



Affective and physiological reactivity to emotional comments in individuals at elevated risk for psychosis



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ABSTRACT

Background: Individuals with schizophrenia are at increased risk of relapse when they live in highly critical (i.e., high expressed emotion; EE) family environments. It remains less clear, however, how individuals at elevated risk for a psychotic disorder react to the social stress of EE. Here we examined whether individuals at elevated risk for developing schizophrenia report greater subjective changes in affect and have increased physiological reactivity after hearing critical, praising and neutral comments.

Method: Measures of heart rate, heart rate variability, skin conductance, and self-reported affective ratings were used to assess differential responses to EE-type stimuli in 38 individuals at elevated-risk for psychosis and 38 low-risk controls.

Results: The elevated-risk group and low-risk controls, did not differ in their initial affective and physiological reactivity to criticism. However, during the recovery period following the criticism, the elevated-risk group demonstrated greater heart rate activation. They also showed more sensitivity to praise. Although elevated-risk participants initially had higher baseline levels of negative affect and heart rate, following praise, these levels reduced and became indistinguishable from the levels of low-risk controls.

Conclusions: These findings suggest that at-risk individuals may have more difficulty recovering from criticism than their self-report data might suggest. They may also derive physiological and affective benefits from praise. Important clinical implications of these findings are discussed.

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1. Introduction

Schizophrenia occurs in about 1% of the population, and the illness often has a lifetime course, high rates of comorbidity with other psychiatric disorders, and is associated with poor psychological well-being (Buckley et al., 2008; Saha et al., 2005; Verdoux and van Os, 2002). Due to the impact schizophrenia has on those with the illness and on society more broadly, evaluating the mechanisms that may lead to the development of the disorder is a clear priority. It is widely believed that psychosis develops as a result of genetic or prenatal vulnerabilities that interact with environmental stressors, exacerbating or unmasking symptoms among individuals at risk (Walker and Diforio, 1997). With regard to environmental stressors, social stress in particular may play

an important role in the development of psychosis (Jones and Fernyhough, 2006; Selten et al., 2013).

One widely studied form of social stress is expressed emotion (EE). EE is a measure of how critical, hostile, or emotionally over-involved a family member is toward the patient (Hooley, 2007). After a period of hospitalization, patients who live in high EE family environments have 9 to 12-month relapse rates that are more than twice those of patients who live in low EE home environments (Butzlaff and Hooley, 1998). Thus, EE is considered a psychosocial stressor that interacts with the patients' diatheses to produce relapse (Hooley and Gotlib, 2000). Of the EE components, the most important component is criticism, as it is most strongly linked to relapse in schizophrenia (Alvarez-Jimenez et al., 2012; Brown et al., 1972).

The social stress of EE appears to have an immediate physiological impact on patients with schizophrenia, as they show heightened physiological responses (measured via blood pressure) when interacting with a high EE relative (Tarrier et al., 1979). Critical statements by relatives have also been linked to increased cardiovascular activity in patients with schizophrenia and bipolar disorder (Altorf et al., 1998).

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Taken together, such findings indicate that interacting with a critical, high EE relative is physiologically stressful for patients with psychotic disorders. Little is known, however, about stress response resulting from criticism in individuals at risk for psychosis. Because the social stress of criticism is such a potent predictor of relapse in patients with psychosis (Butzlaff and Hooley, 1998), it is important to clarify the risk mechanisms for illness progression in at-risk populations.

The research to-date experimentally testing the effects of psychosocial stress on individuals at risk for psychosis reveals mixed findings. Some research has found that at-risk individuals have greater physiological and emotional sensitivity to social stress compared to healthy controls (Mizrahi et al., 2012; Veling et al., 2016). One study found at-risk individuals had an attenuated cortisol stress response relative to healthy controls (Pruessner et al., 2013). Finally, a fourth study did not find differences in physiological and emotional reactivity to a psychosocial stressor between at-risk individuals, individuals with a psychotic disorder, and healthy controls (Lincoln et al., 2015). These studies examined stress responses from the social stressor of performing/interacting with strangers (e.g., giving a speech). Despite Pruessner and colleagues' and Lincoln and colleagues' findings, much research thus far on psychosocial and emotional reactivity suggests that individuals along the psychosis spectrum have heightened reactivity to social stress. However, examining whether there is heightened physiological and emotional reactivity to family stress and, more specifically, criticism in populations at risk for psychosis remains unexplored.

Positive aspects of family interactions in schizophrenia also deserve consideration. Warmth and positive remarks (praise) were both original components of the expressed emotion construct (Brown et al., 1972). However, Brown and colleagues found that they added little to the overall prediction of patients' clinical outcomes. As a result, research has almost exclusively focused on the more negative aspects of the family environment (e.g., criticism; Hooley, 2007). Nonetheless, more recent research suggests that family warmth and positive family environments were protective factors against relapse in patients with schizophrenia (Lee et al., 2014; López et al., 2004) as well as protective factors against social stress in high-risk populations (Bentley et al., 2016). There is also some work to suggest that maternal praise is predictive of reductions in prodromal symptoms over a 3-month period in individuals at high-risk for psychosis (O'Brien et al., 2006). Considering the protective effects of familial warmth on psychotic symptoms as well as previously cited research suggesting that individuals across the psychosis spectrum have greater emotional sensitivity and reactivity relative to controls, a closer examination of whether at-risk individuals have heightened reactions to positive social stimuli in populations is warranted.

The current study compared the self-reported affective changes and physiological consequences of hearing criticism, praise and neutral commentary between individuals with higher versus lower risk for psychosis (based on attenuated psychotic symptoms and/or genetic risk). To accomplish this, we measured self-reported positive and negative affect as well as heart rate, heart rate variability, and skin conductance before and after exposing participants to a standardized set of affectively-challenging EE comments (see Hooley et al., 2009, 2010). We predicted that, relative to baseline and neutral commentary measurement, hearing critical comments would elicit greater levels of self-reported negative affect, lower levels of self-reported positive affect, increased heart rate and skin conductance, and reduced heart rate variability in all participants. We also predicted that, compared to baseline and neutral measurements, hearing praise comments would elicit lower levels of self-reported negative affect, increased levels of self-reported positive affect, reduced heart rate and skin conductance and increased heart rate variability for participants in both groups. Finally, we predicted that responses to both positive and negative stimuli would be most marked for individuals at-risk for psychosis compared to the low-risk controls.

2. Methods

2.1. Participants

Two groups of non-treatment seeking individuals from the community were recruited. The at-risk group (i.e., elevated-risk group) included participants who experienced highly elevated attenuated positive psychotic symptoms OR who have a first-degree relative with schizophrenia/schizoaffective disorder and who had at least moderately elevated attenuated positive symptoms (cut-offs are detailed below). The low-risk group included participants who did not have a first degree relative with schizophrenia/schizophrenia and had attenuated psychotic symptoms that fell below the at-risk cutoff. Both groups reported no current psychiatric medication use, no nicotine use in the last 30 days, no substance abuse or dependence over the past 3 months, as well as no current use of blood pressure medication, steroid-based medications, oral contraceptives, asthma medication, or pain medications. All participants were required to be between the ages of 18 and 30, as this is the peak period of risk for first-onset psychotic disorders in adults (Yung et al., 2005).

2.2. Procedures

Advertisements were placed on the Miami Metrorail, around the Miami community, and on Craigslist. Prior to the participants coming into the laboratory, interested persons were screened over the phone for eligibility into the elevated-risk or low-risk group. Following an eligibility screen over the phone, participants completed questionnaires online within 48 h of coming in to the laboratory. Online questionnaires included measures such as demographics, family variables (not presented here), and a re-assessment of prodromal symptoms. Then, participants came into the laboratory, where they were set up to a BioPac System for heart rate, respiratory, and skin conductance measurement. Baseline measurements were taken for a total of 3 min to match the length of physiological measurements for each comment-block (described below). Additionally, participants provided baseline self-reports of positive and negative affect.

Participants then listened to three blocks of comments (neutral, critical, and praise), which were each 2 min in length. The neutral and critical comment-blocks were counterbalanced throughout the study. Because it was unclear how stressful the critical comments would be for the elevated-risk participants, it was decided that all participants would hear the praise comments in the third block to leave the laboratory after hearing a positively-valenced stimuli. Immediately following each comment-block, the participants sat quietly for a 1 min recording of their recovery from the comments. Then, participants reported on their positive and negative affect and then had a 30 min break (during which they watched a calming nature video) until the next comment-block.

The standardized comments were digitally recorded and played for the participants through speakers. All comments were phrased in the first person, and participants were asked to listen to each comment as if they were being said by a close female relative (preferably their primary female caregiver growing up). These standardized comments have been found to elicit reliable affective changes in healthy controls, people with dysthymia, depressed patients, recovered depressed participants, and patients with borderline personality disorder (Hooley et al., 2010). Participants were compensated \$40 for their time and effort.

2.3. Measures

2.3.1. Prodromal Questionnaire—Brief (PQ-B)

The PQ-B (Loewy et al., 2011) is a 21-item self-report of prodromal symptoms. For this study, participants were asked the questions of the PQ-B over the phone by the primary investigator (MJW) to determine eligibility for the study. This was done in an effort to ensure respondents

were answering questions genuinely. Additionally, the primary investigator was able to determine whether the respondents' positive responses to questions were consistent with symptoms along the psychosis spectrum (e.g., experiencing a visual perceptual abnormality versus seeing things others cannot due to good eyesight). Participants with elevated symptoms (total symptom score ≥ 6 and distress score ≥ 32), but who did not meet full criteria for a psychotic disorder were considered to be at elevated risk. Individuals with moderate symptoms (total symptom score ≥ 3 and distress score ≥ 6), a first-degree relative with a psychotic disorder, and did not meet criteria for a psychotic disorder also met criteria for the elevated-risk group. A total score ≤ 3 and a distress score ≤ 6 on the PQ-B and not having a first-degree relative with a psychotic disorder indicated low risk. (See Loewy et al., 2011 for positive predictive values and specificity of PQ-B values for a diagnosis of the psychosis-risk syndrome.) It is important to note that while the PQ-B was used to approximate the risk criteria outlined by the Structured Interview for Prodromal States (SIPS; Miller et al., 2003), it is not the gold-standard measurement for psychosis risk. Since PQ-B is a self-report screen, the cut-offs used here likely yield a lower risk sample relative to samples using a structured interview like the SIPS. Thus, rather than the term high-risk (which is a label used for individuals with prodromal psychosis based on a structured interview), we use the term "elevated risk." The internal consistency for the PQ-B within this study was excellent (PQ-B symptoms total Cronbach's $\alpha = 0.93$; PQ-B distress Cronbach's $\alpha = 0.93$).

2.3.2. Structured Clinical Interview for DSM-IV Disorders – psychotic module and substance use disorders (SCID)

The SCID (First et al., 2002), a semi-structured instrument, was used to exclude individuals who met DSM-IV criteria for schizophrenia, schizoaffective disorder, or a substance use disorder. All SCID assessments were conducted by the primary investigator of the study (MJW), who was an advanced clinical psychology doctoral student at the time of the study. The study's primary investigator demonstrated reliability for the SCID psychotic screen by rating eight videotaped interviews for a previous research trial (Weisman de Mamani et al., 2014). The assigned diagnoses from the videotapes were compared with the diagnoses assigned by the senior investigator (AWM). Results indicated perfect inter-rater reliability (Cohen's Kappa = 1.0) for the eight interviews.

2.3.3. Family Interview for Genetic Studies – Psychosis Checklist (FIGS)

The FIGS (Maxwell, 1992), a semi-structured interview, was used to determine whether a first-degree relative met criteria for a psychotic disorder.

2.3.4. Positive and Negative Affect Schedule (PANAS)

The PANAS (Watson et al., 1988) was used to measure participants' state level of positive and negative affect in response to the standardized expressed emotion comments.

2.3.5. Relevance and valence of standardized comments

At the end of the study, participants were asked to rate (1) the relevance of each comment to them personally and (2) the valence of each comment. Participants rated on a scale from 1 to 9 the relevance and the valence of each comment. For the relevance ratings, 1 indicated "Did not at all feel this was about me" and 9 indicated "Totally felt this was about me." For the valence ratings, 1 indicated "Very positive" and 9 indicated "Very negative." For both relevance and valence, the four responses per comment-block were summed for a total score.

2.3.6. BioPac MP150 system

The BioPac system was used for the autonomic measurements: heart rate (HR), heart rate variability (measured via respiratory sinus arrhythmia, RSA), and skin conductance levels (SCL). Using a time-course approach, ECG and SCL measurements were continuously recorded for a

3-minute duration, comprised of 2 min of comment presentation and a 1 min recovery period (in which no stimuli were presented).

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., Gahanna, OH). Heart rate was calculated as the average number of heartbeats (measured as the R-R intervals of the heartbeat) separately for each of the 1 min measurement periods. RSA was 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). A natural log transformation was applied to RSA values to normalize the frequency distribution for statistical analyses. The electrical conductance across the skin for SCL response is measured in units of microSiemens (μS), which was similarly averaged over each of the 1 min measurement periods. Following appropriate corrections to ensure data quality, physiological indices were extracted in 1 min segments – an initial reactivity (minute 1), sustained reactivity (minute 2) and recovery (minute 3) variable.

2.4. Statistical analyses

2.4.1. Primary analyses

A series of repeated measures ANOVAs were conducted in SPSS 22. Repeated measures ANOVA was conducted for each outcome, using risk group (elevated-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 physiological indices, measurement time (of which there were 3 – initial reactivity, sustained reactivity, and recovery) was used as another repeated within-subject measure.

2.4.2. Confirmatory analyses

As a manipulation check on whether the comments were (1) relevant to participants and (2) had the expected valence, repeated measures ANOVAs were performed to determine whether the two risk groups differed in their perceptions of the relevance and valence of each comment-block.

3. Results

3.1. Preliminary results

A total of 87 individuals participated in the study. 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 over the phone for the low-risk control group) were excluded from analyses. Further, two individuals were excluded for failing to comply with study procedures (falling asleep or using a cell phone). In total, 76 participants (38 elevated-risk and 38 low-risk controls) were retained for subsequent analyses.

The negative affect subscale of the PANAS had significant skewness and kurtosis across each measurement. A log transformation was performed to correct for high skewness and kurtosis, leading to values within acceptable ranges. All other variables fell within normal limits. The demographic variables did not relate to any of the self-report affective outcome or physiological reactivity outcome variables. Of note, Hispanics did rate marginally higher total scores on the PQ-B relative to other groups ($F(4,71) = 2.21, p = .08$). There was no interaction between ethnicity and risk group on PQ-B total scores ($F(3,67) = 1.12, p = .35$). Ethnicity did not relate to PQ-B distress scores ($F(4,71) = 1.82, p = .14$). There was a marginally significant interaction between ethnicity and risk group on PQ-B distress scores ($F(3,67) = 2.70, p = .06$); the low-risk Hispanic group had marginally lower distress relative to the low-risk Caucasian and African-American/Black groups, whereas there were no differences between ethnicities in the elevated-risk

Table 1
Sample demographics.

	Elevated-risk	Low-risk Controls	Group difference
N	38	38	
Age	23.6 (SD = 3.4)	24.2 (SD = 3.4)	$t(74) = 1.09$, $p = .28$
Gender	21 female 17 male	18 female 20 male	$\chi^2(1) = 0.47$, $p = .49$
Background			$\chi^2(4) = 7.44$, $p = .11$
Caucasian	4 (10.5%)	10 (26.3%)	
African-American/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%)	
PQ-B symptoms	12.0 (SD = 3.2)	0.8 (SD = 1.0)	$t(74) = 20.76$, $p < .001$
PQ-B distress	37.61 (SD = 14.1)	1.1 (SD = 1.5)	$t(74) = 15.83$, $p < .001$
Elevated-risk categories			
Gen. rel. & moderate PQ-B	8 (21.1%)	n/a	
Gen. rel. & elevated PQ-B	7 (18.4%)	n/a	
Elevated PQ-B only	23 (60.5%)	n/a	

group. Considering ethnicity was not a significant predictor of PQ-B scores and because demographic variables did not relate to any outcome variables, no demographic variables were used as control variables in any analyses. Demographic data are presented in Table 1.

3.2. Primary analyses

3.2.1. Self-reported negative affect

The elevated-risk group had greater negative affect across all measurements compared to low-risk controls ($F(1,74) = 29.61, p < .001, \eta^2 = 0.29, 95\% \text{ CI } [0.12, 0.43]$). The comments also significantly impacted negative affect across both groups ($F(3,72) = 13.75, p < .001, \eta^2 = 0.36, 95\% \text{ CI } [0.17, 0.49]$). Relative to baseline negative affect, critical comments increased negative affect whereas praise lowered negative affect ($M_{\text{logdif}} = 0.10, SE = 0.03, p < .001; M_{\text{logdif}} = -0.09, SE = 0.02, p < .001$, respectively). There was no change from baseline in negative affect following the neutral comments. There was also a significant interaction between comments and risk group ($F(3,72) = 4.20, p = .01, \eta^2 = 0.15, 95\% \text{ CI } [0.01, 0.27]$). Compared to the low-risk group, the elevated-risk group showed significantly greater declines in negative affect relative to their baseline measurements following the praise comments ($F(1,74) = 11.98, p = .001$). There were no differences between groups in negative affect reactivity to neutral or critical comments. The effects of comments on negative affect are depicted in Fig. 1.

3.2.2. Self-reported positive affect

The two groups did not have an overall difference in positive affect ($F(1,74) = 1.38, p = .28, \eta^2 = 0.02, 95\% \text{ CI } [0.00, 0.11]$). As expected, hearing the various types of comments significantly impacted positive affect for both groups ($F(3,72) = 15.73, p = .001, \eta^2 = 0.40, 95\% \text{ CI } [0.20, 0.52]$). Compared to baseline, positive affect declined following neutral and critical comments ($M_{\text{dif}} = -3.29, SE = 0.78, p < .001; M_{\text{dif}} = -3.29, SE = 0.73, p < .001$, respectively). There was no change from baseline in positive affect following the praising comments. There was a non-significant trend toward an interaction between comments and risk group ($F(3,72) = 2.45, p = .09, \eta^2 = 0.03, 95\% \text{ CI } [0.00, 0.20]$). The elevated-risk group showed marginally greater declines in positive affect relative to their baseline compared to the control group in response to the neutral comments ($F(1,74) = 3.61, p = .06, \eta^2 = 0.05, 95\% \text{ CI } [0.00, 0.17]$). Contrary to prediction, the high- and low-risk groups did not differ with respect to how much their positive affect changed from baseline following criticism or praise. The effects of comments on positive affect are depicted in Fig. 2.

3.2.3. Heart rate response

The two groups did not have an overall difference in heart rate ($F(1,72) = 2.32, p = .13, \eta^2 = 0.03, 95\% \text{ CI } [0.00, 0.14]$). Both groups also responded to the comments with an overall reduction in heart rate from baseline ($F(3,70) = 5.75, p = .001, \eta^2 = 0.20, 95\% \text{ CI } [0.03, 0.33]$). There was, however, a significant interaction between comments and risk group on heart rate ($F(3,70) = 2.69, p = .047, \eta^2 = 0.04, 95\% \text{ CI } [0.00, 0.22]$). In line with expectations, during the critical comments the elevated-risk group had marginally greater heart rate relative to low-risk controls ($F(1,73) = 2.90, p = .09, \eta^2 = 0.04, 95\% \text{ CI } [0.00, 0.15]$).

There was also a significant interaction between time and risk group on heart rate during the critical comments ($F(2,72) = 3.42, p = .04, \eta^2 = 0.09, 95\% \text{ CI } [0.00, 0.21]$). Specifically, the two risk groups showed no difference in initial reactivity or sustained reactivity; however, the elevated-risk group showed an increase in heart rate during the recovery period ($t(36) = 2.21, p = .03, \text{Cohen's } d = 0.36, 95\% \text{ CI } [0.03, 0.69]$), whereas the low-risk controls showed a maintenance of heart rate ($t(36) = -1.31, p = .20, \text{Cohen's } d = 0.21, 95\% \text{ CI } [-0.11, 0.53]$). The elevated-risk group also showed greater reductions in heart rate from baseline during the praise comments compared to the low-risk control group ($F(1,72) = 4.26, p = .04, \eta^2 = 0.06, 95\% \text{ CI } [0.00, 0.18]$). This reduction in heart rate in the elevated-risk group was maintained at each time point and their heart rate was not different than the heart rate found in the low-risk group throughout the praise measurements (initial reactivity: $t(74) = 0.90, p = .37, \text{Cohen's } d = 0.21, 95\% \text{ CI } [-0.25, 0.67]$; sustained reactivity: $t(74) = 0.61, p = .55, \text{Cohen's } d = 0.14, 95\% \text{ CI } [-0.31, 0.60]$; recovery: $t(74) = 0.79, p = .43, \text{Cohen's } d = 0.18, 95\% \text{ CI } [-0.27, 0.64]$). The neutral comments did not affect the two risk

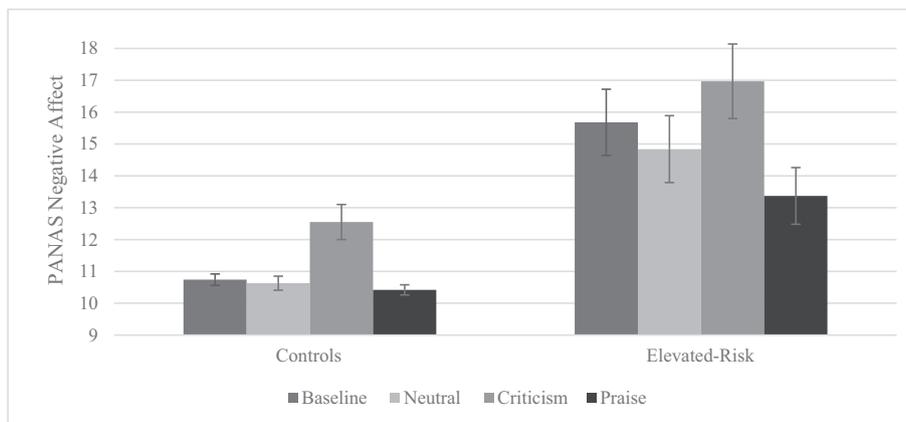


Fig. 1. Negative affect responses to emotional comments. (Error bars represent the within-group error and are equal to ± 1 standard error of the means for the between-group comparisons.)

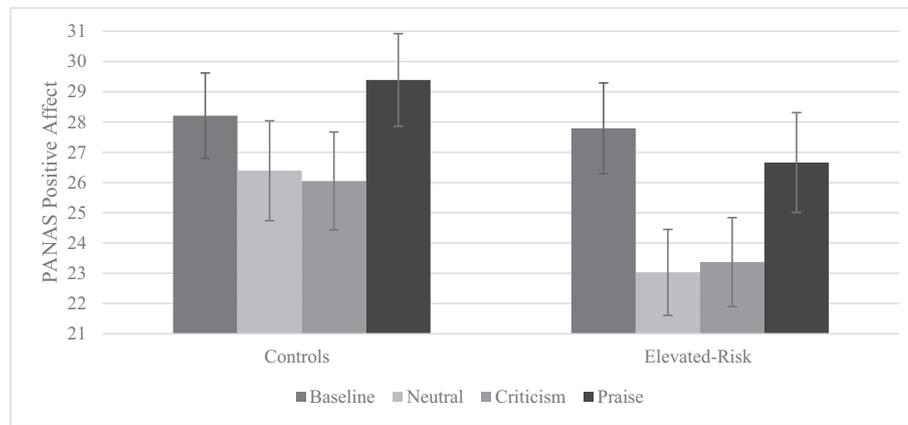


Fig. 2. Positive affect responses to emotional comments. (Error bars represent the within-group error and are equal to ± 1 standard error of the means for the between-group comparisons.)

groups' differently ($F(1,73) = 2.41, p = .13, \eta^2 = 0.03, 95\% \text{ CI } [0.00, 0.14]$). The effects of comments on heart rate are depicted in Fig. 3.

3.2.4. Heart rate variability (RSA)

The two groups did not differ in overall RSA. There were also no interactions between comments and risk group, between time and risk group, or between comment, time, and risk group on RSA (Fig. 4).

3.2.5. Skin conductance levels (SCL)

There was no overall difference in SCL between groups, nor were there any significant interactions between comments and risk group, between time and risk group, or between comment, time, and risk group for SCL (Fig. 5).

3.3. Confirmatory analyses on the relevance and valence of comments

The high- and the low-risk groups did not differ with respect to how personally relevant they rated comments overall. However, there was a significant interaction between comment type and group. The elevated-risk group rated the criticism comments as more relevant to them than the low-risk controls. There was no difference in relevance ratings between the groups for neutral or praise comments. The data comparing each group's rated relevance across comments is presented in Table 2.

In terms of valence, there was a main effect of group such that the elevated-risk group rated the comments overall as being more negative than did the low-risk control group. There was also a main effect of comment type on valence; neutral comments were rated as less positive than the praise comments and the critical comments were rated as

more negative than both the neutral and praise comments. Although we found no significant interaction between risk group and condition for valence, elevated-risk participants rated neutral comments less positively than low-risk participants did. There was no difference in valence ratings between groups for critical or praising comments. The data comparing each group's rated valence across comments is presented in Table 2.

4. Discussion

This study examined the effects of in-the-moment EE comments on self-reported affect and physiological responses of individuals at elevated risk for psychosis. Contrary to expectations, this study did not find that individuals at elevated risk for psychosis are more sensitive/reactive to the social stress of criticism. There were no differences between groups in self-reported affective or physiological reactivity to critical comments. The critical comments had the expected effect of increasing self-reported negative affect relative to baseline measurements and neutral comments. However, these changes in initial reactivity were not different between groups, indicating that elevated-risk individuals may not be more sensitive to criticism compared to low-risk controls. This contradicts some previous research, which has suggested that elevated-risk individuals are more sensitive to stressors compared to low-risk controls (e.g., Mizrahi et al., 2012; Myin-Germeys et al., 2001; Trotman et al., 2014). Considering family stress can contribute to the progression of prodromal symptoms (Schlosser et al., 2010), it appears that criticism serves as a stressor that initially affects all

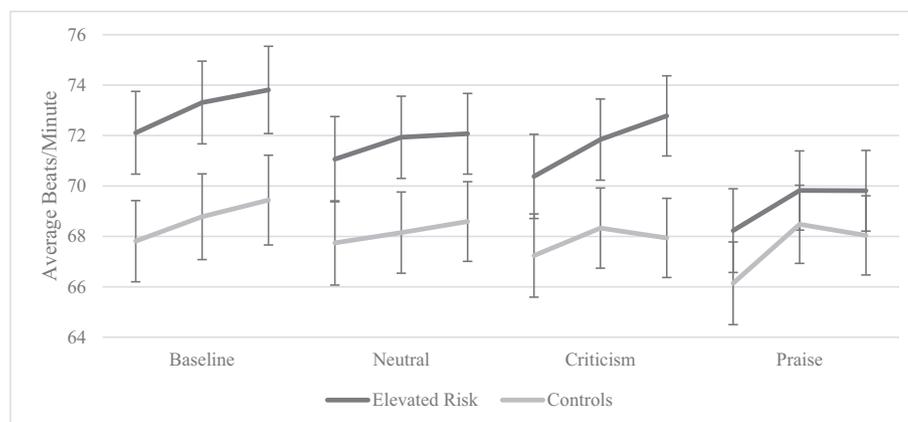


Fig. 3. Heart rate in response to emotional comments. Each measurement-set displays (from left to right) the average heart rate over the first, second, and third minute of measurement. (Error bars represent the within-group error, equal to ± 1 standard error of the mean for the between-group comparisons.)

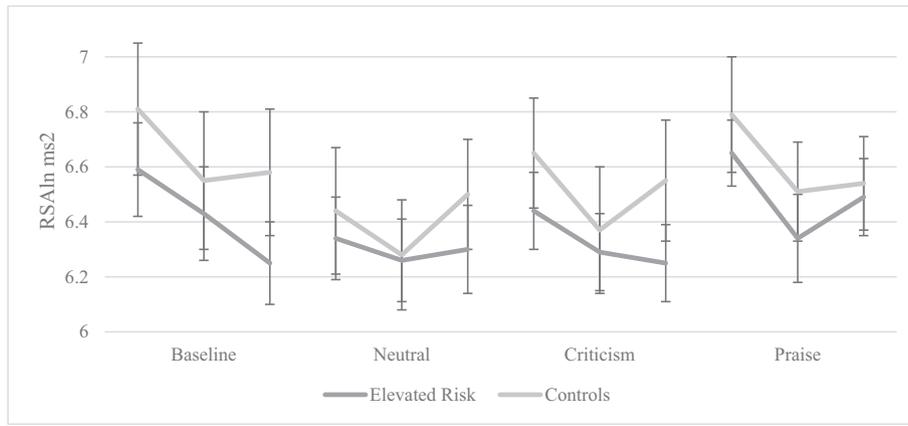


Fig. 4. Heart rate variability in response to emotional comments. Each measurement-set displays (from left to right) the average heart rate variability (RSA_{1m}^2) over the first, second, and third minute of measurement. (Error bars represent the within-group error, equal to ± 1 standard error of the mean for the between-group comparisons.)

individuals to a similar degree, but elevated symptoms among those at risk may place them closer to a threshold of clinical severity.

There was some evidence to suggest that the elevated-risk group had difficulty recovering from criticism; they showed increased heart rate activation following the exposure to criticism relative to the control group. While individuals at elevated risk for psychosis had similar affective and physiological reactivity to stress relative to low-risk controls, they appear to have difficulties regulating their physiological response (and potentially their negative affect and anxiety) following social stress. Heart rate is considered a reliable, non-invasive measure of sympathetic-parasympathetic interplay (Scheer et al., 2003; Oldehinkel et al., 2008). Some measures of physiological response (i.e., skin conductance) characterize the immediate sympathetic stress response; however, the interplay of sympathetic and parasympathetic is a somewhat downstream process that appears to be driven, at least in part, by brain systems which are critical for emotion processing, threat recognition, and social cognition more generally (e.g., Del Giudice et al., 2011; Porges, 2003). Thus, elevated-risk individuals' ability to process negative emotion and/or regulate their stress response following criticism may be impaired. There is a paucity of literature examining emotion regulation in at-risk individuals; however, some work suggests that emotion regulation is a deficit in this population (Amminger et al., 2011; Gee et al., 2012; van Rijn et al., 2011). Overall, these self-report and physiological responses have important implications for elevated-risk individuals, as they suggest that these individuals respond similarly to criticism relative to controls but have difficulties regulating their physiological stress response following stressful social interactions.

Praise had beneficial effects across multiple indices, including reducing negative affect and heart rate, as well as increasing positive affect relative to hearing neutral comments. Additionally, the elevated-risk group had greater reductions in negative affect and heart rate compared to the low-risk control group. Thus, the elevated-risk group showed greater physiological and affective benefit from praise than did the low-risk controls. These results parallel previous research, which found that positive, warm family interactions are predictive of reductions prodromal symptoms (O'Brien et al., 2006). This sensitivity to praise could be due to elevated-risk individuals' increased loneliness and reduced social networks (Robustelli et al., 2017). As a result, receipt of social rewards such as praise may be experienced as particularly rewarding for elevated-risk individuals given the relative rarity of these types of experiences. The psychological and biological mechanisms to explain the elevated-risk group's receptiveness to praise require further research. In summary, the implication from the results related to praise comments is that warm social displays are both well-received by elevated-risk individuals and are associated with affective and physiological benefits.

Contrary to expectations, neutral comments led to reductions in positive affect, and there was some evidence that this relationship was stronger for the elevated-risk group. Additionally, based on the participants' reports of the valence of the comments, elevated-risk individuals felt that the neutral comments were less positive than did the low-risk controls. Previous work on neutral stimuli has previously found that individuals with schizophrenia perceive and interpret neutral stimuli more negatively, experience more negative emotions and show amygdala hyperactivation when presented with neutral stimuli compared

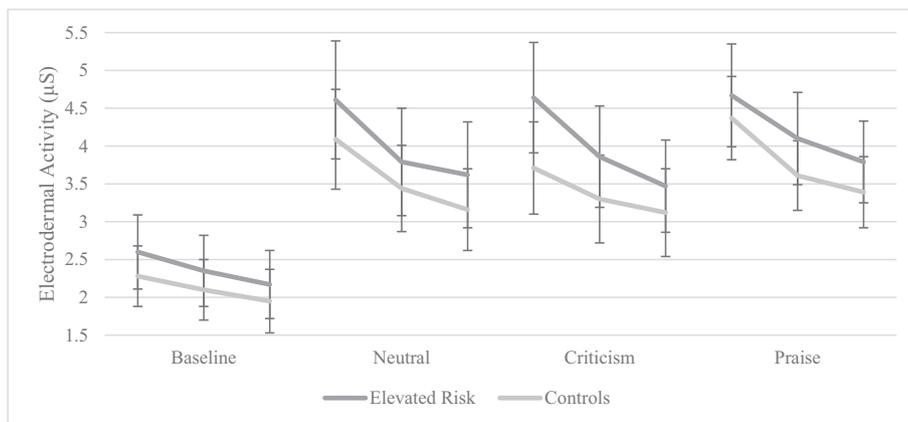


Fig. 5. Skin conductance response to emotional comments. Each measurement-set displays (from left to right) the average electrodermal activity (EDA; as measured by μS) over the first, second, and third minute of measurement. (Error bars represent the within-group error, equal to ± 1 standard error of the mean for the between-group comparisons.)

Table 2
Self-reported valence and relevance of comments.

	Elevated-risk	Controls	Group difference
Relevance			
Neutral	3.3 (<i>SD</i> = 2.0)	3.9 (<i>SD</i> = 1.4)	$F(1,74) = 1.74, p = .19$; Cohen's $d = 0.31$
Criticism**	5.9 (<i>SD</i> = 2.4)	4.3 (<i>SD</i> = 1.9)	$F(1,74) = 10.50, p = .002$; Cohen's $d = 0.75$
Praise	6.5 (<i>SD</i> = 2.1)	6.8 (<i>SD</i> = 1.3)	$F(1,74) = 0.54, p = .47$; Cohen's $d = 0.17$
Valence			
Neutral**	4.7 (<i>SD</i> = 1.2)	4.0 (<i>SD</i> = 1.1)	$F(1,74) = 7.50, p = .008$; Cohen's $d = 0.64$
Criticism	8.0 (<i>SD</i> = 0.9)	7.7 (<i>SD</i> = 1.5)	$F(1,74) = 1.64, p = .21$; Cohen's $d = 0.30$
Praise	1.6 (<i>SD</i> = 1.0)	1.5 (<i>SD</i> = 0.6)	$F(1,74) = 0.65, p = .42$; Cohen's $d = 0.19$

** $p < .01$.

to controls (e.g., Cohen and Minor, 2008; Eack et al., 2009; Seiferth et al., 2008). In sum, these findings on responses to neutral stimuli suggest that individuals with psychosis, or at risk for psychosis, may not perceive neutral stimuli as completely neutral. The reasons for this warrant additional exploration.

This study had several limitations that should be noted. First, the participants were screened for eligibility 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). Although the PQ-B has strong concordance with gold-standard psychosis-risk measurements, the level of risk in this sample may be somewhat reduced relative to studies using more formal assessments of psychosis-risk. Additionally, there was some evidence to suggest that the PQ-B may have differential validity based on ethnicity (specifically Hispanics). Thus, more research needs to be done to determine whether the PQ-B accurately screens Hispanic individuals for elevated risk of psychosis. The standardized comments represent another potential limitation. Standardized comments have the advantage of being consistent across all participants. However, such comments are unlikely to be completely relevant and, thus, emotionally potent to each participant. Finally, while the study had a priori hypotheses for each set of analyses, many comparisons between groups were made. Thus, the lack of correction of multiple comparisons allowed for more power in the statistical findings of this report. Considering the number of analyses relative to the strength of the findings, the results should be interpreted with caution in the absence of replication.

Future research should discern if elevated-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. Also required is a better understanding of the emotion regulation strategies (and potential deficiencies) as well as coping methods characteristic of elevated-risk individuals. Another avenue of future inquiry involves examining the effects of praise on elevated-risk populations. O'Brien et al. (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. Testing the effects of treatments that incorporate praise, warmth, and other positive social interactions may help us identify and increase protective factors for individuals at elevated risk for psychosis.

Conflicts of interest

The authors have no conflicts of interest to declare.

Contributors

Marc J. Weintraub developed the study concept. All authors contributed to the study design. Data collection was done by Marc J. Weintraub and William J. Villano. Marc J. Weintraub and Travis C. Evans performed the data analysis and interpretation. Marc J. Weintraub drafted the paper, and Amy Weisman de Mamani, Jill M. Hooley, Kiara R. Timpano and Zachary B. Millman provided critical revisions. All authors approved the final version of the paper for submission.

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