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Physiological Reactivity in a Community Sample of Sexually Aggressive Young Men: A Test of Competing Hypotheses

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Abstract

Men's sexually aggressive behavior potentially could relate to either physiological hyporeactivity or hyperreactivity, and these two different physiological profiles could be associated with different underlying causes of sexual aggression. Thus, measurement of physiological reactivity could provide insight into mechanisms relevant to the etiology of sexual aggression. The relationship between sexual aggression and physiological reactivity was investigated in 78 community men (38 sexually aggressive and 40 non-aggressive men). In a laboratory protocol, the men were exposed to neutral, negative-affect-inducing, and positive-affect-inducing stimuli. Men's salivary cortisol concentrations and electrodermal activity (EDA) were measured throughout the laboratory procedure. Sexually aggressive men demonstrated (1) lower overall cortisol levels and (2) lower EDA reactivity in some conditions as compared to non-aggressive men. Results of this study were consistent with the idea that men's sexual aggression is associated with physiological hyporeactivity, a physiological profile that has been found to be associated with externalizing behaviors and psychopathic traits.

Keywords

sexual aggression; sexual coercion; rape; psychophysiology; electrodermal activity; skin conductance; cortisol; psychopathy; emotion regulation

Acts of sexual aggression, including rape or sexual assault (i.e., nonconsensual sexual acts obtained through physical force, threats of physical harm, or incapacitation such as through

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alcohol or drugs) and sexual coercion (i.e., sexual acts obtained through verbal pressure or manipulation), are highly prevalent. Based on nationally representative samples, 18–22% of women report an experience with completed rape or sexual assault (Laumann, Gagnon, Michael, & Michaels, 1994; Tjaden & Thoennes, 2000), and as many as 34% report an experience with sexual coercion (Basile, 2002). The potential health consequences of sexual aggression are extensive and can include vaginal and rectal bleeding; bruises, cuts, and scrapes; sexually transmitted infections; pelvic inflammatory disease; and pregnancies, many of which end in elective abortions (Resnick, Acierno, Holmes, Dammeyer, & Kilpatrick,, 2000; U.S. Bureau of Justice Statistics, 2002). Further, in the long-term, victims of sexual aggression are more likely than other women to report physical health problems including gynecological, gastrointestinal, and chronic pain symptoms (Kimerling & Calhoun, 1994; Koss, Koss, & Woodruff, 1991) and psychological problems including depression, anxiety, sexual dysfunction, and substance abuse (McFarlane & Malecha, 2005; Resnick et al, 2000).

Understanding the causes of men's sexual aggression is essential to developing effective rape prevention. Yet, researchers are only in the beginning stages of understanding the factors that motivate men's sexual aggression against women. Further, researching sexually aggressive men is difficult because it typically relies on men's self-reports of sexually aggressive behavior. It has been estimated that the vast majority of rapes (approximately 95%) are not reported to police (Koss, Gidycz, & Wisneiwski, 1987), so most rapists are never identified by the criminal justice system. Thus, research relying exclusively on incarcerated rapists also provides an incomplete picture.

Identifying the physiological correlates of men's sexual aggression could contribute to a better understanding of the etiology of sexually aggressive behavior and thus provide direction for intervention. Once identified, physiological correlates also could function as markers of risk for sexual aggression, providing unique information to augment researchers' reliance on self-report, which is subject to recall and social desirability biases. To our knowledge, no study to date has examined general physiological reactivity in a sample of non-incarcerated adult men who have perpetrated sexual aggression against adult women.

Although there is currently no research on the relationship between sexual aggression and physiological reactivity in community samples of men, there are two ways in which physiological reactivity might be related to men's sexually aggressive behavior. These two opposing physiological patterns also have been suggested to underlie violent behaviors more generally (e.g., see Gottman, 2001). First, sexually aggressive men might demonstrate hyporeactivity compared to non-aggressive men; this pattern would be expected if men are driven to engage in sexually aggressive behavior by traits such as a lack of concern for consequences and deficits in empathy. Alternatively, sexually aggressive men might demonstrate hyperreactivity compared to non-aggressive men; this pattern would be expected if sexual aggression is prompted by high levels of poorly regulated negative affect.

The psychobiological stress response involves activation of both the hypothalamic-pituitary-adrenal (HPA) and the sympathetic-adrenal-medullary (SAM) axes. Although these systems function to help individuals' respond to threatening stimuli, when the systems are challenged

too heavily, pathophysiology may occur. Pathophysiology in the stress response systems has been associated with aggressive and other problematic behaviors (Alink et al., 2008). Investigating responses across both the HPA and SAM axes allows for a more thorough understanding of the physiological correlates of behavior, as it is possible that these two systems may be activated by different types of situational stressors (Bauer, Quas, & Boyce, 2002). In this article, we focus on two measures of physiological activity or stress response: (1) Cortisol is a hormonal product of the HPA axis, with low concentrations of salivary cortisol reflecting an underarousal or hyporesponsivity in response to emotional stress, and high levels reflecting hyperresponsivity (e.g., Van Goozen et al., 1998). (2) Electrodermal activity (EDA) is under control of the sympathetic nervous system as part of the SAM axis; increases in EDA reflect increased emotional arousal (Fowles, 2000).

The Hyporeactivity Hypothesis

Although physiological studies of sexually aggressive community men are lacking, there is reason to believe that sexually aggressive men may display a pattern of physiological hyporeactivity, as measured both at baseline and in response to stressful or negative events. Evidence for the Hyporeactivity Hypothesis comes primarily from findings suggesting a shared link between physiological hyporeactivity and psychopathy and between sexual aggression and psychopathy.

The clinical construct of psychopathy involves a cluster of behaviors and personality characteristics, including exploitation and manipulation of others, lack of remorse and empathy (i.e., callousness), impulsivity, and lack of emotional responsivity, that tend to be associated with antisocial (i.e., violent and criminal) behaviors (Hare, 2003). Based on college and community samples, research has found that men who self-report engaging in sexually aggressive behavior endorse relatively more psychopathic personality traits and antisocial behaviors on self-report measures than men who do not report sexual aggression (e.g., Konsson, Kelly, & White, 1997; Lalumiere & Quinsey, 1996; Malamuth, 1986).

Additionally, across a variety of studies, psychopathy and associated behavioral features, such as antisocial behaviors and conduct problems, have been shown to be associated with physiological under-reactivity, as measured by EDA reactivity and salivary cortisol concentrations, in both children and adults (e.g., Holi, Auvinen-Lintunen, Lindberg, Tani, & Virkkunen, 2006; Lorber, 2004; O'Leary, Loney, & Eckel, 2007). In a meta-analysis, psychopathy was associated with low resting EDA (measured in the absence of stimuli), low task EDA (measured as the participant performed a task), and low EDA reactivity (measured as change from baseline to task; Lorber, 2004). Indeed, based on a review of the literature, Fowles (2000) concluded that "electrodermal hyporeactivity in psychopathy is one of the most reliable psychophysiological correlates of psychopathology" (p. 177). With a few recent exceptions (e.g., Feilhauer, Cima, Korebrits, & Nicholson, 2013; Gowen et al., 2013), hyporeactivity in relation to psychopathy and antisocial behaviors has also been demonstrated in studies examining cortisol concentrations. Low serum cortisol levels have been observed in male criminal psychopaths (Holi et. al., 2006), and salivary cortisol was inversely associated with psychopathic traits in a sample of college men (O'Leary et al., 2007). Several studies have demonstrated that adolescents with conduct disorder symptoms

(a precursor to Antisocial Personality Disorder) have lower resting and task-related salivary cortisol levels than non-conduct disordered controls, and this effect is particularly evident in conduct disordered boys with high levels of aggressive and disruptive behaviors (e.g., McBurnett et al., 2000; Popma et al., 2006). In fact, Loney, Butler, Lima, Counts, and Eckel (2006) observed that low resting cortisol actually appears to be a unique feature of a small group of antisocial individuals with the most severe and persistent conduct disorder presentations; the researchers found that low resting cortisol in adolescent boys was specifically associated with callous-unemotional traits regardless of level of conduct problems. As callous sexual attitudes have consistently been shown to be related to sexual aggression (e.g., Abbey & McAuslan, 2004; Malamuth, 2003), these findings provide particularly strong support for the hyporeactivity hypothesis.

There are two prominent theories to explain the low levels of physiological reactivity observed in individuals with psychopathic traits and antisocial behaviors (see e.g., Raine, 2002). First, a biological predisposition to low physiological arousal may lead to heightened sensation-seeking in psychopathic individuals and thus may motivate the antisocial behavior that is part of the psychopathic profile (e.g., Quay, 1965; Wilson & Scarpa, 2011). Alternatively, psychopathy may be driven by a tendency toward fearlessness or low anxiety (e.g., Fowles, 2000; Lykken, 1957), which is displayed as low physiological reactivity and which results in a lack of inhibition or concern about consequences. Both of these theories would seem to predict that individuals with psychopathic traits would demonstrate physiological underreactivity particularly in response to anxiety- or fear-inducing stimuli; however, some research suggests that the association between physiological hyporeactivity and psychopathy may extend to a variety of emotional stimuli, including fearful stimuli (e.g., Patrick, Cuthbert, & Lang, 1994), stimuli depicting distress in others (e.g., Blair, 1999), and even pleasant stimuli (e.g., Pastor, Molto, Vila, & Lang, 2003). Some researchers (e.g., Blair, 1999) have extended the fearlessness hypothesis to better explain the general hyporeactivity demonstrated by psychopathic individuals across a range of stimuli. For example, it is possible that the tendency toward low anxiety and lack of concern for consequences may lead a developing child to be insensitive to parental and societal attempts at socialization, and thus the child may fail to learn moral and affective skills; this may result in a general lack of emotional depth and responsivity, which likely underpins the lack of empathy and remorse associated with psychopathy (Cleckley, 1976).

Although there is evidence that, on average, sexually aggressive men have more psychopathic and antisocial traits than non-aggressive men and thus might be expected to demonstrate the physiological hyporeactivity that is associated with these traits, it is possible that sexually aggressive men share attitudes or traits with psychopathic or antisocial men, but do not demonstrate the fearlessness or lack of emotional depth that is thought to be associated with physiological hyporeactivity. Indeed, in the meta-analysis described above, Lorber (2004) found that aggressive behavior in adults was *positively* associated with EDA reactivity; this was in opposition to a negative association between EDA reactivity and psychopathy. This illustrates that, although aggression may be one trait in the psychopathic constellation, some aggressive behavior is clearly motived by factors other than psychopathy.

The Hyperreactivity Hypothesis

Just as physiological under-responsiveness may reflect a callous fearlessness, physiological over-responsiveness may reflect a tendency toward strong and unregulated negative affect. For example, in a variety of studies, negative affectivity, or a proneness to depression, anxiety, stress, anger, and hostility, has been associated with elevated cortisol levels during normal daily activities as well as during laboratory tasks (e.g., al'Absi et al., 1997; Pope & Smith, 1991; Steptoe, Cropley, Griffith, & Kirschbaum, 2000). EDA increases have similarly been observed in response to negative emotions, including anger, anxiety, and fear (e.g., see Kreibig, 2010, for a review). Evidence for the Hyperreactivity Hypothesis comes from the apparent association between strong negative affective states and aggressive behaviors and from findings indicating that physiological hyperreactivity is associated with some acts of criminality and violence.

Negative affect proneness has been shown to be associated with a variety of aggressive behaviors, including physical abuse of children, partner violence, and workplace aggression (Douglas & Martinko, 2001; Mammen, Kolko, & Pilkonis, 2002; Margolin, John, & Gleberman, 1988). One particular type of negative affectivity—hostility—has been shown in a variety of studies to be related to sexual aggression (e.g., Malamuth, 2003). Additionally, Peterson, Goodrich, Janssen, Fortenberry & Heiman (2013) found a positive association between trait levels of negative affect, particularly anxiety and anger, and self-reported sexually aggressive behavior in a sample of young men from the community. This is inconsistent with the idea that sexual aggression is driven by a psychopathic fearlessness and perhaps more consistent with suggestions that sexually aggressive men may be insecure and anxious about their relationships with women (Malamuth, Linz, Heavey, Barnes, & Acker, 1995) and/or about their sexual performance (Peterson, Janssen, & Heiman, 2010) and may attempt to reduce their anxiety by taking control of the sexual encounter and eliminating the possibility of rejection.

Further evidence for the Hyperreactivity Hypothesis comes from findings suggesting that physiological hyperreactivity is related to a variety of criminal and aggressive acts. Cima, Smeets, and Jelicic (2008) compared psychopathic and non-psychopathic prison inmates. They found that psychopathic offenders demonstrated lower cortisol levels than non-psychopathic offenders. However, in contrast to the predictions of the Hyporeactivity Hypothesis, their results suggested that this difference could be attributed to higher than typical cortisol levels among non-psychopathic offenders rather than lower than typical levels in psychopathic offenders (p. 82). This raises the possibility that some types of criminal behavior are associated with hyperreactivity rather than hyporeactivity.

Consistent with this, some researchers have found a relationship between hyperreactivity and intimate partner physical violence, a behavior that is analogous to sexual aggression in many respects (e.g., both are typically perpetrated against a well-known victim and intimate partner physical violence and sexual aggression often co-occur). In two different studies, baseline cortisol levels were found to be positively associated with physical aggression against an intimate partner (Feinberg, Jones, Granger, & Bontempo, 2011; Lindman, von der Pahlen, Ost, & Eriksson, 1992). Also, George et al. (2000) noted that some perpetrators of

intimate partner physical violence reported physiological symptoms prior to engaging in aggression that are similar to a panic attack (e.g., heart palpitations, increased respiration rate, and feelings of fear); these symptoms are consistent with physiological hyperarousal. When the researchers administered sodium lactate, a chemical agent that induces fear, to men with and without a history of partner violence, the violent men exhibited more rage and panic and greater changes in speaking, breathing, and motor activity than did the nonviolent men, suggesting that some men's violence may reflect a maladaptive response to heightened fear rather than a psychopathic fearlessness.

The Current Study

The present study tested two competing hypotheses related to men's sexual aggression. We exposed men to positive- and negative-affect-inducing stimuli and measured the impact of the stimuli on men's salivary cortisol concentrations and EDA. If the Hyporeactivity Hypothesis is correct, aggressive men should demonstrate lower levels of emotional arousal (i.e., lower baseline cortisol concentrations and lower EDA reactivity in response to negative affect) than non-aggressive men. If the Hyperreactivity Hypothesis is correct, aggressive men should demonstrate greater emotional reactivity than non-aggressive men as measured by higher baseline cortisol levels and higher EDA reactivity.

Methods

Participants

All participants were recruited from questionnaire studies focusing on a similar topic. Participants were recruited from a variety of sources to ensure a relatively diverse sample. Specifically, participants for the questionnaire studies were recruited (1) at an urban, sexually transmitted infection (STI) clinic in Indianapolis, Indiana that serves low-income patients, (2) from ads placed on craigslist.com for Indianapolis, and (3) from online classified ads posted in an electronic system accessible by students, faculty, and staff in the Indiana University system. After completing the questionnaire studies, participants had the option to provide contact information if they were interested in future paid research opportunities.

Interested participants were eligible to be contacted for this study if they met the following criteria based on their responses to the questionnaire studies: They were between the ages of 18 and 30 inclusive, they were sexually experienced (i.e., had had at least one vaginal intercourse partner), they self-identified as heterosexual, and they were unmarried. Participants were excluded if they were HIV positive or if they had serious dental problems that might impact the reading of their salivary cortisol levels.

Invitations to participate in the laboratory study were made based on responses in the questionnaire studies; specifically, invitations to the laboratory study were issued selectively with the goal of including approximately equal numbers of White/European American and Black/African American participants, as those are the predominant racial groups in Indianapolis. We also selectively issued invitations with the goal of including approximately

equal numbers of sexually aggressive and sexually non-aggressive men (defined based on responses to the questionnaire studies; see below).

Although 90 eligible men began the laboratory study, due to data loss resulting from technical problems (i.e., not enough saliva to allow for cortisol assay, problems with the EDA signal, or technical problems with our computer-administered protocol.), the final sample consisted of 78 men (M_{age} = 24.44; *SD* = 3.27). Of these men, 41 identified as White/European American; 33 identified as Black/African American; 3 identified as bi- or multi-racial; and 1 identified as Moroccan. The majority of the men (65.4%) reported a household income of less than $30,000 per year. The mean years of education for the sample was 14.10 (SD = 2.29; range = 9–23 years), equivalent to a high school diploma plus two years of undergraduate education.

Of the 78 men in our sample, 38 were classified as sexually aggressive and 40 were classified as non-sexually aggressive. Men were classified based on their responses to the Sexual Strategies Scale (SSS; Strang, Peterson, Hill, & Heiman, 2013), which they completed as part of the questionnaire studies. Prior research has found that the SSS is significantly correlated with other measures of sexual aggression; further, in past research, men have reported higher rates of sexual aggression on the SSS than on two other measures of sexually aggressive behavior (Strang et al., 2013). Given that there is strong evidence to suggest that men may tend to underreport sexually aggressive behavior (Strang et al., 2013) and given that the SSS has demonstrated expected correlations with attitudinal measures that are typically associated with sexual aggression (Peterson et al., 2013), higher rates of reporting on the SSS seems to be a strength of the scale, suggesting that the SSS may identify some sexually aggressive men who are missed by other scales. The SSS, like other measures of sexual aggression, is a behavioral sampling measure and does not produce a summed or average score. For this study, men were classified as sexually aggressive if they reported ever using verbal pressure or manipulation, older age or authority, intoxication, threats, or force to obtain oral, anal, or vaginal intercourse from a female partner. Men who denied using all of these strategies were classified as non-aggressive. Notably, the 38 aggressive men in our sample had primarily engaged in relatively less severe forms of sexual aggression; 35 men reported use of verbal coercion or manipulation to obtain sex, 13 men reported exploiting a woman's intoxication, and only 2 men reported use of force. There were no significant differences between our aggressive and non-aggressive groups in terms of age, race, or income category.

Procedure

A researcher contacted participants who were interested and eligible based on their responses in the questionnaire studies. Participants were instructed to avoid smoking and eating for at least four hours prior to their participation, as these behaviors may impact salivary cortisol levels (Kirschabaum & Hellhammer, 1994).

In the lab, participants were met by a male researcher. Informed consent was discussed and signed. Participants were then seated in a small, private room. Physiological measurement devices, including electrodes for the measurement of EDA along with other measurement devices not discussed here (i.e., facial EMG), were placed by the researcher. Participants

were also instructed in the self-placement of genital response measures; data from those measures are not reported here. For the remainder of the study, participants were alone in the room.

First, participants observed a neutral video clip from a documentary about oceans. This video served as a baseline for the EDA measurement. EDA was recorded using Ag/AgCl electrodes attached to the volar surface of the first and second fingers on the nondominant hand (Dawson, Schell, & Filion, 2000).

Following the neutral video, participants provided baseline ratings of their affective state using the state version of the Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS is a widely-used 20-item self-report scale measuring positive and negative affect. Following careful instruction, participants also provided a saliva sample for the baseline assessment of cortisol levels (Kirschabaum & Hellhammer, 1994), and completed a laboratory task measuring sexual risk intent (data not reported here). Saliva was collected using salivette sampling devices. Samples were frozen (−20°C) until the day of assay. Cortisol analyses were performed in duplicate using a commercially available enzyme immunoassay kit (Salimetrics, LLC). The inter- and intra-assay coefficients of variance (CV) were 12.59 and 2.55, respectively.

In the negative affect-induction condition, participants were randomly assigned to watch one of two negative affect-inducing videos—either an anxiety-inducing video (a clip from *Silence of the Lambs*) or a sadness-inducing video (a clip from *Sophie's Choice*)—after which they completed the PANAS and provided a second saliva sample. In the positive-affect-induction condition, all participants watched the same positive-affect-inducing video (a clip from *The Natural*) and then completed the PANAS. EDA measurements were recorded every 20 milliseconds throughout the baseline and affect-inducing videos. The videos used in this study have been shown in prior research (Gross and Levenson, 1995; Janssen, Hahn, & Rullo, 2005; Philippot, 1993) to effectively induce a specific mood (e.g., anxiety) while leading to minimal levels of other moods (e.g., anger).

Order of the two affect inductions (positive and negative) was randomized. Regardless of the order of the inductions, collection of the third saliva sample took place approximately 20 minutes after the negative-affect-inducing video, as cortisol has been shown to peak approximately 20 minutes after exposure to a stressor (Levine, Zagoory-Sharon, Feldman, Lewis, & Weller, 2007). Cortisol was not collected following the positive mood condition because in prior research, positive photos and film clips have had no significant impact on participants' cortisol levels (Codispoti et al., 3002; Hubert, Moller, & de Jong-Meyer, 1993).

Between the two affect inductions, there was a return-to-baseline period consisting of a 10 minute neutral video. Additionally, for the purpose of analyses not presented here, participants watched erotic videos and completed laboratory tasks (i.e., sexual risk intent tasks) following each of the affect-induction conditions; however, the erotic videos and related tasks were always followed by a return-to-baseline period. The laboratory procedure is summarized in Figure 1.

Participation in the laboratory study took approximately 75 minutes. Participants were paid $50 for their participation. The methods of this study were approved by the Institutional Review Board at the Indiana University School of Medicine.

Results

All analyses were conducted using SPSS 20. Notably, some of our analyses, particularly our between-subject analyses, were under-powered. For example, for a 3 (within-subject) × 2 (between-subject) × 2 (between-subject) × 2 (between-subject) mixed Analysis of Variance (ANOVA), with 78 participants, we had 99.9% power to detect medium effect sizes ($f = .25$) for within-subject analyses, but only 41.8% power to detect medium effect sizes ($f = .25$) and 87% power to detect large effect sizes ($f = .40$) for between-subject analyses (Faul, Erdfelder, Lang, & Buchner, 2007). For this reason, we report effect sizes for all non-significant results below. In all analyses reported below, Greenhouse-Geisser corrections were used for within-subject analyses.

Self-Reported Affect

First, we examined whether there were demographic differences in self-reported positive and negative affect at baseline or in response to the affect inductions. There were no differences in PANAS scores across any of the conditions as a function of age, race, or income category.

Next, in order to test the effectiveness of our affect inductions, we conducted two analyses, each consisting of a 3 × 2 × 2 × 2 mixed ANOVA with Affect Induction (neutral baseline, positive affect induction, negative affect induction) as a within-subject variable and Aggression Group (sexually aggressive, nonaggressive), Negative Affect Condition (anxiety, sadness), and Order Condition (negative first, positive first) as between-subject variables. For the first ANOVA, positive affect was the dependent variable, and for the second ANOVA, negative affect was the dependent variable.

For the analysis with positive affect as the dependent variable, there was a significant main effect for Affect Induction, F (2, 140) = 7.70, $p = .001$, $\eta_p^2 = .10$. Follow-up tests revealed that men reported less positive affect in the negative-affect-inducing ($M = 22.46$; $SD = 8.65$) condition than in the neutral baseline ($M = 25.38$; $SD = 8.10$) or the positive-affect-inducing conditions ($M = 25.31$; $SD = 9.77$), t (77) = 4.26; $p < .001$ and t (77) = 3.04 $p = .003$, respectively. There was no difference between the positive and neutral conditions. There were no other significant main effects or interactions, and all effect sizes for the non-significant analyses were fairly small ($\eta_p^2 < .04$), suggesting that the non-significant findings were not merely a result of insufficient power.

For the analysis with negative affect, there was a significant main effect for Affect Induction, F (2, 140) = 38.60, $p < .001$, $\eta_p^2 = .36$, as well as a significant Affect Induction by Negative Affect Condition interaction, F (2, 140) = 4.50, $p = .03$, $\eta_p^2 = .06$. Overall, men reported more negative affect in the negative-affect-inducing condition ($M = 16.09$; $SD = 6.67$) than in the neutral baseline ($M = 11.91$; $SD = 2.24$) or the positive-affect-inducing condition ($M = 11.42$; $SD = 2.01$), t (77) = −5.84, $p < .001$; t (77) = −6.41, $p < .001$,

respectively. Men also reported less negative affect in the positive affect condition than in the neutral baseline condition, t (77) = 2.20, p = .03. Further, men who were presented with the sadness-inducing stimuli in the negative affect condition, reported more negative affect in the negative-affect-inducing condition (M = 17.97; SD = 7.30) than men who were presented with the anxiety-inducing stimuli in the negative affect condition (M = 14.71; SD = 5.88), F (1, 76) = 4.76, p = .03, η_p^2 = .06. There was no difference between the sadness and anxiety conditions in response to the neutral baseline or positive affect inductions; this was expected, as the neutral and positive stimuli were identical for the two negative affect conditions. There were no other significant main effects or interactions. Effect sizes for non-significant results were relatively small ($\eta_p^2 \leq .05$). Results of the analyses on self-report data suggested that our affect inductions were generally successful at producing the intended affective state.

Cortisol

Salivary cortisol was measured in micrograms per deciliter, and transformed using a natural log transformation to normalize the distribution. Although both basal levels of cortisol and changes in cortisol in reaction to a stressor have been found to be related to aggressive and antisocial behaviors, the relationship between basal cortisol and behavioral problems is slightly more robust than the relationship between cortisol reactivity and behavior problems (Alink et al., 2008). Thus, for these analyses we examined cortisol concentrations at each time point rather than using difference scores[1]. The relationship between aggression group and cortisol levels was assessed using a 3 × 2 × 2 × 2 mixed ANCOVA, with Time Point (baseline, immediately after the negative affective stimulus, 20 minutes after the negative stimulus) as a within-subject variable. Aggression Group (sexually aggressive, nonaggressive), Negative Affect Condition (anxiety, sadness), and Order Condition (negative first, positive first) were between-subject variables. Because the time of day in which the cortisol was collected in our study was not consistent and because many, but not all, individuals show diurnal patterns in their salivary cortisol concentrations (Stone et al., 2001), we entered time of data collection as a covariate.

There were no significant main effects for Time Point, Negative Affect Condition, or Order Condition, and there were no significant interactions; all effect sizes for these analyses were relatively small ($\eta_p^2 < .04$). However, there was a significant main effect for Aggression Group, F (1, 69) = 4.63, p = .04, η_p^2 = .06, with aggressive men having lower overall cortisol levels (M = 1.99; SD = 0.68) than non-aggressive men (M = 2.33; SD = 0.55). See Figure 2.

The majority of men in our sample (n = 58; 31 non-aggressive and 27 aggressive) started the study between late morning (10 AM) and early afternoon (1:00 PM). To better control for time of day of cortisol collection, we re-ran the cortisol analysis, including only these 58 men who completed the study during this more limited time frame. The results were consistent with those from the entire sample. Aggressive men demonstrated lower overall cortisol levels (M = 2.05; SD = 0.41than non-aggressive men (M = 2.40; SD= 0.59), F (1,

[1]We also ran the analyses with cortisol reactivity (cortisol 20 minutes after the negative affect induction minus baseline cortisol) as the dependent variable. There were no significant results and all effect sizes were small.

50) = 6.92, p = .01, η_p^2 = .12. There were no other significant main effects or interactions, and all effect sizes for these analyses were relatively small ($\eta_p^2 \leq .05$). Overall, cortisol levels among the sexually aggressive men suggested that they had lower physiological arousal than the non-aggressive men, consistent with the Hyporeactivity Hypothesis.

EDA Reactivity

EDA responses were recorded intermittently during each video presentation, and averages were calculated for the neutral baseline, negative-affect inducing, and positive-affect-inducing conditions. In contrast to cortisol, EDA reactivity in response to a stressor appears to be more strongly associated with psychopathic tendencies and aggressive behaviors than baseline EDA (Lorber, 2004). Thus, for these analyses EDA reactivity was calculated by subtracting the mean EDA reading during the neutral baseline film from the mean EDA reading during each of the affect inductions[2]. The relationship between aggression group, affect condition, and EDA reactivity was then assessed using a 2 × 2 × 2 × 2 mixed ANOVA with Affect Induction (change from baseline during positive video, change from baseline during negative video) as a within-subject variable and Aggression Group (sexually aggressive, nonaggressive), Negative Affect Condition (anxiety, sadness), and Order Condition (negative first, positive first) as between-subject variables.

There was a marginally significant main effect for Affect Induction, F (1, 70) = 3.99, p = .05, η_p^2 = .05, which must be interpreted in light of a significant Affect Induction by Negative Affect Condition interaction, F (1, 70) = 9.66, p = .003, η_p^2 = .12. Thus, in the negative affect condition, men who saw the anxiety-inducing video had greater EDA reactivity (M = 0.03; SD = .06) than men who saw the sadness-inducing video (M = −0.02; SD = .12), F (1, 76) = 5.68, p = .02, η_p^2 = .07. There was no difference between the two conditions in relation to the positive affect film, as would be expected giving that the positive affect induction was identical for the two negative affect conditions. There was a significant main effect for Aggression Group, F (1, 70) = 9.12, p = .004, η_p^2 = .12, with aggressive men demonstrating less reactivity across affect conditions (M = −0.01; SD = .09) than non-aggressive men (M = .04; SD = .05). However, there was also a significant Aggression Group by Negative Affect Condition interaction, F (1, 70) = 6.48, p = .01, η_p^2 = .09. For men who received the sadness-inducing film, the non-aggressive men actually demonstrated *increased* EDA across affect conditions as compared to baseline (M = 0.05; SD = .05), while the aggressive men demonstrated *decreased* EDA across conditions as compared to baseline (M = −0.04; SD = .11). Thus, the aggressive men who were assigned to watch the sadness video showed lower overall reactivity than the non-aggressive men who were assigned to watch the sadness video; this was a statistically significant difference, F (1, 31) = 9.01, p = .005, η_p^2 = .23. There were no significant difference in overall EDA reactivity scores between the aggressive and the non-aggressive groups who were assigned to watch the anxiety-inducing video. See Figure 3. All non-significant results had relatively small effect sizes ($\eta_p^2 \leq .05$). Thus, generally, the aggressive men showed less reactivity than the non-aggressive men; however, that finding was moderated by the type of negative

[2]We also ran the analyses examining EDA during the three separate affect inductions (baseline, positive, negative). There was no main effect for Aggression Group. There was a significant three-way interaction for Affect Induction, Aggression Group, and Negative Affect Condition, which essentially replicated the pattern found in the reactivity analyses reported here.

affect that was induced. Nevertheless, results of the EDA analyses were more consistent with the Hyporeactivity than the Hyperreactivity Hypothesis.

Discussion

In this study, sexually aggressive men demonstrated lower physiological reactivity than non-aggressive men, as measured by overall cortisol levels and EDA reactivity. On average, the aggressive men's physiological responses were consistent with the idea that their sexual aggression was more strongly associated with hyporeactivity than with hyperreactivity. Although there are a few exceptions, (e.g., Feilhauer et al., 2013; Gowen et al., 2013), cortisol and EDA hyporeactivity have been found in an abundance of studies to be related to psychopathy and antisocial behaviors (e.g., Holi et al., 2006; Loney et al., 2006; Lorber, 2005; O'Leary et al., 2007). In contrast, physiological hyperreactivity has been found to be related to negative affectivity, including trait levels of depression, anxiety, anger, and hostility (e.g., Kreibig, 2010; Pope & Smith, 1991; Steptoe et al., 2000). Thus, for the sexually aggressive men in our sample, their history of coercive and forceful sexual behavior appeared to be associated more with a lack of emotional depth and/or a lack of anxiety regarding consequences than with overwhelming and uncontrolled negative affect.

Although several researchers have found evidence of antisocial behaviors and psychopathic traits among community samples of sexually aggressive men based on self-report measures (e.g., Konsson et al., 1997; Lalumiere & Quinsey, 1996; Malamuth, 1986), this is the first study to our knowledge to demonstrate that sexually aggressive community men demonstrate a physiological pattern consistent with psychopathy. This is particularly notable given that the majority (60.5%) of our sexually aggressive group were men who had engaged only in relatively minor forms of sexual aggression that typically would not be illegal (i.e., use of verbal pressure or manipulation). This suggests that even non-forceful sexual coercion is part of a constellation of callous and aggressive sexual behaviors.

Although the results of our study clearly seem in line with the Hyporeactivity Hypothesis rather than the Hyperreactivity Hypothesis, there were some complexities related to our results that are worthy of further discussion. Not surprisingly, EDA reactivity in the negative affect condition was higher for participants receiving the anxiety induction than for participants receiving the sadness induction. Based on past research, anxiety appears to be a stronger and more consistent predictor of EDA reactivity than sadness; indeed, sadness has sometimes been observed to decrease EDA reactivity, but that is typically not found in cases in which the sadness-inducing stimulus involves imminent loss (Kreibig, 2010), as our particular video clip did. What is more noteworthy about our results is that the aggressive men demonstrated lower overall reactivity than the non-aggressive men only when they were assigned to the sadness induction condition and not when they were assigned to the anxiety induction condition. This finding might seem counterintuitive, as EDA underreactivity in individuals with psychopathy and antisocial behaviors often has been suggested to be associated particularly with fearlessness or low levels of anxiety (Fowles, 2000; Lykken, 1957); thus, we might expect the strongest group difference in the anxious rather than the sadness condition. However, lack of reactivity to our sadness-inducing stimuli, which depicted an individual—notably, a woman—facing a severe loss, is

consistent with the lack of empathy that would be expected among individuals with psychopathic traits and among sexually aggressive men.

Related to our findings, Blair (1999) and Blair, Jones, Clark, and Smith (1997) found that psychopathic men did not differ from non-psychopathic men in their EDA responses to threatening or neutral stimuli, but the psychopathic men demonstrated lower reactivity than the non-psychopathic men in response to distress cue stimuli (e.g., photos of people crying). Blair et al. (1997) noted that individuals with psychopathy consistently show lower than typical levels of reactivity when they anticipate *personal experiences* of threatening or adverse stimuli, but they do not consistently demonstrate reduced autonomic reactivity in response to direct visual presentations of threatening stimuli. Blair (1999) suggested several possible explanations for these findings, which extend the fearlessness theory of psychopathy to help explain the tendency for psychopathic individuals to show hyporeactivity to distress cues: (1) It may be that an innate fearlessness leaves children poorly equipped to learn empathy because fearless children may be unresponsive to parental affective cues. (2) It may be that both fearlessness and lack of responsiveness to distress cues reflect a shared neurological pathology. (3) It may be that a combination of fearlessness and lack of response to distress cues are necessary for the development of psychopathy, such that for individuals who are not responsive to distress cues, inhibition of violence may be able to be learned through fear of consequences unless the lack of responsiveness to distress cues is combined with fearlessness.

Consistent with the idea that psychopathy is associated with hypoarousal to distress cues, some studies have demonstrated that individuals with psychopathic traits have lower amygdala activation than controls when processing fearful facial expressions (Jones, Laurens, Herba, Barker, & Viding, 2009; Marsh et al. 2008). Interestingly, however, there is some evidence that, compared to controls, individuals with psychopathic traits and individuals at risk for aggressive behavior demonstrate exaggerated amygdala activation in response to angry facial expressions (Carre, Hyde, Neumann, Viding, & Hariri, 2013; Coccaro, McCloskey, Fitzgerald, & Phan, 2007). Thus, hyporeactivity to distress cues combined with hyperreactivity to anger cues may be particularly associated with psychopathy and aggression. In the future, researchers might benefit from examining amygdala activation in response to fearful and angry facial cues among sexually aggressive men.

Although our findings seem to support the Hyporeactivity rather than the Hyperreactivity Hypothesis, the possibility remains that there are two types of sexually aggressive men—a hyporeactive type, who may be high in psychopathic traits, and a hyperreactive type, who may be high in negative affectivity. Some authors (e.g., Hall & Hirschman, 1991; Ward & Beech, 2006) have proposed that there may be multiple factors that facilitate sexual aggression, such that some individuals may be motivated by strong negative affect (particularly anger) and others may be motivated by trait variables (e.g., psychopathy). Related to this possibility, researchers (McBurnett et al., 2000; Walker et al., 1991) have identified two types of conduct disordered boys—one with comorbid anxiety and high concentrations of cortisol and one without comorbid anxiety and low concentrations of cortisol; the latter group demonstrated more severe and persistent aggression than the

former. If there are two (or more) types of sexually aggressive men, it is possible that the physiologically aroused aggressive men may have self-selected out of our laboratory study due to their anxiety, leaving the aggressive men that are characterized by low arousal. This possibility could explain the fact that other self-report research (Peterson et al., 2013) has found high levels of trait anxiety among some sexually aggressive men, a fact that seems at odds with the Hyporeactivity Hypothesis and with the physiological findings reported here. Future research could compare sexually aggressive men who are high and low in trait anxiety in terms of their physiological arousal responses.

It is also worth noting that, although the aggressive men in our study demonstrated lower physiological reactivity than the non-aggressive men, the groups did not differ in terms of self-reported negative affect. This raises at least two possibilities: (1) The aggressive men may be experiencing a greater disconnect than the nonaggressive men between their subjective sense of affect and their physiological responses to affect. (2) The aggressive men may be reporting higher levels of negative affect than they are actually experiencing in an attempt to meet demand characteristics. Future research potentially could examine both possibilities.

Future research also would benefit from directly measuring antisocial or psychopathic traits in sexually aggressive men and examining how those traits relate to physiological reactivity. Although a preponderance of research suggests a negative association between psychopathy and physiological reactivity, some studies provide evidence for a more complex relationship. For example, examining the relationships among intimate partner violence, EDA reactivity, and antisocial traits, Babcok, Green, Webb, and Yerington (2005) found that for men who engaged in severe violence against their intimate partners and for men who engaged in no violence against their partners, there was a negative relationship between EDA reactivity and antisocial behavior; however, for men who engaged in low level violence against their intimate partners, there was a positive relationship between EDA and antisocial behavior. Thus, it is possible that physiological reactivity has different correlates in men who obtain nonconsensual sex using verbal manipulation than in men who obtain nonconsensual sex using physical force.

Several methodological limitations should be acknowledged. First, it is important to note that we examined only men who sexually aggressed against women in this study. Some men perpetrate sexual aggression against other men, and some women perpetrate sexual aggression against men and women (e.g., Krahe & Berger, 2013); our results may not be generalizable to these other sexually aggressive individuals. More research is needed to explore physiological responses in a more diverse sample of sexually aggressive men and women. Our sample also was relatively small, and several men who completed our laboratory study were not included in the analyses due to technical or experimenter error. Further, although our negative-affect-inducing video appeared to be quite successful at inducing negative emotional states, our positive-affect-inducing video was less successful, as it did not produce greater positive affect than the neutral baseline video. Additionally, the saliva used for cortisol measurements was collected at variable times of the day. Although we did statistically control for time of collection in our analyses, it would have been ideal to have collected samples at a consistent times in relation to participants' awakening. Finally,

these data are part of a larger laboratory study that involved additional stimuli and tasks which may potentially have impacted the results presented here; however, this possibility was minimized by the return-to-baseline period between the conditions and by the fact that the additional tasks and stimuli were identical for the aggressive and non-aggressive groups. Additionally, the fact that we did not find any significant order effects (or any moderate to large effect sizes for order effects) provides some support for the idea that the erotic videos and additional tasks that were involved in this study did not contaminate the results; if the additional tasks had impacted the results, we would have expected a significant Order by Affect Induction interaction, because the first affect induction would presumably have been uncontaminated and the second affect induction would have been contaminated.

Despite the limitations of this study, it represents a first attempt at identifying a pattern of physiological arousal in sexually aggressive men in the community. This study in combination with future research that examines the physiological correlates of men's sexual aggression may contribute to researchers' understanding of the etiology of sexual aggression and eventually may help clinicians to better identify boys and men who are at risk for sexually aggressing in the future.

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Order Condition 1: Negative affect induction followed by positive affect induction (n = 41)

Orientation to lab and placement of devices	Neutral video (baseline EDA measured)	PANAS and **Time 1 saliva sample collection**	Negative affect video anxiety-inducing video (n = 22) OR sadness-inducing video (n = 19)	PANAS and **Time 2 saliva sample collection**	Erotic video	Self-report measures and additional laboratory tasks	Neutral video	**Time 3 saliva sample collection**	Positive affect video	PANAS	Erotic video	Self-report measures and additional laboratory tasks	Post-experimental interview
10 minutes	5 minutes	2 minutes	5 minutes	2 minutes	3 minutes	5 minutes	10 minutes	1 minute	5 minutes	2 minutes	3 minutes	5 minutes	10 minutes

Order Condition 2: Positive affect induction followed by negative affect induction (n = 37)

Orientation to lab and placement of devices	Neutral video (baseline EDA measured)	PANAS and **Time 1 saliva sample collection**	Positive affect video	PANAS	Erotic video	Self-report measures and additional laboratory tasks	Neutral video	Negative affect video anxiety-inducing video (n = 23) OR sadness-inducing video (n = 14)	PANAS and **Time 2 saliva sample collection**	Erotic video	Self-report measures and additional laboratory tasks	Post-experimental interview	**Time 3 saliva sample collection**
10 minutes	5 minutes	2 minutes	5 minutes	2 minutes	3 minutes	5 minutes	10 minutes	5 minutes	2 minutes	3 minutes	5 minutes	10 minutes	1 minute

Figure 1.

Laboratory procedures in each of the two order conditions. Procedures relevant to data presented here are highlighted. Participants were randomly assigned to an order condition and to a negative affect condition (i.e., anxiety-inducing or sadness-inducing video).

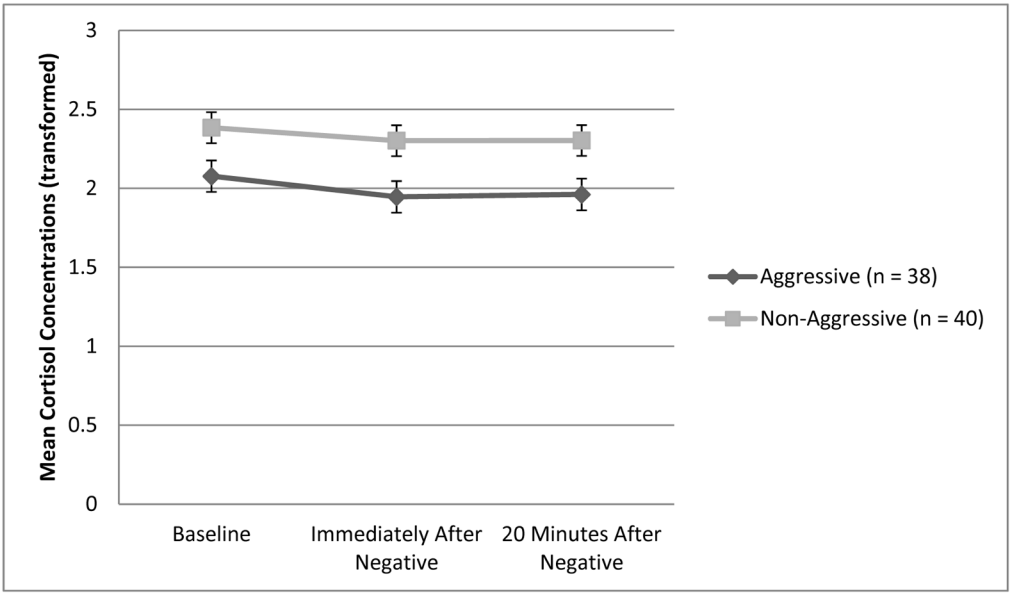

Figure 2.

Mean cortisol levels as a function of Time Point and Aggression Group. Error bars represent standard errors.

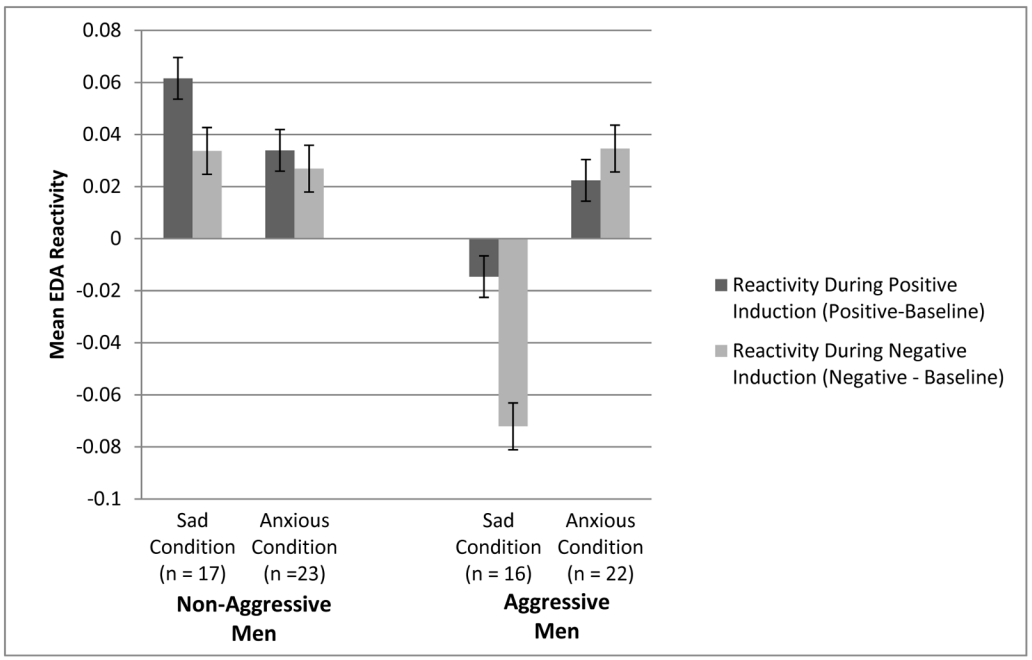

Figure 3.

Mean EDA reactivity as a function of Affect Induction, Negative Affect Condition, and Aggression Group. Scores above 0 indicate greater responsivity in the affect induction conditions than in the neutral baseline condition (i.e., high reactivity). Scores below 0 indicate greater responsivity in the baseline condition than in the affect induction conditions (i.e., low reactivity). Error bars represent standard errors.