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Autonomic Reactivity In Emerging Adulthood : Relationships Between Cyberbullying , The Autonomic Nervous System, And Clinical Outcomes.

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AUTONOMIC REACTIVITY IN EMERGING ADULTHOOD : RELATIONSHIPS BETWEEN CYBERBULLYING , THE AUTONOMIC NERVOUS SYSTEM, AND CLINICAL OUTCOMES.

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Abstract

With the advent of the COVID-19 pandemic, online environments have become critical ways of connecting among college students. With the increase in online interactions, cybervictimization has been identified as a public health issue. This study aims to examine whether cybervictimization among college students is associated with clinical adjustment outcomes such as depression and alcohol consumption. This study also aims to examine whether reactivity in the two branches of the autonomic nervous system (sympathetic and parasympathetic) jointly moderate the relationship between cyberbullying and depression/alcohol consumption, indicating sensitivity to the environment. Participants (n =164, 69% female, M age = 19.92, SD = 1.42; 65% White) completed a stress task in the laboratory, during which participants' ANS reactivity was continuously monitored, and reports of depression and alcohol consumption were obtained. Results indicated that cyberbullying predicted higher levels of both depressive symptoms and alcohol use. Results also showed that PNS and SNS reactivity did not jointly moderate the relationship between cyberbullying and depressive symptoms or alcohol consumption. However, parasympathetic reactivity moderated the relationship between cyberbullying and alcohol consumption. This study corroborates prior research on the relationship between online environmental stressors and clinical outcomes. The results also support the notion that physiological factors are important to our understanding of how social stressors affect clinical outcomes in college students.

Keywords: autonomic nervous system, emerging adults, alcohol consumption, depressive symptoms, polyvagal theory.

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Introduction

Not all stress is equal, and not all individuals are susceptible to stress in the same way. Indeed, individuals with different biological characteristics do not respond to negative or positive environments in the same way. Multiple theories have explored how individual biological differences and the environment interact to predict developmental outcomes. The *diathesis stress model* (Monroe & Simons, 1991) suggests that some individuals may be more susceptible to stressful environments than others. In this model, individuals with a genetic or biological vulnerability are more vulnerable to harsh environments, leading to maladaptive outcomes. In other words, poor experiences/environments will only affect those with pre-existing biological vulnerabilities (Roisman et al., 2012). The *differential susceptibility* theory (Belsky, 1997; Boyce & Ellis, 2005), on the other hand, postulates that vulnerability factors may instead be considered as plasticity factors. Children with a plasticity factor are susceptible "for better" or "for worse" functioning (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007): these children thrive in and benefit from positive environments and exhibit adverse functioning in negative contexts. That is, it is true that children with plasticity factors (referred to as vulnerability factors in the diathesis stress model) are sensitive to harsh environments, but those same children will flourish when placed in positive environments. Children who lack plasticity factors are not necessarily impervious to the environment. They are, however, less reactive; for those children, the effects of a positive or adverse environment are less pronounced compared to their peers who exhibit a "for better or for worse" pattern. These children exhibit a more modest response to positive and negative environments. In both the *diathesis-stress*

model and *differential susceptibility* theories, environmental stressors interact with an individual's physiology to predict their social, psychological or emotional functioning.

With the advent of technology, new stressors have emerged that are affecting individuals in online environments, such as cyberbullying (Bradbury et al., 2018). Cyberbullying is defined by a range of behaviors that communicate hostility with the intention to harm the receiver using social digital media such as SMS, apps, or social media (Langos, 2012; Slojne et.al, 2008, Tokunaga, 2010; Hinduja et al, 2009; Patchin et. al, 2006). The term cybervictimization refers to being victimized or bullied through the use of digital social media (Slonje et al., 2008; Cook et al., 2007). Cybervictimization can be indirect (e.g. in public forums where material may not be seen by the victim) or direct (e.g. sending text messages to the victim) and may be characterized by repetitive behavior and a power differential between the bully and the victim (Langos, 2012; Piccoli et al., 2020). Cyberbullying is now considered a public health concern among children and adolescents by the Center for Disease Control (Aboujaoude et. al, 2015). The National Crime Prevention Council and Harris Interactive have reported that more than 40% of American adolescents are cybervictims (Bhat, 2008). Cyberbullying victimization prevalence rates in other studies range from 1.0% to 61.1% (Piccoli et al., 2020).

Previous literature has shown that cybervictimization affects victims' mental health regardless of their ages: negative clinical outcomes have been found in adolescents (Bottino et al., 2005; Rose & Tynes, 2015) and adults (Brack & Caltabiano, 2014). Cybervictimization is associated with negative mental health outcomes such as lower self-esteem, higher levels of anxiety, depression, suicidal ideation and substance use

(Sibold et al., 2020; Rose & Tynes, 2015; Patchin & Hinduja, 2010, Ybarra et al., 2006; Wright; 2016; Gini & Espelage, 2014; Gamez-Guadix et al., 2013). In fact, Bonnano & Hymel (2013) showed that being involved in cyberbullying as a perpetrator or victim predicted internalizing symptoms of depression and suicidal ideation above and beyond traditional forms of bullying. Cyberbullying thus constitutes a salient social stressor for young people that can have unique effects on their adjustment and functioning.

College students find online relationships particularly relevant to their lives. A study by Manago et al. (2012) emphasized the importance of online platforms (e.g. Facebook) to college students socially; social networking helps satisfy enduring psychosocial needs in a world in which social relationships are constantly geographically moving. Facebook, in the study, was perceived as a tool for social support. College students also use social media to express political views, organize politically (Kushin & Yamamoto, 2013) and express identity (Alruwaili & Ku, 2020). College students have reported high rates of using social media and websites as a form of communication. According to a study by Sheldon (2008), more than 50% of college students use a social networking website multiple times daily. Quan-Haase & Young (2010) found that 82% of college students log on Facebook more than once a day. Furthermore, Sponcil & Gitimu (2013) reported that 59.4% of their student sample visited social media sites multiple times a day.

With the rampant use of social media and other texting applications, there is a higher chance for college students to be exposed to negative experiences online such as cyberbullying. It is important to examine how cyberbullying (a social stressor) can affect

clinical outcomes that are relevant to college students such as depression and alcohol use. Though previous studies have linked cyberbullying to negative clinical outcomes such as depression, anxiety, and substance use, the effect sizes are small to moderate. This suggests that there are individual differences in how college students react to cyberbullying.

In this study, I use the differential susceptibility framework to examine individual differences in the associations between cybervictimization and the clinical outcomes of depressive symptoms and alcohol use in an emerging adult college student population. Specifically, I aimed to examine whether higher rates of cybervictimization predicts higher depressive symptoms and alcohol use, and whether this relationship is moderated by autonomic nervous system reactivity to stress. I examined the questions: (a) is there a pattern of autonomic nervous system reactivity that confers vulnerability or protection to cyberbullying stress for college students? (b) is this pattern consistent with the differential susceptibility model?

The Autonomic Nervous System

The autonomic nervous system (ANS) regulates organ functions to bring the body to homeostatic balance (Jänig, 2008). It also contributes to emotion regulation processes in high stress situations (Porges, 2007, 2011; Kreibig, 2010; Levenson, 2014). The ANS is divided into two branches: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS), which interact dynamically (Fox, 1994).

The PNS controls involuntary responses that allow the body to "rest and digest", thus conserving body energy. For instance, the PNS can slow heart rate or respiration.

The PNS has been shown to be involved in cardioprotective processes (Donato et al., 2013) and emotion regulation (Beauchaine, 2015). Vagus nerve activity has been widely used in previous research as an indicator of PNS activity (Fox, 1994). The vagus nerve is the tenth cranial nerve originating in the nucleus ambiguus part of the brain stem. When the vagus nerve increases its input, the PNS becomes more activated and the body engages in more "rest and digest" functions (e.g. decelerating heart rate and decreasing autonomic arousal; Fox, 1994). In stressful situations, the vagus nerve "withdraws", meaning that it decreases input to the heart. In this case, there is a release of the "vagal brake," which leads to heartbeat acceleration and autonomic arousal (Porges 2007, 2011). When the "vagal brake" is withdrawn (like lifting one's foot off of a brake pedal), the PNS activity decreases and the SNS is recruited for a fight or flight response. When the vagal brake is activated (like pressing down on a brake pedal), the vagal nerve is stimulating rest and digest functions.

The proposed study examined respiratory sinus arrythmia (RSA) as a measure of PNS activity. RSA represents a complex interaction between respiratory and cardiovascular systems (Grossman & Taylor, 2007; Fox, 1994). Specifically, RSA measures cyclical changes in heart beats that correspond to different phases of respiration (expiration vs. inspiration). RSA reactivity (RSAR) represents the RSA response to stress (Porges, 2007). RSA withdrawal refers to when the PNS decreases arousal in response to stressors, which is also referred to as PNS withdrawal. RSA augmentation refers to the activation of the PNS after a stressful event (Porges, 2007).

Previous research suggests that moderate levels of PNS withdrawal is adaptive (Marcovitch et al., 2010). It facilitates attention, metabolic and regulatory activities (Porges, 2007, 2011; Graziano & Derefinko, 2013). When the vagus nerve withdraws, the organism exhibits preparedness to respond to high environmental demands. Consequently, any problems with vagal withdrawal may underlie children's abilities to cope effectively with stress (Mezzacappa et al., 1997; Pine et al., 1998), leading to internalizing or externalizing problems (Thayer & Lane, 2000*)* and social or academic issues (Blair & Peters, 2003). In the case of extreme vagal withdrawal, the organism becomes hypervigilant, which is metabolically taxing for the body and anxiety provoking (Thayer & Lane, 2000). In addition, too little vagal withdrawal is considered maladaptive (Schmitz, Kramer, Tuschen-Caffier, Heinrichs & Blechert, 2011), restricting the organisms' ability to mobilize its resources and deal with stress.

A recent metanalysis by Graziano and Derefinko (2013) suggested that PNS withdrawal yielded fewer internalizing and externalizing problems in both community and clinical samples. This finding however, had a small effect size. One crucial difference was that PNS withdrawal was associated with fewer social problems in community samples but with more social problems in clinical/at-risk children. These mixed results could be due to the fact that at-risk children may already have higher levels of baseline PNS withdrawal compared to community samples, which may exacerbate symptoms. Previous studies have also found that PNS activity was different between healthy and autistic children or children exposed to opiates during fetal development (El Sheikh et al., 2009; Toichi & Kamio, 2003; Hickey, Suess, Newlin, Spurgeon, & Porges,

1995). A recent metanalysis on adult samples (Beauchaine et al., 2019) showed that RSA withdrawal significantly moderated externalizing symptoms. However, the paper reported considerable heterogeneity in findings of the effects of RSA for different psychopathologies. Consequently, previous research suggests that the effects of PNS withdrawal on psychopathology are not fully understood, as the results differ between samples.

The sympathetic nervous system (SNS), in contrast, is responsible for fight or flight responses when the stressor is extreme or prolonged. The SNS prepares the body to flight or flee by mobilizing metabolic resources, and increasing heart rate, respiration rate and perspiration (Boucsein, 1992; Porges, 2011). In this study, I looked at skin conductance level (SCL) as a measurement of SNS activity. SCL quantifies electrodermal activity, as sweat glands are innervated exclusively by the SNS (Dawson, Schell, & Filion, 2007). SCL reactivity (SCLR) reflects the change in SCL activity from baseline, after the occurrence of a stressor or challenge. High SCLR thus represents increased SNS reactivity, and low SCLR represents blunted SNS reactivity.

Previous research established that lower SCL baseline levels were associated with disruptive behaviors (van Goozen, Matthys, Cohen-Kettenis, Buitelaar & van Engeland, 2000), and higher levels of externalizing and conduct problems (El Sheikh et al., 2009; Van Bokhoven et al., 2005; Beauchaine et al., 2007; Crowell et al., 2006). Blunted SCLR is an indicator of poor behavioral and emotional inhibition as well as aggressive and antisocial behavior in children and adolescents (Erath et al., 2011; Gregson, Tu, & Erath, 2014; see Lorber, 2004 for a meta-analysis). In addition, heightened SCLR has been

associated with internalizing problems in children and adolescents (Weems, Zakem, Costa, Cannon, & Watts, 2005; Kagan, Reznick, & Snidman, 1987; El-Sheikh, 2005) such as anxiety and depression symptoms (El-Sheikh, 2005) and externalizing problems (Hubbard et al., 2002; El-Sheikh, 2005; Beauchaine, 2001; Erath, El-Sheikh, Hinnant, & Cummings, 2011; Posthumus, Bocker, Raaijmakers, Van Engeland, & Matthys, 2009).

Previous research on RSA and SCL reactivity suggests a discrepancy in the literature on whether blunted or heightened psychophysiological reactivity (RSAR and/or SCLR) are better predictors of psychopathology. In this study, I hoped to clarify whether the interaction between SNS and PNS can help to make sense of these findings.

Theories of the Autonomic Nervous System

One limitation in psychophysiology research is that most studies consider the effect of one branch of the ANS (SNS or PNS) at a time. The Polyvagal and Doctrine of Autonomic Space theories argue in favor of considering both branches of the autonomic nervous system in interaction and studying how this interaction affects clinical outcomes of functioning and adaptation to the environment.

Polyvagal Theory

The Polyvagal theory, developed by Porges (1995), steers away from a dichotomous understanding of the SNS working against the PNS, instead proposing that the autonomic system is comprised of two branches: an older phylogenetic branch that is reptilian and unmyelinated, promoting freezing behaviors after danger, and another newer

branch that is myelinated and that connects to facial muscles and vocal strings, promoting social engagement.

Porges explains that the SNS does not work antagonistically to the PNS, an idea that has been widely used in our previous understanding of the ANS. Porges proposes that the SNS is one *level* of response to environmental stressors. The first neural circuit that responds to the environment is the ventral vagal complex, which involves social engagement when threat or stress level is low to moderate. When threat or stress increases and the social engagement circuit is overwhelmed, the ANS thus recruits older circuits and begins mobilization of the fight or flight response via the SNS. In this capacity, the SNS leads to increased heart rate, sweating, dilating pupils and inhibiting homeostasis.

According to polyvagal theory, vagal influence on the heart is an adaptive process allowing the nervous system to respond to environmental demands. The vagal brake may thus be withdrawn and reinstated in order to adapt to perceived stress conditions. According to Porges, the systems that protect humans from danger, and the systems of social engagement and communication are interrelated neurophysiological processes that involve both the PNS and SNS. Porges' theory stresses that the physiological response to stress does not operate in a linear fashion. Instead, it is characterized by a complex "blending of physiological activity" (El Sheikh et al., 2009, p.14).

Doctrine of Autonomic Space

Bernston, Caccioppo, & Quigley (1991) proposed a new framework to study how the PNS and SNS interact together. Bernston et al. (1991) proposed that the PNS and

SNS can interact in a coordinated or an uncoordinated manner. According to the doctrine of autonomic space, PNS and SNS activation can be a) *reciprocal SNS activation,* in which SNS activity is high and PNS activity is low, b) *reciprocal PNS activation*, which involves increased PNS activity and decreased SNS activity, c) *coinhibition,* which represents blunted activity for both PNS and SNS, and d) *coactivation,* where PNS and SNS are both activated.

Newer research (e.g. Abaied et al., 2018; Lafko et al., 2015; El-Sheikh et al., 2009) has applied polyvagal theory and the Doctrine of Autonomic Space to explore how both ANS branches work together and interact with the environment to predict adjustment outcomes in youth.

Reciprocal Sympathetic and Parasympathetic Activation

El-Sheikh and colleagues have examined how some ANS profiles may be more adaptive than others. According to El- Sheikh et al. (2009), reciprocal sympathetic activation is an adaptive response to stress because it involves sympathetic activation and parasympathetic inhibition; this leads to a net increase in arousal, and thus provides the metabolic output necessary for taking action in challenging situations. In contrast, El Sheikh (2009) considered reciprocal parasympathetic activation as most adaptive in situations in which "a calm physiological state is beneficial" (p. 12). That is because, in the case of combined sympathetic inhibition and parasympathetic activation, there is a net decrease in arousal, which is appropriate for calm situations (El Sheikh, 2009). In case of high danger, when fighting or fleeing is necessary, reciprocal parasympathetic arousal is not helpful, as it does not provide metabolic resources for action. However, instead of

thinking of different profiles as adaptive or non-adaptive responses to stress as conceptualized by El Sheikh (2009), we may examine these profiles from a differential susceptibility or diathesis stress perspective, in which these profiles interact with different environments to predict positive or negative outcomes.

Reciprocal Activation and Environmental Sensitivity

Reciprocal SNS activation has been shown to confer sensitivity to the environment. In a study of adolescent girls, Lafko et al. (2015) found that unpopularity and rejection were associated with increased relational victimization for girls with a reciprocal SNS activation pattern. In line with the differential susceptibility model, girls with a reciprocal SNS activation profile were more sensitive to the environment for "better" or for "worse". Conversely, for girls exhibiting reciprocal PNS activation, Lafko et al. (2015) found that social rejection was associated with subsequent victimization. However, this effect was weaker for girls with a reciprocal PNS profile. These results indicate that girls with a reciprocal PNS profile were less sensitive to the effects of unpopularity and rejection compared to their peers with a reciprocal SNS profile. In an adolescent sample, Abaied et al. (2018) found that adolescents with a reciprocal SNS activation pattern exhibited more sensitivity to both maternal involvement and psychological control. In a positive environment (maternal involvement), adolescents with a reciprocal SNS pattern exhibited lower depressive symptoms and higher emotion regulation; in a negative environment (maternal psychological control), they exhibited higher depressive symptoms and lower emotion regulation. Conversely, adolescents with coinhibition or coactivation patters remained unreactive to maternal involvement and

psychological control. Quas, Bauer, and Boyce (2004) experimentally manipulated environments of supportiveness by interviewers in the lab to predict children's memory performance. They similarly found that children with a reciprocal SNS activation performed better on the memory task in positive supporting environment and had worse memory performance in the negative low support environment, consistent with the differential susceptibility model.

Breslend et al. (2018) found that, among teenage girls, relational victimization was positively associated with anxiety, depression and anxious rejection sensitivity for girls who exhibited both reciprocal SNS and PNS activation. This study may suggest that having a reciprocal SNS or PNS profiles indicates heightened sensitivity to the environment: individuals with these patterns are negatively affected by the environment when the environment is deleterious. However, most studies (except for Abaied et al., 2018 and Quas et al., 2004) did not compare the functioning of individuals in both positive and negative environments, which would provide us with a clearer idea of whether these profiles indicate differential susceptibility to the environment.

Other studies in the literature found contradictory results that suggest that reciprocal PNS and SNS activation were not consistently sensitive to different types of environments. Wagner and Abaied (2015), in a college student sample, found that relational victimization was not associated with relational aggression for emerging adults who showed reciprocal SNS activation. Thus, in the Wagner and Abaied paper (2015) this profile did not confer sensitivity to the environment following a differential susceptibility model. However, emerging adults with reciprocal PNS activation were

more sensitive to the environment; participants exhibited high relational aggression in a negative environment (high relational victimization), and low relational aggression in a positive environment (low relational aggression). El Sheikh et al. (2009) found that having reciprocal PNS and SNS activation was protective for children in negative marital conflict environments. Specifically, marital conflict was not associated with externalizing behaviors for children with reciprocal SNS and PNS profiles. These results were replicated across three samples with similar results.

Although there are some inconsistencies in the literature, the pattern that we observe reveals that reciprocal SNS activation is often an indicator of sensitivity to the environment, consistent with the differential susceptibility framework, but there is also some evidence that reciprocal PNS activation can indicate sensitivity to the environment as well.

Coactivation and Coinhibition

Bernston et al. (1991) proposed that coactivation and coinhibition responses do not support an adaptive, organized, voluntary response to stress, but rather represent an ambivalent and maladaptive response. According to polyvagal theory, coactivation (i.e., simultaneous PNS and SNS activation) may reflect a sympathetic "override" of the parasympathetic response (El Sheikh, 2009; Porges, 1995, 2001); thus, it is possible that coactivation stimulates "fight-or-flight" responses of anger and dysregulation when faced with a stressor (El Sheikh, 2009). In the case of coinhibition (i.e., simultaneous low PNS and SNS activation), this response allows the parasympathetic branch to withdraw inhibitory defenses while the sympathetic branch fails to metabolically produce enough

resources to fight stress. Coinhibition may thus promote passive vigilance (El Sheikh, 2009).

Indeed, coinhibition responses have been found in children with externalizing behavior problems (Quas, Bauer & Boyce, 2004; Beauchaine, 2007). Coactivation is also associated with emotion dysregulation (Berntson et al., 1991; El-Sheikh et al., 2009), emotional reactivity such as anger and aggression (Scarpa & Raine, 1997) and externalizing outcomes (Nederhof et al., 2015).

Coactivation and coinhibition and sensitivity to the environment

Previous literature has examined coactivation and coinhibition as potential indicators of sensitivity to the environment. In an emerging adult sample, Wagner and Abaied (2015) found that relational victimization was significantly associated with higher reactive relational aggression for individuals who showed ANS coactivation or coinhibition, suggesting that these ANS profiles can confer sensitivity to the environment, indicating a "for better" or "for worse" functioning. Similarly, in another study on children, El-Sheikh et al. (2009) showed that marital conflict predicted higher externalizing symptoms for individuals with coactivation or coinhibition patterns. Consistently, Gordis, Feres, Olezeski, Rabkin, and Trickett (2010) found that coinhibition (conceptualized as low baseline RSA and SCL reactivity) and coactivation (conceptualized as high levels of baseline RSA and SCL reactivity) strengthened the link between childhood maltreatment and aggression in children and adolescents. Suurland et al. (2018) recently found that, in toddlers, cumulative prenatal risk (measured in mothers) predicted elevated physical aggression and non-physical aggression/oppositional

behavior in toddlerhood. This relationship was moderated by PNS and SNS functioning, such that for toddlers exhibiting greater coinhibition or coactivation in response to challenge, higher cumulative risk predicted higher levels of physical aggression. Furthermore, Holterman et al. (2016) showed that, in emerging adults with coactivation and coinhibition patterns to both social and non-social stressor tasks, relational victimization was positively associated with depressive symptoms. Looking at adolescents, Philbrook et al. (2018) found that in high marital conflict environments, coactivation and coinhibition predicted negative outcomes such as increased internalizing symptoms over time.

Overall, the consistent pattern in the literature is that coactivation and coinhibition are often vulnerability factors: individuals with these profiles are not protected from negative environments. One study by Abaied et al. (2018) found that adolescents with a coinhibition or coactivation patterns remained unreactive to maternal involvement and psychological control. Lafko et al. (2015) showed that for girls with coactivation and coinhibition profiles, there was no significant difference in relational victimization when comparing positive and negative social environments (unpopularity and rejection). Thus, coactivation and coinhibition profiles were not sensitive to positive and negative iterations of the social environment. In this study, I hope to elucidate whether coactivation or coinhibition confers insensitivity to the environment.

The current study

Building upon prior theory and research, I examined whether cyberbullying predicted clinical outcomes of depression and alcohol use in an emerging adult sample,

and whether ANS reactivity moderated this relationship. In particular, I explored whether specific ANS profiles are more sensitive to the adverse effects of high cyberbullying and beneficial effects of low cyberbullying.

I proposed four hypotheses:

1) Cyberbullying will predict higher rates of depressive symptoms and alcohol use in college students.

2) The three-way cyberbullying x SNS reactivity x PNS reactivity will significantly predict higher levels of depressive symptoms and alcohol use.

3) Decomposition of the three-way interaction will reveal that college students with reciprocal SNS and PNS activation will be sensitive to cyberbullying consistent with a differential susceptibility model: students will exhibit higher symptoms in a high cyberbullying environment and lower symptoms in a low cyberbullying environment. I predicted that this effect is more pronounced for students with a reciprocal SNS profile compared with their reciprocal PNS profile peers.

4) Cyberbullying will not predict symptoms of depression or alcohol use for college students with coactivation or coinhibition patterns of reactivity.

Methods

One-hundred and ninety-one college students participated in the study; due to physiological equipment malfunctions, data for the current project was available for 164 participants (83.5% Caucasian, 7.9 % Asian; 3.7% Hispanic; 4.9 % Other). Participants' age ranged from 18-25 years (M= 19.92, SD= 1.42). Gender was not equally distributed with 51 men and 113 women participating in this study.

Procedure

Families were recruited via the SONA recruiting system at the University of Vermont. Students received credit towards their research requirements for completing a 3-hour laboratory visit. Participants who arrived at the lab were informed of the procedures and provided informed consent prior to participation.

Measures

Psychophysiology Markers

Skin conductance, heart rate, and respiration were continuously monitored throughout baseline and stress tasks. The data acquisition system includes a Pentium computer, Snapmaster software, and a bioamplifier. We used James Long equipment to measure skin conductance, respiration and heart rate at a sampling rate of 1,000 samples per second.

Heart Rate was assessed using an electrocardiogram (ECG). This noninvasive technique consists of placing three electrodes on participants' torso: two electrodes were placed on the top part of the torso below the collarbone, and one electrode was placed below the rib cage. Respiration was assessed using a respiration belt placed around the waist. Research assistants used the James Long Company's IBI Analysis software to process R waves in the ECG signal. We manually edited and cleaned problematic R waves. Using the Bernston et al. (1997) "peak to valley" method, we calculated RSA values (reported in units of ln(ms)^2) as the difference between the minimum interbeat

intervals (IBI) during inspiration and the maximum IBI during exhalation. IBI refers to the millisecond interval between two consecutive R wave peaks (i.e. two heart beats).

Skin conductance level (SCL) was measured using two electrodes attached to the middle volar surfaces of participants' index and middle fingers on the non-dominant hand. Participants washed their hands before attaching the skin conductance electrodes. We placed isotonic gel on the electrodes to increase conductivity and then attached electrodes to fingers using double-sided adhesive collars. Skin conductance level was measured in microsiemens.

Physiological data was collected continuously, throughout all baseline and stressor tasks. To measure baseline RSA and SCL, I averaged physiological data across a three-minute baseline period during which participants were instructed to sit quietly and relax. To calculate RSAR, I subtracted the mean baseline RSA from the mean RSA during the stressor task. To calculate SCLR, I subtracted the mean baseline SCL from the mean SCL during the stressor task. I manually searched for RSAR and SCLR outlier data, defined as cases that were above or below 3 standard deviations from the respective mean. I winsorized these cases in order to reduce the effect of extreme values on my analysis. Before analyzing the data, missing cases on any of my variables were excluded from my analysis, resulting in a final 164 data points to be analyzed.

Tasks

Subjects participated in three stress tasks. The order of stressor tasks was counterbalanced between participants. One of the stress tasks was The Trier Social Stress Test (TSST), a public speaking task in which participants were asked to speak about a

subject of their choice for five minutes while being videotaped. Participants were told that the videotape would be reviewed by a panel of judges. The goal of this task was to elicit social stress (Dickerson & Kemeny, 2004). The Trier Social Stress Test (TSST) is widely used and has been shown to reliably elicit ANS stress reactivity (Allen et al., 2014). Obradovic, Bush & Boyce (2011) showed that the interaction between ANS reactivity and the environment (marital conflict) was a significant predictor of children's behavior issues; however, the direction of the effect varied with the nature of the challenge task (i.e., interpersonal or cognitive). Consequently, the nature of the lab stressor can have an effect on outcomes. I selected this social stress task since it is relevant to the domain of stress I am examining in the social environment; choosing a social stress is a reasonable approximation of participants' sensitivity to social stress in particular. Both the social environment (cyberbullying) and the social stress task involve a socially evaluative and judgmental component.

At the end of the study, participants were debriefed about the study and were told that there was not a panel of judges that evaluated their speech, as they were led to believe. For the purposes of my project, the two other stress tasks will not be examined. The study included a distractor task before each stress task in which participants viewed a slide show of nature videos.

Questionnaires

After the laboratory visit, participants were asked to complete a series of questionnaires. I will focus on three questionnaires in this project. Participants completed other questionnaires that will not be analyzed in this project.

Cyber Victimization. (CVQ; Study name "My Experiences with Social Media II"; adapted from Calvete, Orue, Estevez, Villardon, Padilla, 2010). Participants completed an adapted reworded version of the cyberbullying questionnaire which contains 14 questions. We used the passive tense in our reworded versions to clarify experiences of cybervictimization as opposed to cyberbullying in the original version. Participants were asked the initial question "How often have you had these experiences happen to you?" Example items included "threatening or insulting messages sent by e-mail" and "someone got your password and sent email messages to others in your name". Participants reported on the extent of cybervictimization they have been exposed to using a 3-point scale (0 = never, 1 = sometimes 2 = often). Scores were calculated by adding the scores for all items. In this sample, the CVQ had an internal consistency of 0.73.

Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977). The CES-D contains 20 items that assess depressive symptoms. Using a 4-point scale (0=rarely or none of the time, 1=some of a little of a time, 2= a moderate amount of time, 3=most or all of the time), participants were asked to rate how often they have experienced depressive symptoms in the previous week. Examples included "I felt depressed," "I enjoyed life," and "I had crying spells". Five items are reverse coded. In this sample, the CES-D had a high internal consistency of 0.90. CES-D scores were calculated by calculating a sum of the items. A score of 16 or higher on the CES-D indicates high depressive levels (Lewinsohn, Seeley, Roberts, & Allen, 1997).

Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, Fuente, Grant, 1993). The AUDIT is a 10-item questionnaire developed by the World

Health Organization. The objective is to identify harmful or hazardous alcohol consumption. The AUDIT comprises an alcohol consumption subscale including 3 items that assess the frequency and amount of drinking. In addition, the AUDIT includes a 7-item alcohol problem subscale, assessing negative consequences of drinking (e.g., alcohol-related injuries). In this sample, the internal consistency of the AUDIT was 0.86. Scores were calculated as a sum of the items. A standard cutoff of 8 places individuals in the risky category (Conigrave, Hall, & Saunders, 1995).

Statistical Analyses

I ran two hierarchical multiple regressions to investigate my hypotheses. Sex, age, race, Baseline SCL and RSA were entered as covariates in the first step of the model. Cyberbullying, SCLR and RSAR were entered in the second step of the model. In the third step of the model, I entered the two-way RSAR x SCLR, Cyberbullying x SCLR, and Cyberbullying x RSAR interactions. Finally, the last step of the model included the three-way Cyberbullying x SCLR x RSAR interaction. Following the Aiken and West (1991) procedure, all predictors in the model were standardized. I decomposed my interaction by computing simple slopes at low (-1 SD) and high (+ 1 SD) levels of SCLR and RSAR (Aiken & West, 1991). I graphed my results using the Jeremy Dawson template.

To alleviate skew and kurtosis in the variable distribution, I transformed all variables. I computed the square root and logarithmic functions for all variables, and then examined the resulting skew and kurtosis statistics after transformations. I also added a

constant of 1 to variables before transforming them to fix any problems with transformations due to negative values. After examining skew and kurtosis statistics, I determined that a logarithmic transformation was suitable for the cybervictimization and RSAR variables. I ran a square root transformation on all other variables.

Results

Descriptive statistics for the study variables appear in Table 1, and intercorrelations appear in Table 2.

Cybervictimization x RSAR x SCLR predicting depressive symptoms.

A hierarchical regression model was used to test the hypothesis that cybervictimization predicts depressive symptoms and that this relationship is moderated by RSAR and SCLR. Results (Table 3) revealed that gender, race, age, baseline RSA and baseline SCL accounted for a non-significant amount of variance in depressive symptoms ($R^2 = .09$, $F [5, 156] = 1.94$, $p > .05$) such that these covariates explained 9.0% of the variance in depressive symptoms in the first step of the model.

When cybervictimization, RSAR and SCLR were entered into the model at step two, the overall model became significant ($R^2 = .20$, $F [8, 153] = 4.80$, $p < .001$). After accounting for covariates, cybervictimization, RSAR and SCLR explained an additional 14.2% of the variability in depressive symptoms ($\Delta R^2 = .14$, $\Delta F [3, 153] = 9.08$, $p < .001$). Together, all the variables in the model accounted for 20.1% of the variance in depressive symptoms. Consistent with my hypothesis, cybervictimization significantly,

positively predicted higher levels of depressive symptoms (b= .40, t[154] = 5.20, p < .001, ß = .40, CI[.25, .55]). This suggests that cybervictimization is driving the significance in step 2 of the model.

When the two way cybervictimization x RSAR, cybervictimization x SCLR, and RSAR x SCLR interactions were entered into the model at step three, the overall model remained significant (R^2 = .22, F [11, 150] = 3.78, p < .001) and explained an additional 1.6 % of the variability in depressive symptoms (ΔR^2 = .02, ΔF [3, 150] = 1.01, p =.39). Together, all the variables in the model accounted for 21.7% of the variance in depressive symptoms. Specifically, none of the two-way interactions significantly predicted depressive symptoms (see Table 3). This suggests that the two-way interactions are not driving the significance at step 3 of the model.

When the three-way cybervictimization x RSAR x SCLR interaction was entered into the model at step four, the overall model remained significant (R^2 = .22, F [12, 149] = 3.54, p < .001) and explained an additional 0.5 % of the variability in depressive symptoms (ΔR^2 = .005, ΔF [1, 149] = 1.00, p =.32). Together, all the variables in the model accounted for 22.2% of the variance in depressive symptoms. However, contrary to my hypothesis, the three-way interaction did not significantly predict depressive symptoms experienced (See Table 3).

Cybervictimization x RSAR x SCLR predicting alcohol consumption.

A hierarchical regression model was used to test the hypothesis that cybervictimization predicts alcohol consumption and that this relationship is moderated by RSAR and SCLR. Results (Table 4) revealed that gender, race, age, baseline RSA and

baseline SCL accounted for a non-significant amount of variance in depressive symptoms (R^2 = .10, F [5, 156] = 3.35, p< .05) such that these covariates explained 9.7% of the variance in alcohol consumption in the first step of the model.

When cybervictimization, RSAR, and SCLR were entered into the model at step two, the overall model became significant (R^2 = .18, F [8, 153] = 4.13, p < .001). After accounting for covariates, cybervictimization, RSAR and SCLR explained an additional 8.1% of the variability in alcohol consumption (ΔR^2 = .08, ΔF [3, 153] = 4.99, p =.002). Together, all the variables in the model accounted for 17.7% of the variance in alcohol consumption. Specifically, consistent with my hypothesis, cybervictimization significantly positively predicted higher levels of alcohol consumption (b= .27, t[154] = 3.49, p = .001, ß = .27, CI[.12, .42]). This suggests that cybervictimization is driving the significance at step 2 of the model.

When the two way cybervictimization x RSAR, cybervictimization x SCLR, and SCLR x RSAR interactions were entered into the model at step three, the overall model remained significant (R^2 = .21, F [11, 150] = 3.55, p < .001) and explained an additional 12.9 % of the variability in alcohol consumption (ΔR^2 = .03, ΔF [3, 150] = 1.84, p =.14). Together, all the variables in the model accounted for 20.7% of the variance in alcohol consumption. Specifically, none of the two-way interactions significantly predicted alcohol consumption (see Table 4). The cybervictimization x RSAR two -way interaction was marginally significant. This suggests that both of the two-way interactions are not driving the significance at step 3 of the model.

When the three-way interaction cybervictimization x RSAR x SCLR interaction was entered into the model at step four, the overall model remained significant (R^2 = .21, F [12, 149] = 3.24, p < .001) and did not explain any an additional variability in alcohol consumption (ΔR^2 = .00, ΔF [1, 149] = .03, p =.87). Together, all the variables in the model accounted for 20.7% of the variance in alcohol consumption. However, contrary to my hypothesis, the three-way interaction did not significantly predict alcohol consumption (See Table 4).

I ran an exploratory follow up analysis to decompose the marginal two-way cybervictimization x RSAR interaction predicting alcohol consumption. Simple slopes analyses revealed that cybervictimization significantly predicted higher levels of alcohol consumption among students with high RSAR, indicating PNS augmentation (b= .36, t[154] =3.34, p = .001, ß = .37, CI[.15, .58]) but not among students with low RSAR, indicating PNS withdrawal (b= .17, t[154] = 1.57, p = .12, ß = .18, CI[-.05, .25]). A graph of this interaction appears in Figure 1.

Discussion

The goal of this study was to apply differential susceptibility theory to examine the relationship between cybervictimization and clinical symptoms with developmental salience for college students (i.e., depression and alcohol use) and whether this relationship is moderated by psychophysiological factors. Results supported my first hypothesis, which posited that cybervictimization would significantly predict higher

levels of depression and alcohol use. My second hypothesis, which posited that the relationship between cybervictimization and clinical outcomes would be jointly moderated by SNS and PNS reactivity, was not supported. However, an unexpected marginally significant two-way interaction emerged between RSAR and cybervictimization predicting alcohol consumption. Specifically, at high levels of RSAR, higher levels of cybervictimization predicted higher levels of alcohol consumption. This suggests that vagal regulation of the PNS may be relevant to the effects of cybervictimization on alcohol consumption.

My analyses revealed that cybervictimization predicted higher levels of both depressive and alcohol consumption symptoms, supporting my first hypothesis. This finding is in line with previous literature that established the relationship between cyberbullying and mental health problems such as depression (Rose & Tynes, 2015), suicidal ideation (Quintana-Orts et al., 2020) and alcohol use (Rodríguez-Enríquez et al., 2019). Although I analyzed data from a high functioning non-clinical college student sample, mostly exhibiting low levels of cybervictimization, the relationship between cybervictimization and clinical outcomes (depression and alcohol consumption) was still significant. This suggests that the relationship between cybervictimization and depression and alcohol use is robust. This has implications for college life. Since cybervictimization had a significant impact on adjustment even in a high functioning sample, programs that support college students, such as programs targeting college retention or academic advising, may benefit from prevention models that seek to address cybervictimization from a preventative public health perspective to foster wellbeing on campus.

Contrary to my hypotheses, the three-way cybervictimization x SNS reactivity x PNS reactivity interaction did not significantly predict depressive or substance use symptoms. These results are inconsistent with previous literature that found a relationship between environmental stressors and clinical outcomes jointly moderated by SNS and PNS reactivity (e.g., Lafko et al., 2015). However, there are multiple factors in my study that are inconsistent with the cited literature and that may have contributed to seeing a null result. First, previous literature such as Abaied et al.'s (2018) paper operationalized both positive and negative environments separately as maternal psychological control (negative environment) and maternal involvement (positive environment). My study, on the other hand, did not include a separate measure for positive and negative environments. I conceptualized a positive environment as a low cyberbullying environment and a negative outcome as a high cyberbullying environment. One problem with this conceptualization is that the absence of cybervictimization is not necessarily a positive environment and could be conceptualized as a neutral environment. The differential susceptibility "for better or for worse" model theoretically considers positively and negatively valanced environments, yet, I did not capture a broad range of positive and negative environments when considering cybervictimization. This is a limitation of my study that should be considered if this analysis were to be replicated. Future studies could, for instance, measure cybervictimization (conceptualized as a negative environment) as well as praise and validation from peers (e.g. encouraging validating comments online and praise, conceptualized as a positive environment) and examine their relation to clinical outcomes as moderated by psychophysiology. There is

currently no measure in the literature that measures a positive online environment, thus we need more research that can validate a reliable measure for this construct.

Second, I had access to a convenience sample of college students, which was skewed towards low cyberbullying, low depression, and low alcohol consumption. Due to the low variability in the constructs I chose, it is harder to detect interaction effects in my sample. In addition, having low variability in my sample does not constitute a robust test of differential susceptibility. The differential susceptibility model is based on examining differences between individuals with high and low "plasticity factors", which are also conceptualized as vulnerability factors in the diathesis stress model. In my sample, there is not much variability in predisposing plasticity factors that are theorized to render individuals more sensitive to different environments. In the future, I could recruit a wider, more diverse community sample, beyond a single college campus, to capture better variability and better examine the interaction between PNS augmentation and alcohol use.

Third, the laboratory task is another factor that could have limited my ability to detect physiological reactivity relevant to the differential susceptibility to cybervictimization. Previous studies found different physiological reactions to stress depending on the nature of laboratory task (Abaied et al., 2014). I used a laboratory task that was relevant to social stress but that did not include responses to a virtual online environment. Future studies could design a validated stress task that tests online social interactions, such as an interactive online texting task where participants receive "likes" and "dislikes" for comments, and measure autonomic stress reactivity to these tasks. This

may allow us to better capture differential susceptibility to cybervictimization specifically.

A novel marginal two-way interaction

Although I did not pose specific hypotheses regarding two way interactions, I found that the two way cybervictimization x RSAR was marginally significant predicting problematic alcohol consumption. Cybervictimization significantly predicted more alcohol consumption when RSAR was high (i.e. in the case of PNS augmentation or blunted PNS withdrawal). In the context of polyvagal theory, when the vagal break does not withdraw to increase physiological arousal, this renders individuals unprepared to adaptively respond to stressful environments (Porges, 1995).

As seen in Figure 1, the two-way interaction is not consistent with the differential susceptibility model. At high levels of cybervictimization, individuals with high RSAR still had lower levels of cybervictimization than their low RSAR counterparts. Had the finding been consistent with the differential susceptibility model, in which high RSAR confers sensitivity to the environment, we would have observed a cross-over effect where individuals with high RSAR exhibited worse outcomes than their low RSAR peers. The results do not confer to the diathesis-stress model either. In the diathesis stress model, in a positive environment, there is no difference in functioning between individuals with high vs. low vulnerability factors. In this study, I observed a difference in the low cybervictimization environment between individuals exhibiting high vs. low RSAR. Polyvagal theory posits that the PNS is further dissected into a ventral vagal and dorsal vagal branch. The ventral vagal system is the **first** line of defense whereas the dorsal

vagal system is the **third** line of defense in case the SNS fails to adequately respond to stress. Examining RSA is an indicator of PNS functioning but does not provide us with specific information as to which **level** of the PNS is being recruited. Perhaps, detailing which level of the PNS is being recruited may help us understand underlying PNS mechanisms that may help elucidate my findings, These findings should thus be interpreted with caution. Future studies could find ways to physiologically operationalize the different levels of PNS functioning. This could help us deepen our understanding of how the PNS moderates relationships between social stressors and alcohol consumption.

No previous research has examined whether RSA moderates the link between the social environment and alcohol consumption. My two-way interaction finding is thus a novel finding in the field. Some theories may help explain why cybervictimization predicted greater alcohol consumption for individuals with RSA augmentation. From the aggression literature, we know that a blunted fight or flight response (characterized by RSA augmentation) is an indicator of poor behavioral and emotional inhibition as well as aggressive and antisocial behavior in children and adolescents (Erath et al., 2011; Gregson, Tu, & Erath, 2014; see Lorber, 2004 for a meta-analysis). Some theories have attempted to explain the association between blunted arousal and externalizing symptoms. Stimulation-seeking theory (Zuckerman, 1980) hypothesizes that individuals with low arousal seek high risk activities to increase their arousal states to homeostasis. Fearlessness theory (Raine, 2002) posits that low arousal during stress is an indicator of insensitivity to the environment, which indicates failure to inhibit externalizing behavior.

These theories linking blunted withdrawal to externalizing behavior such as aggression may also explain alcohol consumption as an externalizing behavior in my findings.

Few studies in the literature have examined RSAR as a moderator of the association between social environments and clinical outcomes. Previous research with children has supported RSAR as a moderator of the effects of the social environment (e.g. parenting effects) on a variety of adjustment outcomes in children and adolescents, including internalizing and externalizing symptoms, social functioning, and academic achievement, but not including alcohol consumption (Katz & Gottman, 1995; Katz & Gottman, 1997; Leary & Katz, 2004; Obradovic et al., 2010; for a review, see El-Sheikh & Erath, 2011). For instance, Abaied et al. (2014) showed that emerging adults' sensitivity to parental encouragement to engage versus disengage from stress varied as a function of RSAR profile (withdrawal vs. augmentation) as well as the nature of the laboratory challenge (interpersonal vs. noninterpersonal). However, the literature shows discrepancies in the ways RSAR moderates the relationship between environmental factors and clinical outcomes. Some studies showed that individuals with high RSAR/PNS augmentation are more sensitive to the adverse effects of negative social environments, which was in line with my findings. For example, in a study by Khurshid et al. (2019), high marital conflict was associated with internalizing problems for adolescents with RSA augmentation. Other studies, however, showed the opposite pattern, in which youth with RSA withdrawal were most sensitive to the negative effects of an adverse environment (Tabachnik et al., 2020; Caldwell et al., 2019; Dyer et al., 2016) The discrepancy in these findings can be due to using different stress tasks (see

Abaied et al., 2014) or to the various operationalizations of environmental stressors. Although my findings are in line with part of the literature, more research is needed to better understand the observed discrepancies.

Polyvagal theory might help explain why PNS reactivity and not SNS reactivity moderated the association between a social stressor and alcohol consumption. According to Porges (1995), the PNS is the first line of response to stress through the ventral vagal complex. Phylogenetically, Porges considers the PNS to be an older structure than the SNS. The PNS's "vagal brake" can rapidly be withdrawn or reapplied to generate an instant change in cardiovascular output in order to respond to environmental demands **without activating the SNS** (Porges, 2007, 2011). The second line of defense, if the PNS vagal withdrawal is not sufficient to respond to stress, is the SNS. Finally, if the SNS does not meet the demands of the stressful environment, the organism resorts to the dorsal vagal complex of the PNS (oldest phylogenetic branch of the ANS). Theory thus suggests that problems in vagal withdrawal of the PNS might hinder recruiting the SNS, which in turn will disrupt the way the organism responds to stress. In other words, any malfunction with the PNS will have effects on SNS recruitment. In this case, the PNS does not exhibit normal functioning with RSA augmentation and that may have repercussions on SNS functioning. Polyvagal theory may help explain my findings; however, more empirical research is needed to provide us with a more specific explanation concerning why the PNS but not SNS moderated the relationship between social stress and problematic alcohol use. Alcohol consumption may be considered an externalizing clinical symptom as a result of cybervictimization, or can also be explained

as an alternative self-regulatory response (Sher & Grekin, 2007) when PNS regulation fails to adequately respond to stress. Previous studies (Stifter et al., 2011) operationalized ANS reactivity as related to the ability to regulate emotions. In this case, when the ANS fails to facilitate emotion regulation in response to cybervictimization, we may also conceptualize alcohol consumption as a way to calibrate emotional responses.

Limitations and Future Directions

The study has additional limitations that should be taken into account. First, this study used a homogeneous, non-racially diverse sample. Additionally, my sample mainly consisted of predominantly white middle and upper-middle class college students. Recent research suggested that race might confer different psychophysiological responses to stress (Hill & Thayer, 2019). Consequently, my results may not be representative of other populations. Another factor that may have affected my results is that my sample was not evenly distributed between male and female participants. Previous findings (Beauchaine et al., 2008) found differences in psychophysiological reactivity between male and female participants. My sample was predominantly female, and thus my findings may not extend to both male and female individuals. More specifically, El Sheikh (2009) considered that evidence for sex differences in RSA and SCL is inconclusive. More recently, in Beauchaine's (2019) metanalysis, females showed greater RSA reactivity than males. When it comes to SCL, Boucsein (1992) indicated higher levels of tonic electrodermal activity in women in comparison to men, however, other studies reported disparate findings (Venables and Mitchell, 1996; El Sheikh, 2009). It is thus difficult to hypothesize about the effect of sex on the four different profiles. Previous research noted

differences in the links between psychophysiological and clinical outcomes between clinical and community samples (see Graziano & Derefinko, 2013 for a metanalysis). Thus, the type of samples (e.g., community vs. clinical) being analyzed will affect psychophysiological responses to stress. My access to a community sample does not allow me to generalize findings to the broader population including clinical populations. Due to the small sample size I used, this study was likely underpowered to detect small effects. Finally, the study design that does not include a control and intervention group does not allow for inferring causality.

Conclusion

Given the limitations of my study, I suggest that future studies should not only look at psychophysiological responses to stress cross sectionally, but also longitudinally. The literature has not yet examined whether an autonomic profile (Bernston et al., 1991) is fixed throughout development, or whether it is plastic and changes across development. Another question that future studies have yet to answer is whether patterns of activation change or fluctuate across situations for the same individual. To examine these questions, I suggest future intensive longitudinal designs of ambulatory assessments of behavior, clinical characteristics and psychophysiology.

One promising clinical approach to address the negative outcomes of cybervictimization is just-in-time adaptive interventions (JITAIs) (Nahum-Shani, 2015), which represents interventions that vary over time to accommodate an individual's changing status and circumstances in order to provide better support. For instance, future

studies can use a JTAIs through a mobile app, which can target alcohol consumption after cybervictimization by allowing individuals to log and track alcohol consumption.

In conclusion, this study showed that cybervictimization is associated with higher depressive symptoms and alcohol use in college students. However, the study did not find evidence for the differential susceptibility model showing that some types of autonomic activation patterns confer sensitivity to the environment. The novel finding of this study, however, is that high RSAR moderated the relationship between cyberbullying and problematic alcohol consumption.

Table 1.

Descriptive Statistics among the Study Variables

Variable	M(SD)	Min	Max	Skewness
Cybervictimization	1.91 (2.30)	.00	11.00	1.70 (.19)
RSAR	.002 (.13)	-.21	.81	3.87 (.19)
SCLR	2.49 (1.99)	-1.27	10.00	.85 (.19)
Depression (CES-D)	15.63 (10.11)	.00	47.00	.79 (.19)
Alcohol Consumption (AUDIT)	7.27 (.39)	.00	23.00	.69 (.19)

Note. RSAR = respiratory sinus arrhythmia reactivity. SCLR = skin conductance level reactivity.

Table 2.

Intercorrelations Among Study Variables

	1	2	3	4	5
1.Depression (CES-D)	—	.10	.37**	.01	.02
2.Alcohol Consumption (AUDIT)		—	.23**	-.07	.10
3.Cybervictimization (CVQ)			—	.17*	.13
4.RSAR				—	.04
5.SCLR					—

* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).

Table 3.

Regression Coefficients for the Models Predicting Depressive symptoms

Predictors	Unstandardized Estimate [95% CI]	β	*t*	R^2
Step 1				.058
Sex (Male=0, Female=1)	.18 [-.16, .51]	.08	1.04	
Age	-.11 [-.22, .002]	-.16	-1.95	
Race/Ethnicity (White=1, Nonwhite=0)	-.25 [-.67, .16]	-.10	-1.21	
Baseline RSA	1.74 [-.75, 4.22]	.11	1.38	
Baseline SCL	.003 [-.04, .04]	.01	.16	
Step 2				.201*
Cybervictimization	.40 [.25, .55]	.40	5.20	
RSAR	-.05 [-.20, .10]	-.05	-.70	
SCLR	-.03 [-.18, .12]	-.03	-.42	
Step 3				.217*
Cybervictimization x RSAR	.13[-.04, .30]	.15	1.53	
Cybervictimization x SCLR	.04[-.11, .19]	.04	.52	
RSAR x SCLR	-.04[-.18, .10]	-.05	-.58	
Step 4				.222*
Cybervictimization x RSAR x SCLR	-.07[-.07, .20]	.10	.99	

Note. Model 2 contains all predictor variables listed in Model. Model 3 contains all predictor variables listed in Models 1 and 2. Model 4 contains all predictor variables listed in Models 1, 2 and 3 RSAR = respiratory sinus arrhythmia reactivity. SCLR = skin conductance level reactivity.
* $p < .05$.

Table 4

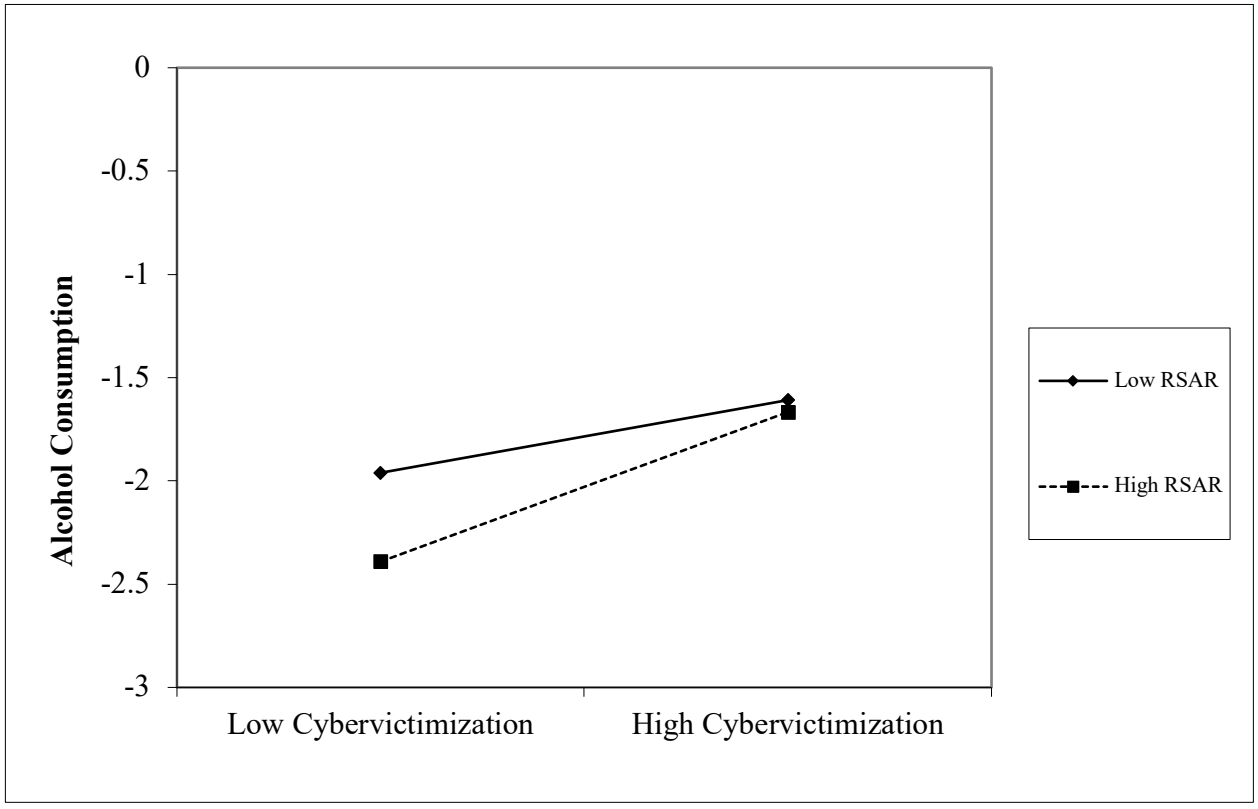
Regression Coefficients for the Models Predicting Alcohol Consumption

Predictors	Unstandardized Estimate [95% CI]	β	*t*	R^2
Step 1				.097*
Sex (Male=0, Female=1)	.14 [-.19, .46]	.06	.83	
Age	.02 [-.09, .13]	-.03	.34	
Race/Ethnicity (White=1, Nonwhite=0)	.74 [.34, 1.14]	-28	3.66	
Baseline RSA	-1.50 [-3.90, .90]	-.10	-1.24	
Baseline SCL	.02 [-.02, .06]	.08	1.03	
Step 2				.177*
Cybervictimization	.27 [.12, .42]	.27	3.49	
RSAR	-.07[-.21, .08]	-.07	-.88	
SCLR	-.08 [-.07, .23]	.08	1.06	
Step 3				.207*
Cybervictimization x RSAR	.16[-.002, .33]	.20	1.95	
Cybervictimization x SCLR	-.07[-.22, .07]	-.08	-1.00	
RSAR x SCLR	-.13[-.26, .01]	-.14	-1.78	
Step 4				.207*
Cybervictimization x RSAR x SCLR	-.01[-.14, .12]	.02	-.16	

Note. Model 2 contains all predictor variables listed in Model. Model 3 contains all predictor variables listed in Models 1 and 2. Model 4 contains all predictor variables listed in Models 1, 2 and 3 RSAR = respiratory sinus arrhythmia reactivity. SCLR = skin conductance level reactivity.
* $p < .05$.

Figure 1

Cybervictimization levels predicting alcohol consumption at high (+1 SD) and low (-SD) levels of RSAR (respiratory sinus arrhythmia reactivity).



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