Revictimization: A Multi-Method Approach to Understanding Risk Detection

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Revictimization: A Multi-method Approach to Understanding Risk Detection

A Dissertation

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by

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ABSTRACT

Research has focused on identifying mechanisms that increase revictimization risk in women, such as risk detection (i.e., ability to identify threat). While risk detection deficits have been linked to revictimization in college samples, individual differences that might predict risk detection deficiencies remain unclear. In this study, 94 women recruited from the community performed a risk detection task by listening to an audiotape of a risky dating situation. We obtained parasympathetic (e.g., vagal tone) and sympathetic (e.g., pre-ejection period) activation, heart rate, and self-reported emotional responding while participants completed the task. We also assessed participants’ trauma histories and relevant symptoms. Results suggested that community women with less self-reported reactivity detect risk faster than women with more self-reported reactivity. Women who detected risk faster also displayed a discrepancy between sympathetic versus self-reported reactivity. For women who detected risk at a slow rate, an interaction between trauma and symptom levels significantly predicted risk detection latency. Implications of reactivity associated with risk detection abilities are discussed.
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Chapter One

Introduction

Background

Fifty-nine percent of women with victimization histories report sexual assaults in both childhood and adulthood, a phenomenon that has been referred to as revictimization (Cloitre, 1998; Wyatt, Guthrie, & Notgrass, 1992). In fact, women with history of childhood sexual abuse (CSA) are 2.5 to 3 times more likely than women without CSA to be sexually assaulted in adulthood, with a meta-analysis showing an overall effect size of 0.59 (e.g., Cloitre, Tardiff, Marzuk, Leon, & Potera, 1996; Roodman & Clum, 2001; Urquiza & Goodlin-Jones, 1994; Wyatt et al., 1992). The increased risk for revictimization has important public health implications. Revictimization is associated with more severe physical, psychological, and social problems than single victimizations (e.g., Follette, Polusny, Bechtle, & Naugle, 1996; Marx, Heidt, & Gold, 2005). While researchers know relatively little about the mechanisms underlying revictimization risk, risk detection (the ability to detect cues that may indicate danger in various situations) has received increasing support as a potential mediator (see Marx, Heidt, & Gold, 2005). In one experimental methodology, participants listen to an audiotaped situation involving a man and a woman that culminates in sexual assault. Participants are asked to indicate when the man has gone too far with the woman; the latency of the button-press has been viewed as a measure of risk detection ability (Marx & Gross, 1995). Sexually revictimized college women take
significantly longer to indicate when the man has become inappropriate than singly- or non-victimized college women (e.g., Marx, Calhoun, Wilson, & Meyerson, 2001; Wilson et al., 1999). Furthermore, a larger percentage of revictimized college women than their singly- or non-victimized peers waited until the man used verbal threats and physical force before indicating that the woman was in danger (Wilson et al., 1999).

One prospective study found that college women with poorer risk detection abilities reported rape episodes at follow-up, despite having participated in a sexual assault intervention program (Marx et al., 2001). Using a written-format of a rape scenario, another prospective study found that delayed risk perception substantially increased risk of subsequent rape for college women regardless of previous rape histories (Messman-Moore & Brown, 2006).

Additional findings suggest that college women with abuse histories may view threat cues merely as indications of discomfort whereas their peers without abuse histories view those same cues as signals of danger. While viewing a videotape depicting a potential sexual assault scenario, college participants were asked to write down cues that made them feel uncomfortable (Breitenbecher, 1999). The number of identified threat cues did not relate to sexual victimization history or to reported revictimization at 5 months follow-up. Meadows, Jaycox, Webb, & Foa (1996) also asked college-aged participants to indicate when they felt discomfort and when they would leave the situation (as cited in Marx et al., 2001). Interestingly, while groups did not differ on discomfort ratings, the sexually victimized group reported they would leave the situation at a significantly later time-point than the non-sexually victimized group. These findings have been replicated subsequently with college samples (Meadows et al., 1997 as cited in Wilson et al., 1999; Messman-
Moore and Brown, 2006). These findings suggest that risk detection differences in college women are associated with risk for additional sexual assaults; however, we know little about these abilities in community and older samples of women.

Based on findings from college samples, disruptions in risk detection abilities appear to offer promising insights into processes associated with revictimization; however, we are aware of no studies to date that have examined risk detection abilities in community samples. For several reasons, extending findings from college samples to community samples is important. First, college samples are typically comprised of predominantly Caucasian, educated, young women (approximately ages 18 to 22) who are middle- to upper-class (see Messman-Moore and Long, 2003). The degree to which findings on risk detection generalize from college participants to more racially, educationally, or socioeconomically diverse community samples remains an empirical question. Further, we do not know the extent to which risk detection abilities are disrupted among women living in high-crime neighborhoods who are also at increased risk for gang or community violence. Second, college participants tend to report lower symptom levels, psychological distress, and/or impairment than community women (see Messman-Moore and Long, 2003). To the extent that symptoms interfere with risk detection, college samples may not adequately tap the severe end of symptom spectrums; therefore, our understanding of the relationship between severe psychological distress and disruptions in risk detection may be limited in college samples. Given the limitations of college samples, a primary aim of the current study was to extend previous findings on risk detection from college women to a heterogeneous community sample. The present study tested a sample of community
women between the ages of 18 and 40 with varied educational and demographic backgrounds who reported a range of trauma experiences.

A second area of research associated with risk detection has focused on physiological correlates to understand individual differences that predict risk detection deficiencies. In a sample of undergraduate women, PTSD arousal was found to moderate revictimization risk, perhaps due to difficulty recognizing or responding to danger cues after being chronically hypervigilant (Risser, Hetzel-Riggin, Thomsen, & McCanne, 2006). In a sample of multiply-victimized college women, PTSD arousal symptoms were positively correlated with risk detection using the audiotape paradigm (Wilson et al., 1999). However, when examined across groups of non-victimized, singly-victimized, and multiply-victimized women in the same study, PTSD arousal symptoms were no longer associated with risk detection. Marx & Soler-Baillo (2005) also failed to find a relationship between PTSD arousal symptoms and risk detection in a sample of college women with sexual assault history.

Dissociation was also found to relate to suppression of autonomic physiological responses in adolescents (Koopman et al., 2004) as well as to risk detection deficiencies in college-aged women (Sandberg, Lynn, & Matorin, 2001), suggesting that hypo-reactivity may contribute to risk detection problems. College women with CSA history, as compared to women without CSA, exhibited significantly decreased heart rate reactivity while at the same time reporting more arousal and feeling more unpleasant when listening to audiotapes of a potential date rape situation (Soler-Baillo et al., 2005). The same group of women with CSA history was significantly slower in determining when the man in the audiotape scenario had ‘gone too far’ than women without CSA history. Another sample of college
women who acknowledge their status as sexual assault victims also self-reported more arousal when listening to the same audiotape as compared to non-victims (Marx & Soler-Baillo, 2005). These results point to an incongruity between physiological reactivity and self-reported reactivity (where risk detection is negatively associated with self-reported reactivity yet positively associated with physiological reactivity) in college-aged victims of sexual assault in response to a date rape scenario.

One explanation for the inconsistent relationship between reactivity and risk detection may be that objective physiological reactivity affects risk detection differently depending on the individual’s subjective experience of reactivity. While many victims of abuse exhibit deviations from normal physiological functioning, neither hypo-reactivity nor hyper-reactivity appears to contribute definitively to deficiencies in risk detection. Perhaps women at risk for revictimization recognize threat at the same time as other women. However, due to a disconnect between their physiological reactivity and their subjective experience, they do not activate their defensive system to escape the dangerous situation. When a woman cannot read her own reactions accurately or expends energy regulating her physiological responses, she may not have sufficient resources to deal with dangerous situations.

Many trauma-related research studies obtain heart rate as a measure of cardiovascular reactivity because of its sensitivity to the arousal component of emotional reactivity more so than other physiological indices (see Soler-Baillo, Marx, and Sloan, 2005). However, as the sole index of cardiovascular reactivity, heart rate has limited ability to discriminate the underlying autonomic processes that result in these changes (Berntson, Cacioppo, & Quigley, 1991). There are many underlying factors that influence
global cardiovascular reactivity, such as autonomic nervous system reactivity (ANS). Autonomic reactivity, leading to greater heart rate, is comprised of parasympathetic withdrawal, sympathetic activation, or a combination of the two (Berntson et al., 1991). Therefore, obtaining measures of sympathetic (i.e., pre-ejection period) and parasympathetic reactivity (i.e., respiratory sinus arrhythmia) in addition to heart rate reactivity provides a more comprehensive understanding of the physiological correlates of risk detection than heart rate reactivity alone.

Although we obtained multiple physiological indices to provide a comprehensive measure of cardiovascular reactivity, we did not expect to find main effects of victimization on any of the three measures of physiological reactivity. Rather, we expected heterogeneity of responses across our physiological indices, consistent with the mixed post-trauma reactivity patterns observed in previous research. For example, some women exhibit hyper-reactivity post-trauma (e.g., Cloitre, 1998; Perry et al., 1995; van der Kolk, 1989), while others exhibit hypo-reactivity (e.g., Cloitre, Miranda, Stovall-McClough, & Han 2005; Koopman et al., 2004; Marx et al., 2005; Polusny, Rosenthal, Aban, & Follette, 2004). Thus, the focus of this study was on the discrepancy between physiological and self-reported subjective reactivity rather than main effects within physiological reactivity. We expected this discrepancy to be present across physiological indices.

**Current Study**

The current study extended previous studies of risk detection to examine whether survivors of multiple sexual victimizations in a heterogeneous community sample have longer response latencies than non-victims or survivors of single-victimizations. Second, this study examined multiple measures of reactivity (physiological and self-report) as
potential contributors to risk detection. The influence of physiological reactivity on women’s risk detection abilities may vary depending on the level of women’s self-reported reactivity. Women whose self-reported and physiological reactivity do no match were expected to have longer response latencies on the risk detection task than individuals with comparable levels of physiological and self-reported reactivity. Third, the current study expanded upon traditional physiological measures that focused only on heart rate, or cardiovascular reactivity, to also include assessments of autonomic nervous system (ANS) reactivity. ANS reactivity includes indices of pre-ejection period (PEP), which measures sympathetic activation, and vagal tone - respiratory sinus arrhythmia (RSA) - which measures parasympathetic withdrawal. Thus, the study more comprehensively explored a range of physiological correlates that might contribute to differing risk detection abilities.
Chapter Two

Method

Participants

Ninety-four women between the ages of 18 and 40 (Age $M = 30.70; SD = 6.24$) were recruited in the Denver metro area through flyers placed at community agencies (e.g., public housing/shelters, mental health clinics, social services agencies, police department, colleges) and web-based list-serves or bulletins. Of 93 women who recalled the source of where they saw information on the study, 53.8% responded to flyers at community agencies, 36.6% responded to web-based postings, and 9.7% received information from friends or family members. Participants were screened on gender and age but not other demographic variables. Participants were accepted based on self-reports of having experienced childhood physical and/or sexual abuse (occurring before age of 14) or a recent interpersonal crime. Participants were excluded from the study if they made a suicide attempt and/or were hospitalized for psychiatric reasons in the previous six months. Inclusion and exclusion criteria were assessed during initial phone contact with potential participants. Participants received $25 for participation.

Seven participants did not provide racial information; the remaining women reported the following racial backgrounds: 66% Caucasian, 18% African-American, 3% Asian-American, 1% Native-American/Alaska Native, and 12% other race or bi/multi-racial. Of the 75 women who provided ethnicity information, 25.3% identified as
Hispanic/Latina. Of the 93 women who provided education information, 19.4% completed some grade school up to part of high school, 17.2% obtained a high school diploma, 40.9% completed partial college or specialized training, 12.9% obtained a college degree, and 9.7% had some graduate or professional training. 89 women provided the following income information: 40.71% <$10,000; 12.3% $10,000-$20,000; 14.8% $20,000-$30,000; 9.9% $30,000-$40,000; 6.2% $40,000-$50,000; 16.0% >$50,000. See Table 1 for demographic information by Victimization Status Groups.

Procedure

The project coordinator or trained research assistants screened women who called the lab. Callers who met inclusion criteria were invited to schedule an appointment at the research offices. Upon arrival, consent information was carefully explained to participants; participants then reviewed and signed consent forms. The experimenter then administered a consent quiz to insure understanding of consent information. All participants passed the consent quiz.

After consenting, participants completed non trauma-relevant surveys (see Materials section) and lab tasks that were part of a larger study (only tasks analyzed for this study are described here). After finishing these tasks, participants were seated in a comfortable chair in front of a computer monitor; physiological sensors were attached. A PC-based computer in an adjacent control room monitored all physiological data. Participants were then instructed on the tasks they were to complete while attached to the sensors (see Materials section for details on the instructions). The experimenter then left the room, began recording physiological activity, and initiated the visual and audio stimuli (see Materials section for details on contents of stimuli). Participants first viewed neutral
photographs (210 seconds) to establish baseline physiological activation and to bring them to a comparable and relatively neutral mood state. Participants then listened to one ‘neutral’ audiotape (190 seconds), one ‘target’ audiotape (length dependent on response latency), and one ‘recovery’ audiotape (180 seconds). After the recovery audiotape concluded, sensors were removed and participants escorted into another room to finish the testing session. The experimenter then conducted the trauma history interview. Trauma-relevant symptom questionnaires were completed last to minimize any influence the questionnaires might have had on participants’ emotional reactivity during the audiotapes. After finishing all tasks, all participants were fully debriefed and compensated monetarily for their time.

**Measures**

*Trauma History.* Sexual victimization history was assessed by a two-stage interview strategy used in the National Crime Victims Survey (see Fisher & Cullen, 2000). In the first stage, each woman is asked a series of behaviorally specific screening questions that describe sexual victimization, ranging from threatened sexual contact to completed rape. These screening questions cue participants to relevant incidents and prompt them to remember any victimization that may have occurred in their lifetime (Fisher & Cullen, 2000). Participants who answer “yes” to any of the first series of screening questions are then be asked a series of detailed questions about the incidents in the second stage of an in-depth incident report; questions asked are about the characteristics of the victim, offenders, and incidents. The information gathered in the second stage, not the first set of screening questions, is used to categorize victimization. This two-step process builds upon previous research, which typically relies exclusively on screening questions to measure whether
victimization occurred. Fisher and Cullen (2000) suggest that this two-step interview methodology allows for a more accurate assessment of sexual victimization.

Screening questions for both sexual and physical victimization events were based on location (e.g., home, work, on the street or in a parking lot), method of attack (e.g., with a weapon; something thrown; grabbing, punching, or choking; rape, attempted rape, or other type of sexual attack), relationship with perpetrator (e.g., someone at work or school, neighbor or friend, relative or family member). One set of screening questions focused specifically on sexual victimization (have you been forced or coerced to engage in unwanted sexual activity by someone you didn’t know before, a casual acquaintance, or someone you know well); all other screening questions did not distinguish between sexual or physical victimization – information gathered during the second stage of the incident report was used to categorize sexual versus physical victimizations. A total of 21 screening questions were asked; participants were able to report multiple events for each screening question.

Definitions of child and adult sexual victimization as well as revictimization were based on previous studies that used the risk detection paradigm (e.g., Wilson et al., 2001; Marx & Soler-Baillo, 2005; Soler-Baillo et al., 2005). Specifically, childhood sexual victimization was defined as any attempted or completed sexual contact (e.g., exposure, fondling, or penetration) prior to the age of 14. Adult sexual victimization was defined as unwanted attempted or completed sexual contact by use of drugs, coercion, threat, or actual force at or after the age of 14. Childhood physical victimization was defined as physical assault or abuse with or without an object by a caregiver or adult figure before age 14. Adult physical victimization was defined as physical assault or abuse by a romantic
partner, caregiver, or family member after age 14. In addition to assessing the number of victimization events experienced, information including age at onset, chronicity, frequency, and perpetrator relationship was obtained.

Based on their interviews, women were assigned to one of three groups. The no-sexual victimization (NV) group consisted of women who reported no sexual victimizations; the single adult victimization (SV) group included women who reported one sexual victimization in adulthood and no childhood events; the multiple lifetime victimization (MV) group included women who reported either a) childhood sexual abuse and subsequent adult sexual victimization or b) multiple sexual victimization events in adulthood. Women who reported childhood sexual victimization only with no subsequent adult victimization events \( (n = 17) \) were not included in the replication analyses because previous studies also excluded this group of victimization status (e.g., Wilson et al., 2001; Marx & Soler-Baillo, 2005; Soler-Baillo et al., 2005). Additionally, if we categorized victim status groups by counting both sexual and physical victimizations, 77 women would fall into the MV group with only 2 women in the SV group (no women in the NV group). Thus, due to the unequal cell sizes, we did not include physical victimizations in the victim status groups. However, childhood victimizations as well as physical victimizations are counted in the continuous Trauma Composite variable, described below.

Given the heterogeneity in and complexity of the women’s trauma histories in this community sample, we examined additional variables to capture the range of types and chronicity of trauma. We created: 1) sum of times sexual and physical victimizations occurred; 2) sum of different types of interpersonal trauma experienced (e.g., sexual abuse, physical abuse, verbal abuse, witnessing abuse, threats of violence, sexual harassment,
forced sexual activity, and kidnapping); 3) closeness of victim-perpetrator relationship from least close to most close (0 = stranger, 1 = outside family, 2 = relative, 3 = parent figure or romantic partner; when multiple perpetrators are reported, the closest relationship was coded); and 4) frequency (1 = once, 2 = few times over short period, 3 = many times chronically; when multiple events are reported, the most frequent event was counted).  We conducted a principal-components analysis (PCA) on these four trauma variables.  All four trauma variables loaded on to a single factor (loadings > .70) when using both the oblique oblimin and the orthogonal varimax rotation methods.  Since the factor analysis demonstrated that the measures tapped an underlying trauma construct, we transformed all four trauma variables into z-scores and calculated a mean to create a continuous Trauma Composite score.  We used the Trauma Composite variable in our exploratory analyses.

Symptom Measures. PTSD symptomatology was assessed with the Posttraumatic Stress Diagnostic Scale (PDS; Foa, Cashman, Jaycox, & Perry, 1997).  The PDS is a 49-item measure based on the DSM-IV criteria for PTSD.  This measure is unique among PTSD symptom self-reports in that Criterion A (traumatic event and response to the event) is assessed.  Coefficient alpha for this measure was .93 in this community sample.

Dissociative symptomatology was assessed using the Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986), a widely-used 28 item self-report measure.  The DES has been shown to have good validity and reliability and is scored by taking an average across items.  Coefficient alpha for this measure was .93 in this community sample.

Depression symptomatology was assessed with the Beck Depression Inventory – 2 (BDI-II; Beck, Steer, Ball, Ranieri, 1996), which is among the most widely used self-report measures of depression with demonstrated validity and reliability.  This 21-item measure
assesses depression symptoms based on DSM-IV criteria. Sample items range from choices of “I do not feel sad” to “I am so sad or unhappy that I can’t stand it”. Coefficient alpha for this measure was .90 in this community sample.

Given that all symptom measures were relevant to post-trauma distress, we conducted a principal-components analysis (PCA) with the three symptom variables. All three symptom variables loaded onto a single factor (loadings > .68) with the oblique oblimin and the orthogonal varimax rotations. Therefore, we created a Symptom Composite variable for eighty-eight of the women (one woman did not complete symptom measures; two women were missing data for the dissociation measure) by transforming the symptom variables into z-scores and calculating a mean. We include the Symptom Composite in our exploratory analyses.

**Visual and Audio Stimuli.** Neutrally valenced pictures from the *International Affective Pictures System* (IAPS; Lang, Bradley, & Cuthbert, 1999) were used for the visual stimuli during the initial neutral baseline. Seventy pictures were presented for three seconds each, totaling 210 seconds. The 190 second ‘neutral’ audiotape depicted dialogue between a man and a woman discussing the rules of the game “Go Fish” and then playing a game together. The dialogue was created as a comparable audio stimuli to the target audiotape in length and interpersonal exchange but containing valence-neutral tone and wording. The ‘recovery’ audiotape consisted of another 180-second dialogue between a man and a woman discussing 2 comedic movies.

The ‘target’ audiotape consisted of The Response Latency Measure (Marx & Gross, 1995), which assesses participants’ detection of danger cues during a sexual scenario. Participants listen to a portrayal of a man and woman in a sexual encounter that results in
date rape. Participants are asked to press a button when they thought the man had gone too far. While listening to the audiotapes, a prompt was present at all times on the computer screen instructing participants to “press the left button if you feel that the man has gone too far”. The potential acquaintance rape audiotape was stopped as soon as participants pressed the button to indicate that the man has gone too far. The vignette contains strong inhibiting and disinhibiting cues for sexual contact that increase in intensity as the tape progresses. The physical intimacy in the audiotape is demonstrated through dialogue and breathing and kissing sounds. The man then uses verbal persuasion, argument, threats, and then physical force to obtain intercourse. The woman responds with verbal refusals and resistance that increase in intensity. The audiotape consists of 6 distinct segments: mutual interaction (0-74s), polite refusals from the woman (75-97s), verbal refusals from the woman and apologies by the man (98-136s), continued verbal pressure from the man and refusals from the woman (137-179s), verbal threats and adamant refusals (180-276s), and forced sex (277-325s) (Marx, Gross, & Adams, 1999). Risk detection was operationalized as the latency of the button-press.

As a manipulation check, participants rated how realistic the interaction was on a scale from 0 (completely unrealistic) to 10 (completely realistic) after each of the three audiotapes. One-sample t-test comparing realistic ratings to a neutral point on the rating scale (5) indicated that participants rated all audiotapes as realistic (Neutral $M = 6.61$, $SD = 2.22$, $t(86) = 6.75$, $p < .01$; Target $M = 7.09$, $SD = 2.00$, $t(85) = 9.69$, $p < .01$; Recovery $M = 7.47$, $SD = 1.77$, $t(86) = 13.02$, $p < .01$). Participants also rated the emotional valence of each audiotape on a scale from 1 (extremely negative) to 9 (extremely positive). The emotional valence ratings corresponded to the contents of each audiotape such that the
neutral and recovery audiotapes were rated as significantly more positive than the neutral point (5) while the target audiotape was rated as significantly more negative than the neutral point (Neutral $M = 6.07$, $SD = 1.83$, $[t(86) = 5.45, p < .01]$; Target $M = 2.66$, $SD = 1.38$, $[t(85) = -15.74, p < .01]$; Recovery $M = 6.69$, $SD = 1.97$, $[t(86) = 7.99, p < .01]$).

Measures of Emotional Responding. During the experimental session, physiological channels collected data on heart rate, pre-ejection period, respiratory sinus arrhythmia as measures of central ANS responding. Data were sampled continuously at 400 Hz using laboratory software. Later, customized analysis software (Wilhelm, Grossman & Roth, 1999) was used to reduce physiological data, control for artifacts, and ensemble-average data for the neutral picture and audiotape segments. The physiological data were ensemble averaged within time-windows comprising each visual or audio stimuli, resulting in averages for each of the neutral picture, neutral audiotape, target audiotape, and recovery audiotape segments.

A MindWare 200D impedance cardiograph was used to measure electrocardiogram (ECG), basal thoracic impedance ($Z_0$), and the first derivative of the impedance signal ($dZ/dt$) using disposable spot electrodes. Disposable spot electrodes were placed on participants’ right clavicle, left rib, and right rib to obtain electrocardiograph (ECG) data. Two current electrodes were placed over the fourth cervical vertebra and the ninth thoracic vertebra, while two recording electrodes were placed 4 cm above the clavicle and over the sternum at the fourth rib to obtain impedance data. A 4-mA AC current at 100 kHz was passed through the two current electrodes, and $Z_0$ and $dZ/dt$ was recorded from the two recording electrodes. A respirometer belt was also used, placed above the umbilicus and below the diaphragm, to monitor respiratory rate.

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Heart rate (HR) was calculated from interbeat intervals (R-R intervals), measured in milliseconds, in the electrocardiogram. Pre-ejection period (PEP) was quantified as the time interval in milliseconds from the onset of the ECG Q-wave to the B-point of the $dZ/dt$ wave. To obtain vagal tone (respiratory sinus arrhythmia; RSA), the R-R interbeat intervals from the ECG was converted into a time series of instantaneous R-R intervals with a resolution of 4 Hz. RSA was quantified as the magnitude of the transfer function relating respiratory rate interval oscillations to lung volume oscillations (resampled to 4 Hz) at the peak respiratory frequency (Saul et al., 1991), thus adjusting RSA for tidal volume changes. The natural log of this variance was calculated and RSA is reported in units of $\ln(\text{ms})^2$. The peak respiratory frequency was the greatest local maximum in the lung volume power spectral density. Spectral coherence at this frequency was required to be at least 0.5. Epochs with peak respiratory frequency below the 0.5 Hz band were excluded. RSA and PEP scores were obtained for each audiotape segment. Smaller RSA scores indicate greater parasympathetic withdrawal; smaller PEP scores (i.e., shorter Q-B lags) reflect greater sympathetic activation.

Self-reported emotional responding was obtained using a continuous rating dial. The rating dial consisted of a turning knob that controlled a series of lights along a scale underneath the knob (similar to that used by Gottman & Levenson, 1985). The label “none” was placed at the left end-point of the scale and the label “all” was placed at the right end-point of the scale. The knob was attached to a potentiometer in a voltage-dividing circuit that was monitored by a computer to enable determination of the light position at a sample rate of 200 Hz. Participants were instructed that turning the knob changed the amount of lights being lit on the scale, which in turn indicated the amount of
feelings they were experiencing at each moment. They were asked to continuously turn the knob so that the lights continuously reflected *how much* they were feeling at each moment while listening to the audiotapes. They were instructed not to pay attention to the specific feeling, but instead to report on how much of a feeling they were experiencing. While listening to the audiotapes, a prompt was present at all times on the computer screen instructing participants to “adjust the dial with your right hand as often as necessary to reflect how much you feel at the moment”.

Reactivity scores. PEP and RSA were reverse scored. The mean reverse-scored RSA for the neutral audiotape was subtracted from the mean reverse-scored RSA for the target audiotape to create the RSA reactivity score. The PEP reactivity score was obtained with the same computations. The self-report reactivity score was assessed by the rating dial, quantified as the maximum rating dial output. One maximum score was obtained for the neutral picture segment and one for each of the audiotapes. The maximum score for the neutral audiotape was subtracted from the maximum score for the target audiotape to create a self-report reactivity score. In order to examine whether the influence of physiological reactivity on risk detection abilities varies by self-reported reactivity, an interaction term was calculated for each physiological measure by multiplying the physiological reactivity z-score with the self-reported reactivity z-score.
Chapter Three

Results

Preliminary Examination of Response Latency

Response latency data for 5 women were not obtained due to equipment malfunction. Data for the remaining 89 women were included in the final analyses. Inspection of response latencies indicated a non-normal, multi-modal distribution (see Figure 1). The breaks in the distribution corresponded to specific segments of the audiotape dialogue. Although previous studies using the audiotape identified six segments based on level of refusal and resistance (see Marx & Gross, 1995), the audiotape can also be divided into four broader segments of dialogue. Each segment begins with verbal appeals and threat that escalate negatively until the characters reach a temporary resolution. A period of kissing without dialogue follows each resolution until the next dialogue segment begins. The modes for each segment indicate that the majority of participants pressed the button during the segment when the female character resisted the sexual pressure of the male character.

Because we could not analyze the non-normal reaction time data continuously, we created two new variables for analysis. First, women were assigned to Segment Groups that corresponded to the four segments of the dialogue: Segment Group 1: reaction time <120 seconds, n = 38; Segment Group 2: 120-190 seconds, n = 21; Segment Group 3: 190-260 seconds, n = 18; Segment Group 4: >260 seconds, n = 12. We used this categorical variable to test a priori hypotheses about button press latency. Second, we
looked at continuous reaction time data within each segment group (Segment Group 1 $M = 83.52$; $SD = 16.76$; Segment Group 2 $M = 140.35$; $SD = 9.46$; Segment Group 3 $M = 207.16$; $SD = 11.66$; Segment Group 4 $M = 299.13$; $SD = 16.34$). Notably, distributions within group were normally distributed. We conducted exploratory linear regression analyses to examine additional predictors (e.g., trauma, symptom, and reactivity variables) of continuous response latencies within each Segment Group.

**Descriptive Statistics: Predictor Variables**

*Trauma Variables.* 10 women reported no sexual victimizations (NV group), 15 women reported one sexual victimization in adulthood and no childhood events (SV group), and 47 women reported multiple lifetime victimizations (MV group) for a total of 72 women included in analyses involving Sexual Victimization Status groups. As part of the Trauma Composite variable, participants reported an average of 2.2 ($SD = 1.8$; Range = 0 – 10) lifetime sexual victimizations and an average of 1.7 ($SD = 1.6$; Range = 0 – 10) lifetime physical victimizations. Participants also reported having experienced an average of 2.70 ($SD = 1.15$; Range = 1 – 6) different types of interpersonal trauma events (e.g., sexual abuse, physical abuse, verbal abuse, witnessing abuse, threats of violence, sexual harassment, forced sexual activity, and kidnapping).

To check the validity of the Trauma Composite and Sexual Victimization Status grouping, we examined links between the trauma variables and Symptom Composite. Consistent with research demonstrating that multiple-victimizations are consistently associated with more severe symptomatology than single-victimizations (e.g., Follette et al., 1996; Marx et al., 2005), one-way ANOVA’s indicated that Sexual Victimization Status Groups significantly differed on the Symptom Composite (see Table 2 for mean
Symptom Composite scores by Sexual Victimization status groups). Post-hoc tests showed that the MV group exhibited significantly greater Symptom Composite scores than the SV or the NV group (the SV and NV groups were not significantly different from each other). Similarly, the Trauma Composite was also significantly positively correlated with the Symptom Composite ($r = .29, p < .01$).

**Emotional Responding Variables.** PEP data for three participants were excluded due to excessive data artifacts. One additional participant was excluded due to excessive movement during physiological data collection. Mean emotional responding scores during the neutral audiotape were 101.75 ms ($SD = 21.91$; Range = 40.00-140.00) for PEP, 6.78 ln(ms)$^2$ ($SD = 1.60$; Range = 1.43 – 11.01) for RSA, 80.05 ms ($SD = 12.77$; Range = 56.25 – 118.62, and 2.26 volts ($SD = 1.39$; Range = .01 – 5.27) for self-reports. Mean emotional responding scores during the target audiotape were 100.99 ms ($SD = 22.45$; Range = 40.00-141.00) for PEP, 6.86 ln(ms)$^2$ ($SD = 1.60$; Range = 1.79 – 11.01) for RSA, 79.60 ms ($SD = 12.99$; Range = 57.20 – 125.34, and 3.71 volts ($SD = 1.29$; Range = .28 – 5.45) for self-reports. To test for group differences in baseline functioning, we compared the emotional responding scores for the neutral audiotape across Sexual Victimization Status (see Table 2). Sexual Victimization Status did not significantly differ from one another.

**Group Differences Between and Correlations Among Predictor Variables.**

Prior to inferential analyses, we checked for group differences and correlations among predictor variables. Mean physiological and self-reported reactivity scores by Sexual Victimization Status groups are reported in Table 2. One-way ANOVA’s indicated no Sexual Victimization Status group differences on any reactivity variables. See Table 3 for zero-order correlations among reactivity variables. There were no significant
correlations between the physiological or self-reported reactivity scores and the Trauma or Symptom Composites (see Table 3).

**Predicting Response Latency Segment Groups**

To test our primary hypotheses, we first attempted to replicate the finding that women who are survivors of multiple sexual victimizations take longer to press the button on the response latency task than women reporting single- or no-victimization. We used a chi-square analysis to determine whether frequency of segment group membership differed across Sexual Victimization Status Groups. Results did not indicate a significant relationship between Sexual Victimization Status and Segment Group membership ($\chi^2(6) = 4.93, p > .05$).

To test the hypothesis that the influence of physiological reactivity on risk detection abilities varies by self-reported reactivity, we ran a multinomial logistic regression with physiological reactivity variables (PEP, RSA, HR), self-reported reactivity, and interaction terms between each physiological variable and self-reported reactivity as predictors of Segment Group membership. When entering self-reported reactivity, physiological reactivity and interaction terms between self-reported and physiological reactivity scores as predictors of Segment Group membership, multinomial logistic regression results indicated a trend toward reaching statistically significant levels ($\chi^2 = 30.69, df = 21, p = .08$, Cox and Snell Pseudo $R^2 = .30$). See Table 4 for regression coefficients and Wald chi-square tests of the coefficients along with odds ratios. In particular, self-reported reactivity significantly distinguished women in Segment Group 1 from Segment Group 2 and Segment Group 4. Interestingly, although PEP reactivity did not appear as a significant individual predictor of Segment Group membership, the interaction term between PEP
reactivity and self-reported reactivity also significantly distinguished women in Segment Group 1 from Segment Group 2 and Segment Group 4.

Response Latency Segment Groups: Exploratory Within-Group Analyses

Additional exploratory linear regression analyses were conducted within each Segment Group to examine whether predictors of response latency might function differently for women of different Segment Groups. While the multinomial logistic regressions focused on reactivity variables to test a priori hypotheses, we used these analyses as an opportunity to conduct additional exploratory analysis with trauma and symptom variables. Thus, for each Segment Group, we ran two linear regression models: one model containing reactivity variables (PEP, RSA, HR, reactivity variables, self-reported reactivity, and interaction terms between physiological and self-reported reactivity variables) as predictors and one model containing the Trauma and Symptom Composites and an interaction term as predictors. Regression models containing reactivity variables as predictors did not reach statistically significant levels for any of the Segment Groups. The regression model with Trauma and Symptom Composites and an interaction term was statistically significant for Segment Group 4 ($F(3,11) = 30.19, p < .01; R^2=.92$). Table 5 provides individual regression coefficients for the model. Parameter estimates indicated that the Trauma Composite uniquely explained variance in response latencies for women belonging to Segment Group 4 such that the higher the Trauma Composite score, the shorter the response latency. Similarly, the Symptom Composites uniquely explained variance in response latencies for women belonging to Segment Group 4 such that the higher the Symptom Composite score, the longer the response latency. The interaction term between Trauma and Symptom also significantly predicted response latencies. A
graphical representation of the interaction effect (See Figure 2) was obtained following procedures recommended by Cohen et al. (2003) where the regression line for the dependent variable on the independent variable was plotted based on one SD above and below the mean as the high and low levels of the moderator (in this case, the Symptom Composite). Regression models containing Trauma and Symptom Composites as predictors were not significant for the other Segment Groups.
Chapter Four

Discussion

Previous studies that utilized the response latency task described here found that college women with multiple victimizations exhibited longer response latencies than their peers with single or no victimizations (Wilson et al., 2001; Soler-Baillo et al., 2005). The current study extended these findings to a community sample of women with a range of victimization histories. We attempted to replicate the previous findings with non-parametric analyses since the response latencies exhibited by this community sample of women was not normally distributed as in previous studies. Despite replicating methods for assigning women to Sexual Victimization Status Groups, these groups did not exhibit significantly different response latencies on the risk detection task. Results did not support a replication of the finding that women with multiple sexual victimizations exhibit longer response latencies than women with single- or no-victimizations. In addition to categorical groupings, we also examined participants’ trauma histories as a continuous Trauma Composite variable to capture the heterogeneity of trauma experienced by these women. However, we failed to find a relationship between the Trauma Composite and risk detection task performance as well.

The divergence between our findings in a community sample and previous findings in college samples may be due to several factors. Definitions of childhood sexual abuse (CSA) vary widely across studies (as reviewed in Messman & Long, 1996). While we attempted to define revictimization and sexual abuse events similarly to studies by Marx
and colleagues, differences in definition and methodology remained. We obtained trauma history information through an interview based on Fisher and Cullen’s (2000) strategy; previous response latency studies utilized self-report questionnaires. The different methodologies may have yielded differing prevalence rates of victimization. Furthermore, previous studies defined sexual victimization events as unwanted attempted or completed oral, anal, or vaginal intercourse, while our definition also included unwanted exposure and fondling.

The community sample in the present study was likely exposed to more severe and multiple forms of victimization than college samples. While our primary analyses focused on sexual victimization, many of the women in the current sample also reported physical victimizations (19 women reported no physical victimizations, 30 women reported one physical victimization, and 43 women reported multiple physical victimizations). For replication purposes, we did not count physical victimizations in our Sexual Victimization Status groups. However, women in the current sample may have been miscategorized due to additional experiences of physical victimization (e.g., a woman in the Single Victimization group based on one sexual victimization could have experienced additional physical victimizations); physical victimization may also be important to revictimization. To deal with this issue, we created a Trauma Composite to include physical victimizations; we also wanted to capture the heterogeneity of trauma experiences reported by the women in our community sample. However, given we failed to find relationships between the Trauma Composite and risk detection latencies, this likely does not explain our failure to replicate.
In addition to the relationship between trauma and risk detection abilities, we also examined the relationship between reactivity and risk detection. Specifically, we tested whether the influence of physiological reactivity (e.g., RSA, PEP, and HR) on risk detection abilities varies by self-reported reactivity. Analyses did not reach conventional statistical significance levels to fully support this hypothesis. However, the full model testing for Segment Group membership using physiological and self-reported reactivity as predictor variables was marginally significant, suggesting that we can distinguish women’s membership in Segment Group 1 versus the other Segment Groups. Specifically, women in Segment Group 1 were more likely than women in Segment Group 2 and Segment Group 4 to self-report lower levels of reactivity. This finding may demonstrate that women who are less emotionally reactive based on self-reports are more likely to say that a man has gone too far in his sexual advances than women who are more subjectively emotionally reactive. Women who pressed the button after the first refusal may be using their self-awareness more effectively to react to danger cues than women who pressed the button later. Women who waited to press the button after additional refusals may be more dysregulated in that they are perhaps overly concerned with internal emotion reactivity and are less able to pay attention to external cues, placing them at greater danger.

Importantly, women in Segment Group 1 were more likely to display a discrepancy between their PEP versus self-reported reactivity than women in Segment Groups 2 and 4. This finding does not support the hypothesis that women whose physiological and self-reported reactivity do not match will have longer risk detection response latencies. In fact, the findings are opposite to predictions because the discrepancy between physiological and self-reported reactivity was linked with membership in the fastest response group versus
the slower response groups. Possibly, a discrepancy between one’s physiological versus self-reported reactivity may prompt women to be more aware of when a man has pushed her comfort zone too far with his sexual advances rather than leading to a risk detection deficit. Future studies could examine the relationship between discrepancy of reactivity and readiness for action in a risky situation.

Notably, PEP was the only physiological measure that showed this pattern of results. We had expected all three measures of physiological reactivity (PEP, RSA, HR) to show similar patterns of results. PEP acts as a marker of central sympathetic system activity – which enhances cardiac performance – and has been linked to threat responses (Mendes, Blascovich, Lickel, & Hunter, 2002). RSA as a measure of parasympathetic withdrawal and HR as a global measure of cardiac reactivity may not be specific to threat responses. Therefore, PEP may reflect the women’s responses to the threat of the man’s advances, versus RSA or HR. Future research using specific measures of threat and challenge responses (e.g., cardiac output, total peripheral resistance) or additional physiological indices (e.g., blood pressure, skin conductance) could further investigate more specific aspects of women’s physiological responses while detecting danger.

We also examined variables within each Segment Group instead of the full sample to better understand more fine-tuned individual differences in reactivity. For women in Segment Group 4 (the women slowest to press the button), reactivity variables did not predict reaction time to press the button; rather, trauma and symptom levels explained unique variance in reaction time. In particular, an interaction effect between Trauma and Symptom Composite in this Segment Group suggests that at lower levels of trauma, higher levels of symptomatology were significantly related to slower reaction times relative to the
opposite pattern at higher levels of trauma (see Rosnow and Rosenthal, 1989). Perhaps this pattern of findings points to different post-trauma reactivity patterns for certain women (in this case, the women who showed risk detection problems based on their membership in Segment Group 4). For example, women with low levels of trauma and high levels of symptoms may be more dysregulated internally (as compared to women with similarly low levels of trauma and low levels of symptomatology) so that they are less able to identify external danger cues. However, for women with high levels of trauma, perhaps the ongoing cycle of experiencing additional traumas has set them up to be vigilant for danger cues, thus displaying faster reaction times regardless of symptom levels. These preliminary findings point to the importance of teasing apart individual variability in post-trauma functioning in more nuanced ways rather than focusing on broad main effects.

The main effect in Segment Group 4 of higher symptoms associated with slower reaction times may also help explain why previous research has produced mixed evidence for the role of post-trauma symptomatology in mediating revictimization (e.g., Filipas & Ullman, 2006; Risser, Hetzel-Riggin, Thomsen, & McCanne, 2006). Trauma-related symptoms may only have an impact on risk detection at certain levels of risk detection impairment. For women who already show some impairment in risk detection (e.g., Segment Group 4), symptom severity may then further amplify these problems. However, at lower levels of risk detection impairment (e.g., Segment Groups 1-3), symptoms may not exert influence. To the extent that studies on risk detection have utilized undergraduate samples reporting lower levels of post-trauma symptoms, we may be limiting our understanding of women who function less adaptively post-trauma. However, these findings remain exploratory and require future replications.
Limitations and Future Direction

Several limitations in the current study can be addressed in future studies. Extant research on revictimization primarily focuses on women, given that women are at higher risk for sexual assaults than men (Rich et al., 2004). The current study only recruited women; however, future studies should examine these processes among men. Men with childhood abuse are also at greater risk of victimization later in life compared to those without childhood abuse (Cloitre et al., 1996). The current study also depended on retrospective self-reported trauma histories, which can suffer from both false positive and false negative reports. Longitudinal, prospective studies are needed to establish causal links between child maltreatment and revictimization.

The instruction for the risk detection task utilized in this study asked women to press the women when the man has “gone too far.” While this may capture risk detection in the sense that women indicate when they no longer feel comfortable or safe with the man, this instructional phrase may not capture action tendencies of the woman. Some previous studies have asked college-aged women to indicate “when you would leave” the risky situation (for a review, see Marx et al., 2005) rather than “if the man has gone too far” and found similar results where multiply-victimized college women stayed longer in the risky situation than their singly- or non-victimized peers. However, no studies have examined whether community women view the two phrases as similar instructions of being asked to detect and act upon danger cues. Future research should further examine potential differences in providing various instructions for the risk detection task in community samples. Better understanding of women’s risk detection abilities will in turn provide additional knowledge on increased risk for revictimization.
Conclusion

Experiencing traumatic experiences leads to heterogeneous outcomes and responses in survivors. For example, both hyper-reactivity (e.g., posttraumatic stress disorder arousal symptoms, hypervigilance) and hypo-reactivity (e.g., dissociation, avoidant coping) are common post-trauma responses. Theorists have proposed that trauma survives either subjectively minimize or exaggerate danger (and sometimes do both) due to these deviations from normal patterns of physiological responding (e.g., Chu, 1992; Cloitre, 1998). Importantly, findings from the current study extend this idea by suggesting a relationship between women’s subjective awareness of their body’s objective reactivity and their responses to external danger cues. Additionally, results also indicate that symptom severity interacts with trauma experiences to further exaggerate deficits in detecting risk. Therefore, this study contributes to the growing literature on the emotional responding of women in the context of their ability to detect risk. Although perpetrators are always responsible for sexually aggressive acts toward victims, better understanding of ways we can empower women with defensive strategies will also help prevent additional assault from occurring.
References


Appendix

Table 1.  
*Demographic Information by Victimization Status Groups.*

<table>
<thead>
<tr>
<th></th>
<th>No Victimization</th>
<th>Adult Single Victimization</th>
<th>Multiple Victimization</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 10</td>
<td>n = 15</td>
<td>n = 47</td>
</tr>
<tr>
<td>Age</td>
<td>29.30 (5.95)</td>
<td>30.15 (6.49); n = 13</td>
<td>30.15 (6.55)</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
</tr>
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<td>2</td>
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</tr>
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</tr>
<tr>
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</tr>
<tr>
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<td>0</td>
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<td>2</td>
<td>9</td>
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<tr>
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<td></td>
</tr>
<tr>
<td>Education</td>
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<td></td>
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<td>High School or less</td>
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<td>2</td>
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<td>5</td>
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</tr>
<tr>
<td>Income</td>
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<tr>
<td>&lt;10,000</td>
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<td>3</td>
<td>18</td>
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<tr>
<td>10,000-30,000</td>
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<td>6</td>
<td>13</td>
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<tr>
<td>30,000-50,000</td>
<td>2</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>&gt;50,000</td>
<td>3</td>
<td>3</td>
<td>6</td>
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Table 2.  
*Mean (SD) Physical Victimization Events and Symptom Scores by Victimization Status Groups (N = 72)*

<table>
<thead>
<tr>
<th></th>
<th>No Victimization (n=10)</th>
<th>Adult Single Victimization (n=15)</th>
<th>Multiple Victimization (n=47)</th>
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<tr>
<td>Symptom Composite (z scores)</td>
<td>- .49 (.62)</td>
<td>- .54 (.48)</td>
<td>.25 (.80)</td>
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<tr>
<td></td>
<td>n = 14</td>
<td></td>
<td></td>
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<tr>
<td>PEP baseline</td>
<td>92.40 (22.82)</td>
<td>99.07 (22.18)</td>
<td>104.19 (21.32)</td>
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<tr>
<td></td>
<td>n = 43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RSA baseline</td>
<td>6.36 (1.55)</td>
<td>6.98 (1.95)</td>
<td>6.77 (1.50)</td>
</tr>
<tr>
<td></td>
<td>n = 46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR baseline</td>
<td>82.42 (10.68)</td>
<td>85.12 (14.26)</td>
<td>78.92 (12.15)</td>
</tr>
<tr>
<td></td>
<td>n = 46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-report baseline</td>
<td>2.08 (1.31)</td>
<td>2.86 (1.16)</td>
<td>2.30 (1.44)</td>
</tr>
<tr>
<td>PEP reactivity</td>
<td>1.30 (2.54)</td>
<td>1.33 (4.27)</td>
<td>1.16 (5.75)</td>
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<td></td>
<td>n = 43</td>
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<tr>
<td>RSA reactivity</td>
<td>-.16 (.47)</td>
<td>-.05 (.55)</td>
<td>-.05 (.53)</td>
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<td></td>
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<tr>
<td>Self-report reactivity</td>
<td>2.08 (1.30)</td>
<td>.94 (1.51)</td>
<td>1.57 (1.48)</td>
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*Note.* Sample sizes are given if missing data.
Table 3. *Bivariate Correlations Among Predictor Variables*

<table>
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<tr>
<th></th>
<th>PEP Reactivity</th>
<th>RSA Reactivity</th>
<th>HR Reactivity</th>
<th>Self-report Reactivity</th>
<th>Trauma Composite</th>
<th>Symptom Composite</th>
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<tr>
<td>PEP Reactivity</td>
<td>--</td>
<td>.02</td>
<td>.22*</td>
<td>.15</td>
<td>.19</td>
<td>.02</td>
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<tr>
<td>RSA Reactivity</td>
<td>--</td>
<td>--</td>
<td>.44**</td>
<td>.08</td>
<td>-.05</td>
<td>-.02</td>
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<tr>
<td>HR Reactivity</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>.05</td>
<td>.08</td>
<td>.06</td>
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<tr>
<td>Self-Report Reactivity</td>
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<td>--</td>
<td>--</td>
<td>--</td>
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<td>.06</td>
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<tr>
<td>Trauma Composite</td>
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<td></td>
<td></td>
<td></td>
<td>.29**</td>
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* = p < .05; ** = p < .01
Table 4. Multinomial Logistic Regressions with Reactivity Variables Predicting Segment Group Membership for the PEP and the RSA Measures.

<table>
<thead>
<tr>
<th>Segment Group 2 (versus Group 1)</th>
<th>B</th>
<th>SE(B)</th>
<th>Wald</th>
<th>Sig.</th>
<th>OR</th>
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<tr>
<td>PEP Reactivity</td>
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<td>.02</td>
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<table>
<thead>
<tr>
<th>Segment Group 3 (versus Group 1)</th>
<th>B</th>
<th>SE(B)</th>
<th>Wald</th>
<th>Sig.</th>
<th>OR</th>
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<tbody>
<tr>
<td>PEP Reactivity</td>
<td>-.04</td>
<td>.08</td>
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<td>.51</td>
<td>1.12</td>
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<tr>
<td>Self-reported Reactivity</td>
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<td>2.74</td>
<td>.10</td>
<td>1.51</td>
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<td>.81</td>
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<td>.65</td>
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<td>RSA Interaction Term</td>
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<td>.43</td>
<td>2.79</td>
<td>.10</td>
<td>2.06</td>
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<td>.50</td>
<td>1.27</td>
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<td>.57</td>
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<table>
<thead>
<tr>
<th>Segment Group 4 (versus Group 1)</th>
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<th>SE(B)</th>
<th>Wald</th>
<th>Sig.</th>
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<tr>
<td>PEP Reactivity</td>
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<td>.01</td>
<td>.93</td>
<td>1.01</td>
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<td>HR Reactivity</td>
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<td>.22</td>
<td>.05</td>
<td>.83</td>
<td>1.05</td>
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<td>3.57</td>
<td>.06</td>
<td>1.91</td>
</tr>
<tr>
<td>PEP Interaction Term</td>
<td>-1.65</td>
<td>.66</td>
<td>6.15</td>
<td>.01</td>
<td>.19</td>
</tr>
<tr>
<td>RSA Interaction Term</td>
<td>.36</td>
<td>.76</td>
<td>.23</td>
<td>.64</td>
<td>1.43</td>
</tr>
<tr>
<td>HR Interaction Term</td>
<td>-.91</td>
<td>.71</td>
<td>1.67</td>
<td>.20</td>
<td>.40</td>
</tr>
</tbody>
</table>

*p<.05, **p<.01
Table 5. Linear Regression Predicting Response Latency for Segment Group 4.

<table>
<thead>
<tr>
<th>Regression Term</th>
<th>B</th>
<th>SE(B)</th>
<th>Beta</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma Composite</td>
<td>-20.89</td>
<td>2.64</td>
<td>-.94</td>
<td>-7.92**</td>
</tr>
<tr>
<td>Symptom Composite</td>
<td>14.64</td>
<td>2.22</td>
<td>.71</td>
<td>6.59**</td>
</tr>
<tr>
<td>Interaction Term</td>
<td>-11.91</td>
<td>4.56</td>
<td>-.31</td>
<td>-2.61*</td>
</tr>
</tbody>
</table>

*p<.05; **p<.01
Figure 1.

Distribution of Response Latencies Across Segment Groups.
Figure 2.
*Interaction Effect Between Symptom and Trauma Composites in Segment Group 4.*