Specifically, despite higher baseline levels of negative affect and heart rate, these levels became nearly indistinguishable to controls following praise. There was also some evidence, that high-risk individuals perceived neutral comments as more negative than did their low-risk control counterparts. Overall, these results suggest that high-risk individuals are not more reactive to criticism than controls. High-risk individuals do, however, start at higher levels of negative affect, anxiety and heart rate, and their heart rate is slower to recover than controls. Additionally, praise comments appeared to benefit high-risk individuals as the praise made them nearly indistinguishable from the control subjects on multiple indices. Study findings have important clinical implications. They suggest that attending to regulatory strategies for stressors (such as criticism) and increasing positive social interactions (such as praise) may be helpful in reducing physiological hyperactivity and affect symptoms.

Keywords: at-risk, criticism, praise, negative affect, positive affect, heart rate, heart rate variability, cortisol, perceived criticism, loneliness

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CHAPTER 1: INTRODUCTION

Individuals with schizophrenia show sensitivity to family criticism and hostility (often measured by Expressed Emotion; EE), leading to a greater likelihood of relapse and worse prognosis when living in high EE environments. The overall aim of the current project is to investigate whether patterns of heightened sensitivity to EE evident in full-blown schizophrenia are also present in individuals who are high-risk for a psychotic disorder (based on genetic risk and moderate-level prodromal symptoms OR elevated prodromal psychotic symptoms). The proposed study will provide valuable knowledge about underlying vulnerabilities of high-risk populations and information about how certain familial interactional styles affect these vulnerabilities. Currently, no research has examined the immediate physiological or affective impact of negative family interactions (e.g., criticism) or positive family interactions (e.g., praise) on high-risk populations for psychosis.

As such, this project aims to fill an important gap in the existing literature by comparing the subjective arousal (affect and anxiety self-reports) and physiological arousal (heart rate, heart rate variability, skin conductance and cortisol) of high-risk individuals and low-risk controls in response to critical comments, as well as neutral and praising comments. The high-risk group was recruited based on either combined genetic risk (i.e. report of a first-degree relative with schizophrenia) and moderately heightened prodromal psychotic symptoms OR elevated prodromal psychotic symptoms. There are five specific aims for the current project. The first two aims are to compare the effect of (1) critical comments and (2) praise comments on subjective affect and anxiety ratings in the high-risk group versus the low-risk control group. The next two aims are to compare

the effect of (1) critical comments and (2) praise comments on physiological arousal in the high-risk group versus the control group. The final aim is to evaluate whether participants' perceptions of their key relative's criticalness and their self-rated feelings of loneliness moderate the effects of the emotional comments on affect, anxiety, and physiological arousal.

Psychosis Spectrum & High-Risk Populations

Schizophrenia is a chronic and severe mental illness, characterized by the presence of positive and negative psychotic symptoms (APA, 2013), and it occurs in about 1 in a 100 people (Saha et al., 2005). The human suffering and societal costs of schizophrenia are immense, as individuals with the illness often have a lifetime course of the illness, high rates of comorbidity with other psychiatric disorders, and poor psychological well-being (Buckley et al., 2009; Verdoux & van Os, 2002). Family members also experience significant emotional, psychological, and physical distress when caring for a loved one with schizophrenia (Awad & Voruganti, 2008). Total costs for the illness in 2002 were estimated to be $62.7 billion in the U.S., and these costs have been projected to grow continually in the foreseeable future (Wu et al., 2005). Due to the major impact schizophrenia has on society and individuals with the illness, it is crucial to evaluate the mechanisms that lead to the disorder.

One method to better understand mechanisms that may lead to the onset of schizophrenia is through the examination of sub-clinical psychosis populations. Psychotic symptoms and the occurrence of unusual experiences (e.g. false beliefs and hallucinations) that do not reach threshold for a psychotic disorder are considered psychosis proneness or "at-risk mental states" (Claridge, 1997). The psychosis phenotype

is conceptualized as a continuum, with symptoms ranging from sub-clinical manifestations to full-blown schizophrenia (e.g. Chapman et al., 1994; van Os et al., 2000). These unusual, psychotic-like experiences are fairly common. The prevalence of one lifetime delusional or hallucinatory experience by the age of 26 is estimated to be 20.1% and 13.2%, respectively (Poulton et al., 2000). Overall, about 5-8% of the general population experiences repeated sub-clinical symptoms of psychosis (Kelleher & Cannon, 2011). The distinction between these varying levels of the psychosis continuum, then, is based on the severity, duration, and impairment caused by the symptoms (van Os et al., 2009).

The ability to accurately identify high-risk individuals for a psychotic disorder has improved greatly in recent years. The most widely recognized method of classifying individuals as "high-risk" involves assessing for genetic risk for schizophrenia (i.e., having a first degree relative with a psychotic disorder) as well as elevated levels of attenuated positive psychotic symptoms (e.g., prodromal symptoms; Cannon et al., 2008). Using this assessment approach, individuals can be classified into three "psychosis-risk syndrome states" – (1) genetic risk and deterioration state, (2) attenuated positive symptom state, or (3) brief intermittent psychotic state (i.e., clinical psychotic symptoms emerging in the recent past that occur too briefly to meet official criteria for a diagnosis of psychosis), of which criteria 1 and 2 make up approximately 99% of the risk states (McGlashan, Walsh, & Woods, 2010). This methodology builds upon the initial risk-classification process, which mainly sought out individuals at genetic risk for psychosis (Lichtenstein et al., 2006). Having a first-degree relative with a psychotic disorder places an individual at approximately 9% risk of developing a psychotic disorder (8.5% in full

siblings, 10.3% in offspring), which greatly improves the ~1% prediction rate seen in the general population (Lichtenstein et al., 2006). However, with the current psychosis-risk syndrome classifications, Cannon and colleagues have been able to increase prediction rates to 41% after 2½ years of follow-up (2008). The current study will use the genetic risk and elevated attenuated positive symptoms states (the two most commonly seen psychosis-risk states) to base identification for high-risk status – assessing for the presence of a first-degree relative with schizophrenia and moderately elevated prodromal symptoms OR greatly elevated prodromal symptoms.

Studying individuals who are at high-risk for psychosis can provide information about whether patterns that are witnessed in full-blown schizophrenia are also evident at sub-clinical levels. This type of work can be a crucial step in elucidating the mechanisms that are associated with conversion to a psychotic disorder, which is immensely important from a mental health and social welfare perspective. For example, examining whether individuals at-risk for psychosis show a greater sensitivity to stressors (compared to low-risk controls) that are also seen in patients with psychosis can provide insights into the psychosocial pathways that lead to psychosis. Finding early mechanisms in which to intervene is crucial, as a longer duration of untreated psychotic symptoms is associated with worse response to therapy and functional outcomes (Marshall et al., 2005). Thus, investigating individuals who are at high-risk for psychosis can provide important information about the full psychosis spectrum and elucidate the mechanisms that lead to full-blown schizophrenia.

Stress Response and Psychosis

Daily stressors and stressful life events are positively correlated with psychotic and affective symptoms in patients with psychosis (Lataster et al., 2012; Phillips et al., 2007). Some research has found a "threshold effect" for stressful life events and the exacerbation of symptoms (i.e. when the number or severity of stressors exceeds a particular threshold, symptom onset occurs). For example, Lataster and colleagues (2012) found in a longitudinal population study that 10 or more recent negative life events significantly increased the risk of developing psychotic symptoms. Further, a study on the largest cohort of high-risk individuals to-date has found that high-risk participants who progressed to having a psychotic disorder reported a greater frequency of stressful life events than those whose prodromal symptoms remitted (Trotman et al., 2014).

Other research has not found this effect, as their results have shown that clinically high-risk individuals do not experience (quantitatively) more stressors than low-risk controls (Phillips et al., 2012; Tessner, Mittal, & Walker, 2012). Rather, high-risk individuals who convert to a psychotic disorder experience more serious/traumatic stressors. Some research has found a link between the development of psychotic symptoms and the experience of trauma during childhood (Bechdolf et al., 2010; Janssen et al., 2004) or extreme stress before migration in studies on refugees (Bhui et al., 2003; Zolkowska, Cantor-Graae, & McNeil, 2003). Further, a study examining childhood trauma found that individuals who had experienced serious childhood trauma (e.g. neglect, abuse) were three times more likely to exhibit psychotic symptoms than those who did not (Varese et al., 2012).

It is unclear whether the development of psychosis is dependent upon the number of stressful events versus the severity of stressful events (or both). In fact, some findings indicate that patients with schizophrenia report fewer stressful events, but they appraise these events as less controllable and more difficult to manage (Horan et al., 2005). Both patients and high-risk individuals perceive their stressors as more stressful and rate themselves as having greater responses to the stressors than healthy controls (Myin-Germeys et al., 2001; Phillips et al., 2012; Tessner et al., 2011; Trotman et al., 2014). This tendency for patients to appraise stressors as more stressful has been a consistent finding throughout the psychosis literature. It is believed that these individuals are more sensitive to stress because they have greater emotional reactivity, as indexed by self-report measures of reactivity and arousal (Docherty et al., 2009). Docherty and colleagues found that emotional reactivity moderates the relationship between stressful life events and psychotic symptoms, such that only individuals who are high in emotional reactivity showed symptom exacerbations in response to stressful events. Overall, these findings indicate that patients with schizophrenia and individuals at high risk for psychosis experience stressors as more subjectively stressful.

It is often difficult to make conclusions about studies collecting subjective ratings of stress, particularly when comparing healthy and clinical populations. It could be that individuals who are prone to psychotic disorders perceive similar events as more stressful than healthy controls, and/or it could be possible that individuals who have a psychotic disorder experience(d) more serious or severe stressors in their lives compared to healthy controls. To objectively examine stress along the psychosis continuum, much research has examined the hypothalamic-pituitary-adrenal (HPA) axis both in its resting state, as

well as in response to stressors. Studying the HPA axis largely originated from the diathesis-stress model of psychosis, which has been the prominent theory on the etiology of psychotic disorders (Walker & Diforio, 1997; Walker, Mittal, & Tessner, 2008). The diathesis stress model posits that psychosocial and biological factors interplay such that there is an initial biological vulnerability/genetic predisposition that interacts with environmental stressors and leads to the onset of the disorder. The response to stress in the human body is commonly measured through the HPA axis' cascade of hormones and neuroendocrine signals (Stevens & White, 2010). Following the exposure to a stressor, cortical input to the hypothalamus signals the release of a corticotrophin-releasing hormone (CRH), which then results in the secretion of adrenocorticotrophic hormone (ACTH) from the anterior pituitary gland into the bloodstream. ACTH then stimulates the adrenal gland's production of glucocorticoids, which is primarily cortisol (the "stress hormone") in humans. Once cortisol is secreted into the bloodstream, it acts as a catalyst for the sympathetic nervous system, crossing the blood-brain barrier and activating the body's arousal system (e.g., increasing heart-rate and skin conductance; Holtzman et al., 2013).

There has been a growing body of literature on the HPA axis and psychosis (Walker et al., 2008). In particular cortisol, the "stress hormone" and proxy for sympathetic arousal, has received much attention. There is evidence indicating that patients with psychosis (especially patients who are not receiving medication) have higher baseline cortisol levels and decreased heart rate variability compared to healthy controls (e.g. Garner et al., 2010; Ieda et al., 2014; Kale et al., 2010). These findings indicate that baseline HPA activity (i.e. when in the absence of acute stressors) is higher

in patients with psychosis than in healthy controls. However, research examining at-risk populations have found mixed results when comparing daytime basal cortisol levels in high-risk individuals and low-risk controls (Day et al., 2014; Walker et al., 2013). Further, some research has suggested that patients' psychotic symptoms are positively related to cortisol levels (Gamer et al., 2010; Murri et al., 2012; Walker et al., 2013), but other research has not fully replicated these findings (e.g., Corcoran et al., 2012). With such varied results in the literature, it is difficult to determine whether there are differences in basal cortisol and whether cortisol plays a direct role in the worsening of the at-risk state.

One theory to explain the varied cortisol findings is that psychiatric medications are commonly used in individuals at-risk for psychosis, and these medications alter HPA activity. For example, antipsychotics reduce the secretion of cortisol and ACTH, and the reduction of symptom severity as a result of antipsychotic medication has been linked with the magnitude of cortisol reduction (Mondelli et al., 2010; Venkatasubramanian et al., 2010; Walker et al., 2008). On the other hand, medication that increases cortisol levels (e.g. combining a benzodiazepine agonist and a serotonin agonist) has been found to increase psychotic symptoms in patients with schizophrenia and healthy controls (D'Souza et al., 2006; Lindenmayer et al., 1997). Early studies on high-risk psychosis populations have also been plagued with small sample sizes and poor methods (e.g. assessing cortisol at only one time point; Dowd et al., 2009 & Thompson et al., 2007). More conclusive results have emerged from recent research with larger samples, and these results seem to indicate that high-risk individuals have elevated baseline cortisol levels compared to low-risk controls (Walker et al., 2010; Walker et al., 2013). Further,

Walker and colleagues (2010) found that individuals who subsequently converted to a psychotic disorder had consistently higher cortisol levels throughout the year preceding the onset of their disorder.

The research on the HPA axis indicates that patients along the psychosis continuum tend to have an elevated 'set point' of stress hormone secretion and HPA activity. Further, as mentioned above, it has been suggested that patients with psychotic disorders are more sensitive to stressors. Much theoretical work and some recent empirical findings suggest that high-risk populations have dysregulated HPA reactivity to stressors as well (Walker et al., 2008). However, very little research has examined the HPA axis' response to acute *social* stressors in patients with schizophrenia. A study examining the effects of the Trier Social Stress Test (which involves having the participant give a public speech and perform mental arithmetic) found that patients with schizophrenia had a blunted cortisol response compared to healthy controls (Brenner et al., 2009). Other studies have also found that patients with schizophrenia have blunted cortisol responses to psychological and psychosocial stressors (Albus et al., 1982; Jansen et al., 2000). However, Brenner and colleagues (2009) note that both antipsychotic medication and smoking are negatively associated with cortisol response (both of which are common in patients with schizophrenia) and are likely to contribute to these results.

Similar to the literature on psychotic disorders, little work has examined the social stress response in individuals at high risk for psychosis. However, the literature on stress reactivity in high-risk populations thus far suggests that their stress response is more pronounced than that of low-risk controls. In a study that used the Montreal imaging stress task, a mental arithmetic assignment, high-risk participants had increased stress-

induced dopamine secretion as compared to low-risk controls (Mizrahi et al., 2012). Further, a study using a virtual reality paradigm found heightened sensitivity to environmental social stressors in individuals at high risk for and individuals diagnosed with a psychotic disorder (grouped together in the analyses) compared to low-risk controls and siblings of patients with psychosis (Veling et al., 2016). However, the effects of social stress on cortisol and other psychophysiological reactivity have not yet been tested in high-risk populations.

Expressed Emotion

There is no evidence that families cause schizophrenia, but families can affect the course of the patient's illness. One of the most consistent predictors of psychiatric relapse is a measure of social/family stress called expressed emotion (EE; Hooley, 2007). EE is a measure of how critical, hostile, and overly-involved a family member is towards the patient, and is thought to represent relational disturbances and transactional patterns between the family member and the patient (Miklowitz, 2004). Critical remarks demonstrate disapproval of the patient's actions (e.g., "it's very annoying that she's always sleeping and doing nothing"). Hostile remarks also demonstrate disapproval or dislike, but in a more generalized way (e.g., "he's very lazy and I have to make him do everything"). Finally, emotional over-involvement reflects a devoted, but over-protective style with the patient (e.g., I need to stay with her at all times. She may need me while I'm away"). While an individual who is elevated on any one of these three relational styles would be classified as high EE, the most important component of EE is criticism (e.g., Brown, Birley, & Wing, 1972; Hooley & Parker, 2006; Vaugn & Leff, 1976). This is because it is most strongly linked to relapse in patients with schizophrenia, as

determined by the number of critical comments made during the semi-structured interview that measures EE (Hooley, 2007).

Efforts to understand the familial factors that are associated with differing clinical outcomes were first studied in patients diagnosed with schizophrenia (Rutter & Brown, 1966). As a result, most of the research on the EE construct has been done with patients that have this disorder (Hooley, 2007). A meta-analysis of 26 studies on EE and relapse showed that patients with schizophrenia who live in a high EE environment have more than twice the relapse rates of patients who live in a low EE environment, within 9 to 12 months after hospitalization (Butzlaff & Hooley, 1998). While the direction of this effect is difficult to determine (since it is hypothetically possible that patients who are at high-risk for relapse elicit high EE comments from relatives), a review of the literature supports the assumption that high EE is causally related to worse symptom outcomes (Hooley & Gotlib, 2000). For example, even when important patient variables (e.g., duration of illness, transactional processes) are controlled statistically, levels of familial EE still make a significant and independent contribution to relapse (Nuechterlein, Snyder, & Mintz, 1992). Additionally, intervention studies that have reduced levels of EE within the family have shown decreased rates of relapse for patients with schizophrenia (e.g., Hogarty et al., 1991; Miklowitz & Tompson, 2003).

Although it may be expected that patients who are more severely ill or have more symptoms of psychopathology would have relatives who are more critical of them, this does not appear to be the case. Levels of EE and levels of psychopathology are uncorrelated, meaning that high EE is found equally both in families with severely and mildly symptomatic patients (e.g. Brown et al., 1972; Cutting, Aakre, & Docherty, 2006;

Miklowitz, Wendel, & Simoneau, 1998). Therefore, EE is present at all levels of psychosis spectrum. What remains unclear, however, is whether high EE comments affect individuals at all levels of the psychosis spectrum in the same way. Investigating the effect criticism has on sub-clinical levels of psychosis will provide insight into whether criticism (and high EE more generally) may play a role in the conversion to full-blown schizophrenia. Thus, this study will ascertain whether individuals at high-risk for psychosis are more sensitive to critical comments than a general, low-risk population.

High Criticism as a Stressor

Expressed emotion is considered a psychosocial stressor that interacts with the patients' diatheses to produce relapse (Hooley & Gotlib, 2000). Hearing critical or hostile comments is not a pleasant experience for anyone, but individuals with schizophrenia appear to be particularly sensitive to these types of comments (Butzlaff & Hooley, 1998). High EE relatives tend to talk more and listen less (Kuipers et al., 1983). Additionally, they make critical remarks directly to patients more often than do low EE relatives (Miklowitz et al., 1984). They also are more prone to disagree with patients and attribute more control over the patients' behavior (e.g. they believe the patient can control more aspects of their illness than do low EE relatives; Hooley, 1986; Weisman et al., 2000).

Patients with schizophrenia also have heightened physiological responses when interacting with a high EE relative. When patients were tested in their own homes, being in the presence of a high EE relative resulted in elevated diastolic blood pressure compared to their own baseline heart rate and to healthy controls who were also in the presence of a high EE relative (Tarrier et al., 1979). On the other hand, patients with low EE relatives had a decrease in the frequency of non-specific skin conductance responses

(NS-SCRs). Subsequent studies found similar results, suggesting that greater electrodermal arousal is associated with high EE relatives (e.g. Tarrier & Turpin, 1992; Sturgeon et al., 1984). An extension of this research found that critical and stressful statements by relatives were connected with increased cardiovascular activity in patients with schizophrenia and bipolar disorder (Altorfer, Kaeserrmann, & Hirsbrunner, 1998). The cortisol response to high EE environments in patients with schizophrenia has not been examined. Overall, these results indicate that patients with schizophrenia are physiologically stressed when interacting with a high EE relative. However, it is uncertain whether individuals who are at high-risk for psychosis have similar physiological responses. This study will elucidate the physiological and emotional effects of critical comments on individuals at high-risk for psychosis.

Moderators of Stress Response

Much research has found that perceptions of stress are important indicators of how an individual responds to stress (reviewed above). This effect was shown in patients with psychosis, as only individuals who were reactive to and perceived an event as stressful had exacerbations in their psychotic symptoms (Docherty et al., 2009). Further, patients' perceptions of criticism have been found to be an even stronger predictor of relapse than objective measures of EE (Hooley & Teasdale, 1989). A similar pattern is believed to take place in individuals at high risk for psychosis, as they have been shown to face a similar number of daily stressors as low-risk controls, yet rate them as more stressful (e.g. Tessner et al., 2011). Therefore, getting the participants' perspective by measuring their perception of a stressor is important. Clinician rated measures (like the Camberwell Family Interview) have been created to assess the degree of expressed

emotion within the family (Leff & Vaughn, 1985); however, these objective measures of EE do not always match up with the participants' subjective perception of the family climate. For example, relapse rates in objectively high EE families do not always predict a poorer course of illness (Rosenfarb, Bellack, & Aziz, 2006). The reason for this finding is that some patients do not perceive comments as being critical, despite them being designated as critical through EE coding systems (Weisman, Rosales, Kymalainen, & Armesto, 2006). It would seem, then, that patients are not as likely to be aversively affected by comments that they do not perceive as critical. These results highlight the importance of gathering information on participants' own perceptions of their relatives' criticism.

With the importance of participant perceptions in mind, a specific measure was created to assess an individual's perception of their relative's expressed emotion, and it has been shown to be a robust predictor of psychiatric outcomes (Hooley & Teasdale, 1989). The measure of perceived criticism (PC) was specifically designed to target the key component of EE, criticism, and has been found to be highly predictive of depressed patients' psychiatric relapse over the course of a 9-month follow-up. PC has also shown predictive validity in anxiety disorders, obsessive-compulsive disorder, and bipolar disorder (Chambless & Steketee, 1999; Miklowitz et al., 2005). There is no known predictive validity data of PC for patients with psychotic disorders; however, a similar measure of perceived criticism used with patients with schizophrenia has shown good concordant validity with the CFI (Weisman et al., 2006).

Another potential moderator of response to a social stressor is perceived loneliness. Loneliness has also been linked to a variety of poor outcomes, including

increases in anxiety and depressive symptoms as well as the worsening of psychotic symptoms (Freeman & Garety, 2003; Sündermann et al. 2014). Many studies have found that individuals with a psychotic disorder report smaller social networks and lower levels of social support than others (e.g. Neelman & Power, 1994; Norman, Malla, & Manchanda, 2005). Little research has examined whether perceived loneliness plays a role in the lives of individuals at high risk for psychosis, but the work that has been done suggests that these individuals also have smaller social networks, fewer close friends, and diminished social support relative to the general population (Gayer-Anderson & Morgan, 2013). Overall, it appears that loneliness and reduced social support are present across the psychosis spectrum.

As described above, perceptions of one's social interactions can be an important indicator of how that individual responds (both affectively and physiologically) to social situations. In terms of perceived loneliness and social support, perceptions of being supported socially (i.e., not feeling alone) may be an important buffer for stress, as social support has been shown to be negatively associated with emotional reactivity towards stressful events (Affleck et al., 1994; DeLongis et al., 1988). On the other hand, it is believed that loneliness and a lack of social support may be increase stress responses and contribute to the worsening of the prodromal state (Gayer-Anderson & Morgan, 2013). It is theorized that loneliness may worsen prodromal psychosis as these individuals (1) may not have family or peer support to help "reality test" unusual experiences and/or (2) may not have support in sharing their stressful experiences (French & Morrison, 2004). Together, this body of work suggests it is important to assess whether feelings of

loneliness may act as a moderator of physiological and affective response to a social stressor in individuals at high risk for psychosis.

Praise as an Exploratory Buffer

Much work has found that criticism is a psychosocial stress that interacts with a patient's diathesis to produce relapse (Hooley, 2007); however, the effects of positive interpersonal interactions on individuals with psychosis has not been well studied. Warmth is included as a rating that is calculated when expressed emotion is measured, and was an original component of the expressed emotion construct (Brown et al., 1972). However, because warmth is not included in the overall rating (high versus low) of expressed emotion and because Brown and colleagues (1972) found such a strong negative association with criticism and emotional overinvolvement, research has largely neglected to examine the relationship between warmth and patient outcomes (Hooley, 2007). In addition, warmth is often overlooked as a worthwhile variable when examining the progression and course of psychosis.

Studying the effects of positive social interactions may yield valuable insight into potential buffers against the progression of psychosis. Two studies have found that family warmth and positive family environments can be protective factors for relapse in patients with schizophrenia (Lee, Barrowclough, & Lobban, 2014; López et al., 2004). However, the study of warmth and its effects on individuals at high risk for psychosis is a large gap in the literature. Examining positive social interactions, like warmth, in individual at high risk for psychosis can help us understand whether these individuals can benefit from positive social interactions. Although there is limited evidence to draw upon, based on the research by Lee and colleagues and López and colleagues, it is possible that warmth

is a protective factor for individuals at high-risk for psychosis. More specifically, warm social interactions may be protective in that (1) positive, praising social interactions can feel nice, thereby increasing positive affect, as well as (2) help reduce negative affect and anxiety. Warmth may also help by calming the recipient's physiological state and reduce physiological arousal.

The Current Study

This study has five main objectives. (1) To determine whether high-risk participants respond to critical comments with greater subjective affect and anxiety compared to their baseline measurements, their responses to neutral comments, and compared to low-risk controls' responses. (2) To determine whether high-risk participants respond to praise comments with greater subjective affect and anxiety compared to their responses to their baseline measurement, their responses to neutral comments, and compared to low-risk controls' responses. (3) To determine whether high-risk participants respond to critical comments with increased physiological arousal (measured by heart rate, heart rate variability, skin conductance, and salivary cortisol secretion) compared to their baseline measurement, their responses to neutral comments, and compared to low-risk controls' responses. (4) To determine whether high-risk individuals for a psychotic disorder respond to praise comments with reduced physiological arousal (measured by heart rate, heart rate variability, skin conductance) compared to their baseline measurements, their responses to neutral comments, and compared to low-risk controls' responses. (5) To investigate whether high-risk individuals' perceptions of their relative's criticalness and feelings of loneliness moderate these effects on their physiological arousal, affective and anxiety reactivity. To

accomplish these objectives, this study used an established paradigm of standardized neutral, critical, and praising comments previously employed with remitted depressed patients (Hooley et al., 2010). The neutral comments are helpful in providing an alternative comparison to the baseline measurements for the critical and praise comments. They allow for the examination (as noted above) of whether the "neutral" comments are perceived as neutral by the participants. The neutral comments also allow for an examination of whether hearing comments at all creates affective, anxiety, and physiological changes relative to one's baseline.

Summary of Hypotheses

Drawing from the research reviewed above, the current study tested eight primary hypotheses:

- *Hypothesis 1*: All participants will have increased negative affect after hearing critical comments and lower negative affect after hearing praising comments compared to their baseline negative affect and negative affect following the neutral comments. Additionally, these responses will be greater for the high-risk group compared to the low-risk group.
- *Hypothesis 2*: All participants will have lower positive affect after hearing critical comments and increased positive affect after hearing praising comments compared to their baseline positive affect and their positive affect following the neutral comments. Additionally, these responses will be greater for the high-risk group compared to the low-risk group.
- *Hypothesis 3:* All participants will have increased heart rate after hearing critical comments and lower heart rate after hearing praising comments compared to their

baseline heart rate and their heart rate following the neutral comments. Additionally, these responses will be greater for the high-risk group compared to the low-risk group.

- *Hypothesis 4:* All participants will have lower heart rate variability after hearing critical comments and increased heart rate variability after hearing praising comments compared to their baseline heart rate variability and their heart rate variability following the neutral comments. Additionally, these responses will be greater for the high-risk group compared to the low-risk group.
- *Hypothesis 5:* All participants will have increased skin conductance responses after hearing critical comments and reduced skin conductance responses after hearing praising comments compared to their baseline skin conductance response and their skin conductance response following the neutral comments. Additionally, these responses will be greater for the high-risk group compared to the low-risk group.
- *Hypothesis 6:* All participants will have higher cortisol levels after hearing critical comments compared to their cortisol levels following the neutral comments. Additionally, these responses will be greater for the high-risk group compared to the low-risk group.
- *Hypotheses 7 & 8:*
    - Perceptions of their relative's criticalness will moderate the relationship between emotional comments and arousal, such that individuals from both groups who perceive their relative as more critical will have greater physiological arousal, greater negative affect, reduced positive affect, and

greater anxiety after hearing emotional comments compared to individuals who perceive their relative as less critical.

- Feelings of loneliness will moderate the relationship between emotional comments and arousal, such that individuals from both groups who report feeling more lonely will have greater physiological arousal, greater negative affect, reduced positive affect, and greater anxiety after hearing emotional comments compared to individuals who report feeling less lonely.

CHAPTER 2: METHODS

Participants

Two groups of participants were recruited from the South Florida (i.e. Miami) area. Group 1 (high-risk group) included individuals at high-risk for psychosis. Participants were eligible for the high-risk group if they reported having highly elevated prodromal symptoms OR reported having a first-degree relative with schizophrenia/schizoaffective disorder and moderately elevated subclinical psychotic symptoms (measures to determine cut-offs are detailed below). Group 2 (low-risk control group) included participants who did not have a first degree relative with schizophrenia/schizophrenia and had a total score below the at-risk cutoff on the clinical risk measure. Both groups also had to report being free of any current psychiatric medication use, nicotine use in the last 30 days, substance abuse and dependence over the past 3 months, current post-traumatic stress disorder (PTSD), as well as current use of other commonly known medications that affect the measurement of cortisol (see cortisol section below). All participants were between the ages of 18 and 30 (defined as the peak period of risk for first-onset psychotic disorders; Yung et al., 2005).

Procedures

To recruit participants, advertisements were placed on the Miami Metrorail, around the Miami community, and on Craigslist. Two fliers were used to recruit for high-risk participants. One said, “In the last month: Have you felt that other people are watching you or talking about you? Have you felt that you’re not in control of your own ideas or thoughts? Are you between 18 and 30 years old? If so, you may be eligible to participate in a paid, 3-hour research study at the University of Miami.” The second high-

risk flier said, “Do you have a parent or sibling who has been diagnosed with schizophrenia or schizoaffective disorder? Are you between 18 and 30 years old? If so, you may be eligible to participate in a paid, 3-hour research study at the University of Miami.” The flier for low-risk participants said, “Are you between 18 and 30 years old? If so, you may be eligible to participate in a paid research study at the University of Miami.”

Prior to the participants coming into the laboratory, interested persons were screened over the telephone for a family history of a first-degree relative with schizophrenia/schizoaffective disorder and prodromal symptoms to classify them into the “high-risk” or “low-risk” groups (see measures section below). Participants were also screened for substance use, abuse and dependence, PTSD, and medication use. Individuals who scored in the high-risk range on the prodromal symptoms measure were also given the Structured Clinical Interview for DSM-IV Disorders (SCID) psychotic screening module to ensure they do not meet criteria for a psychotic disorder. If the interested individual was eligible for the study (matching either the high-risk or low-risk criteria), the participant was scheduled to participate in the study at the University of Miami’s Department of Psychology. Verbal and written instructions (sent via email) were provided with dietary and behavioral instructions to ensure proper measurement of cortisol (detailed below). Participants were then provided with an online link of questionnaires to complete prior to arriving to the study, which included measures of the participants’ perceptions of their relative’s criticalness, their feelings of loneliness, as well as trait-like levels of depression and anxiety symptoms over the previous two weeks.

Upon arrival at the University of Miami, participants were questioned to determine their compliance with the dietary and behavioral instructions for the cortisol measurement (described below). All participants confirmed their compliance with the dietary and behavioral instructions before being able to begin the study. Participants were then set up to a BioPac System for heart rate, respiratory, and skin conductance measurement. Participants waited in silence for five minutes before taking baseline measurements. Baseline heart rate, heart rate variability and skin conductance measurements were then taken for a total of three minutes to match the length of physiological measurements for each comment-set (described below). Participants were instructed to sit as still as possible and wait silently for the three minute recording period. Following the baseline physiological measurement, the baseline self-report affective and anxiety measurements were obtained. Finally, a baseline salivary cortisol measurement was taken.

After the baseline measurements were taken, the participants were asked to listen to three sets of comments (neutral comments, critical comments, and praise comments; Hooley et al., 2010). They were instructed to imagine that a close female relative was saying each of the comments to them. Each participant listened to one comment-set, followed by a 30-minute break, then another comment-set, followed by a 30-minute break, and then the final set of comments. The neutral and critical comment-sets were counterbalanced throughout the study. Because it was unclear how stressful the critical comments would be for the high-risk participants, it was decided that it would be most ethical to play the praise comments last for each participant. That way, participants would leave the laboratory following exposure to a positively valenced stimuli.

The 30 minute breaks allowed for the collection of subjective affect and anxiety ratings as well as salivary samples for cortisol analysis. Participants were asked to fill out the self-report affect and anxiety questionnaires directly following the comments and the physiological recordings. Once participants were done completing these questionnaires, they were shown a calming nature video that lasted for the entirety of the between-comments period (about 25 minutes). Reactivity and recovery salivary cortisol measurements were also taken during the break period, as well as a second baseline cortisol measurement before the next comment-set (more detail is given below). Participants were instructed not to use any electronics (e.g. cell phones) throughout the study to ensure consistency across all participants. Participants were compensated $40 each for their time and effort. See Table 1 for the progression of study procedures.

Table 1. Study procedures

	Pre-Study
	Eligibility Screen
	Study
9:00AM	Consent & Attach to Bio Pac System
9:20AM	Baseline measurements gathered
9:30AM	First comment-set played
9:33AM	Self-report affect & anxiety
9:45AM	Reactivity cortisol measurement
9:55AM	Recovery cortisol measurement
10:05AM	Pre-comment cortisol measurement
10:05AM	Second comment-set played
10:08AM	Self-report affect & anxiety
10:20AM	Reactivity cortisol measurement
10:30AM	Recovery cortisol measurement
10:35AM	Praise comments played
10:38AM	Self-report affect & anxiety

Measures and Measurements

All measures are described below and self-report measures are included in the Appendix.

Prodromal Questionnaire—Brief (PQ-B). The PQ-B (Loewy et al., 2011) is a self-report of prodromal symptoms adapted from the original Prodromal Questionnaire (see below). The PQ-B only contains positive symptom items, as these symptoms are the basis of the psychosis-risk syndrome diagnoses (McGlashan, Walsh, & Woods, 2010). Further, the positive symptoms that were retained from the original Prodromal Questionnaire were the approximately 33% of items that were least likely to be endorsed by a general undergraduate university sample. Participants first rated their agreement for each of the 21 items (yes/no). If an item was rated as "yes," the participant then rated their distress on that item on a five point Likert-scale (ranging from "strongly disagree" to "strongly agree") to a secondary item which reads: "When this happens, I feel frightened, concerned, or it causes problems for me." A combination of a greater total score in addition to a greater distress score is indicative of greater risk for psychosis. Sample items on the PQ-B are "Have you heard unusual sounds like banging, clicking, hissing, clapping, or ringing in your ears?" and "Do you find yourself feeling mistrustful or suspicious of other people?"

With cutoff values of a total score greater than or equal to 3 and a distress score of greater than or equal to 6, the PQ-B has over a 90% positive predictive value and over 85% sensitivity for a diagnosis of the psychosis-risk syndrome (Loewy et al., 2011). Therefore, a total score of greater than 3 *and* a distress score of greater than 6 on the PQ-B (but not meeting full criteria for a psychotic disorder as measured by the SCID psychotic screening module; discussed below) indicated clinical high risk for the

individuals who also have a first degree relative with schizophrenia/schizoaffective disorder. With cutoff values of a total score greater than or equal to 6 and a distress score of greater than or equal to 32, the PQ-B has a 100% positive predictive value and over 95% sensitivity for a diagnosis of the psychosis-risk syndrome (Loewy et al., 2011). Thus, a total score of greater than or equal to 6 *and* a distress score of greater than or equal to 32 on the PQ-B (but not meeting full criteria for a psychotic disorder as measured by the SCID psychotic screening module indicated clinical high risk for the individuals who do not have a first degree relative with schizophrenia/schizoaffective disorder. A total score of less than 3 *and* a distress score of less than 6 on the PQ-B indicated low risk. The internal consistency for the PQ-B within this study was excellent (PQ-B total Cronbach's $\alpha = .93$; PQ-B distress Cronbach's $\alpha = .93$).

Structured Clinical Interview for DSM-IV Disorders – psychotic screen II, *PTSD section of the anxiety disorders module, and substance use disorders* (SCID). The SCID (First et al., 2002) is a widely used semi-structured instrument to identify individuals who meet criteria for DSM-IV disorders. The PTSD section within the anxiety disorders module and the substance use disorders module were administered to each participant to ensure he/she did not meet criteria for any of these disorders. The psychotic screen was also administered to each high-risk participant to ensure he/she did not meet criteria for these disorders. The SCID has demonstrated strong convergent validity with other standard clinical interviews (Basco et al., 2000; Fennig et al., 1994). All SCID assessment were conducted by the primary investigator of the study. The study's primary investigator demonstrated reliability for the SCID psychotic screen by rating and determining an overall diagnosis for eight videotaped interviews (in six of the training

tapes a psychotic disorder diagnosis was present, and in two it was absent). The assigned diagnoses from the videotapes were compared with the diagnoses assigned by the dissertation chair (Amy Weisman de Mamani, Ph.D., Licensed Clinical Psychologist). Results indicated perfect inter-rater reliability (Cohen's Kappa = 1.0) for the eight interviews.

Family Interview for Genetic Studies – Psychosis Checklist (FIGS). The FIGS (Maxwell, 1992) is a semi-structured interview used to collect diagnostic information about relatives of study participants. The primary study investigator collected diagnostic information on the first-degree relative that was reported to have schizophrenia in the high-risk group and all first-degree relatives in the low-risk group. Within the Psychosis Checklist subsection, subjects are asked about symptoms, including delusions, hallucinations, bizarre behavior, catatonia, and avolition. Sample items include, "Did he/she believe someone was reading his/her mind?" and "Did he/she see things that were not really there?" Symptoms endorsed by participants about their first-degree relative(s) were matched with the DSM-IV-TR to determine whether the relative met criteria for a psychotic disorder. The FIGS has been found to have good reliability for assigning accurate diagnosis of a relative (k = .73) and good test-retest reliability (k = .75; Nurnberger et al., 1994).

Depression, Anxiety and Stress Scale. The Depression, Anxiety and Stress Scale (DASS; Henry & Crawford, 2005) is a measure of emotional well-being that was used to measure trait-like symptoms of depression and anxiety for the two weeks prior to the study. The DASS is a self-report questionnaire with 21 items that make up three factors: depression, anxiety, and stress. Only the depression and anxiety factors were used, as

affect (via depressive symptoms) and anxiety were the constructs of interest for this study. Each factor was measured by 7 items on a 4-point Likert scale (0 = did not apply to me at all, 1 = applied to me somewhat, 2 = applied to me a considerable degree, or a good part of the time, and 3 = applied to me very much, or most of the time). Sample items from the DASS include, "I felt like I had nothing to look forward to" and "I couldn't seem to experience any positive feelings." In this study, the anxiety subscale showed good internal reliability (Cronbach's $\alpha = .88$). The depression subscale showed excellent internal reliability (Cronbach's $\alpha = .96$).

Standardized Expressed Emotion Comments (Hooley et al., 2010). The standardized comments were recorded onto a digital disk and will be played for the participant through headphones. The same female voice was used for each comment. Each comment within the neutral, critical, and praise conditions lasted approximately 20-25 seconds each (with ~5 seconds of silent space between comments), and there were a total of four comments for each comment-set. Thus, each comment-set lasted 2 minutes. All comments were phrased in the first person, and participants were asked to listen to each comment as if it were being said to them by a close, female relative (preferably their primary female caregiver growing up). The comments were written by Jill Hooley, D.Phil., and are based on actual comments made by relatives of patients with severe mental illnesses. These standardized comments have been found to elicit affective changes and higher activation in prefrontal brain regions in patients with borderline personality disorder (Hooley et al., 2010). An example of one of the standardized critical comments is, "Another thing that really bothers me is how lazy and apathetic you can be sometimes. You often tend to just sit around basically doing nothing but vegetating. And

you can watch mindless stuff on T.V. for hours and hours. You say you're bored but that's because you have no life. How can you have a life if you don't make any effort? You need to try much harder than you do." An example of one of the neutral standardized comments is, "One thing you did last week was to go out for a walk. It was a nice sunny day, so you are pleased to be outside. You walked from the house to the post office, a distance of about a half of a mile. When you got there, you waited in line, and then bought some stamps. You also mailed a package to a friend of yours. The whole trip took less than an hour." An example of one of the praise standardized comments is, "One of the things I really like about you is your sense of humor. It's not that you are always telling jokes or anything like that but you can be really, really funny. You have such a positive way of seeing the world that I just love. And when you laugh your whole face lights up and you bring an energy to the room that everyone wants to be around. It's just so great to see you like that."

Positive and Negative Affect Schedule (PANAS). The PANAS (Watson, Clark, & Tellegen, 1988) was used to measure participants' state level of positive and negative affect in response to standardized expressed emotion comments. It contains ten positive (e.g. "Strong," "Proud," and "Enthusiastic") and ten negative affective descriptors (e.g. "Distressed," "Upset," "Scared"). Each item is rated on a 1-5 Likert scale (1 = very slightly or not at all; 5 = extremely). Scores for the two sub-sections were summed to give a total score for positive and negative affect. The internal consistency for the positive PANAS subscale was excellent across each comment-set in this study (baseline Cronbach's $\alpha = .91$; neutral Cronbach's $\alpha = .94$; criticism Cronbach's $\alpha = .93$; praise Cronbach's $\alpha = .94$). The internal consistency for the negative PANAS subscale was

good across each comment-set in this study (baseline Cronbach's α = .88; neutral Cronbach's α = .92; criticism Cronbach's α = .91; praise Cronbach's α = .90).

State-Trait Anxiety Inventory (STAI). The STAI (Speilberger et al., 1983) is a measure of 20 state and 20 trait anxiety items, each of which is measured on a 4-point scale, ranging from "not at all" to "very much so." Only the state anxiety items were used in the current study to measure participants' state level of anxiety in response to critical versus neutral comments. In the instructions, participants were asked to indicate the extent to which they feel this way "right now, that is, at the present moment." Sample items are "I feel calm," "I feel tense," and "I feel upset." The STAI state version has strong test-retest reliability (ICC's = .88; Barnes, Harp, & Jung, 2002). In this study, the STAI showed good internal consistency across each comment-set (baseline Cronbach's α = .93; neutral Cronbach's α = .94; criticism Cronbach's α = .93; praise Cronbach's α = .94).

Perceived Criticism (PC). PC (Hooley & Teasdale, 1989) is a measure designed to assess perceptions of the degree to which participants perceive their relative as critical. A modified version of the PC was used in this study, which includes the original single question, "How critical is your relative of you?" (severity of criticism) as well as an additional question, "When your relative criticizes you, how upset do you get?" (participant's distress from criticism). Ratings were made on a scale of 1 (not critical) to 10 (very critical) for each question, and the responses to both questions were multiplied together to get a single value for PC. Previous research suggests that the PC scale has adequate test-retest reliability (r = .75; Hooley & Teasdale, 1989). While this measure

only has two items, the internal consistency in this study was adequate (Cronbach's α = .66).

UCLA Loneliness Scale (UCLA-R). The UCLA-R (Russell, 1996) was used to assess participants' feeling of loneliness. It is measure of social isolation and loneliness that asks the respondent how often he/she feels (1) a lack of companionship, (2) left out, and (3) isolated from others. The measure contains 20 items and each item is measured on a 5-point scale from "never" to "often." Sample items for this measure are "I lack companionship" and "I am unhappy being so withdrawn." The UCLA_R had excellent internal consistency in this study (Cronbach's α = .96).

Relevance and Valence of Standardized Comments. At the end of the study, participants were asked to rate (1) the relevance of each comment for them and (2) the valence of each comment. Each of the twelve comments (four per comment-set) was presented in text to the participants, and participants rated on a scale from 1 – 9 the relevance the valence of each comment. Anchors were provided for the valence scale such that 1 was "very positive," 3 was "somewhat positive," 5 was "neither positive nor negative," 7 was "somewhat negative," and 9 was "very negative." The anchors for the relevance scale were: 1 was "did not at all feel this was about me," 5 was "somewhat felt this was about me," and 9 "totally felt this was about me." For both relevance and valence, the four responses per comment-set were summed for a total score. The valence scores had acceptable to good internal consistency for the neutral, critical and praise comments (Cronbach's α = .78, Cronbach's α = .86, Cronbach's α = .69, respectively). The relevance scores also had acceptable to good internal consistency for the neutral,

critical, and praise comments (Cronbach's α = .73, Cronbach's α = .87, Cronbach's α = .80).

BioPac MP150 System. The BioPac system was used for three physiological measurements: heart rate, heart rate variability (HRV), and skin conductance response (SCR). Heart rate is considered a reliable, non-invasive measure of autonomic activity, which is especially related to emotional arousal (Scheer et al., 2003; Oldehinkel et al., 2008). HRV measures sympathetic-parasympathetic arousal and the flexibility of the autonomic nervous system. Greater variability is considered more adaptive/resilient, while low variability suggests that the sympathetic and parasympathetic nervous systems are not coordinating properly (Appelhans & Luecken, 2006). SCR is another widely used and reliable measure of sympathetic response and emotional arousal (Elfering & Simone, 2011).

To measure heart rate and heart rate variability, electrocardiographic and respiratory data were gathered. Electrocardiagraphic data was gathered using three Disposable Multipurpose EKG/ECG electrodes (Model 93-0100-00; Mindware Technologies, Ghana, OH). The electrodes were attached to participants, placing one on their right collarbone, one on the lower left rib, and a ground on the lower right rib. Respiration was measured using the girth method, in which a strain gauge (i.e., Respiration Belt with Pulse Lock [BioNex pl500]; Model 50-4504-00; Mindware Technologies, Ghana, OH) was attached around the torso and positioned at the base of the sternum. As participants inhale and exhale, the degree of strain placed on the belt clasp is measured. To measure SCR, Disposable GSC Electrodes (Model 93-0102-00; Mindware Technologies, Ghana, OH) were used. Two GSC electrodes were attached to

the palm of participants' non-dominant hand (thus allowing participants to use their dominant hand to fill out questionnaires). One GSC electrode was placed on the thenar eminence and the other electrode was placed on the hypothenar eminence of the participants' palms.

Physiological measurements of heart rate, heart rate variability and skin conductance response were measured during the 2 minute playing of each set of comments and the 1 minute immediately following each set of recordings. This allowed data to be collected for the participant's immediate physiological reactivity (minute 1), sustained reactivity (minute 2), and the participants' recovery (minute 3) following the affective comments. Participants were asked to sit as still as possible and wait silently for the entire three minute recording period.

The data were cleaned and analyzed using Mindware's Heart Rate Variability Analysis Software (Version 3.0.25) and Electrodermal Activity Analysis Software, (Version 3.0; MindWare Technologies Ltd., 2010). Data were collected continuously across a three minute period (the two minutes of each comment set and the one minute following each comment set). Prior to conducting the analyses, data files were visually examined to determine whether valid data were gathered for electrocardiography, respiration, and skin conductance. Data files were then cleaned and analyzed in 1-minute segments. For each psychophysiological index, an initial reactivity, sustained reactivity and recovery variable was outputted. The initial reactivity was represented by the first minute of measurement. Sustained reactivity was represented by the second minute of measurement. Finally, the recovery period was represented as the third minute of measurement.

Heart rate was calculated as the average number of heartbeats (measured as the R-R intervals of the heartbeat) over each of the 1-minute measurement periods. HRV is calculated as the overall variance between heartbeats (in ms^2) that falls within the respiratory frequency range (0.12 – 0.40 hz) to isolate parasympathetic influence over the autonomic system (vagal tone). Then, a natural log transformation is applied to ensure a normal distribution. Through this natural log transformation, the range of output values is generally between about 5 $RSA_{ln\ ms}^2$ and 9 $RSA_{ln\ ms}^2$. HRV was outputted for each of the 1-minute measurement periods. The electrical conductance across the skin for SCR response is measured in units of microSiemens (μS), which was averaged over each of the 1-minute measurement periods.

Salivette for Cortisol Testing.

Salivary cortisol levels were collected using Salivettes (roll-shaped cotton saliva collectors). Salivary cortisol measurements were taken at three time points for both the neutral and critical comments – Time 1 was immediately prior to the neutral and critical comments, Time 2 was 15 minutes after these comments were made, and Time 3 was 25 minutes after these comments were made. These measurements allowed for the measurement of peak cortisol following the stressor as well as recovery following the stressor (Miller & O'Callahaghan, 2002). Participants chewed on the collector for approximately one minute and then placed the collector in a plastic tube. All samples were stored in a -20°C freezer until ready for assay. Samples were then assayed by the Dresden Lab Service in Dresden, Germany. Because diet and exercise can affect cortisol (Hill et al., 2008), participants were asked to follow dietary and behavioral instructions in order to take part in the study.

Participants were asked to refrain from alcohol and dairy products the evening and morning before sampling, and also not to eat at all, drink caffeine, or exercise 2 hours prior to the study. All subjects were run in the morning between the hours of 9am and 10am to help control for diurnal variations of cortisol (Nicolson, 2008). It is believed that morning values are more reliable and consistent, as the situational factors (e.g. diet, stressful daily events, and exercise) that affect cortisol measurements have less cumulative effects for participants in the morning (Kirschbaum et al., 1990). Further, medications, including psychiatric medications (e.g., anti-depressant, beta-blockers), medications for hypertension and blood pressure, steroid-based medications (hydrocortisone), sleeping pills, asthma medication, pain killers, oral contraceptives, and compounded progesterone have all been found to affect diurnal cortisol levels (Granger et al., 2009). Thus, all participants were screened before the start of the study to ensure they were not taking any of the aforementioned classes of medication. Finally, both wake time and sleep quality (via the Pittsburgh Sleep Quality Index; Buysse et al., 1989) were measured as potential covariates for cortisol analyses.

Statistical Analyses

Preliminary analyses. First, all variables were examined for normality by assessing skewness and kurtosis values. Skewness values between -1 and 1 and kurtosis values between -2 and 2 were considered acceptable (George & Mallery 2003). Next, to identify potential covariates, differences between the two groups of participants (high-risk and low-risk controls) among demographic variables (gender, age, and ethnicity) were examined. An independent samples T-test was used to determine if there were group differences in age. A Chi-Square test was used to determine if the groups were different in proportion of gender and ethnicity. Pearson correlations were used to test for

significance between each of these continuous variables. Similarly, the relationships among baseline depression and anxiety, and all dependent variables were examined to determine if statistical controls are necessary.

Primary analyses. A series of two-way repeated measures ANOVASs were conducted in SPSS 22. Repeated measures ANOVA demonstrate multiple relationships. First, any differences in responses between groups (high-risk versus low-risk control) from the standardized comments is shown for each of the dependent variables (i.e., state negative and positive affect, state anxiety, heart rate, heart variability, skin conductance, and cortisol). Additionally, within subject differences between standardized comment-type is shown for each of the dependent variables. Further, the interaction between group and comment-type is analyzed. Finally, time within comments (i.e., differences in initial response, sustained response and recovery) for heart rate, heart variability, skin conductance, and cortisol was also included as a within subjects variable. In sum, the repeated measures ANOVA was conducted for each outcome, using risk group assignment (high-risk or low-risk control) as a between-subjects independent variable and comment type (baseline, neutral, critical, praise) as a repeated within-subject measure. For the physiological indices, measurement time (of which there were 3 – initial reactivity, sustained reactivity, and recovery) was used as another repeated within-subject measure.

Cortisol was also analyzed using area under the curve (AUC) analyses. Since multiple measurements of cortisol were taken, AUC analyses are often used to incorporate multiple time-points into one set of analyses (Pruessner et al., 2003). Mathematically, AUC is calculated using algebraic equations that convert multiple

measurements for each participant into one value (the area under the curve for that participant; Pruessner et al., 2003). Then, an ANCOVA is used to compare groups. Pruessner and colleagues describe two types of AUC analyses that are most frequently used – AUC with respect to ground (AUC_G) and AUC with respect to increase (AUC_I), both of which will be used in this study's analyses. AUC_G was used to measure the total area under the curve. It takes into account the differences between each measurement (sensitivity) as well as the distance of these measures from the ground/from zero (intensity; Fekedulegn et al., 2007). Thus, AUC_G analysis was used to determine the difference in total cortisol secretion between groups and between comment conditions within groups. AUC_I was used to measure the increase in cortisol release compared to baseline. It measures change over time while ignoring the area under the curve between the baseline measurement and the ground (Fekedulegn et al., 2007). Thus, AUC_I was used to assess the difference in sensitivity to each type of comment between and within groups.

Finally, participants' perceptions of their relative's criticalness and their feelings of loneliness as moderators of the effects between criticism and physiological arousal, affect, and anxiety was evaluated. A moderation analysis using a repeated measures ANOVA was conducted by examining the interaction between risk group and the perception of their relative's criticalness in predicting physiological, affect, and anxiety responses. A second moderation analysis using a repeated measures ANOVA was conducted by examining the interaction between risk group and feelings of loneliness in predicting physiological, affective, and anxiety responses.

Exploratory Analyses

As a check on whether the comments were (1) relevant to participants and (2) had the expected valence, independent samples T-tests were performed to determine whether the two risk groups differed in their perceptions of the relevance and valence of each comment-set.

CHAPTER 3: RESULTS

Preliminary Results

Between July 2015 and July 2016, 454 individuals were screened for participation in this study. Of the 454 screened, 121 met criteria for the study and 87 individuals participated. Due to elevated self-reported prodromal symptoms that were gathered on the online questionnaire between the phone screen and the in-person portion of the study, nine individuals (originally screened for the low-risk control group) were excluded from analyses. Further, two individuals were distracted (one fell asleep and another was found using his telephone) during the study, so they were excluded from analyses as well. In total, 76 participants (38 high-risk and 38 low-risk controls) completed the study and had data that was viable for analyses. See Figure 1 for more detail about participants screened for this study.

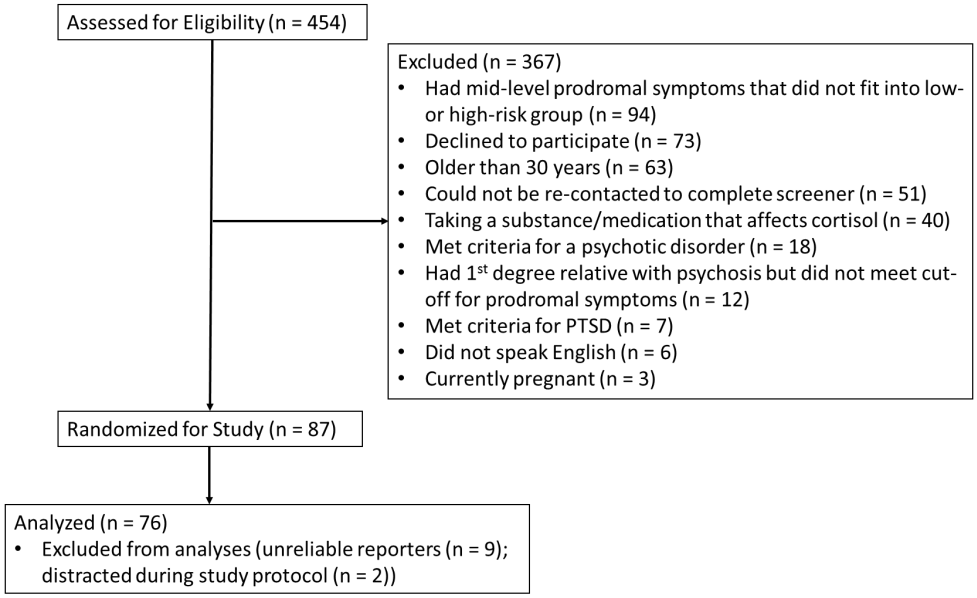

Figure 1. CONSORT diagram of all individuals in contact with study team

First, all variables were examined to determine if their skewness and kurtosis values fell within normal limits according to criteria outlined by George & Mallery (2003). The negative affect subscale of the PANAS had significant skewness and kurtosis across each measurement (baseline skewness = 3.56, kurtosis = 16.96; neutral skewness = 4.054, kurtosis = 21.66; criticism skewness = 2.19, kurtosis = 5.93; praise skewness = 3.80, kurtosis = 16.29). A log transformation was performed to correct for high skewness and kurtosis, and the variable's values fell within acceptable ranges. All other variables fell within normal limits, so no other transformations were necessary to perform.

Then, the high-risk group and the low-risk control group were compared on primary demographic variables. The two risk groups showed no difference in gender ($\chi^2(1) = 0.47$, $p = 0.49$), age ($F(1,74) = 1.18$, $p = .28$), ethnicity ($\chi^2(4) = 7.44$, $p = 0.11$), or socioeconomic status (measure via family income; $F(1,74) = 1.03$, $p = .31$). Also, the demographic variables did not relate to any of the affective outcome or physiological reactivity outcome variables. Thus, no demographic variables were used as control variables in any analyses. Demographics are presented in Table 2.

The relationships between groups on baseline depression and anxiety as well as the relationships among baseline depression and anxiety and all dependent variables were examined. The high-risk group had greater pre-study depression ($F(1,74) = 80.34$, $p < .001$) and greater anxiety ($F(1, 74) = 96.15$, $p < .001$) compared to the low-risk control group. This replicates much previous research, suggesting that individuals at high risk for psychosis have increased rates of anxiety and depression compared to the general population (e.g., Fusar-Poli et al., 2014). Also, as expected, pre-study depression and anxiety levels were positively related to in-study levels of baseline negative affect ($r =$

.55, $p < .001$; $r = .48$, $p < .001$, respectively) and anxiety ($r = .64$, $p < .001$; $r = .43$, $p < .001$, respectively). Because depression and anxiety are such common components of the high-risk state (and even represent a component of the Structured Interview for the Psychosis-Risk Syndrome; Miller et al., 2003) and because affect and anxiety are core outcomes in the study, pre-study differences in these variables were not controlled. Further, by including baseline measurements in the repeated measures ANOVAs for affect and anxiety, group differences in baseline affect and anxiety are controlled for in these analyses. Additionally, in terms of physiological measurements, there were no significant associations found between pre-study depression and anxiety and any of the in-study psychophysiological measurements. Baseline psychiatric symptoms for the sample as well the breakdown of high-risk group are presented in Table 3.

Table 2. Sample demographics

	High-Risk (HR)	**Low-Risk Controls**
N	38	38
Age	23.6 (SD = 3.4)	24.2 (SD = 3.4)
Gender	21 female 17 male	18 female 20 male
Background		
Caucasian	4 (10.5%)	10 (26.3 %)
AA/Black	10 (26.3%)	3 (7.9%)
Asian	0 (0%)	1 (2.6%)
Hispanic	22 (57.9%)	19 (50.0%)
Other	2 (5.3%)	5 (13.2%)

Table 3. Psychiatric symptom severity and high-risk breakdown of sample

	High-Risk (HR)	**Low-Risk Controls**
PQ-B symptoms	12.0 (*SD* = 3.2)	0.8 (*SD* = 1.0)
PQ-B distress	37.61 (*SD* = 14.1)	1.1 (*SD* = 1.5)
DASS_Depression	10.13 (*SD* = 5.94)	1.11 (*SD* = 1.80)
DASS_Anxiety	8.34 (*SD* = 4.43)	0.92 (*SD* = 1.46)
High-Risk Categories:	*n* (%)	
Genetic Relative + Moderate PQ-B	8 (21.1%)	n/a
Genetic Relative + Elevated PQ-B	7 (18.4%)	n/a
Elevated PQ-B Symptoms Only	23 (60.5%)	n/a

Primary Analyses

PANAS Negative Affect

The effects of comments on negative affect were examined first (depicted in Figure 2). Results of the repeated measures ANOVA indicated that the comments had an overall within-subjects effect on negative affect ($F(3,72) = 13.75$, $p < .001$, $\eta^2 = .36$). Further, there was a significant interaction between the effect of comments and risk group on negative affect ($F(3,72) = 4.20$, $p = 0.01$, $\eta^2 = .15$), suggesting that negative affect was impacted differently between risk groups for at least one comment-type. There was also a significant between-group difference in negative affect such that the high-risk group had greater negative affect than did the control group ($F(1, 74) = 29.61$, $p < .001$, $\eta^2 = .29$).

Follow-up analyses were then conducted to determine the main effect of comments on negative affect. As expected, the neutral comments did not cause any

significant change in negative affect relative to the baseline measurement (M_{logdif} = 0.04, SE = .02, p = .10). However, as expected, negative affect was significantly greater following critical comments compared to the baseline measurement and neutral comments (M_{logdif} = .10, SE = .03, p < .001; M_{logdif} = .13, SE = .03, p < .001, respectively). Finally, in line with hypotheses, negative affect was significantly lower following the praise comments relative to the baseline, neutral, and criticism measurements (M_{logdif} = -.09, SE = .02, p < .001; M_{logdif} = -0.06, SE = .02, p = .01; M_{logdif} = -.19, SE = .03, p < .001, respectively).

Finally, follow-up analyses were conducted to examine the interaction effect of comments and risk group on negative affect. In line with the hypothesis, the two-groups did not differ in their magnitude of response to neutral comments; however, contrary to expectations, the two groups also did not differ in their magnitude of response to critical comments relative to their baseline measurements (F(1,74) = 1.27, p = .26; F(1,74) = 1.61, p = .21, respectively). In line with expectations, the high-risk group did show significantly greater declines in negative affect relative to their baseline measurements following the praise comments compared to the low-risk control group (F(1,74) = 11.98, p = .001).

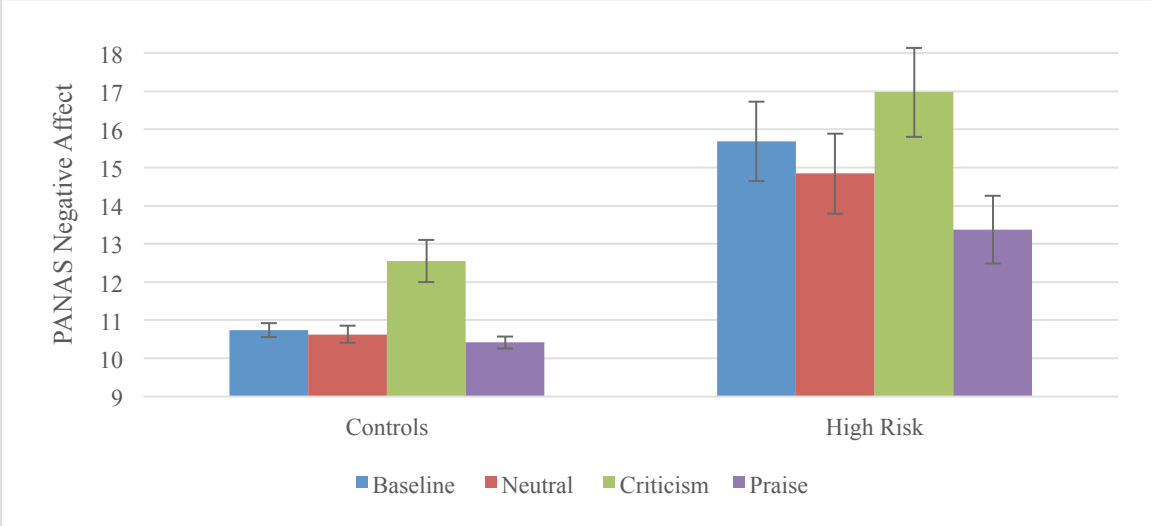

Figure 2. Negative affect responses to emotional comments. (Error bars are equal to +/- 1 standard error of the between-groups means.)

PANAS Positive Affect

The effects of comments on positive affect are depicted in Figure 3. There was a significant within-subjects effect of the comments on negative affect ($F(3,72) = 15.73$, $p = .001$, $\eta^2 = .40$). There was a marginally significant interaction between comments and risk group on positive affect ($F(3,72) = 2.45$, $p = .09$, $\eta^2 = .03$), suggesting that positive affect was impacted differently between risk groups for at least one comment-type. There was no between-group difference in positive affect ($F(1,74) = 1.38$, $p = .28$, $\eta^2 = .02$).

Analyses were then conducted to determine the main effects of comments on positive affect. Contrary to expectations, positive affect was significantly lower following the neutral comments relative to the baseline measurement ($M_{dif} = -3.29$, $SE = .78$, $p < .001$). In line with the hypothesis, the positive affect was significantly lower following the critical comments compared to the baseline measurement; however, contrary to the hypothesis, critical comments did not significantly differ from positive affect compared

to the neutral comments (M_{dif} = -3.29, SE = .73, p < .001; M_{dif} = .00, SE = .57, p = 1.00, respectively). Finally, contrary to the hypothesis, the praise comments did not change positive affect relative to the baseline measurement (M_{dif} = .03, SE = .97, p = .98). However, in line with expectations, positive affect was significantly higher following the praise comments relative to the neutral and criticism comments (M_{dif} = 3.32, SE = .78, p < .001; M_{dif} = 3.32, SE = .67, p < .001, respectively).

Follow-up analyses were also conducted to examine the marginally significant interaction between comments and risk group on positive affect. Unexpectedly, the high-risk group showed marginally greater declines in positive affect relative to their baseline compared to the control group in response to the neutral comments (F(1,74) = 3.61, p = .06). Also, contrary to predictions, the two groups did not differ in changes in positive affect relative to their baseline measurements following the critical or praise comments (F(1,74) = 2.40, p = .13; F(1,74) = 1.43, p = .24, respectively).

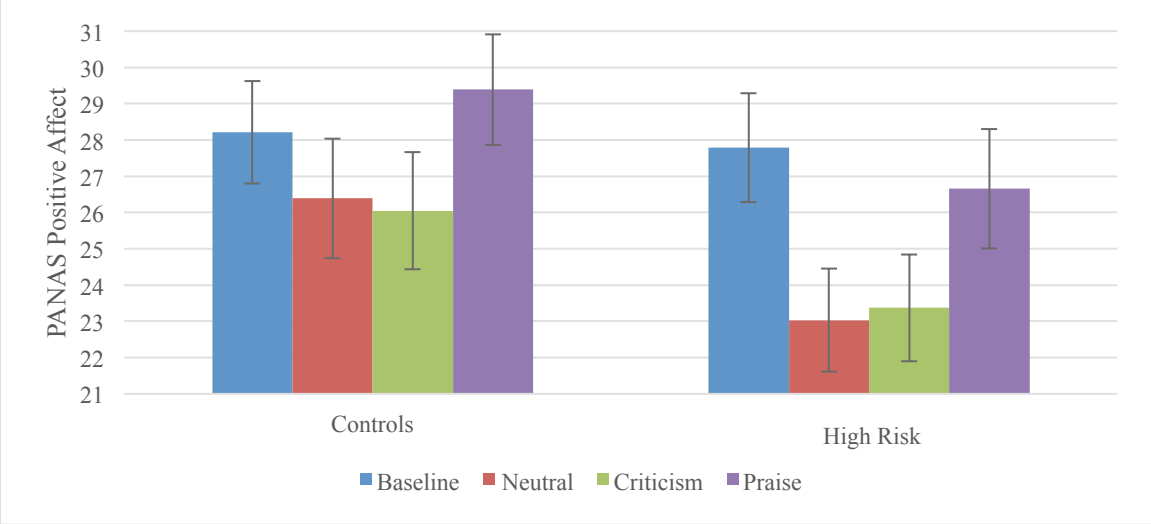

Figure 3. Positive affect responses to emotional comments. (Error bars are equal to +/- 1 standard error of the between-groups means.)

STAI Anxiety

The effects of comments on anxiety are depicted in Figure 4. There was a significant within-subjects effect of the comments on anxiety ($F(3,72) = 11.42$, $p < .001$, $\eta^2 = .32$). There was no interaction between comments and risk group on anxiety ($F(3,72) = 1.14$, $p = .34$, $\eta^2 = .05$). There was a between-group difference in anxiety such that the high-risk group had greater anxiety than the control group ($F(1,74) = 19.40$, $p < .001$, $\eta^2 = .21$).

Follow-up analyses were then conducted to determine the main effects of comments on anxiety. Contrary to the hypothesis, the neutral comments led to significantly greater anxiety relative to the baseline measurement ($M_{dif} = 2.25$, $SE = .66$, $p = .001$). In line with the hypothesis, anxiety was significantly greater following the critical comments compared to the baseline measurement and the neutral comments ($M_{dif} = 4.36$, $SE = .93$, $p < .001$; $M_{dif} = 2.11$, $SE = .95$, $p < .001$, respectively). Finally, contrary to expectations, the praise comments did not change anxiety relative to the baseline measurement ($M_{dif} = -.95$, $SE = .74$, $p = .20$), but, in line with hypotheses, did lead to significantly lower anxiety relative to the neutral and criticism comments ($M_{dif} = 3.20$, $SE = .80$, $p < .001$; $M_{dif} = 5.30$, $SE = 1.06$, $p < .001$, respectively).

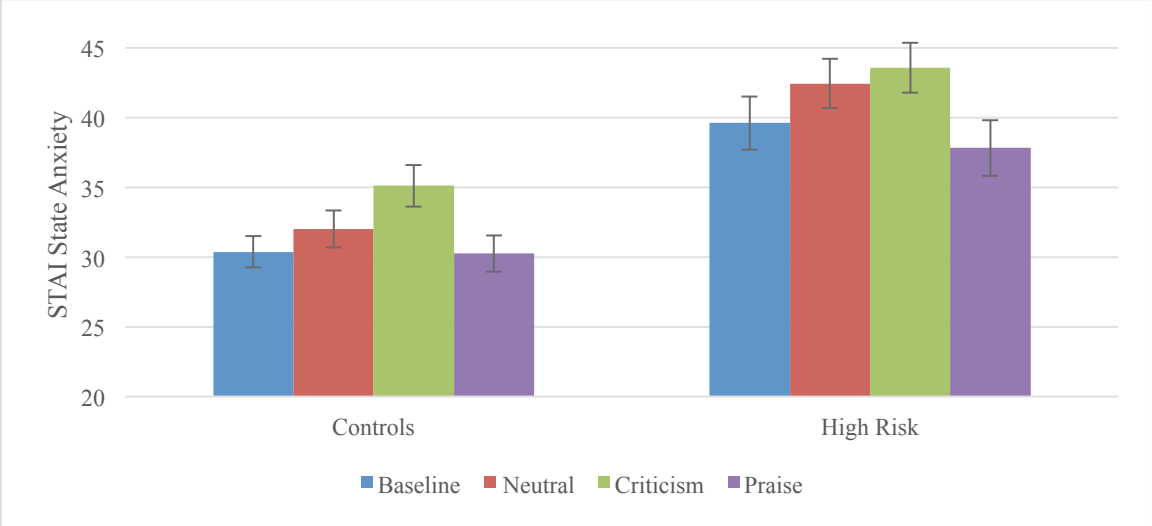

Figure 4. Anxiety responses to emotional comments (Error bars are equal to +/- 1 standard error of the between-groups means.)

Heart Rate Response

A repeated measures analysis was conducted to examine the effects of comments over time (initial reactivity, sustained response, recovery) and between risk group on heart rate (depicted in Figure 5). Results indicated a significant effect of comments on heart rate ($F(3,70) = 5.75$, $p = .001$, $\eta^2 = .20$) and of time on heart rate ($F(2,71) = 23.06$, $p < .001$, $\eta^2 = .39$). There was also a significant interaction between comments and risk group on heart rate ($F(3,70) = 2.69$, $p = .047$, $\eta^2 = .04$). There was no interaction between time and risk group on heart rate ($F(2,71) = 0.43$, $p = .66$, $\eta^2 = .01$), between comments and time on heart rate ($F(6,67) = 1.73$, $p = .13$, $\eta^2 = .13$), or between comments, time and risk group on heart rate ($F(6,67) = 1.21$, $p = .31$, $\eta^2 = .10$). There was also no between group difference in heart rate (on aggregate) throughout the study ($F(1, 72) = 2.32$, $p = .13$, $\eta^2 = .03$).

The main effect of comments on heart rate was examined further. Each comment set – neutral, criticism, and praise – had significantly lower heart rate than the baseline measurement (M_{dif} = -.91, SE = .41, p = .03; M_{dif} = -1.11, SE = .42, p = .009; M_{dif} = -2.34, SE = .57, p < .001, respectively). Further, as expected, praise comments led to significantly lower heart rate relative to neutral and criticism comments (M_{dif} = -1.43, SE = .42, p = .001; M_{dif} = -1.22, SE = .46, p = .01, respectively). Contrary to the hypothesis, there was no difference in heart rate between the neutral and the criticism comments (M_{dif} = -.21, SE = .40, p = .60).

The main effect of time (initial reactivity, sustained response, recovery) on comments was examined next. Contrary to expectations, the initial reactivity period showed significantly lower heart rate compared to the sustained response and recovery periods (M_{dif} = -1.29, SE = .20, p < .001; M_{dif} = -1.48, SE = .24, p < .001, respectively). There was no difference in heart rate between the sustained response and recovery periods (M_{dif} = .19, SE = .17, p = .26).

The simple effects of the interaction between comments and risk group on heart rate was also examined. First, the between risk group differences in heart rate at baseline were examined. In line with the expected direction, the high-risk group did have a marginally higher heart rate relative to the control group during the overall baseline measurement (F(1,72) = 3.36, p = .07, η^2 = .05). This marginally significant difference was maintained at each of the time intervals (initial reactivity, sustained response, recovery) of the baselines measurement (F(1,73) = 3.50, p = .07; F(1,72) = 3.67, p = .06; F(1,72) = 3.10, p = .08, respectively). There was no interaction of risk group and time on baseline heart rate (F(2,71) = .29, p = .75, η^2 = .01).

The differences in heart rate between risk groups during the neutral condition was examined next. In line with expectations, there was no significant difference between risk groups in change in heart rate between neutral comments and baseline measurement ($F(1,72) = 0.60$, $p = .44$). There was also no overall difference between risk groups in heart rate throughout the neutral condition ($F(1,73) = 2.24$, $p = .13$, $\eta^2 = .03$), nor was there an interaction of risk group and time on heart rate in response to neutral comments ($F(2,72) = .27$, $p = .76$, $\eta^2 = .01$). There was also no significant difference between groups in change from baseline heart rate to heart rate during the neutral comments ($F(1,72) = 0.60$, $p = .44$).

For the critical comments, contrary to hypotheses, there was no significant difference between risk group in change in heart rate between critical comments and baseline measurement ($F(1,72) = 1.48$, $p = .23$). In line with expectations, during the critical comments, the high-risk group had marginally higher heart rate relative to low-risk controls ($F(1,73) = 2.90$, $p = .09$, $\eta^2 = .04$). There was also a significant interaction between time and risk group on heart rate during the critical comments, suggesting that the two groups had different heart rate responses over time ($F(2,72) = 3.42$, $p = .04$, $\eta^2 = .09$). The two risk groups showed no difference in initial reactivity or sustained reactivity ($F(1,73) = 1.80$, $p = .18$; $F(1,73) = 2.40$, $p = .13$, respectively). However, in line with expectations, the high-risk group continued to show an activation in heart rate response during the recovery period ($t(36) = 2.21$, $p = .03$), whereas the low-risk controls showed a maintenance of heart rate ($t(36) = -1.31$, $p = .20$). This change in heart rate was significantly different between groups ($F(1,73) = 6.61$, $p = .01$). As a result of this

increase in heart rate, the high-risk group had significantly greater heart rate during the recovery measurement point compared to low-risk controls ($F(1,73) = 4.73$, $p = .03$).

Finally, the differences between risk conditions in heart rate during the praise condition was examined. Contrary to expectations, there was no overall difference between risk groups in heart rate throughout the praise condition ($F(1,73) = 0.61$, $p = .44$, $\eta^2 = .01$). There was also no interaction of risk group and time on heart rate in response to praise comments ($F(2,72) = .49$, $p = .62$, $\eta^2 = .01$). However, in line with expectations, there was a significant difference between risk groups in change from baseline, as the high-risk group showed greater reductions in heart rate from baseline during the praise comments compared to the low-risk control group ($F(1,72) = 4.26$, $p = .04$, $\eta^2 = .06$).

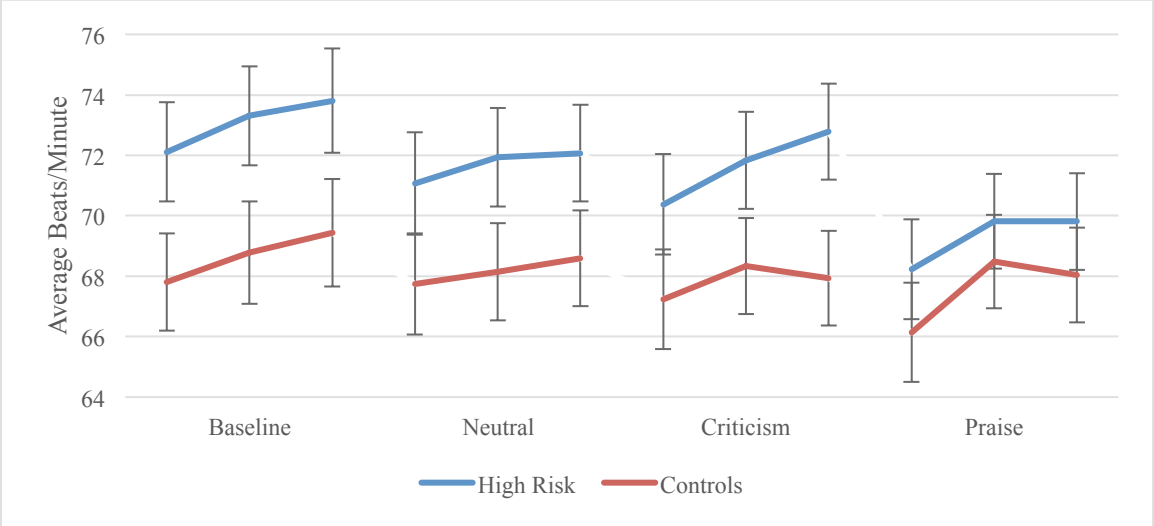

Figure 5. Heart rate in response to emotional comments. Each measurement-set displays (from left to right) the average heart rate over the first, second, and third minute of measurement. (Error bars are equal to +/- 1 standard error of the between-groups means.)

Heart Rate Variability

Repeated measures analysis were conducted to examine the effects of comments over time (initial reactivity, sustained response, recovery) and between risk groups on heart rate variability (depicted in Figure 6). Results indicated a significant effect of comments on heart rate variability ($F(3,68) = 4.20$, $p = .009$, $\eta^2 = .16$) as well as a significant effect of time on heart rate variability ($F(2,69) = 15.27$, $p < .001$, $\eta^2 = .31$). There was also a significant two-way interaction between comments and time ($F(6,65) = 2.33$, $p = .04$, $\eta^2 = .18$). However, there was no interaction between comments and risk group ($F(3,68) = .18$, $p = .91$, $\eta^2 = .01$), between time and risk group ($F(2,69) = 1.23$, $p = .30$, $\eta^2 = .03$), or between comment, time, and risk group on heart rate variability ($F(6,65) = .70$, $p = .65$, $\eta^2 = .06$). There was also no between-group difference in heart rate variability across comments ($F(1,70) = .51$, $p = .48$, $\eta^2 = .01$).

The main effect of comments on heart rate variability was examined further. Unexpectedly, heart rate variability was significantly lower in response to neutral comments compared to the baseline measurement ($M_{dif} = -.20$, $SE = .08$, $p = .01$) and the praise comments ($M_{dif} = -.18$, $SE = .06$, $p = .004$). In line with the hypothesized direction, heart rate variability was also marginally lower during the critical comments compared to the baseline measurement ($M_{dif} = -.13$, $SE = .08$, $p = .09$) and compared to the praise comments ($M_{dif} = -.11$, $SE = .06$, $p = .09$). Contrary to expectations, there were no differences between the baseline measurements and praise responses ($M_{dif} = .02$, $SE = .09$, $p = .82$) nor were there differences between the responses to the neutral comments and the criticism comments ($M_{dif} = .07$, $SE = .06$, $p = .21$).

The main effect of time was also examined further. The sustained response and recovery showed significant reductions in heart rate variability compared to the initial reactivity (M_{dif} = -.22, SE = .04, p < .001; M_{dif} = -.16, SE = .04, p < .001). There was also a marginal increase in heart rate variability between the sustained response and recovery period (M_{dif} = .06, SE = .03, p = .07).

The interaction between time and comments was examined next. First, the effect of time within each measurement set was examined. For the baseline comments, contrary to expectations, results indicated that heart rate variability was reduced at both time points following the initial reactivity. Both the sustained reactivity and recovery were reduced compared to initial reactivity (M_{dif} = -.22, SE = .07, p = .001; M_{dif} = -.29, SE = .08, p < .001). There was no difference between baseline heart rate variability during sustained reactivity and recovery (M_{dif} = .08, SE = .07, p = .26). The neutral comments were examined next, and the neutral comments showed a different trend over time. Unexpectedly, results indicated that heart rate variability was marginally reduced from initial reactivity to sustained reactivity (M_{dif} = -.12, SE = .07, p = .08); however, in line with expectations, there was no difference between initial reactivity and recovery (M_{dif} = .01, SE = .08, p = .89). Further, the recovery heart rate variability was significantly increased compared to the sustained reactivity for neutral comments (M_{dif} = .13, SE = .06, p = .03). A similar pattern as seen for the baseline measurement was seen when examining the heart rate variability for critical comments. As expected, the sustained reactivity was reduced compared to initial reactivity (M_{dif} = -.21, SE = .08, p = .01) and the recovery was marginally reduced compared to initial reactivity (M_{dif} = -.15, SE = .08, p = .057). The sustained reactivity was not different than the recovery for critical

comments (M_{dif} = .07, SE = .06, p = .27). The heart rate variability responses for praise comments were also similar in pattern to the baseline and critical measurements. Both the sustained reactivity and recovery measurements were reduced compared to initial reactivity (M_{dif} = -.29, SE = .08, p = .001; M_{dif} = -.20, SE = .08, p = .01). There was no difference between the sustained reactivity and recovery responses for the praise comments (M_{dif} = .09, SE = .07, p = .22).

The simple effects between measurement sets was also examined by comparing initial reactivity, sustained reactivity, and recovery between measurements. First, comparisons between initial reactivity were examined. Participants had lower heart rate variability during the initial reactivity of neutral comments compared to their baseline measurement and the initial reactivity of praise comments (t(72) = -3.11, p = .003; t(72) = -4.07, p < .001, respectively). Participants also had marginally lower heart rate variability during the initial reactivity of neutral comments compared to their initial reactivity of criticism comments (t(72) = -1.86, p = .07). Additionally, participants had lower heart rate variability during the initial reactivity of criticism comments compared to their initial reactivity of praise comments (t(74) = -2.32, p = .02). There were no differences between initial reactivity during criticism comments and baseline measurement or between praise comments and baseline measurement (t(74) = 1.55, p = .13; t(74) = .22, p = .83, respectively).

During the sustained reactivity, the participants had significantly lower heart rate variability during the neutral condition compared to their baseline measurement (t(71) = -2.68, p = .01). Participants also had marginally lower heart rate variability during the criticism comments compared to their baseline measurement (t(73) = -1.70, p = .09).

There was no difference in heart rate variability during the sustained reactivity between the baseline measurement and praise comments ($t(73) = .76$, $p = .45$). There was also no difference in sustained reactivity between the criticism comments and the neutral comments or the praise comments ($t(72) = .88$, $p = .38$; $t(72) = .98$, $p = .33$, respectively). There was no difference in sustained reactivity between the neutral comments and the praise comments ($t(72) = -1.65$, $p = .10$).

During the recovery period, the participants showed no difference in heart rate variability between measurements. There was no difference between baseline measurement and neutral comments, criticism comments, or praise comments ($t(71) = .27$, $p = .79$; $t(73) = .31$, $p = .76$; $t(73) = -.87$, $p = .39$, respectively). There was no difference between neutral comments and criticism comments or praise comments ($t(72) = -.25$, $p = .81$; $t(72) = -1.10$, $p = .27$). There was no difference between criticism comments and praise comments ($t(74) = -1.26$, $p = .21$).

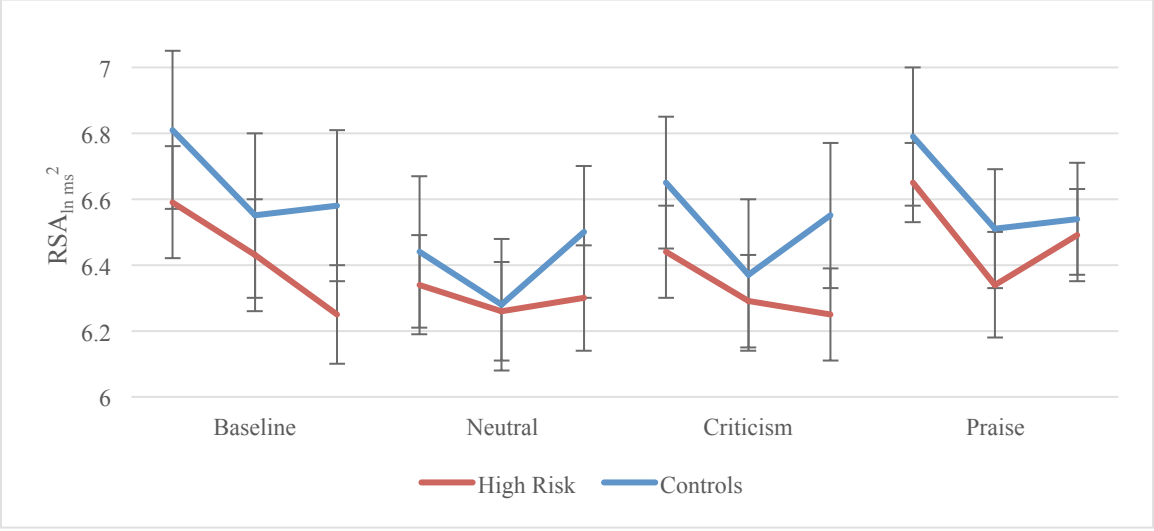

Figure 6. Heart rate variability in response to emotional comments. Each measurement-set displays (from left to right) the average heart rate variability over the first, second,

and third minute of measurement. (Error bars are equal to +/- 1 standard error of the between-groups means.)

Skin Conductance Response

A repeated measures analysis was conducted to examine the effects of comments over time (initial reactivity, sustained response, recovery) and between risk groups on skin conductance response (SCR; depicted in Figure 7). Results indicated a significant effect of comments on SCR ($F(3,71) = 23.73$, $p < .000$, $\eta^2 = .50$) as well as a significant effect of time on SCR ($F(2,72) = 36.73$, $p < .001$, $\eta^2 = .51$). There was also a significant two-way interaction between comments and time ($F(6,68) = 5.27$, $p < .001$, $\eta^2 = .32$) as well as a three-way interaction between risk group, time and comments ($F(6,68) = 2.41$, $p = .036$, $\eta^2 = .18$). However, there was no interaction between comments and risk group ($F(3,71) = .30$, $p = .82$, $\eta^2 = .01$) or between time and risk group on SCR ($F(2,72) = .30$, $p = .74$, $\eta^2 = .01$). There was also no between-group difference in SCR across comments ($F(1,73) = .31$, $p = .58$, $\eta^2 = .004$).

The main effect of comments on SCR was examined further. As expected, SCR was significantly lower during baseline measurements compared to the neutral comments ($M_{dif} = -1.54$, $SE = .23$, $p < .001$), the critical comments ($M_{dif} = -1.44$, $SE = .19$, $p < .001$), and the praise comments ($M_{dif} = -1.75$, $SE = .24$, $p < .001$). Contrary to expectations, there were no differences between the responses to neutral comments and critical comments ($M_{dif} = .10$, $SE = .17$, $p = .57$) nor were there differences between the responses to the neutral comments and the praise comments ($M_{dif} = -.20$, $SE = .18$, $p =$

.27). Also contrary to expectations, there were also no differences between the responses to the critical comments and the praise comments (M_{dif} = -.30, SE = .22, p = .16).

The main effect of time on SCR was also examined further. The sustained response and recovery showed significant reductions in SCR compared to the initial reactivity (M_{dif} = -.55, SE = .06, p < .001; M_{dif} = -.79, SE = .10, p < .001, respectively). There was also a significant decrease in SCR between the sustained response and recovery period (M_{dif} = -.23, SE = .05, p < .001).

The two-way interaction between time and comments was examined next. First, the effect of time within each measurement set was examined. For the baseline comments, results indicated that SCR was reduced at each time point following the initial reactivity. Both the sustained reactivity and recovery SCRs were reduced compared to initial reactivity (M_{dif} = -.21, SE = .06, p < .001; M_{dif} = -.38, SE = .10, p < .001). Further, the recovery SCR was reduced compared to the sustained reactivity (M_{dif} = -.16, SE = .05, p = .002). The neutral comments were examined next. Similar to the baseline measurement, results indicated that SCR was reduced at each time point following the initial reactivity. Both the sustained reactivity and recovery SCRs were reduced compared to initial reactivity (M_{dif} = -.73, SE = .11, p < .001; M_{dif} = -.96, SE = .16, p < .001). Further, the recovery SCR was reduced compared to the sustained reactivity (M_{dif} = -.22, SE = .07, p = .002). A similar pattern was seen when examining the SCRs in response to critical comments. Both the sustained reactivity and recovery SCRs were reduced compared to initial reactivity (M_{dif} = -.60, SE = .09, p < .001; M_{dif} = -.88, SE = .15, p < .001). Further, the recovery SCR was reduced compared to the sustained reactivity (M_{dif} = -.29, SE = .08, p = .001). The SCRs in response to praise showed a

similar pattern as well. Both the sustained reactivity and recovery SCRs were reduced compared to initial reactivity (M_{dif} = -.67, SE = .10, p < .001; M_{dif} = -.93, SE = .16, p < .001). Further, the recovery SCR was reduced compared to the sustained reactivity (M_{dif} = -.27, SE = .09, p = .004).

While the pattern of reactivity was similar across measurements, the rate of SCR reduction between baseline measurements and each comment set were statistically different. The magnitude of reduction between the initial reactivity and the sustained reactivity was greater for each comment set (neutral, criticism, and praise) compared to the baseline measurement (t(74) = 4.24, p < .001; t(74) = 4.21, p < .001; t(74) = 4.21, p < .001, respectively). There were no differences in SCR reduction from initial reactivity to sustained reactivity between neutral comments and critical comments (t(74) = 1.37, p = .18), between neutral comments and praise comments (t(74) = .82, p = .42), or between critical comments and praise comments (t(74) =.61, p = .55). There were no differences in SCR reduction from sustained reactivity to recovery between baseline measurement and neutral, critical, or praise comments (t(74) = 97, p = .34; t(74) = 1.44, p = .16; t(74) = 1.05, p = .30, respectively). And there were no differences in SCR reduction from sustained reactivity to recovery between neutral and critical comments (t(74) = .21, p = .84), between neutral comments and praise comments (t(74) = .29, p = .78), or between critical comments and praise comments (t(74) =.19, p = .85).

The simple effects between measurement sets was also examined by comparing initial reactivity, sustained reactivity, and recovery between measurements. First, comparisons between initial reactivity were examined. Participants had lower SCR during the initial reactivity of baseline measurement compared to their neutral, critical,

and praise measurements ($t(74) = -6.83$, $p < .001$; $t(74) = -7.38$, $p < .001$, ($t(74) = -7.74$, $p < .001$, respectively). There were no differences between initial reactivity during neutral comments and critical comments ($t(74) = .84$, $p = .41$), between neutral comments and praise comments ($t(74) = .92$, $p = .36$), or between critical comments and praise comments ($t(74) = 1.50$, $p = .14$).

During the sustained reactivity, participants had lower SCR during baseline measurement compared to their neutral, critical, and praise measurements ($t(74) = -6.19$, $p < .001$; $t(74) = -6.95$, $p < .001$, ($t(74) = -6.92$, $p < .001$, respectively). There were no differences between sustained reactivity during neutral comments and critical comments ($t(74) = .21$, $p = .84$), between neutral comments and praise comments ($t(74) = -1.33$, $p = .19$), or between critical comments and praise comments ($t(74) = -1.29$, $p = .20$).

During the recovery period, participants had lower SCR during the initial reactivity of baseline measurement compared to their neutral, critical, and praise measurements ($t(74) = -5.66$, $p < .001$; $t(74) = -7.25$, $p < .001$, ($t(74) = -6.33$, $p < .001$, respectively). There were no differences between recovery during neutral comments and critical comments ($t(74) = .54$, $p = .59$), between neutral comments and praise comments ($t(74) = .96$, $p = .34$), or between critical comments and praise comments ($t(74) = 1.22$, $p = .24$).

Finally, the three-way interaction between comment, time and risk group on SCR was examined. First, differences between risk groups in SCR at baseline were examined. There was no difference in SCR between risk groups on baseline measurements ($F(1,73) = .19$, $p = .66$, $\eta^2 = .003$). The interaction of time and risk group on baseline

measurements was then examined. There was no interaction between time and risk group on baseline SCR ($F(2,72) = .12$, $p = .88$, $\eta^2 = .003$).

The neutral comments were examined next. There was no difference between risk group in changes in SCR from baseline to neutral comments ($F(1,73) = .14$, $p = .71$). There was also no difference between risk group in overall SCR for the neutral comments ($F(1,73) = .23$, $p = .64$, $\eta^2 = .003$). Finally, there was no interaction between time and risk group on SCR to neutral comments ($F(2,72) = 1.56$, $p = .22$, $\eta^2 = .07$).

For the critical comments, there was no difference between risk group in changes in SCR from baseline to critical comments ($F(1,73) = 1.71$, $p = .20$). There was also no difference between risk group in overall SCR for the critical comments ($F(1,73) = .48$, $p = .49$, $\eta^2 = .01$). However, there was a significant interaction between time and risk group on SCR to critical comments ($F(2,72) = 3.73$, $p = .049$, $\eta^2 = .05$). The high-risk group had a significantly steeper negative slope between initial reactivity and sustained reactivity compared to the low-risk control group ($F(1,73) = 4.56$, $p = .04$). There was no difference between risk group in the slope between sustained reactivity and recovery ($F(1,73) = 1.78$, $p = .19$). It should be noted that there was no differences between groups in initial reactivity to criticism ($F(1,73) = .95$, $p = .33$), in sustained reactivity ($F(1,73) = .40$, $p = .53$), or in recovery ($F(1,73) = .18$, $p = .68$).

For praise comments, there was no difference between groups in changes in SCR from baseline to praise comments ($F(1,73) = .002$, $p = .97$). There was also no difference between risk group in overall SCR for the neutral comments ($F(1,73) = .27$, $p = .61$, $\eta^2 = .004$). Finally, there was no interaction between time and risk group on SCR to neutral comments ($F(2,72) = 1.32$, $p = .27$, $\eta^2 = .04$).

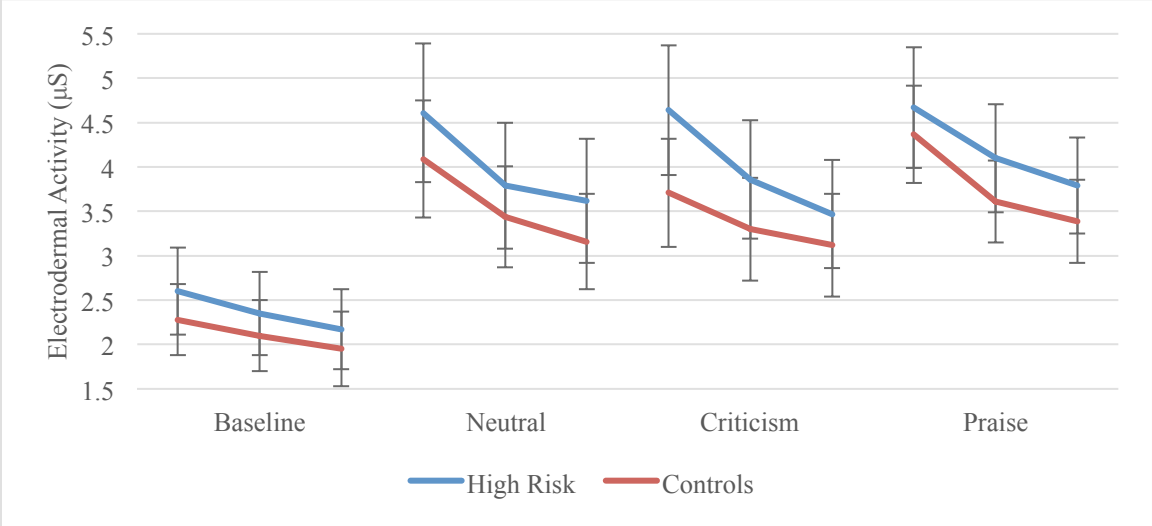

Figure 7. Skin conductance response to emotional comments. Each measurement-set displays (from left to right) the average skin conductance response over the first, second, and third minute of measurement. (Error bars are equal to +/- 1 standard error of the between-groups means.)

Cortisol Response

Sleep quality and wake-time were both examined as potential covariates for cortisol analyses. Sleep quality did not relate to any of the cortisol measurements. However, wake time did relate to baseline cortisol measurements such that individuals who woke up later had greater baseline cortisol; $r = .33$, $p = .008$). Since wake time related to one of the cortisol measurements, the primary cortisol analyses were examined twice, first without wake time as a covariate and then with wake time as a covariate.

Repeated measures analysis (without wake time as a covariate) were conducted to examine the effects of comments over time (initial reactivity, sustained response, recovery) and between risk groups on cortisol secretion (depicted in Figure 8). Within the repeated-measures design, there was no effect of comments on cortisol secretion ($F(1,72)$

= .01, p = .92, η^2 = .00). There was a significant effect of time on cortisol (F(2,71) = 27.67, p < .001, η^2 = .44). There was no interaction between comment-type and risk group, on cortisol (F(1,72) = 0.42, p = .52 η^2 = .01). There was also no interaction between comments and time on cortisol (F(2,71) = 0.23, p = .80 η^2 = .01), nor was there an interaction between comments, time, and risk group on cortisol (F(2,71) = 1.06, p = .35, η^2 = .03). Further there was no between-group difference in cortisol (F(1,72) = 1.60, p = .21, η^2 = .02).

The effect of time on cortisol secretion was then examined. Cortisol levels decreased significantly between time 1 measurement and time 2 measurement (M_{dif} = -1.17, SE = .20, p < .001) and between time 2 and time 3 measurement (M_{dif} = -0.74, SE = .12, p < .001).

The area under the curve analyses (without wake time as a covariate) were then conducted for cortisol response. Within the neutral condition, the two risk groups showed no difference in AUI_G (F(1,73) = 0.87, p = .36), nor was there a difference in AUI_I (F(1,73) = 0.60, p = .44). Within the critical comments, there was a marginal difference between the high-risk group and the low-risk control group in AUI_G (F(1,73) = 2.85, p < .10). Contrary to expectations, the high-risk group had lower cortisol secretion compared to the low-risk control group (M = 162.25, SE = 17.98; M = 206.67, SE = 19.20, respectively). This marginal difference was driven by the high-risk group having marginally significantly lower cortisol secretion at the criticism time 1 measurement (i.e., just prior to the critical comment; F(1,73) = 3.25, p = .08), as well as marginally lower cortisol secretion at the criticism time 2 measurement (i.e., 15 minutes following the

critical comment; $F(1,73) = 3.26$, $p = .08$). There was no difference between the two risk groups in the AUI_I for cortisol in response to critical comments ($F(1,73) = 1.76$, $p = .19$)

The repeated measures analyses were then re-performed with wake time as a covariate. Results indicated that the effect of time on cortisol secretion was no longer significant ($F(2,60) = 1.87$, $p = .16$, $\eta^2 = .06$). All other relationships within the repeated measures analysis remained non-significant. The area under the curve analyses were also re-performed with wake time as a covariate. The results pertaining to the effects of neutral comments on cortisol secretion remained non-significant (AUI_G: ($F(1,62) = .00$, $p = .97$); AUI_I ($F(1,62) = 0.93$, $p = .34$). Further, there no difference between the two groups in cortisol AUC measurement in response to critical comments (AUI_G: ($F(1,62) = 1.27$, $p = .26$); AUI_I ($F(1,62) = 0.94$, $p = .34$).

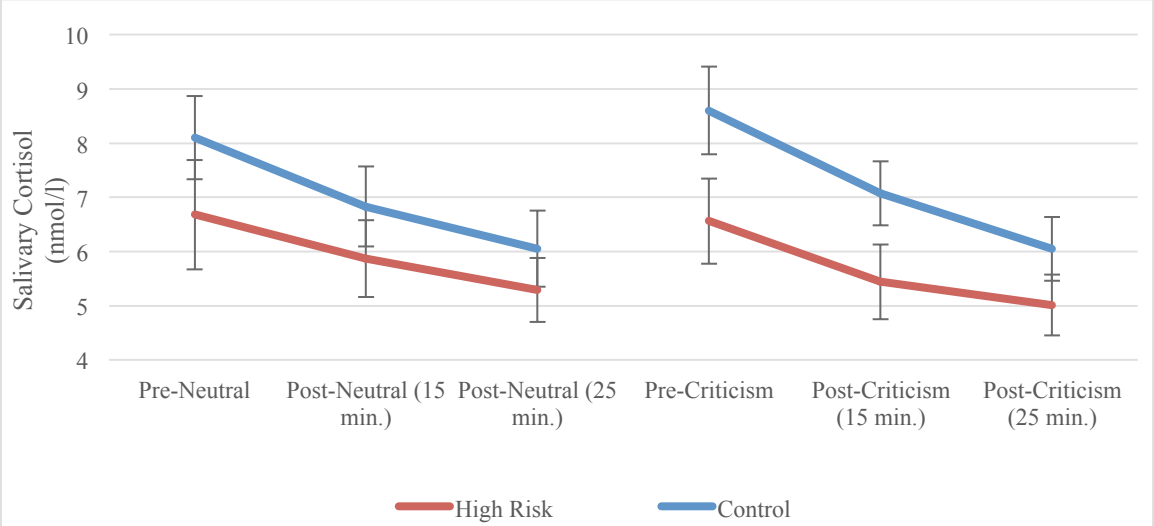

Figure 8. Cortisol response to neutral and critical comments. (Error bars are equal to +/- 1 standard error of the between-groups means.)

Perceived criticism as a moderator

Before examining the role of perceived criticism (PC) as a moderator in the relationship between risk group, comments, and outcomes, the two groups were compared on PC. The high-risk group rated PC significantly higher (M = 67.34, SD = 26.61) than did the low-risk control group (M = 32.47, SD = 26.11; $F(1,74) = 33.24$, $p < .001$). PC was then examined as a moderator in the relationship between risk group, comments and each outcome variable (i.e., negative affect, positive affect, anxiety, heart rate, heart rate variability, skin conductance, and cortisol) using the same aforementioned analyses (repeated measures ANOVA).

For negative affect, there was no significant main effect of PC on negative affect ($F(1,72) = 1.77$, $p = .19$, $\eta^2 = .02$). There was no two-way interaction between PC and comments ($F(3,70) = 1.28$, $p = .29$, $\eta^2 = .05$), nor was there a two-way interaction between PC and risk group on negative affect ($F(1,72) = 2.17$, $p = .15$, $\eta^2 = .03$). There was also no three-way interaction between PC, comments, and risk group on negative affect ($F(3,70) = .54$, $p = .66$, $\eta^2 = .02$).

For positive affect, there was no main effect of PC on positive affect ($F(1,72) = .91$, $p = .34$, $\eta^2 = .01$). There was no two-way interaction between PC and comments ($F(3,70) = .46$, $p = .71$, $\eta^2 = .02$), nor was there a two-way interaction between PC and risk group on positive affect ($F(1,72) = .02$, $p = .88$, $\eta^2 = .00$). There was also no three-way interaction between PC, comments, and risk group on positive affect ($F(3,70) = .31$, $p = .82$, $\eta^2 = .01$).

For anxiety, there was a main effect of PC on anxiety such that greater perceived criticism was associated with greater anxiety across comments ($F(1,72) = 4.11$, $p = .046$,

$\eta^2 = .05$). There was no two-way interaction between PC and comments ($F(3,70) = .68$, $p = .57$, $\eta^2 = .03$), nor was there a two-way interaction between PC and risk group on anxiety ($F(1,72) = .37$, $p = .54$, $\eta^2 = .01$). There was also no three-way interaction between PC, comments, and risk group on anxiety ($F(3,70) = .73$, $p = .54$, $\eta^2 = .03$).

For heart rate, there was no main effect of PC on heart rate ($F(1,71) = .07$, $p = .79$, $\eta^2 = .00$). There was no two-way interaction between PC and comments ($F(3,68) = .75$, $p = .53$, $\eta^2 = .03$), between PC and time ($F(2,69) = .13$, $p = .88$, $\eta^2 = .00$), or between PC and risk group on heart rate ($F(1,70) = .70$, $p = .41$, $\eta^2 = .01$). There was also no three-way interaction between PC, comments, and risk group ($F(3,68) = 2.11$, $p = .11$, $\eta^2 = .09$), between PC, time, and risk group ($F(2,69) = .05$, $p = .96$, $\eta^2 = .00$), or between PC, comments, and time on heart rate ($F(6,65) = 1.37$, $p = .24$, $\eta^2 = .11$). Finally, there was no four-way interaction between PC, comments, time, and risk group on heart rate ($F(6,65) = .72$, $p = .63$, $\eta^2 = .06$).

For heart rate variability, there was no main effect of PC on heart rate variability ($F(1,68) = .14$, $p = .71$, $\eta^2 = .00$). There was no two-way interaction between PC and comments ($F(3,66) = 2.02$, $p = .12$, $\eta^2 = .08$), between PC and time ($F(2,67) = .14$, $p = .87$, $\eta^2 = .00$), or between PC and risk group on heart rate variability ($F(1,68) = .37$, $p = .54$, $\eta^2 = .01$). There was also no three-way interaction between PC, comments, and risk group ($F(3,66) = 1.50$, $p = .22$, $\eta^2 = .06$), between PC, time, and risk group ($F(2,67) = 1.08$, $p = .35$, $\eta^2 = .03$), or between PC, comments, and time on heart rate variability ($F(6,63) = 1.44$, $p = .22$, $\eta^2 = .12$). Finally, there was no four-way interaction between PC, comments, time, and risk group on heart rate variability ($F(6,63) = 1.09$, $p = .38$, $\eta^2 = .09$).

For SCR, there was no main effect of PC on SCR ($F(1,71) = .64$, $p = .43$, $\eta^2 = .01$). There was no two-way interaction between PC and comments ($F(3,69) = .66$, $p = .58$, $\eta^2 = .03$), between PC and time ($F(2,70) = 1.14$, $p = .33$, $\eta^2 = .03$), or between PC and risk group on SCR ($F(1,71) = 1.08$, $p = .30$, $\eta^2 = .02$). There was also no three-way interaction between PC, comments, and risk group ($F(3,69) = .33$, $p = .81$, $\eta^2 = .01$), between PC, time, and risk group ($F(2,70) = .30$, $p = .74$, $\eta^2 = .01$), or between PC, comments, and time on SCR ($F(6,66) = 1.60$, $p = .16$, $\eta^2 = .13$). Finally, there was no four-way interaction between PC, comments, time, and risk group on SCR ($F(6,66) = 1.91$, $p = .09$, $\eta^2 = .15$).

For cortisol response, there was no main effect of PC on cortisol ($F(1,70) = 2.59$, $p = .11$, $\eta^2 = .04$). There was no two-way interaction between PC and comments ($F(1,70) = .70$, $p = .41$, $\eta^2 = .01$), between PC and time ($F(2,69) = .08$, $p = .92$, $\eta^2 = .00$), or between PC and risk group on cortisol ($F(1,70) = .94$, $p = .34$, $\eta^2 = .01$). There was also no three-way interaction between PC, comments, and risk group ($F(1,70) = 1.82$, $p = .18$, $\eta^2 = .03$), between PC, time, and risk group ($F(2,69) = .64$, $p = .53$, $\eta^2 = .02$), or between PC, comments, and time on cortisol ($F(2,69) = .39$, $p = .68$, $\eta^2 = .01$). Finally, there was no four-way interaction between PC, comments, time, and risk group on cortisol ($F(2,69) = 1.42$, $p = .25$, $\eta^2 = .04$).

Cortisol was also examined using area the curve analyses. The examination of the AUC_G revealed no significant main effect of PC on cortisol ($F(1,70) = 2.56$, $p = .11$, $\eta^2 = .04$). There was also no two-way interaction between PC and comments ($F(1,70) = .68$, $p = .41$, $\eta^2 = .01$) or between PC and risk group on AUC_G cortisol ($F(1,70) = .84$, $p = .36$, $\eta^2 = .01$). There was also no three-way interaction between PC, comments, and risk group

on AUC_G cortisol ($F(1,70) = 2.01$, $p = .16$, $\eta^2 = .03$). The examination of the AUC_I revealed no significant main effect of loneliness on cortisol ($F(1,70) = .05$, $p = .83$, $\eta^2 = .00$). There was also no significant two-way interaction between PC and comments ($F(1,70) = .74$, $p = .39$, $\eta^2 = .01$) or between PC and risk group on AUC_I cortisol ($F(1,70) = .77$, $p = .38$, $\eta^2 = .01$). There was no three-way interaction between PC, comments, and risk group on AUC_I cortisol ($F(1,70) = .21$, $p = .65$, $\eta^2 = .00$).

Loneliness as a moderator

Before examining the role of loneliness as a moderator in the relationship between risk group, comments, and outcomes, the two groups were compared on loneliness. The high-risk group had significantly higher loneliness scores ($M = 57.74$, $SD = 9.85$) than did the low-risk control group ($M = 33.16$, $SD = 7.93$; $F(1,74) = 143.47$, $p < .001$). Loneliness was then examined as a moderator in the relationship between risk groups, comments and each outcome variable (i.e., negative affect, positive affect, anxiety, heart rate, heart rate variability, skin conductance, and cortisol) using the same aforementioned analyses (repeated measures ANOVA).

For negative affect, there was a marginally significant main effect of loneliness on negative affect such that greater loneliness was associated with greater negative affect across comments ($F(1,72) = 3.16$, $p = .08$, $\eta^2 = .04$). There was no two-way interaction between loneliness and comments ($F(3,70) = .09$, $p = .96$, $\eta^2 = .00$), nor was there a two-way interaction between loneliness and risk groups on negative affect ($F(1,72) = .03$, $p = .87$, $\eta^2 = .00$). There was also no three-way interaction between loneliness, comments, and risk group on negative affect ($F(3,70) = .53$, $p = .67$, $\eta^2 = .02$).

For positive affect, there was a marginally significant main effect of loneliness on positive affect such that greater loneliness was associated with reduced positive affect across comments ($F(1,72) = 3.00$, $p = .09$, $\eta^2 = .04$). There was no two-way interaction between loneliness and comments ($F(3,70) = .72$, $p = .55$, $\eta^2 = .03$), nor was there a two-way interaction between loneliness and risk group on positive affect ($F(1,72) = .05$, $p = .83$, $\eta^2 = .00$). There was also no three-way interaction between loneliness, comments, and risk group on positive affect ($F(3,70) = 1.67$, $p = .18$, $\eta^2 = .07$).

For anxiety, there was a main effect of loneliness on anxiety such that greater loneliness was associated with greater anxiety across comments ($F(1,72) = 8.86$, $p = .004$, $\eta^2 = .11$). There was no two-way interaction between loneliness and comments ($F(3,70) = .79$, $p = .50$, $\eta^2 = .03$), nor was there a two-way interaction between loneliness and risk group on anxiety ($F(1,72) = .16$, $p = .69$, $\eta^2 = .00$). However, there was a three-way interaction between loneliness, comments, and risk group on anxiety ($F(3,70) = 3.34$, $p = .02$, $\eta^2 = .13$). Follow-up analyses were done to examine this three-way interaction. For the baseline measurement, neutral comments, and praise comments, there was no interaction between risk group and loneliness ($F(1,72) = .72$, $p = .40$, $\eta^2 = .01$; $F(1,72) = .11$, $p = .74$, $\eta^2 = .00$; $F(1,72) = .38$, $p = .54$, $\eta^2 = .01$, respectively). However, for the critical comments there was a significant interaction between risk group and loneliness ($F(1,72) = 3.83$, $p = .05$, $\eta^2 = .05$). The high-risk group showed a significant relationship between loneliness and anxiety following the critical comments ($F(1,36) = 7.05$, $p = .012$), whereas the low-risk control group showed no relationship between loneliness and anxiety following the critical comments ($F(1,36) = .09$, $p = .77$).

For heart rate, there was no main effect of loneliness on heart rate ($F(1,70) = 1.31$, $p = .26$, $\eta^2 = .02$). There was no two-way interaction between loneliness and comments ($F(3,68) = 1.61$, $p = .20$, $\eta^2 = .07$), between loneliness and time ($F(2,69) = 1.70$, $p = .27$, $\eta^2 = .04$), or between loneliness and risk group on heart rate ($F(1,70) = 1.51$, $p = .22$, $\eta^2 = .02$). There was also no three-way interaction between loneliness, comments, and risk group ($F(3,68) = .21$, $p = .89$, $\eta^2 = .01$), between loneliness, time, and risk group ($F(2,69) = .72$, $p = .49$, $\eta^2 = .02$), or between loneliness, comments, and time on heart rate ($F(6,65) = .44$, $p = .85$, $\eta^2 = .04$). Finally, there was no four-way interaction between loneliness, comments, time, and risk group on heart rate ($F(6,65) = .80$, $p = .57$, $\eta^2 = .07$).

For heart rate variability, there was no main effect of loneliness on heart rate variability ($F(1,68) = 1.00$, $p = .32$, $\eta^2 = .01$). There was no two-way interaction between loneliness and comments ($F(3,66) = .46$, $p = .71$, $\eta^2 = .02$), between loneliness and time ($F(2,67) = 1.11$, $p = .34$, $\eta^2 = .03$), or between loneliness and risk group on heart rate variability ($F(1,68) = .05$, $p = .82$, $\eta^2 = .00$). There was also no three-way interaction between loneliness, comments, and risk group ($F(3,66) = .25$, $p = .86$, $\eta^2 = .01$), between loneliness, time, and risk group ($F(2,67) = .66$, $p = .52$, $\eta^2 = .02$), or between loneliness, comments, and time on heart rate variability ($F(6,63) = .58$, $p = .74$, $\eta^2 = .05$). Finally, there was no four-way interaction between loneliness, comments, time, and risk group on heart rate variability ($F(6,63) = .25$, $p = .96$, $\eta^2 = .02$).

For SCR, there was no main effect of loneliness on SCR ($F(1,71) = 1.25$, $p = .27$, $\eta^2 = .02$). There was no two-way interaction between loneliness and comments ($F(3,69) = .31$, $p = .82$, $\eta^2 = .01$), between loneliness and time ($F(2,70) = .56$, $p = .58$, $\eta^2 = .02$), or between loneliness and risk group on SCR ($F(1,71) = .03$, $p = .86$, $\eta^2 = .00$). There was

also no three-way interaction between loneliness, comments, and risk group ($F(3,69) = .49$, $p = .69$, $\eta^2 = .02$), between loneliness, time, and risk group ($F(2,70) = 1.47$, $p = .24$, $\eta^2 = .04$), or between loneliness, comments, and time on SCR ($F(6,66) = 1.60$, $p = .16$, $\eta^2 = .13$). Finally, there was no four-way interaction between loneliness, comments, time, and risk group on SCR ($F(6,66) = 1.62$, $p = .16$, $\eta^2 = .13$).

For cortisol response, there was no main effect of loneliness on cortisol ($F(1,70) = .22$, $p = .64$, $\eta^2 = .00$). There was no two-way interaction between loneliness and comments ($F(1,70) = .26$, $p = .61$, $\eta^2 = .00$), between loneliness and time ($F(2,69) = .47$, $p = .63$, $\eta^2 = .01$), or between loneliness and risk group on cortisol ($F(1,70) = .95$, $p = .33$, $\eta^2 = .01$). There was also no three-way interaction between loneliness, comments, and risk group ($F(1,70) = .65$, $p = .42$, $\eta^2 = .01$), between loneliness, time, and risk group ($F(2,69) = .35$, $p = .70$, $\eta^2 = .01$), or between loneliness, comments, and time on cortisol ($F(2,69) = .07$, $p = .93$, $\eta^2 = .00$). Finally, there was no four-way interaction between loneliness, comments, time, and risk group on cortisol ($F(2,69) = .55$, $p = .58$, $\eta^2 = .02$).

Cortisol was also examined using area the curve analyses. The examination of the AUC_G revealed no significant main effect of loneliness on cortisol ($F(1,70) = .22$, $p = .64$, $\eta^2 = .00$). There was also no two-way interaction between loneliness and comments ($F(1,70) = .26$, $p = .61$, $\eta^2 = .00$) or between loneliness and risk group on AUC_G cortisol ($F(1,70) = .97$, $p = .33$, $\eta^2 = .01$). There was also no three-way interaction between loneliness, comments, and risk group on AUC_G cortisol ($F(1,70) = .66$, $p = .42$, $\eta^2 = .01$). The examination of the AUC_I also revealed no significant main effect of loneliness on cortisol ($F(1,70) = .91$, $p = .35$, $\eta^2 = .01$). There was also no two-way interaction between loneliness and comments ($F(1,70) = .10$, $p = .75$, $\eta^2 = .00$) or between loneliness and risk

group on AUC_I cortisol ($F(1,70) = .25$, $p = .62$, $\eta^2 = .00$). There was no three-way interaction between loneliness, comments, and risk group on AUC_I cortisol ($F(1,70) = .94$, $p = .34$, $\eta^2 = .01$).

Exploratory Analyses on the Relevance and Valence of Comments

The participants' self-reported relevance and valence of each comment set was tested. Results indicated that the high-risk group felt the criticism comments were more relevant to their lives than did the low-risk controls ($M = 5.9$ ($SD = 2.4$), $M = 4.3$ ($SD = 1.9$), $t(74) = 3.24$, $p = .002$). There was no difference in relevance ratings between groups on the neutral or praise comments ($M = 3.3$ ($SD = 2.0$), $M = 3.9$ ($SD = 1.4$), $t(74) = 1.32$, $p = .19$; $M = 6.5$ ($SD = 2.1$), $M = 6.8$ ($SD = 1.3$), $t(74) = .73$, $p = .47$, respectively).

In terms of valence, the high-risk group rated the neutral comments as less positive than did the low-risk control group ($M = 4.7$ ($SD = 1.2$), $M = 4.0$ ($SD = 1.1$), $t(74) = 3.24$, $p = .008$). There was no difference in valence ratings between groups on the criticism or praise comments ($M = 8.0$ ($SD = 0.9$), $M = 7.7$ ($SD = 1.5$), $t(74) = 1.28$, $p = .20$; $M = 1.6$ ($SD = 1.0$), $M = 1.5$ ($SD = 0.6$), $t(74) = .81$, $p = .42$, respectively).

CHAPTER 4: DISCUSSION

This study aimed to test whether high-risk individuals were more sensitive to emotionally-laden comments (both critical and praise comments) compared to low-risk controls. Primary findings indicate that individuals at high risk for psychosis are not more reactive to criticism compared to low-risk controls. Across all self-report indices and physiological measurements, the high-risk group was not different from the low-risk control group in reactivity to critical comments. Instead, what was seen was the high-risk group demonstrated (1) baseline elevations in negative affect, anxiety and heart rate compared to low-risk controls, and (2) the two groups showed similar reactivity to criticism. The two groups (relative to their own baseline and the neutral comments) showed equal increases in negative affect and anxiety and equal decreases in positive affect and heart rate. However, there is some evidence to suggest that the high-risk group had slower recovery to criticism compared to low-risk controls, as their heart rate showed slower recovery following critical comments.

The high-risk group also appeared to be particularly receptive to praise comments. The high-risk group's negative affect had greater reductions from their baseline than the low-risk controls' following praise, and the two group's heart rate became indistinguishable from one another throughout heart rate measurement during and following praise. Overall, this suggests that individuals at high risk for psychosis are not more sensitive to criticism, but may have trouble regulating their arousal to criticism compared to low-risk controls. These findings also suggest that high-risk individuals are particularly responsive (in a beneficial way) to praise.

Stress Reactivity to Neutral and Critical Comments

This is the first study to examine the effects of in-the-moment expressed emotion comments on the affective and physiological state of individuals at high risk for psychosis. Contrary to hypotheses, there were no differences between groups in reactivity to critical comments. The critical comments did have the expected effect of increasing negative affect and anxiety relative to baseline measurements and neutral comments. Also, in line with expectations, skin conductance was elevated and positive affect and heart rate variability were reduced for the critical comments compared to baseline (although they were not different than the neutral comments). However, these changes were not different between groups, indicating that high-risk individuals are not more sensitive to criticism compared to low-risk controls. This contradicts previous research, which has suggested that high-risk individuals are more sensitive to stressors compared to low-risk controls (e.g., Myin-Germeys et al., 2001; Trotman et al., 2014). It is important to note that previous studies relied on ecological momentary assessment (EMA) or hindsight self-reporting practices, which do not take into account the severity of the stressor(s) or hindsight bias. Further, unlike this study, previous studies measured nonspecific life stressors, so it is unclear whether high-risk individuals face unique and/or more stress-inducing life events. To control for severity and type of stress exposure, this study focused exclusively on a standardized criticism as a social stressor. Rather than supporting the sensitivity hypothesis (as hypothesized), these results support the threshold hypothesis (Meehl, 1962; Zubin & Spring, 1977).

The threshold hypothesis is based on two main principles. First, it states that each individual has a threshold at which stressors exceed that person's ability to maintain a

level of psychological "equilibrium" or "adaptation," at which point a psychological disorder/episode ensues. Second, it suggests that every individual's threshold for developing/transitioning to psychosis is based on a function of two variables – one's vulnerability for the disorder and the number of life stressors an individual faces. This threshold is thought to be lower for individuals who have a greater vulnerability and/or a greater number of life stressors. Therefore, individuals at high risk for psychosis have two factors that often lower their threshold for developing psychosis – their vulnerability for psychosis is higher and, some research suggests, their daily life stressors are greater (e.g., Tessner et al., 2011).

In relation to this study, the two groups did not differ in their response to criticism based on self-reported affect or anxiety. However, they did enter the study with greater stress (as measured by negative affect and anxiety), as well as a greater vulnerability for psychosis. The criticism then served as a further stressor, pushing all individuals to a similar degree closer to a "threshold" of psychopathology, with the high-risk individuals entering the study already more proximal to that threshold relative to low-risk controls. This suggests that, when the severity of the social stressor is held constant, high-risk individuals respond with a similar sensitivity compared to low-risk controls. However, it appears that would take less life-stress for the high-risk individuals to cross a threshold of psychopathology, as they have a lowered threshold compared to the general population. Considering the prior evidence that high-risk individuals face more stressful events in their lives, the EMA studies may be demonstrating that multiple stressors do not affect individuals in a linearly progressive way, but rather in an exponential or multiplicative way. This is substantiated by work done by Trotman and colleagues (2014), suggesting

that individuals at high risk for psychosis (1) have more daily stressors than low-risk controls and (2) that there is a sensitization effect in response to life stressors. Based on the results of this current study, however, it appears that the reactivity of one standardized social stressor is not different between high-risk individuals and low-risk controls.

Although reactivity to criticism was not different between groups, there was some evidence to suggest that the high-risk group had difficulty recovering (based on heart rate results) from the criticism relative to the control group. Heart rate showed an unexpected response to comments whereby heart rate was lowered for each comment-set compared to baseline measurements (discussed below), and this reduced heart rate was maintained across neutral and critical comments for the low-risk control group. The high-risk group followed the same trend for each of the measurement points during the neutral comments; however, their heart rate increased throughout the criticism measurement and became significantly elevated relative to the control group during the recovery period of measurement. These findings suggest that high-risk individuals have difficulties regulating their physiological response (and potentially their negative affect and anxiety) that follow from social stress. There is a paucity of literature examining emotion regulation in high-risk individuals. The research that has been done has found that high-risk individuals have difficulties recognizing emotions (an ability that is important for regulation emotions; Amminger et al., 2012; van Rijn et al., 2011). Imaging research has also found that high-risk individuals have difficulties regulating brain areas that are used for emotional processing (including the amygdala and ventrolateral prefrontal cortex; Gee et al., 2012). The current study's findings demonstrate further support for the presence of emotion regulation difficulties within individuals at high risk for psychosis.

Also contrary to expectations, neutral comments led to reductions in positive affect, and there was some marginal evidence that this relationship was stronger for the high-risk group. Additionally, based on the participants' reports of the valence of the comments, high-risk individuals felt that the neutral comments were less positive than did the low-risk controls. Together, these findings suggest that neutral social comments may not be perceived neutrally, and are actually perceived somewhat negatively, by individuals at high risk for psychosis. Research on neutral stimuli has previously found that individuals with schizophrenia experience more negative emotions and show amygdala hyperactivation when presented with neutral stimuli compared to controls (e.g., Cohen & Minor, 2010; Hall et al, 2008). There is also some prior work to suggest that individuals at risk for psychosis over-attribute negative emotions to neutral faces and have increased neural responses to them compared to controls (Seiferth et al., 2008; Eack et al., 2010).

These previous findings on responses to neutral stimuli suggest that individuals with psychosis, or at risk for psychosis, may have increased fear signaling and/or social cognitive deficits that make the interpretation of neutral faces more negative. Previous research has not directly linked reductions in positive affect to neutral stimuli. Reduction in positive affect may serve as another factor that affects at-risk individuals' ability to effectively engage with their environment. In addition to feeling fearful or negative of neutral social stimuli, at-risk individuals may also feel that neutral social stimuli are less positive and worth engaging with. In other words, while low-risk controls were able to retain some positive affect in neutral situations, high-risk individuals appear to lose positive affect when exposed to neutral social stimuli. This could have implications for

the high-risk individuals' ability or desire to remain in neutral social situations, as a neutral setting may (1) appear to pose threats as well as (2) appear to provide no positive reinforcement for remaining in that setting.

Participant's heart rate responses were also unexpected for each of the comment-sets. Heart rate was expected to increase during and following critical comments; however, each of the comments led to decreased heart rate, with neutral and critical comments showing no difference in overall heart rate reactivity. It is possible that the timing of the baseline measurement led to increased heart rate. Baseline measurements were conducted immediately following consent to the study and prior to any of the study activities. Thus, the anticipation of the study events may have increased heart rate. Anticipatory stress has been found to increase heart rate and the increase is even greater for individuals with major depressive disorder (Davidson et al., 2000; Joormann, Waugh, & Gotlib, 2015). Overall, while the comments did elicit various stress responses, it may have been the anticipation of the comments that elevated heart rate before participants began any study activities.

The idea of anticipatory stress can also help explain the null results for cortisol reactivity in this study. Contrary to hypotheses, cortisol levels were elevated prior to the neutral and critical comments in both groups, and cortisol levels steadily declined for each of the measurements following the neutral and critical comments. In other words, the stress response appeared to occur before the presentation of any stressor, and then the stress hormone was steadily reduced at the follow-up measurements. This pattern was the same for both the neutral and critical comments and for both risk groups. Considering that participants were not told what they were going to hear prior to the presentation of

the stimuli, it is possible that anticipation of the stimuli was more stressful than the presentation of the stimuli itself. A similar anticipatory stress response as evidence with heart rate has been found with cortisol. Individuals in the anticipation phase of the Trier Stress Test consistently show increased cortisol levels (e.g., Kirschbaum, Pirke, & Hellhammer, 1993). Taken together, the increased heart rate and cortisol levels prior to the comments indicate some degree of anticipatory stress that was experienced across groups in this study.

Reactivity to Praise

In line with hypotheses, praise had beneficial effects across a range of indices. For both groups, praise comments reduced negative affect and heart rate relative to baseline measurements and relative to neutral comments. Praise also increased positive affect and heart rate variability and reduced anxiety relative to responses from the neutral comments. The hypotheses for praise comments were further substantiated in the examination of group differences, which found that the high-risk group had greater reductions in negative affect and heart rate compared to the low-risk control group. This indicated that the high-risk group was more responsive to praise and showed a greater physiological and affective benefit from praise than did the low-risk controls. A possible explanation for this group difference could be due to the high-risk individuals' increased loneliness and reduced social networks (Robustelli et al., 2017). Because the high-risk group was lonelier than the healthier control group, it is possible that they do not have as many positive social interactions. Therefore, when they are the recipients of praising comments, it may feel especially nice and not as commonplace compared to low-risk controls. Additionally, the high-risk group had increased family conflict (i.e., increased

perceived criticism) and found the criticism to have greater relevance to their lives compared to low-risk controls. This difference between groups also suggests that positive remarks and warmth may not be as common for the high-risk individuals. Thus, exposure to positive remarks, such as praise, may be particularly welcomed by high-risk individuals.

An alternative perspective to explain the results for the praise comments is that the high-risk group is not more sensitive or responsive to praise. Rather, it is possible that the low-risk control group represented a relative floor for negative affect and heart rate, and the high-risk group's response to praise brought them to levels at (or closely matching) the low-risk control group. For example, the low-risk control group had minimal negative affect at the outset of the study. Thus, the low-risk control group did not have much (if any) room to have reductions in positive affect. On the other hand, the high-risk group had elevated negative affect at the outset of the study, which allowed for a much greater decline in negative affect. With either perspective, it is encouraging to see that the high-risk group is receptive to praise. The implication is that warm social displays are (1) well-received by high-risk individuals in a beneficial way and (2) that there are both affective and physiological benefits that are produced from a positive social display.

This is the first study to examine how these benefits of warmth and praise are received in the moment (both physiologically and affectively) by individuals at high risk for psychosis. The literature on positive social interactions, such as warmth and praise, is limited in the prodromal psychosis literature; however, the research that has been conducted does support the benefits of praise that were found in this study. Caregiver

warmth has been found to be predictive of reductions in negative and disorganized symptoms over a 3 month period (O'Brien et al., 2006). There is also some research to suggest that caregiver warmth is beneficial for family functioning (O'Brien et al., 2008; Schlosser et al., 2010). In sum, the growing literature, including the findings from this study, consistently suggest that positive interactions, such as being praised, are well-received within this population and can be beneficial for physiological, psychiatric, and family functioning outcomes.

Loneliness and Perceived Criticism as Moderators

The high-risk individuals reported higher levels of loneliness compared to low-risk controls. There was also some evidence that loneliness related to affective outcomes, as expected. By-in-large, loneliness did not moderate the effect of comments on stress response outcomes. However, it did moderate the effect of criticism and risk group on anxiety. Loneliness was connected to an increase in anxiety following criticism for the high-risk group but not for the low-risk group. Previous work has found that loneliness is related to affective and anxiety symptoms (e.g., Joiner, 1997; Roekel et al., 2014). And the effect of loneliness on anxiety appears to be magnified for high-risk individuals when they are faced with a social stressor (criticism). This suggests that high-risk individuals are more sensitive than low-risk individuals to social stress when they are also lacking social support. French and Morrison (2004) have theorized that loneliness and a lack of social support may contribute to high-risk individuals' sensitivity to social stress because (1) they may not have family or peer support to help "reality test" unusual experiences and/or (2) they may not have support in sharing their stressful experiences. This theory is, in part, supported by this study's findings. It appears that the combined effect of social

stressors (criticism in tandem with loneliness) leads to a greater stress response in high-risk individuals compared to low-risk individuals.

PC was also rated higher in the high-risk group relative to the low-risk group. However, PC did not relate to any of the stress response outcomes except anxiety. Similarly to loneliness, greater PC was associated with greater anxiety across all participants. Further, this study did not find evidence that perceived criticism has an effect on affective or physiological responses to emotional comments that is different between high-risk and low-risk groups. The bulk of the literature on PC has examined the effects of perceived criticism on symptoms and clinical outcomes over time. These results have robustly shown that greater PC is linked to poorer outcomes for a range of clinical presentations (Hooley, 2007). However, the research that has examined concurrent symptom severity has found that PC is unrelated to concurrent anxiety and OCD symptoms (Renshaw, Chambless, & Steketee, 2003). Further, the current study suggests that, for low-risk controls and individuals at risk for a psychotic disorder, PC does not relate to concurrent affective and anxiety symptoms. Thus, PC may play an important role in the progression of symptoms and influence the long-term outcomes of individuals' psychiatric outcomes; however, it does not appear that PC plays a role in the state-level measurement of affect and anxiety. PC also does not seem to affect the sensitivity of individuals' affective or physiological responses to emotional comments.

Baseline Cortisol Measurements

Based on the research and theoretical work suggesting that individuals at high-risk for psychosis have an overactive/dysregulated HPA system (Walker, Mittal, & Tessner, 2008; Walker et al., 2013), it was expected that cortisol would be elevated at

baseline in high-risk individuals compared to low-risk controls. However, this study did not find significant differences between groups in baseline cortisol. While the preponderance of literature has shown differences in cortisol between high-risk individuals and low-risk controls, other studies that have examined baseline cortisol using single time-point, morning measurements in a laboratory setting (as was performed in this study) have failed to find differences in baseline cortisol between high-risk and low-risk controls (Labad et al., 2015; Chaumette et al., 2016). Another study that collected morning cortisol measurements (collecting three measurements) also found no differences between high-risk individuals and low-risk controls (Carol, Spencer, & Mittal, 2016). A variety of reasons for the varying results have been proposed, including medication use, sex-driven differences, time of day, and single versus repeated measurements (Pruessner, Cullen, Aas, & Walker, 2017).

A potential explanation for the null results with cortisol in this study could pertain to the individual, morning measurement. As noted previously, experts in cortisol research note that morning values are more reliable and consistent, as situational/daily stressors have had less effect on individuals (Kirschbaum et al., 1990; Nicolson, 2008). Individuals at high risk for psychosis have been found to have greater daily stressors compared to low-risk controls (Tessner, Mittal, & Walker, 2011), thus cortisol measurements that are collected throughout the day or at a later point in the day may be measuring this increase in stressors and/or dysregulation in response to daily stressors. This speaks again to the threshold hypothesis – individuals at high risk for psychosis may be starting their days with similar concentrations of biological stress hormones (e.g., cortisol levels); however, the number of stressors in their day and/or difficulties coping with the stressors may have

a larger impact on them (leading to greater levels of cortisol) compared to low-risk controls.

Limitations

This study had several limitations that should be noted. First, the sample size was somewhat limited, with 38 individuals per group. While this sample size appeared to appropriately power most of the study (particularly the relationships with a medium effect size or larger) it appears that the achieved power for some of the study (i.e., the moderation analyses) was a bit low. The effect sizes for the moderation analyses were small to small/medium, and a post-hoc achieved power analysis in G*Power 3.1.7 found that the achieved power for these analyses was between 0.47 and 0.82. This suggests that the study was not adequately powered to robustly demonstrate an effect (or a lack thereof) for much of the moderation analyses.

Another important limitation to note was the method of symptom assessments throughout the study. Due to financial and time constraints, the participants were screened with the PQ-B (Loewy et al., 2011), rather than a gold-standard measurement of the psychosis-risk syndrome (such as the Structured Interview for Prodromal Symptoms (SIPS); Miller et al., 2003). While the PQ-B has good concordance with the SIPS, it is considered inferior to the SIPS. Thus, the level of risk for psychosis within the high-risk group is somewhat reduced relative to studies that measure participants using the gold-standard ultra-high-risk measurements. Further, the assessment of baseline depression and anxiety as well as state affect and anxiety symptom severity throughout the study were all measured using self-report. While self-report has some advantages to clinician-rated measurement (such as prioritizing participants' perspective over an outside rater's

perspective), the lack of consistency in rater makes the ratings more variable and subjective.

The standardized comments represent another potential limitation, as each participant was presented with pre-written/recorded comments using a voice that was not familiar to them. This stimulus presents a couple potential issues. First, the pre-made statements were not completely relevant for each participant, which was evidenced by the ratings of comment relevance. This could mean that for some participants, the content (e.g., "Another thing that really bothers me is how lazy and apathetic you can be…") did not apply to their lives. For other participants, it could be that those comments would not be said to them by their relatives. In either case, the potency of the comments would be diminished for individuals that did not feel the comments applied to them.

Further, participants were asked to imagine an older female relative saying the comments about them. It is possible that it was difficult for participants to imagine their relative saying the comments, since the voice was not their relative's and the language/verbiage used may not match their relative's general word-choice. Additionally, when it is a stranger as opposed to a loved one that is criticizing an individual, it could be easier to discount the emotional content of the comment. The comments' limited stress-potency could explain why there were not changes in skin conductance responses (a measure of sympathetic reactivity; Bach, 2014) between comments within participants. With reduced potency comes a restricted breadth of conclusions that can come from this study. For example, there may be differences in stress reactivity between high-risk and low-risk individuals at higher levels of criticism that were not visible at this study's level of criticism.

It is important to mention that the standardized comments did have some advantages, however. First and foremost, having participants' actual relatives criticize them poses a serious ethical and clinical issue, as these comments are likely to be much more hurtful and could create, or intensify, family conflict. Additionally, using standardized comments allows for a standard unit of measurement by which to compare individuals. Much of the literature compares high-risk individuals to low-risk controls through ecologically-valid methods; however, these methods may be comparing different stimuli that are acting upon the participant (e.g., the strength and/or type of the stressors may be different between groups). Therefore, the standardized comments provided the most ethical and efficient way of scientifically testing the effects of a "unit" of social stress on individuals.

Playing the praise comments last for each participant is another limitation of this study. The ordering of comments such that praise was played last was purposely done so that the high-risk participants left the laboratory following a positive mood induction, rather than a stressor. However, this ethical consideration could also be considered a scientific limitation because participants' responses (e.g., reductions in negative affect and heart rate) could be due to increased time in the study (e.g., becoming more relaxed by watching more of the calming nature videos) versus a function of the comments themselves.

There are also important limitations to consider with the cortisol measurement and results. The literature has demonstrated that cortisol is very sensitive to a range of dietary, behavior, and physiological processes (Nicolson, 2007). While this study attempted to control for many of these issues (e.g., having dietary and behavior

restrictions; collecting data at the same time for each participant), the study may not have allowed enough time for participants to settle in so that a true baseline measurement could be gathered. It is possible that the elevated cortisol response prior to the comments was indicative of an anticipatory stress response. It is also possible that the study design did not allow for participants to reach a cortisol baseline at the outset of the study (and possibly during the study as well).

Future Directions

The findings from this study have multiple basic science and clinical implications. From a basic science perspective, the reactivity results indicate that high-risk individuals are not more sensitive to criticism than low-risk controls. Considering some of the literature suggests that high-risk populations are more sensitive to stress (e.g., Myin-Germeys et al., 2001), it is important for future research to discern if high-risk individuals and low-risk controls face different stressors (both in magnitude and type) that make their stress response more reactive outside of the lab compared to in the lab. Determining whether there are differences not only in number of life stressors, but also in type (e.g., social, financial, psychiatric) and severity, will be an important step in piecing together the various findings in the literature and ascertaining the details of stress response for individuals at high risk for psychosis. This can be done by more closely tracking the stressors (coding for severity and type) within participants' lives. It is possible that (per unit of stress) high risk individuals do not show more reactivity. Rather, with increased number, type, and severity of stressors in their daily lives, high-risk individuals demonstrate greater stress responses outside of the lab. This presents a more environmental and social justice perspective and implication to deal with stressors. To

that end, future work should attempt to more closely dissect the real-life stressors and stress-responses in the lives of individuals at high-risk for psychosis.

Another future direction of research that should be undertaken is better understanding emotion regulation strategies (and potential deficiencies) as well as methods of coping with stress in high-risk individuals. High-risk individuals demonstrated some difficulties in recovering from critical comments; however, not much research has examined details of emotion regulation and coping strategies within the high-risk population. The preponderance of work has examined brain regions and circuitry that are altered during emotional processing in high-risk individuals (e.g., Gee et al., 2012), but not much work has examined the behavioral and cognitive coping styles that are engaged in response to negative emotions (as well as positive emotions) and life stressors. It would be valuable to understand if individuals at high risk for psychosis use different/maladaptive coping and regulatory strategies compared to low-risk controls. It would also be helpful to know, in tandem with the previous future direction, whether high-risk individuals use similar coping and regulatory strategies as the general population, but just have more stress to cope with or regulate. Considering the work to show that they report greater stress in their lives (Tessner et al., 2011), as well as the threshold hypothesis, it is possible that high-risk individuals use similar coping and regulatory strategies compared to the general population. However, it is also likely that maladaptive coping and regulatory practices are employed as well, in addition to facing an "overload" of stressors.

Further examining the effects of praise on high-risk populations is another important future direction. Praise has been a component of developmental, educational,

and industrial/organizational psychological interventions for some time now (e.g., Crowell et al., 1988; Jones, Young, & Friman, 2000; Myers, Simonsen, & Sugai, 2011). However, praise and positive social interactions as buffers or protective factors has not been as commonly examined in the severe mental illness literature. O'Brien and colleagues (2006; 2008) have found that positive family interactions predict better clinical and family outcomes, but the relationship between positive social interactions and improved outcomes is unclear. Future research should seek to better understand why and how praise and warmth create clinical and familial benefits for individuals at risk for psychosis. Additionally, implementing more protective factors in treatment (in addition to reducing risk factors) has the potential to greatly improve outcomes. Thus, testing the effects of treatments that incorporate praise, warmth, and other positive social interactions can help to increase the protective factors in the lives of individuals at high risk for psychosis.

Conclusion

This study sought to test the affective, anxiety, and physiological effects of emotional comments (neutral, critical, and praise) on individuals at high-risk for psychosis relative to low-risk controls. Results indicated that, by-in-large, the comments successfully induced affective, anxiety, and physiological responses in the expected directions. Further, high-risk individuals did not show a greater sensitivity to critical comments relative to controls; however, they did show slower recover following criticism. There was also some evidence to suggest that the high-risk group perceived the neutral comments as less neutral and more negative than did low-risk controls. Finally, the high-risk group responded strongly to the praise comments, suggesting that positive

social interactions are well-received and beneficial to these individuals. Overall, these findings have important clinical implications, as they suggest that helping individuals at high risk for psychosis cope with negative social interactions and increasing positive social interactions in their lives can help reduce their affective, anxiety, and physiological stress responses.

REFERENCES

Affleck, G., Tennen, H., Urrows, S., & Higgins, P. (1994). Person and contextual features of daily stress reactivity: individual differences in relations of undesirable daily events with mood disturbance and chronic pain intensity. *Journal of Personality and Social Psychology*, *66*(2), 329-340.

Albus, M., Ackenheil, M., Engel, R.R., Muller, F., 1982. Situational reactivity of autonomic functions in schizophrenic patients. *Psychiatry Research*, *6*, 361—370.

Altorfer, A., Kaeserrmann, M.-L., & Hirsbrunner, H. (1998). Arousal and communication: 1. The relationship between nonverbal, behavioral, and physiological indices of the stress response. *Journal of Psychophysiology*, *12*, 40-59.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5*. Washington, D.C: Author.

Amminger, G. P., Schäfer, M. R., Papageorgiou, K., Klier, C. M., Schlögelhofer, M., Mossaheb, N., ... & McGorry, P. D. (2012). Emotion recognition in individuals at clinical high-risk for schizophrenia. *Schizophrenia Bulletin*, *38*(5), 1030-1039.

Appelhans, B. M., & Luecken, L. J. (2006). Heart rate variability as an index of regulated emotional responding. *Review of General Psychology*, *10*(3), 229-240.

Awad, A.G., & Voruganti, L.N. (2008). The burden of schizophrenia on caregivers. *Pharmacoeconomics*, *26(2)*, 149-162.

Bach, D. R. (2014). Sympathetic nerve activity can be estimated from skin conductance responses—A comment on Henderson et al.(2012). *NeuroImage*, *84*, 122-123.

Barnes, L. L. B., Harp, D., & Jung, W. S. (2002). Reliability generalization of scores on the Spielberger State–Trait Anxiety Inventory. *Educational and Psychological Measurement, 62*, 603– 618.

Basco, M. R., Bostic, J. Q., Davies, D., Rush, A. J., Witte, B., Hendrickse, W., & Barnett, V. (2000). Methods to improve diagnostic accuracy in a community mental health setting. *American Journal of Psychiatry*, *157*(10), 1599-1605.

Bechdolf, A., Thompson, A., Nelson, B., Cotton, S., Simmons, M. B., Amminger, G. P., ... & Yung, A. R. (2010). Experience of trauma and conversion to psychosis in an ultra-high-risk (prodromal) group. *Acta Psychiatrica Scandinavica*, *121*(5), 377-384.

Bhui, K., Abdi, A., Abdi, M., Pereira, S., Dualeh, M., Robertson, D., ... & Ismail, H. (2003). Traumatic events, migration characteristics and psychiatric symptoms among Somali refugees. *Social Psychiatry and Psychiatric Epidemiology*, *38*(1), 35-43.

Brenner, K., Liu, A., Laplante, D. P., Lupien, S., Pruessner, J. C., Ciampi, A., ... & King, S. (2009). Cortisol response to a psychosocial stressor in schizophrenia: blunted, delayed, or normal?. *Psychoneuroendocrinology*, *34*(6), 859-868.

Brown, G. W., Birley, J. L., & Wing, J. K. (1972). Influence of family life on the course of schizophrenic disorders: a replication. *The British Journal of Psychiatry*, *121*(562), 241-258.

Buckley, P. F., Miller, B. J., Lehrer, D. S., & Castle, D. J. (2009). Psychiatric comorbidities and schizophrenia. *Schizophrenia Bulletin*, *35*(2), 383-402.

Butzlaff, R. L., & Hooley, J. M. (1998). Expressed emotion and psychiatric relapse: a meta-analysis. *Archives of General Psychiatry*, *55*(6), 547-552.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Research*, *28*(2), 193-213.

Cannon, T. D., Cadenhead, K., Cornblatt, B., Woods, S. W., Addington, J., Walker, E., ... Heinssen, R. (2008). Prediction of psychosis in youth at high clinical risk: a multisite longitudinal study in North America. [Multicenter Study Research Support, N.I.H., Extramural Research Support, Non-U.S. Gov't]. *Archives of General Psychiatry, 65*(1), 28-37. Doi: 10.1001/archgenpsychiatry.2007.3

Chambless, D. L., & Steketee, G. (1999). Expressed emotion and behavior therapy outcome: A prospective study with obsessive compulsive and agoraphobic outpatients. *Journal of Consulting and Clinical Psychology*, *67*, 658 – 665.

Chapman, L., Chapman, J., Kwapil, T., Eckblad, M., Zinser, M. (1994). Putatively psychosis-prone subjects 10 years later. *Journal of Abnormal Psychology*, *103*, 171–183.

Chaumette, B., Kebir, O., Mam-Lam-Fook, C., Morvan, Y., Bourgin, J., Godsil, B. P., ... & Krebs, M. O. (2016). Salivary cortisol in early psychosis: New findings and meta-analysis. *Psychoneuroendocrinology*, *63*, 262-270.

Claridge, G.S. (1997). *Schizotypy: Implications for illness and health*. Oxford: Oxford University Press.

Cohen, A. S., & Minor, K. S. (2010). Emotional experience in patients with schizophrenia revisited: meta-analysis of laboratory studies. *Schizophrenia Bulletin*, *36*(1), 143-150.

Corcoran, C. M., Smith, C., McLaughlin, D., Auther, A., Malaspina, D., & Cornblatt, B. (2012). HPA axis function and symptoms in adolescents at clinical high risk for schizophrenia. *Schizophrenia Research*, *135*(1), 170-174.

Crowell, C. R., Anderson, D. C., Abel, D. M., & Sergio, J. P. (1988). Task clarification, performance feedback, and social praise: Procedures for improving the customer service of bank tellers. *Journal of Applied Behavior Analysis*, *21*(1), 65-71.

Cutting, L. P., Aakre, J. M., & Docherty, N. M. (2006). Schizophrenic patients' perceptions of stress, expressed emotion, and sensitivity to criticism. *Schizophrenia Bulletin*, *32*(4), 743-750.

Davidson, R. J., Marshall, J. R., Tomarken, A. J., & Henriques, J. B. (2000). While a phobic waits: Regional brain electrical and autonomic activity in social phobics during anticipation of public speaking. *Biological Psychiatry*, *47*(2), 85-95.

Day, F. L., Valmaggia, L. R., Mondelli, V., Papadopoulos, A., Papadopoulos, I., Pariante, C. M., & McGuire, P. (2014). Blunted cortisol awakening response in people at ultra high risk of developing psychosis. *Schizophrenia Research*, *158*(1), 25-31.

DeLongis, A., Folkman, S., & Lazarus, R. S. (1988). The impact of daily stress on health and mood: psychological and social resources as mediators. *Journal of Personality and Social Psychology*, *54*(3), 486.

Docherty, N. M., St-Hilaire, A., Aakre, J. M., & Seghers, J. P. (2009). Life events and high-trait reactivity together predict psychotic symptom increases in schizophrenia. *Schizophrenia Bulletin*, *35*(3), 638-645.

Dowd, J. B., Simanek, A. M., & Aiello, A. E. (2009). Socio-economic status, cortisol and allostatic load: a review of the literature. *International Journal of Epidemiology*, *38*(5), 1297-1309.

D'Souza, D. C., Gil, R. B., Zuzarte, E., MacDougall, L. M., Donahue, L., Ebersole, J. S., ... & Krystal, J. H. (2006). γ-aminobutyric acid–serotonin interactions in healthy men: implications for network models of psychosis and dissociation. *Biological Psychiatry*, *59*(2), 128-137.

Eack, S. M., Mermon, D. E., Montrose, D. M., Miewald, J., Gur, R. E., Gur, R. C., ... & Keshavan, M. S. (2010). Social cognition deficits among individuals at familial high risk for schizophrenia. *Schizophrenia Bulletin*, *36*(6), 1081-1088.

Elfering, A., & Grebner, S. (2011). Ambulatory assessment of skin conductivity during first thesis presentation: lower self-confidence predicts prolonged stress response. *Applied Psychophysiology and Biofeedback*, *36*(2), 93-99.

Fekedulegn, D. B., Andrew, M. E., Burchfiel, C. M., Violanti, J. M., Hartley, T. A., Charles, L. E., & Miller, D. B. (2007). Area under the curve and other summary indicators of repeated waking cortisol measurements. *Psychosomatic Medicine*, *69*(7), 651-659.

Fennig, S., Craig, T., Lavelle, J., Kovasznay, B., & Bromet, E. J. (1994). Best-estimate versus structured interview-based diagnosis in first-admission psychosis. *Comprehensive Psychiatry*, *35*(5), 341-348.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. (2002). Structured clinical interview for DSM-IV-TR axis I disorders, research version, patient edition. *New York: Biometrics Research, New York State Psychiatric Institute*.

Freeman, D., & Garety, P. A. (2003). Connecting neurosis and psychosis: the direct influence of emotion on delusions and hallucinations. *Behaviour Research and Therapy*, *41*(8), 923-947.

French, P., & Morrison, A. P. (2004). *Early detection and cognitive therapy for people at high risk of developing psychosis: A treatment approach*. John Wiley & Sons.

Fusar-Poli, P., Nelson, B., Valmaggia, L., Yung, A. R., & McGuire, P. K. (2014). Comorbid depressive and anxiety disorders in 509 individuals with an at-risk mental state: impact on psychopathology and transition to psychosis. *Schizophrenia Bulletin*, *40*(1), 120-131.

Garner, B., Phassouliotis, C., Phillips, L. J., Markulev, C., Butselaar, F., Bendall, S., ... & McGorry, P. D. (2011). Cortisol and dehydroepiandrosterone-sulphate levels correlate with symptom severity in first-episode psychosis. *Journal of Psychiatric Research*, *45*(2), 249-255.

Gayer-Anderson, C., & Morgan, C. (2013). Social networks, support and early psychosis: a systematic review. *Epidemiology and Psychiatric Sciences*, *22*(2), 131-146.

Gee, D. G., Karlsgodt, K. H., van Erp, T. G., Bearden, C. E., Lieberman, M. D., Belger, A., ... & Woods, S. W. (2012). Altered age-related trajectories of amygdala-prefrontal circuitry in adolescents at clinical high risk for psychosis: a preliminary study. *Schizophrenia Research*, *134*(1), 1-9.

George, D., & Mallery, P. (2003). Frequencies. *SPSS for Windows step by step: A simple guide and reference*, *11*, 20-52.

Granger, D. A., Hibel, L. C., Fortunato, C. K., & Kapelewski, C. H. (2009). Medication effects on salivary cortisol: Tactics and strategy to minimize impact in behavioral and developmental science. *Psychoneuroendocrinology*, *34*(10), 1437-1448.

Hall, J., Whalley, H. C., McKirdy, J. W., Romaniuk, L., McGonigle, D., McIntosh, A. M., ... & Sprengelmeyer, R. (2008). Overactivation of fear systems to neutral faces in schizophrenia. *Biological Psychiatry*, *64*(1), 70-73.

Henry, J. D., & Crawford, J. R. (2005). The short-form version of the Depression Anxiety Stress Scales (DASS-21): Construct validity and normative data in a large non-clinical sample. *British Journal of Clinical Psychology*, *44*(2), 227-239.

Hill, E.E., Zack, E., Battaglini, C., Viru, M., Viru, A., Hackney, A.C., 2008. Exercise and circulating cortisol levels: the intensity threshold effect. *Journal of Endocrinological Investigation 31*, 587–591.

Hogarty, G. E., Anderson, C. M., Reiss, D. J., Kornblith, S. J., Greenwald, D. P., Ulrich, R. F., & Carter, M. (1991). Family psychoeducation, social skills training, and maintenance chemotherapy in the aftercare treatment of schizophrenia: II. two-year effects of a controlled study on relapse and adjustment. *Archives of General Psychiatry*, *48*(4), 340.

Holtzman, C. W., Trotman, H. D., Goulding, S. M., Ryan, A. T., Macdonald, A. N., Shapiro, D. I., ... & Walker, E. F. (2013). Stress and neurodevelopmental processes in the emergence of psychosis. *Neuroscience*, *249*, 172-191.

Hooley, J. M. (1986). Expressed emotion and depression: Interactions between patients and high-versus low-expressed-emotion spouses. *Journal of Abnormal Psychology, 95,* 237-246.

Hooley, J. M. (2007). Expressed emotion and relapse of psychopathology. *Annual Review of Clinical Psychology*, *3*, 329-352.

Hooley, J. M., & Gotlib, I. H. (2000). A diathesis-stress conceptualization of expressed emotion and clinical outcome. *Applied and Preventive Psychology*,*9*(3), 135-151.

Hooley, J. M., Gruber, S. A., Parker, H. A., Guillaumot, J., Rogowska, J., & Yurgelun-Todd, D. A. (2010). Neural processing of emotional overinvolvement in borderline personality disorder. *The Journal of Clinical Psychiatry*, *71*(8), 1017-1024.

Hooley, J. M., & Parker, H. A. (2006). Measuring expressed emotion: an evaluation of the shortcuts. *Journal of Family Psychology*, *20*(3), 386.

Hooley, J. M., & Teasdale, J. D. (1989). Predictors of relapse in unipolar depressives: expressed emotion, marital distress, and perceived criticism. *Journal of Abnormal Psychology*, *98*(3), 229.

Horan, W. P., Ventura, J., Nuechterlein, K. H., Subotnik, K. L., Hwang, S. S., & Mintz, J. (2005). Stressful life events in recent-onset schizophrenia: reduced frequencies and altered subjective appraisals. *Schizophrenia Research*, *75*(2), 363-374.

Ieda, M., Miyaoka, T., Wake, R., Liaury, K., Tsuchie, K., Fukushima, M., ... & Horiguchi, J. (2014). Evaluation of autonomic nervous system by salivary alpha-amylase level and heart rate variability in patients with schizophrenia. *European Archives of Psychiatry and Clinical Neuroscience*, *264*(1), 83-87.

Jansen, L. M., Gispen-de Wied, C. C., & Kahn, R. S. (2000). Selective impairments in the stress response in schizophrenic patients. *Psychopharmacology*, *149*(3), 319-325.

Janssen, I., Krabbendam, L., Bak, M., Hanssen, M., Vollebergh, W., Graaf, R. D., & Os, J. V. (2004). Childhood abuse as a risk factor for psychotic experiences. *Acta Psychiatrica Scandinavica*, *109*(1), 38-45.

Joiner, T. E. (1997). Shyness and low social support as interactive diatheses, with loneliness as mediator: Testing an interpersonal-personality view of vulnerability to depressive symptoms. *Journal of Abnormal Psychology*, *106*(3), 386-394.

Jones, K. M., Young, M. M., & Friman, P. C. (2000). Increasing peer praise of socially rejected delinquent youth: Effects on cooperation and acceptance. *School Psychology Quarterly*, *15*(1), 30-39.

Joormann, J., Waugh, C. E., & Gotlib, I. H. (2015). Cognitive bias modification for interpretation in major depression effects on memory and stress reactivity. *Clinical Psychological Science*, *3*(1), 126-139.

Kale, A., Naphade, N., Sapkale, S., Kamaraju, M., Pillai, A., Joshi, S., & Mahadik, S. (2010). Reduced folic acid, vitamin B 12 and docosahexaenoic acid and increased homocysteine and cortisol in never-medicated schizophrenia patients: implications for altered one-carbon metabolism. *Psychiatry Research*, *175*(1), 47-53.

Kelleher, I., & Cannon, M. (2011). Psychotic-like experiences in the general population: characterizing a high-risk group for psychosis. *Psychological Medicine*, *41*(1), 1-6.

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test'–a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, *28*(1-2), 76-81.

Kirschbaum, C., Steyer, R., Eid, M., Patalla, U., Schwenkmezger, P., & Hellhammer, D. H. (1990). Cortisol and behavior: 2. Application of a latent state-trait model to salivary cortisol. *Psychoneuroendocrinology*, *15*(4), 297-307.

Kuipers, L., Sturgeon, D., Berkowitz, R., & Left, J. (1983). Characteristics of expressed emotion: Its relationship to speech and looking in schizophrenic patients and their relatives. *British Journal of Clinical Psychology, 22,* 257-264.

Labad, J., Stojanovic-Pérez, A., Montalvo, I., Solé, M., Cabezas, Á., Ortega, L., ... & Gutiérrez-Zotes, A. (2015). Stress biomarkers as predictors of transition to psychosis in at-risk mental states: roles for cortisol, prolactin and albumin. *Journal of Psychiatric Research*, *60*, 163-169.

Lataster, J., Myin-Germeys, I., Lieb, R., Wittchen, H. U., & Van Os, J. (2012). Adversity and psychosis: a 10-year prospective study investigating synergism between early and recent adversity in psychosis. *Acta Psychiatrica Scandinavica*, *125*(5), 388-399.

Lee, G., Barrowclough, C., & Lobban, F. (2014). Positive affect in the family environment protects against relapse in first-episode psychosis. *Social Psychiatry and Psychiatric Epidemiology*, *49*(3), 367-376.

Leff, J. P., & Vaughn, C. E. (1985). Expressed emotion in families. New York: Guilford Press

Lichtenstein, P., Bjork, C., Hultman, C. M., Scolnick, E., Sklar, P., & Sullivan, P. F. (2006). Recurrence risks for schizophrenia in a Swedish national cohort. *Psychological Medicine*, *36*(10), 1417-1426.

Lindenmayer, J. P., Vital-Herne, M., Bark, N., Grochowski, S., & Moynihan, N. (1997). Heterogeneity of serotonergic response in treatment-refractory schizophrenia patients. *Biological Psychiatry*, *42*(1), 6-12.

López, S. R., Nelson Hipke, K., Polo, A. J., Jenkins, J. H., Karno, M., Vaughn, C., & Snyder, K. S. (2004). Ethnicity, expressed emotion, attributions, and course of schizophrenia: family warmth matters. *Journal of Abnormal Psychology*, *113*(3), 428.

Loewy, R. L., Pearson, R., Vinogradov, S., Bearden, C. E., & Cannon, T. D. (2011). Psychosis risk screening with the Prodromal Questionnaire—brief version (PQ-B). *Schizophrenia Research*, *129*(1), 42-46.

Marshall, M., Lewis, S., Lockwood, A., Drake, R., Jones, P., & Croudace, T. (2005). Association between duration of untreated psychosis and outcome in cohorts of first-episode patients: a systematic review. *Archives of General Psychiatry*, *62*(9), 975.

Maxwell, M. E. (1992). Family Interview for Genetic Studies (FIGS): a manual for FIGS. *Clinical Neurogenetics Branch, Intramural Research Program, National Institute of Mental Health, Bethesda, MD*.

McGlashan, T., Walsh, B., & Woods, S. (2010). *The psychosis-risk syndrome: Handbook for diagnosis and follow-up*. Oxford University Press.

Meehl, P. E. (1962). Schizotaxia, schizotypy, schizophrenia. *American Psychologist*, *17*(12), 827-838.

Miklowitz, D.J. (2004). The role of family systems in severe and recurrent psychiatric disorders: A developmental psychopathology view. *Development and Psychopathology*, 16, 667–688.

Miklowitz, D. J., Goldstein, M. J., Falloon, I. R., & Doane, J. A. (1984). Interactional correlates of expressed emotion in the families of schizophrenics. *British Journal of Psychiatry, 144,* 482-487.

Miklowitz, D. J., & Tompson, M. C. (2003). Family variables and interventions in schizophrenia. *Textbook of Marital and Family Therapy*, 585-617.

Miklowitz, D. J., Wendel, J. S., & Simoneau, T. L. (1998). Targeting dysfunctional family interactions and high expressed emotion in the psychosocial treatment of bipolar disorder. *In Session,* 4, 25-38.

Miklowitz, D. J., Wisniewski, S. R., Miyahara, S., Otto, M. W., & Sachs, G. S. (2005). Perceived criticism from family members as a predictor of the 1-year course of bipolar disorder. *Psychiatry Research*, *136*, 101–111.

Miller, D. B., & O'Callaghan, J. P. (2002). Neuroendocrine aspects of the response to stress. *Metabolism*, *51*(6), 5-10.

Miller, T.J., McGlashan, T.H., Rosen, J.L., Cadenhead, K., Cannon, T., Ventura, J., . . . Woods, S.W. (2003). Prodromal assessment with the structured interview for prodromal syndromes and the scale of prodromal symptoms: predictive validity, interrater reliability, and training to reliability. *Schizophrenia Bulletin*, 29(4), 703–715. [PubMed: 14989408]

Mizrahi, R., Addington, J., Rusjan, P. M., Suridjan, I., Ng, A., Boileau, I., ... & Wilson, A. A. (2012). Increased stress-induced dopamine release in psychosis. *Biological Psychiatry*, *71*(6), 561-567.

Mondelli, V., Dazzan, P., Hepgul, N., Di Forti, M., Aas, M., D'Albenzio, A., ... & Pariante, C. M. (2010). Abnormal cortisol levels during the day and cortisol awakening response in first-episode psychosis: the role of stress and of antipsychotic treatment. *Schizophrenia Research*, *116*(2), 234-242.

Murri, M. B., Pariante, C. M., Dazzan, P., Hepgul, N., Papadopoulos, A. S., Zunszain, P., ... & Mondelli, V. (2012). Hypothalamic–pituitary–adrenal axis and clinical symptoms in first-episode psychosis. *Psychoneuroendocrinology*, *37*(5), 629-644.

Myers, D. M., Simonsen, B., & Sugai, G. (2011). Increasing teachers' use of praise with a response-to-intervention approach. *Education and Treatment of Children*, *34*(1), 35-59.

Myin-Germeys, I., van Os, J., Schwartz, J. E., Stone, A. A., & Delespaul, P. A. (2001). Emotional reactivity to daily life stress in psychosis. *Archives of General Psychiatry*, *58*(12), 1137-1144.

Nicolson, N. A. (2008). Measurement of cortisol. *Handbook of Physiological Research Methods in Health Psychology*, 37-74.

Norman, R. M., Malla, A. K., Manchanda, R., Harricharan, R., Takhar, J., & Northcott, S. (2005). Social support and three-year symptom and admission outcomes for first episode psychosis. *Schizophrenia Research*, *80*(2), 227-234.

Nuechterlein, K. H., Snyder, K. S., & Mintz, J. (1992). Paths to relapse: possible transactional processes connecting patient illness onset, expressed emotion, and psychotic relapse. *The British Journal of Psychiatry*, *161*(18), 88-96.

Nurnberger, J. I., Blehar, M. C., Kaufmann, C. A., York-Cooler, C., Simpson, S. G., Harkavy-Friedman, J., ... & Reich, T. (1994). Diagnostic interview for genetic studies: rationale, unique features, and training. *Archives of General Psychiatry*, *51*(11), 849-859.

O'Brien, M. P., Gordon, J. L., Bearden, C. E., Lopez, S. R., Kopelowicz, A., & Cannon, T. D. (2006). Positive family environment predicts improvement in symptoms and social functioning among adolescents at imminent risk for onset of psychosis. *Schizophrenia Research*, *81*(2), 269-275.

O'Brien, M. P., Zinberg, J. L., Bearden, C. E., Lopez, S. R., Kopelowicz, A., Daley, M., & Cannon, T. D. (2008). Parent attitudes and parent adolescent interaction in families of youth at risk for psychosis and with recent-onset psychotic symptoms. *Early Intervention in Psychiatry*, *2*(4), 268-276.

Oldehinkel, A. J., Verhulst, F. C., & Ormel, J. (2008). Low heart rate: a marker of stress resilience. The TRAILS study. *Biological Psychiatry*, *63*(12), 1141-1146.

Phillips, L. J., Edwards, J., McMurray, N., & Francey, S. (2012). Comparison of experiences of stress and coping between young people at risk of psychosis and a non-clinical cohort. *Behavioural and Cognitive Psychotherapy*, *40*(01), 69-88.

Phillips, L. J., Francey, S. M., Edwards, J., & McMurray, N. (2007). Stress and psychosis: towards the development of new models of investigation. *Clinical Psychology Review*, *27*(3), 307-317.

Poulton, R., Caspi, A., Moffitt, T., Cannon, M., Murray, R., Harrington, H. (2000). Children's self-reported psychotic symptoms and adult schizophreniform disorder. *Archives of General Psychiatry*, 57, 1053– 1058.

Pruessner, M., Cullen, A. E., Aas, M., & Walker, E. F. (2017). The neural diathesis-stress model of schizophrenia revisited: An update on recent findings considering illness stage and neurobiological and methodological complexities. *Neuroscience & Biobehavioral Reviews*, *73*, 191-218.

Pruessner, J. C., Kirschbaum, C., Meinlschmid, G., & Hellhammer, D. H. (2003). Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology*, *28*(7), 916-931.

Renshaw, K. D., Chambless, D. L., & Steketee, G. (2003). Perceived criticism predicts severity of anxiety symptoms after behavioral treatment in patients with obsessive-compulsive disorder and panic disorder with agoraphobia. *Journal of Clinical Psychology*, *59*(4), 411-421.

Robustelli, B. L., Newberry, R. E., Whisman, M. A., & Mittal, V. A. (2017). Social relationships in young adults at ultra high risk for psychosis. *Psychiatry Research*, *247*, 345-351.

Roekel, E., Goossens, L., Verhagen, M., Wouters, S., Engels, R. C., & Scholte, R. H. (2014). Loneliness, affect, and adolescents' appraisals of company: An experience sampling method study. *Journal of Research on Adolescence*, *24*(2), 350-363.

Rosenfarb, I. S., Bellack, A. S., & Aziz, N. (2006). Family interactions and the course of schizophrenia in African American and White patients. *Journal of Abnormal Psychology*, *115*(1), 112.

Russell, D. W. (1996). UCLA Loneliness Scale (Version 3): Reliability, validity, and factor structure. *Journal of Personality Assessment*, *66*(1), 20-40.

Rutter, M., & Brown, G. W. (1966). The reliability and validity of measures of family life and relationships in families containing a psychiatric patient. *Social Psychiatry*, *1*(1), 38-53.

Saha, S., Chant, D., Welham, J., McGrath, J. (2005). A systematic review of the prevalence of schizophrenia. *PLoS Medicine, 2*, 413-433.

Scheer, F. A., Kalsbeek, A., & Buijs, R. M. (2003). Cardiovascular control by the suprachiasmatic nucleus: neural and neuroendocrine mechanisms in human and rat. *Biological Chemistry*, *384*(5), 697-709.

Schlosser, D. A., Zinberg, J. L., Loewy, R. L., Casey-Cannon, S., O'Brien, M. P., Bearden, C. E., ... & Cannon, T. D. (2010). Predicting the longitudinal effects of the family environment on prodromal symptoms and functioning in patients at-risk for psychosis. *Schizophrenia Research*, *118*(1), 69-75.

Seiferth, N. Y., Pauly, K., Habel, U., Kellermann, T., Shah, N. J., Ruhrmann, S., ... & Kircher, T. (2008). Increased neural response related to neutral faces in individuals at risk for psychosis. *Neuroimage*, *40*(1), 289-297.

Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., & Jacobs, G. A. (1983). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.

Stevens, A., & White, A. (2010). ACTH: cellular peptide hormone synthesis and secretory pathways. In *Cellular Peptide Hormone Synthesis and Secretory Pathways* (pp. 121-135). Springer Berlin Heidelberg.

Sturgeon, D., Turpin, G., Kuipers, L., Berkowitz, R., & Left, J. (1984). Psychophysiological responses of schizophrenic patients to high and low expressed emotion relatives: A follow-up study. *British Journal of Psychiatry*, *145*, 62-69.

Sündermann, O., Onwumere, J., Kane, F., Morgan, C., & Kuipers, E. (2014). Social networks and support in first-episode psychosis: exploring the role of loneliness and anxiety. *Social Psychiatry and Psychiatric Epidemiology*, *49*(3), 359-366.

Tarrier, N., & Turpin, G. (1992). Psychosocial factors, arousal and schizophrenic relapse: The psychophysiological data. *British Journal of Psychiatry, 161,* 3-11.

Tarrier, N., Vaughn, C., Lader, M. H., & Left, J. P. (1979). Bodily reactions to people and events in schizophrenics. *Archives of General Psychiatry*, *36*, 311- 315.

Tessner, K. D., Mittal, V., & Walker, E. F. (2011). Longitudinal study of stressful life events and daily stressors among adolescents at high risk for psychotic disorders. *Schizophrenia Bulletin*, *37*(2), 432-441.

Thompson, K. N., Phillips, L. J., Komesaroff, P., Yuen, H. P., Wood, S. J., Pantelis, C., ... & McGorry, P. D. (2007). Stress and HPA-axis functioning in young people at ultra high risk for psychosis. *Journal of Psychiatric Research*, *41*(7), 561-569.

Trotman, H. D., Holtzman, C. W., Walker, E. F., Addington, J. M., Bearden, C. E., Cadenhead, K. S., ... & McGlashan, T. H. (2014). Stress exposure and sensitivity in the clinical high-risk syndrome: Initial findings from the North American Prodrome Longitudinal Study (NAPLS). *Schizophrenia Research*, *160*(1), 104-109.

van Os, J., Hanssen, M., Bijl, R. V., & Ravelli, A. (2000). Strauss (1969) revisited: a psychosis continuum in the general population?. *Schizophrenia Research*, *45*(1), 11-20.

van Os, J., Linscott, R. J., Myin-Germeys, I., Delespaul, P., & Krabbendam, L. (2009). A systematic review and meta-analysis of the psychosis continuum: evidence for a psychosis proneness-persistence-impairment model of psychotic disorder. *Psychological Medicine*, *39*(2), 179.

van Rijn, S., Schothorst, P., van't Wout, M., Sprong, M., Ziermans, T., van Engeland, H., ... & Swaab, H. (2011). Affective dysfunctions in adolescents at risk for psychosis: emotion awareness and social functioning. *Psychiatry Research*, *187*(1), 100-105.

Varese, F., Smeets, F., Drukker, M., Lieverse, R., Lataster, T., Viechtbauer, W., ... & Bentall, R. P. (2012). Childhood adversities increase the risk of psychosis: a meta-analysis of patient-control, prospective-and cross-sectional cohort studies. *Schizophrenia Bulletin*, *38*(4), 661-671.

Vaughn, C. E., & Leff, J. P. (1976). The influence of family and social factors on the course of psychiatric illness. A comparison of schizophrenic and depressed neurotic patients. *The British Journal of Psychiatry*, *129*(2), 125-137.

Veling, W., Pot-Kolder, R., Counotte, J., van Os, J., & van der Gaag, M. (2016). Environmental Social Stress, Paranoia and Psychosis Liability: A Virtual Reality Study. *Schizophrenia Bulletin*, *42*(6), 1363-1371.

Venkatasubramanian, G., Chittiprol, S., Neelakantachar, N., Shetty, T., & Gangadhar, B. N. (2010). Effect of antipsychotic treatment on Insulin-like Growth Factor-1 and cortisol in schizophrenia: a longitudinal study. *Schizophrenia Research*, *119*(1), 131-137.

Verdoux, H., & van Os, J. (2002). Psychotic symptoms in non-clinical populations and the continuum of psychosis. *Schizophrenia Research, 54*(1-2), 59-65.

Walker, E. F., Brennan, P. A., Esterberg, M., Brasfield, J., Pearce, B., & Compton, M. T. (2010). Longitudinal changes in cortisol secretion and conversion to psychosis in at-risk youth. *Journal of Abnormal Psychology*, *119*(2), 401.

Walker, E. F., & Diforio, D. (1997). Schizophrenia: a neural diathesis-stress model. *Psychological Review*, *104*(4), 667.

Walker, E., Mittal, V., & Tessner, K. (2008). Stress and the hypothalamic pituitary adrenal axis in the developmental course of schizophrenia. *Annual Review of Clinical Psychology*, *4*, 189-216.

Walker, E. F., Trotman, H. D., Pearce, B. D., Addington, J., Cadenhead, K. S., Cornblatt, B. A., ... & Tsuang, M. T. (2013). Cortisol levels and risk for psychosis: initial findings from the North American prodrome longitudinal study. *Biological Psychiatry*, *74*(6), 410-417.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology*, *54*(6), 1063.

Weisman, A. G., Nuechterlein, K. H., Goldstein, M. J., & Snyder, K. (2000). Controllability perceptions and reactions to symptoms of schizophrenia: A within family comparison of high-EE and low-EE relatives. *Journal of Abnormal Psychology, 109,* 167-171.

Weisman, A. G., Rosales, G. A., Kymalainen, J. A., & Armesto, J. C. (2006). Ethnicity, expressed emotion, and schizophrenia patients' perceptions of their family members' criticism. *The Journal of nervous and mental disease*, *194*(9), 644-649.

Wu, E. Q., Birnbaum, H. G., Shi, L., Ball, D. E., Kessler, R. C., Moulis, M., & Aggarwal, J. (2005). The economic burden of schizophrenia in the United States in 2002. *Journal of Clinical Psychiatry*, *66*(9), 1122-1129.

Yung, A. R., Yuen, H. P., McGorry, P. D., Phillips, L. J., Kelly, D., Dell'Olio, M., ... & Buckby, J. (2005). Mapping the onset of psychosis: the Comprehensive Assessment of At-Risk Mental States. *Australian and New Zealand Journal of Psychiatry*, *39*(11-12), 964-971.

Zolkowska, K., Cantor-Graae, E., & McNeil, T. F. (2003). Psychiatric admissions for psychosis in Malmö during the NATO bombing of Kosovo. *The Journal of Nervous and Mental Disease*, *191*(12), 820-826.

Zubin, J., & Spring, B. (1977). Vulnerability: A new view of schizophrenia. *Journal of Abnormal Psychology*, *86*(2), 103-126.

Appendix A: MEASURES

PQ-B (Rachel Loewy, PhD and Tyrone D. Cannon, PhD**)** ©University of California, 2010

Please indicate whether you have had the following thoughts, feelings and experiences **in the past month** by checking "yes" or "no" for each item. **Do not include experiences that occur only while under the influence of alcohol, drugs or medications that were not prescribed to you.** If you answer "YES" to an item, also indicate how distressing that experience has been for you.

1. Do familiar surroundings sometimes seem strange, confusing, threatening or unreal to you? ☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

2. Have you heard unusual sounds like banging, clicking, hissing, clapping or ringing in your ears? ☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

3. Do things that you see appear different from the way they usually do (brighter or duller, larger or smaller, or changed in some other way)? ☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

4. Have you had experiences with telepathy, psychic forces, or fortune telling?

☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

5. Have you felt that you are not in control of your own ideas or thoughts?

☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

6. Do you have difficulty getting your point across, because you ramble or go off the track a lot when you talk? ☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

7. Do you have strong feelings or beliefs about being unusually gifted or talented in some way? ☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

8. Do you feel that other people are watching you or talking about you?

☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

9. Do you sometimes get strange feelings on or just beneath your skin, like bugs crawling? ☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

10. Do you sometimes feel suddenly distracted by distant sounds that you are not normally aware of? ☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

11. Have you had the sense that some person or force is around you, although you couldn't see anyone? ☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

12. Do you worry at times that something may be wrong with your mind?

☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

13. Have you ever felt that you don't exist, the world does not exist, or that you are dead?

☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

14. Have you been confused at times whether something you experienced was real or imaginary? ☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

15. Do you hold beliefs that other people would find unusual or bizarre?

☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:

☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

16. Do you feel that parts of your body have changed in some way, or that parts of your body are working differently? ☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:
☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

17. Are your thoughts sometimes so strong that you can almost hear them?
☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:
☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

18. Do you find yourself feeling mistrustful or suspicious of other people?
☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:
☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

19. Have you seen unusual things like flashes, flames, blinding light, or geometric figures?
☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:
☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

20. Have you seen things that other people can't see or don't seem to see?
☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:
☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

21. Do people sometimes find it hard to understand what you are saying?
☐ YES ☐ NO

If YES: When this happens, I feel frightened, concerned, or it causes problems for me:
☐ Strongly disagree ☐ disagree ☐ neutral ☐ agree ☐ strongly agree

DASS 21 (Abridged – Depression and Anxiety subscales only)

Please read each statement and give the number 0, 1, 2 or 3 that indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1 _____ I was aware of dryness of my mouth

2 _____ I couldn't seem to experience any positive feeling at all

3 _____ I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)

4 _____ I found it difficult to work up the initiative to do things

5 _____ I experienced trembling (eg, in the hands)

6 _____ I was worried about situations in which I might panic and make a fool of myself

7 _____ I felt that I had nothing to look forward to

8 _____ I felt down-hearted and blue

9 _____ I felt I was close to panic

10 _____ I was unable to become enthusiastic about anything

11 _____ I felt I wasn't worth much as a person

12 _____ I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)

13 _____ I felt scared without any good reason

14 _____ I felt that life was meaningless

SELF-EVALUATION QUESTIONNAIRESTAI Form Y-1

DIRECTIONS:

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel *right* now, that is, *at this moment.* There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

	NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
1. I feel calm	1	2	3	4
2. I feel secure	1	2	3	4
3. I am tense	1	2	3	4
4. I feel strained	1	2	3	4
5. I feel at ease	1	2	3	4
6. I feel upset	1	2	3	4
7. I am presently worrying over possible misfortunes	1	2	3	4
8. I feel satisfied	1	2	3	4
9. I feel frightened	1	2	3	4
10. I feel comfortable	1	2	3	4
11. I feel self-confident	1	2	3	4
12. I feel nervous	1	2	3	4
13. I am jittery	1	2	3	4
14. I feel indecisive	1	2	3	4
15. I am relaxed	1	2	3	4
16. I feel content	1	2	3	4
17. I am worried	1	2	3	4
18. I feel confused	1	2	3	4
19. I feel steady	1	2	3	4
20. I feel pleasant	1	2	3	4

PANAS Questionnaire

This scale consists of a number of words that describe different feelings and emotions. Read each item and then list the number from the scale below next to each word. **Indicate to what extent you feel this way right now, that is, at the present moment.**

1	2	3	4	5
Very Slightly or Not at all	A Little	Moderately	Quite a Bit	Extremely

_____1. Interested

_____2. Distressed

_____3. Excited

_____4. Upset

_____5. Strong

_____6. Guilty

_____7. Scared

_____8. Hostile

_____9. Enthusiastic

_____10. Proud

_____11. Irritable

_____12. Alert

_____13. Ashamed

_____14. Inspired

_____15. Nervous

_____16. Determined

_____17. Attentive

_____18. Jittery

_____19. Active

_____20. Afraid

Perceived Criticism

1. How critical is your relative of you?

1	2	3	4	5	6	7	8	9	10
Not at all critical									Very critical

2. When your relative criticizes you, how upset do you get?

1	2	3	4	5	6	7	8	9	10
Not at all upset									Very upset

Revised UCLA Loneliness Scale:

INSTRUCTIONS: Indicate how often each of the statements below is descriptive of you.

Statement	Never	Rarely	Sometimes	Often
1. I feel in tune with the people around me	1	2	3	4
2. I lack companionship	1	2	3	4
3. There is no one I can turn to	1	2	3	4
4. I do not feel alone	1	2	3	4
5. I feel part of a group of friends	1	2	3	4
6. I have a lot in common with the people around me	1	2	3	4
7. I am no longer close to anyone	1	2	3	4
8. My interests and ideas are not shared by those around me	1	2	3	4
9. I am an outgoing person	1	2	3	4
10. There are people I feel close to	1	2	3	4
11. I feel left out	1	2	3	4
12. My social relationships arc superficial	1	2	3	4
13. No one really knows me well	1	2	3	4
14. I feel isolated from others	1	2	3	4
15. I can find companionship when I want it	1	2	3	4
16. There are people who really understand me	1	2	3	4
17. I am unhappy being so withdrawn	1	2	3	4
18. People are around me but not with me	1	2	3	4
19. There are people I can talk to	1	2	3	4
20. There are people I can turn to	1	2	3	4