THE EFFECTS OF VICTIMIZATION, POST-TRAUMATIC STRESS DISORDER, AND HEART RATE REACTIVITY ON ANTISOCIAL BEHAVIOR

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ABSTRACT

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Understanding factors associated with antisocial behavior and substance use is paramount within the field of criminology to better understand correlates of crime and criminal behavior. A growing literature concerning risk factors that increase the propensity for antisocial behavior and substance use has explained the relationship between many traits and these outcomes. However, many risk factors are correlated not only to antisocial behavior and substance use, but also to one another. The risk factors of victimization, post-traumatic stress disorder (PTSD), and increased heart rate reactivity (HRR) have all been shown to be related to one another as well as to antisocial behavior and substance use. The current dissertation seeks to examine if the three risk factors of victimization, increased PTSD, and increased HRR increase the propensity of antisocial behavior and substance use when all three risk factors are present. In addition, the current dissertation also seeks to better understand if the interaction between these three risk factors on antisocial behavior and substance use vary based on gender. The sample for the current dissertation is 486 college students from a southwestern state university. To test the effects of victimization (property and personal), increased PTSD, and increased HRR on antisocial behavior and substance use (both for the full and gender split sample) Tobit regression models were estimated. The findings in part, support that the interaction of victimization, increased PTSD, and increased HRR increase the propensity for antisocial behavior for males only. Herein, the specific findings and future directions suggested by the current dissertation are further discussed.

KEY WORDS: Victimization, Post-traumatic stress disorder, Heart rate reactivity, Antisocial behavior, Substance use

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CHAPTER I

Introduction

Antisocial behavior is characterized by behaviors that are outside of the norms for a given social setting. Antisocial behaviors can range from speaking too loud in certain situations that are normally quiet, such as attending a movie theater, to aggressive and hostile actions committed with the intent to do harm to an individual or their property. Past literature has linked the risk factors of victimization, post-traumatic stress disorder (PTSD), and heart rate reactivity (HRR) to increased participation in antisocial behavior (Gottman, Jacobson, Rushe, & Shortt, 1995; Sullivan, Farrell, & Kliewer, 2006; Jakupcak et al., 2007). Although all three risk factors have been discussed independently of one another and can have independent influences on antisocial behavior, past literature has also shown these three risk factors may be associated with one another, thereby exacerbating their effects (Echeburua, De Corral, Zubizarreta, & Sarasua, 1997). For example, individuals who have experienced victimization are more likely to exhibit symptoms of PTSD (Resnick, Kilpatrick, & Lipovsky, 1991); both victims and individuals who exhibit PTSD (even without the presence of victimization) are more likely to exhibit abnormal HRR (Escheburua et al., 1997); and finally those who exhibit abnormal HRR are more likely to experience worsening symptoms of PTSD and are at a higher risk of victimization events (Moshe-Kotler, Matar, & Kaplan, 2000). In addition, males and females have been shown to differ based on all three risk factors as well as rates of antisocial behavior (Ullman & Filipas, 2005; Odgers et al., 2008). Finally, a specific type of antisocial behavior, substance use, is a common side effect related to all three risk factors (Acierno, Kilpatrick, Resnick, Saunders, & Best, 1996; Koob & Franz,

2004; Dutton, Green, Kaltman, Roesch, Zeffiro, & Krause, 2006). Thus, the primary purpose of the current dissertation is to examine the relationship between biological, psychopathological, and environmental factors that have been shown to be empirically related to general antisocial behavior as well as substance use specifically based on the interrelationship between these three risk factors. Moreover, the current dissertation seeks to better understand the influence of each risk factor on antisocial behavior independently and how these risk factors interact together. Further, given that each risk factor has been shown to impact males and females differently, the current dissertation will also explore these important gender differences.

Considering the range of information, relationships, and effects that will be discussed herein it is important to set up a logical approach for the information presented in the current dissertation. First, the independent relation between victimization, PTSD, and HRR related to general antisocial behavior and substance use will be discussed. Second, the relation and interactions between the risk factors themselves will be discussed. Third, a theoretical argument will be presented as to why the interactive effects among all three risk factors is important concerning antisocial behavior, rather than each factor on its own. Finally, the effects of the risk factors on general antisocial behavior and substance use will be looked at based on differences between males and females.

Victimization

Victimization has been linked as a risk factor to the onset of antisocial behaviors that were not present before the victimization event occurred (Snyder, Brooker, Patrick, Snyder, Schrepferman, & Stoolmiller, 2003). Specifically, the trauma involved with a

victimization event has been shown to have several negative consequences for victims and alter behavioral trajectories, ultimately increasing the risk for antisocial behaviors (Tjaden & Thoennes, 2006). It is important to note that protective factors such as individual traits like high general intelligence or social factors such as increased parental supervision have reduced the risk of antisocial behavior associated with victimization (Kandel et al., 1988; Morrison, Robertson, Laurie, & Kelly, 2002). However, even though victimization does not always lead to antisocial behavior it does increase the chances of an individual exhibiting antisocial behavior, especially with decreased protective factors present (Kim & Cicchetti, 2010).

An example of how victimization can act as a risk factor for antisocial behavior is through the onset of substance use and risky lifestyles. For instance, after a victimization event, some individuals begin relying on substances (e.g., alcohol and drugs) to deal with the negative effects of the trauma they experienced. The use of substances has been linked to increased risky lifestyle outcomes and both factors increased antisocial behaviors (Kilpatrick, Acierno, Saunders, Resnick, Best, & Schnurr, 2000). Hence, the use of substances may initially begin as a way to cope with trauma, but the onset of substance use and a risky lifestyle subsequent to victimization can lead to an increased propensity to exhibit antisocial behavior (Kilpatrick et al., 2000; Bina, Graziano, & Bonino, 2006; Schreck, Stewart, & Fisher, 2006).

In addition, substance use has been linked to a momentary decrease in judgment and increased impulsivity; hence it is not surprising that substance use is associated with an increase in serious antisocial behaviors such as physical aggression and criminal behavior (Luengo, Carrillo-de-la-Peña, Otero, & Romero, 1994; Loney, Frick, Clements,

Ellis, & Kerlin, 2003; Bina, Graziano, & Bonino, 2006). Further, if the substance being abused is illegal, it is likely the individual using the substance would have to break the law in order to acquire the substance as well as be in direct contact with criminal elements and antisocial peers, which have also increased antisocial behavior (Barnes & Farrell, 1992; Bina, Graziano, & Bonino, 2006). Thus, victimization can provoke the onset of substance use and risky lifestyles that lead to an increase in other antisocial behaviors.

In that same vein, growing evidence from both psychological and biosocial perspectives suggest that, while there is variation in responses to experiences of traumatic events, as the frequency of victimization events increase so does the likelihood of antisocial behavior (Snyder et al., 2003). Moreover, as subsequent victimization events increase so does the likelihood of more severe forms of antisocial behavior being exhibited (e.g., aggression and violence) (Turner, Finkelhor, & Ormrod, 2006). Specifically, the ability to regulate emotions associated with social interactions has been found to decrease after multiple victimization events, which is related to increased levels of aggression, violence, and other antisocial behaviors (Turner, Finkelhor, & Ormrod, 2006; Kim & Cicchetti, 2010). However, aspects of victimization and the effects of victimization can be influenced by several factors. One factor that has been shown to influence both victimization and the effects of victimization is gender.

For example, females are much more likely, on average, to experience sexual assault, intimate partner violence (IPV), stalking, and physical abuse from close friends or family members as compared to males (NCADV, 2014). Also, these types of victimization events have been shown to exhibit increased amounts of trauma compared

to other forms of victimization (e.g., burglary) (Dansky, Brady, Saladin, Killeen, Becker, & Roitzsch 1996; Pimlott-Kubiak & Cortina, 2003). Moreover, females are more likely to identify victimization events as traumatic and experience negative effects of victimization, such as PTSD, and increased HRR, when compared to males (Cutler & Nolen-Hoeksema, 1991). Considering these documented differences, the current dissertation will test to see if there are gender differences in overall victimization rates, levels of PTSD, and HRR, and how these risk factors influence general antisocial behavior and substance use in males and females separately.

PTSD

Post-traumatic stress disorder (PTSD) is also a risk factor for antisocial behavior, particularly for those who have experienced trauma associated with victimization (Kilpatrick, Saunders, Veronen, Best, & Von, 1987; Duncan, Saunders, Kilpatrick, Hanson, & Resnick, 1996). Post-traumatic stress disorder is generally characterized by three major categories, that although can be defined separately, tend to be exhibited simultaneously and are highly intertwined in a symptomatic feedback loop. The three major symptom components of PTSD are intrusive thoughts, avoidance behaviors, and hyperarousal (NIH, 2016). At the core of PTSD symptoms is the first factor of intrusive thoughts (Shipherd, Stafford, & Tanner, 2005). Intrusive thoughts are memories, dreams, or associated ideas dealing with the traumatic event or other stress-related events that individuals are trying to forget (Shipherd, Stafford, & Tanner, 2005; NIH, 2016). More specifically, intrusive thoughts are not part of individuals trying to work through traumatic events as part of the healing process, but rather random negative thoughts that occur at any time during the individuals' day. Further, intrusive thoughts cause increased

stress to individuals that have experienced trauma. In many cases it is due to increased levels of stress brought on by the intrusive thoughts that individuals alter their behaviors to reduce the effects or frequency of the intrusive thoughts (Shipherd, Stafford, & Tanner, 2005; NIH, 2016). A common behavior associated with reducing intrusive thoughts and the second major component of PTSD is avoidance behaviors (Asmundson, Stapleton, & Taylor, 2004; NIH, 2016).

Avoidance behaviors are any behavioral changes to an individuals' average pre-trauma behavior or routine that reduce the potential of experiencing stimuli that could induce intrusive thoughts or stress (Asmundson, Stapleton, & Taylor, 2004; NIH, 2016). Avoidance behaviors can be exhibited on a behavioral spectrum from small changes such as taking a new path to commonly visited locations or extreme lifestyle changes such as agoraphobic tendencies (Asmundson, Stapleton, & Taylor, 2004; NIH, 2016). While any behavioral shift due to trauma can have negative influences on an individual's life, agoraphobic tendencies can have detrimental effects and lead to worsening symptoms associated with trauma and PTSD (Tarrier et al., 1999). It is important to understand that avoidance behaviors are generally exhibited as a precautionary behavior and, as such, are not behaviors based on logic or known information by the individual. Thus, this symptom has been shown to worsen with time as individuals come into increased contact with triggering stimuli (Tarrier et al., 1999; NIH, 2016). Unfortunately, triggering stimuli can be somewhat random; hence individuals who suffer from trauma can start to view any interaction as a possibility for experiencing triggering stimuli. By reducing all social interactions, individuals not only decrease the potential negative triggers but also the prosocial interactions that act as a resiliency factor against PTSD (Haroz, Murray,

Bolton, Betancourt, & Bass, 2013; NIH, 2016). The third factor, hyperarousal, also plays a key role in avoidance behaviors as individuals suffering from PTSD often report a feeling of foreboding and choose avoidance as way to reduce their fear (Jakupcak et al., 2007).

Hyperarousal is often discussed by individuals suffering from PTSD as a feeling of being keyed up, being overly reactive to stimuli, and experiencing a tendency to exhibit negative opinions of the future for themselves and others (Kendall-Tackett, 2000). While the physiological aspects of hyperarousal will be discussed below, it is important to explain the common psychological aspects felt among individuals suffering from PTSD. Hyperarousal causes individuals to be reluctant to deal with the stresses of day-to-day life, such as running errands or engaging in social activities (NIH, 2016). Many times the sensation of hyperarousal reduces motivation to complete daily tasks in order to reduce the chances of intrusive thoughts and an overall feeling of being on edge (NIH, 2016). However, the lack of motivation can become frustrating to individuals and even spur guilt or shame of not being able to complete daily tasks or contribute to their own lives or families' needs which can increase the sensations of hyperarousal leading to anxiety and panic attacks (NIH, 2016). Ultimately, PTSD is a term that envelopes all of the major symptoms described above into a single intertwined outcome where individuals try to reduce the effects of intrusive thoughts and hyperarousal through avoidance, but in part, unconsciously increase the effects of hyperarousal and intrusive thoughts through avoidant behavior (Kendall-Tackett, 2000; NIH, 2016).

The description above is aligned with a diagnosis of PTSD, but in reality there is variability in expression of symptoms (NIH, 2016). Either way, the guiding aspect of the

psychological development of PTSD is useful to understand the complexity of such a psychopathology. Any trauma or victimization event that yields symptoms of PTSD can act as antecedents to antisocial behavior. For example, the onset of symptoms associated with PTSD has been linked to several negative behavioral outcomes such as substance use, depression, and aggression, all of which have been linked to antisocial and criminal behavior (North, Kawasaki, Spitznagel, & Hong, 2004; Jakupcak et al., 2007). For example, postwar combat veterans diagnosed with PTSD have been shown to be more likely to exhibit alcohol abuse and increased aggressive and violent behavior, which decreases prosocial support factors such as employment and romantic relationships (Jakupcak et al., 2010). Further, Begic and Jokić-Begić (2001) found that individuals who develop PTSD are more likely to exhibit onset physical aggression toward themselves and others and verbal aggression toward loved ones. Post-traumatic stress disorder has also been associated with increased levels of reactive anger resulting in physical and psychological spousal abuse (Taft, Street, Marshall, Dowdall, & Riggs, 2007). Unfortunately, subsequent stress has been shown to exacerbate both the symptoms of PTSD, as well as antisocial behaviors associated with the onset of the PTSD symptoms (Jakupcak et al., 2007; Vasterling, Street, Marshall, Dowdall, & Riggs, 2010).

Moreover, PTSD has been shown to occur at different rates within genders. While females are more likely to experience severe victimization events (e.g., sexual assault), males are more likely to experience general trauma (Tolin & Foa, 2002). This is discussed in the literature as a product of trauma related events that males are more likely to experience due to an overall riskier nature than females, especially during their teen to young adult years (Tolin & Foa, 2002). For example, males are more likely to experience

events such as accidents involving machinery or vehicles, occupational trauma such as military service, and physical conflict (Bachman, Segal, Freedman-Doan, & O'malley, 2000; Harris, Jenkins, & Glaser, 2006). It is interesting to note that although males are more likely to experience various forms of trauma, females are more likely to experience negative effects associated with trauma such as PTSD (Tolin & Foa, 2002). This difference is thought to be due to the way females and males process traumatic events (Tolin & Foa, 2002). Compared to males, females are more likely to allocate most of the blame on themselves for trauma and also experience an increase in fearfulness of future trauma (Tolin & Foa, 2002). Some scholars suggest that these differences in trauma processing may exist on a neurological level based on areas of the brain associated with processing emotions (Tolin & Foa, 2002). Further, these areas of the brain are also thought to be more developed early on in females more so than males (Tolin & Foa, 2002). Hence, it will be important to test trends between genders for the current dissertation based on reported symptoms of PTSD and victimization and its effects on antisocial behavior.

Heart Rate Reactivity

The physiological trait in the current dissertation is heart rate reactivity (HRR), and it will be discussed as a risk factor for antisocial behaviors.[1] To better understand the role HRR plays in behavior and how experiences can alter HRR, the current dissertation will discuss HRR within the larger system of the autonomic nervous system (ANS). The ANS controls physiological reactions to stressful and excitatory stimuli (Low, 1993). The

[1] The current dissertation is not discussing antisocial behavior associated with static inherent traits such as low resting heart rate that act as life course risk factors (Moffitt, 1993; Armstrong Keller, Franklin, & Macmillan, 2009). Instead the focus is on changes in heart rate reactivity that could be associated with victimization or trauma (Finkelhor & Browne, 1985; Chamberlain & Moore, 2002).

ANS has two broad roles, to induce excitatory responses and to bring the body back to homeostasis after the excitatory stimuli is over (Low, 1993). Both roles of the ANS are equally important and if not functioning correctly can have negative consequences for an individual (Low, 1993; Thayer, Friedman, & Borkovec, 1996). For example, an underactive ANS could result in under reactivity to dangerous environments putting an individual in harm's way (Low, 1993). In an opposite scenario an overactive ANS could cause unnecessary psychological and physiological stress causing problems such as sleep issues, substance use, and cardiac system problems (Haller & Benowitz, 2000; Heim et al., 2000; Brady, Back, & Coffey, 2004). Hence, a functional ANS is important for the physical, psychological, and behavioral well-being of an individual.

Traumatic events and exposure to long-term or extreme stress has been linked to increased dysfunction of the ANS (Chrousos & Gold, 1992). The relationship between PTSD and increased ANS dysfunction makes sense given the similarity of some of the symptoms individuals with each problem exhibit. For example, both PTSD and ANS dysfunction are associated with hyperarousal, increased aggression, higher propensity for substance use, and over reactions to stressful stimuli (Van der Kolk, 1994). Hyperarousal and over reactions to stimuli have been linked to behavioral shifts after the onset of the symptoms generally associated with aggression (Van der Kolk, 1994; Raine, 1996). More specific to the current dissertation, individuals who exhibit PTSD and a dysfunctional ANS are also more likely to exhibit antisocial behaviors (Van der Kolk, 1994; Raine, 1996). Taken together, trauma associated with victimization increases the propensity for the development of PTSD which increases the propensity for ANS dysfunction.

Further, gender has been shown to be a factor that influences victimization, how trauma is experienced, and ANS responses to stress. As early as adolescence, females have been shown to exhibit increased reactions to stress and trauma due in part to physiological differences in hypothalamus pituitary adrenal axis (HPA-axis) sensitivity and ANS function (Ordaz & Luna, 2012). Adolescent females also show an increased verbal ability to understand stress which may indicate increased brain activity of certain regions that differ from males (Ordaz & Luna, 2002). Hence, being aware of and able to understand that certain events are traumatic and stressful, stress and trauma can have increased negative effects at younger ages for females rather than males (Ordaz & Luna, 2002). These findings supports that gender differences may occur on a physiological level concerning ANS function and reactions to stress.

Victimization & PTSD

Victimization, especially sexual or violent victimization, has been linked to several negative outcomes and psychological changes. Some of the negative outcomes, such as the onset of psychopathologies have been linked to increased alterations in behaviors, specifically antisocial behaviors (Widom, 2001). One such negative outcome of victimization associated with behavioral shifts and antisocial behavior is the development of PTSD (Crowe & Blair, 2008). Victimization events ranging from mild abuse to sexual assault have been linked to the onset of PTSD symptoms in that individuals who have experienced victimization are more likely to exhibit PTSD symptoms in their lifetime (Elklit, 2002).

Further, both victimization and the development of PTSD have been linked to behavioral shifts associated with the inherent psychological and physiological alterations

caused by PTSD (Crowe & Blair, 2008). For example, women who have suffered victimization events and exhibited PTSD symptoms have been shown to exhibit higher scores on the Addiction Severity Index, and exhibit non-compliant behaviors such as aggression during substance use treatment more so than women who had not experienced victimization (Brady, Killeen, Saladin, Dansky, & Becker, 1994). Moreover, mental illnesses associated with past violent victimizations such as PTSD have been shown to increase the risk of addiction, risky lifestyles, and increase the risk of serious violent behavior (Scarpa, Haden, & Hurley, 2006). Similar to the above findings, females who experience severe IPV, either sexual, physical, or verbal, have been shown to be more likely to exhibit onset aggressive and abusive behaviors (Kuijpers, Van der Knaap, & Winkel, 2012).

The aforementioned studies are focused primarily on samples made up of predominately female participants. Although males do experience victimization and develop PTSD, females have been shown to experience victimization more often and are twice as likely to exhibit PTSD associated with victimization than males (Elklit, 2002). Thus, much of the literature concerning behaviors associated with both victimization and PTSD focuses on female samples. However, child abuse has been shown to have more equal impacts on individuals, regardless of gender (Ford, Chapman, Mack, & Pearson, 2006). For example, Ford and colleagues (2006) found that all individuals within their sample who had experienced physical or sexual child abuse were both more likely to exhibit PTSD as well as delinquent behaviors compared to individuals who had not been abused (Ford, Elhai, Connor, & Frueh, 2010). Thus, individuals who exhibit both past

victimization and PTSD are potentially at a higher risk for antisocial behavior than individuals who only exhibit one of these risk factors.

PTSD & Heart Rate Reactivity

Although there are different factors that can increase the chances of a dysfunctional ANS, one of the most documented factors related to ANS dysfunction is trauma (Chrousos & Gold, 1992). The major function of the ANS is to respond to stressful stimuli and return the body to homeostasis. In some cases, trauma can cause the system to decrease this regulatory ability (Chrousos & Gold, 1992). Beyond only physiological changes, trauma is also a well-documented antecedent to PTSD and behavioral shifts (Chrousos & Gold, 1992). In addition, individuals who have experienced traumatic events and develop PTSD have been more likely to experience ANS dysfunction (Hoehn-Saric, & McLeod, 1988; Chrousos & Gold, 1992). Further, the overlap between ANS dysfunction and PTSD becomes more apparent when comparing symptoms common to both issues, such as more severe HRR to stimuli, extended periods of hyperarousal, constant feeling of foreboding, unprovoked outbursts, irritable bowel syndrome, increased anxiety and fear, and trouble sleeping (Hoehn-Saric, & McLeod, 1988; Aggarwal, Cutts, Abell, Cardoso, Familoni, Bremer, & Karas, 1994; NIH, 2016).

Individuals who exhibit PTSD have been shown to exhibit increased cardiac reactions to stressful stimuli compared to non-PTSD individuals (Buckley, Holohan, Greif, Bedard, & Suvak, 2004). In addition, individuals who suffer from PTSD have overall higher resting heart rates (RHR) than non-PTSD individuals and also exhibit increased sympathetic system function when at rest (i.e., sitting still for a long period of time) (Cohen, Kotler, Matar, Kaplan, Miodownik, & Cassuto, 1997). The order of how

these two systems influence each other is not solidified and may vary on a case by case basis. In that, the symptoms of hyperarousal, anxiety, intrusive thoughts, and avoidant behaviors may be based on the physiological reaction of the ANS or the dysfunction of the ANS may be based on the presence of the PTSD symptoms. The etiology and order of the association between PTSD and ANS dysfunction is not central to the current dissertation, but instead that the relationship between PTSD and increased levels of HRR present. Specifically, since individuals who suffer from PTSD have higher RHR, exhibit increased autonomic responses to stress, and experience increased sympathetic function of the ANS, they exhibit increased risk of antisocial behaviors, such as substance use, aggression, and violence (Gottman et al., 1995; Kilpatrick et al. 2000; NIH, 2016).

Victimization, PTSD, & Heart Rate Reactivity

Each of the three focal risk factors discussed here (i.e., victimization, PTSD, HRR) share a relation to one another. Although the theoretical aspects of this relation are well known there has been little empirical work in the area of studying the effect that all three risk factors together have on antisocial behavior. This, in part, could be due to the amount of overlap thus these three risk factors share and that it is hard to tease apart the effects each factor has on an individual's behavior, especially antisocial outcomes. Said another way, these three risk factors may be so closely related and intertwined that the effect each factor exhibits is difficult to isolate. In addition, all three factors: victimization, PTSD, and HRR have been shown to influence behavioral changes and increase the onset of general antisocial behaviors and substance use (Pineles et al., 2011). Thus, although the current dissertation is unable to isolate the temporal ordering of each

factor, the primary goal is to better understand the effect of the presence of all three risk factors on general antisocial behavior and substance use.

Current Dissertation

To date, studies have mainly focused on understanding how victimization, PTSD and HRR influence antisocial behavior individually or for associations between two of the three risk factors interacting to influence antisocial behavior. Thus, the current dissertation extends this body of literature by examining how victimization, PTSD, and HRR interact together as risk factors for increased general antisocial behaviors and substance use. Second, the current dissertation explores differences between how victimization, PTSD, and HRR affect general antisocial behaviors and substance use between males and females.

Specifically, the proposed dissertation will use a purposive sample of college students enrolled in class at a southwestern state university. The data includes measures of victimization events, PTSD symptoms, HRR to a stressful stimuli (to measure ANS function), and a bevy of control variables common to the study of general antisocial behavior and substance use, such as gender, race, age, socioeconomic status, levels of self-control and delinquent peers.

CHAPTER II

Literature Review

There is a major focus in the field of criminology to better understand antecedents to antisocial behavior. Antisocial behaviors are intentional negative behaviors generally targeting toward another individual or their property (Eisner & Malti, 2015). Antisocial behavior is one of the most well-documented correlates of crime and criminal behavior (Moffitt, 1993, Coie & Dodge, 1998; Raine, 2002; Eisner & Malti, 2015), but unlike crime, the definition of antisocial behavior does not have to be defined as illegal or involve formal contact with the criminal justice system (Rutter, Giller, & Hagell, 1998). For example, consumption of alcohol in the United States is legal for individuals at or over the age of 21. However, the frequency of consumption, location, the time of day when alcohol is being consumed, and behaviors resulting from drinking could be described as antisocial even though the acts are legal. On the other end of the spectrum antisocial behavior can also be discussed as behaviors that are violent in nature and clearly criminal such as shootings and sexual assault (Mayer, 1995). Considering that antisocial behavior is more strongly associated with context, has a more pliable and broad definition, and is highly related to criminal behavior, it is important to understand what factors increase the risk of antisocial behavior.

Past research has shown that there are several correlated traits and factors that increase the propensity for antisocial behaviors (Moffitt, 1993). For example, individual traits and environmental factors such as increased impulsivity, low self-control, psychopathy, low resting heart rate, underactive serotonin systems, delinquent peers, being male, growing up and/or living in low socioeconomic environments, and having

antisocial parents have been risk factors for increased antisocial behaviors (Vitaro et al., 1998; Antonaccio et al., 2010; Shin et al., 2006). However, it is unlikely that the above listed factors exist as single attributes for individuals. Instead, they are more likely to co-occur as constellations of traits and factors that influence personality, temperament, preferences, and behavior (Chamorro-Premuzic & Furnham, 2003). Beyond inherent traits that individuals exhibit, life events can also influence behavioral outcomes and increase the risk for antisocial behavior. Experiencing a traumatic criminal victimization is one life event that has been linked to both negative psychological outcomes and behavioral changes (Coker, Davis, Arias, Desai, Sanderson, Brandt, & Smith, 2002). More specifically, aspects associated with the trauma of victimization, such as PTSD, and physiological factors, such as a dysfunctional ANS (i.e., response of HRR) have been identified as risk factors for increased antisocial behavior (Gottman et al., 1995; Sullivan, Farrell, & Kliewer, 2006; Jakupcak et al., 2007).

However, some traits such as gender can influence how victimization impacts individuals via the development of psychopathologies such as PTSD, and physiological factors such as altered HRR to stressful stimuli. For instance, females are more likely to experience more severe forms of victimization such as sexual assault and IPV (Coker et al., 2002; Catalano, Smith, & Rand, 2009), are roughly twice as likely to experience negative psychological effects such as PTSD from trauma compared to males (Elklit, 2002), and on average experience increased physiological reactions to stress (Ordaz & Luna, 2012). Although the differences in gender are important, it is also instructive to note that, regardless of the degree of impact, victimization generally produces some negative outcome for both genders. For example, Coker and colleagues (2002) found that

while females were significantly more likely than males to experience physical and verbal IPV, both males and females suffered negative outcomes from experiencing IPV. Specifically, they found that both males and females who experienced physical IPV were more likely to neglect taking care of their health or hygiene, exhibit increased symptoms of depression, and were more likely to develop a chronic mental or physical disease than individuals who had not experienced IPV (Coker et al., 2002). Beyond IPV, experiencing traumatic criminal victimization of any kind, including sexual assault, being robbed at gun or knife point, or being physically beaten have all been associated with negative psychopathologies such as PTSD, and dysfunctional ANS and HRR responses to stress (Koss, Koss, & Woodruff, 1991; Kilpatrick et al., 2000; Coker et al., 2002). In addition, like victimization, shifts in overall health, such as the onset of PTSD symptoms and increased HRR to stress have been linked to increased levels of general antisocial behaviors and the more specific antisocial behavior substance use (Gottman et al., 1995; Kilpatrick et al., 2000). Further, generally the more traumatic the victimization event, the worse the negative outcomes such as PTSD symptoms and increased HRR to stress are for individuals, hence the increased risk of increased general antisocial behavior and substance use (Koss, Koss, & Woodruff, 1991; Shalev et al., 1998). Substance use is being singled out from general antisocial behaviors for the current dissertation given the prevalence of substance use associated with victimization, PTSD, and HRR (Acierno, Kilpatrick, Resnick, Saunders, & Best, 1996; Kilpatrick et al., 2000; Koob & Franz, 2004; Bina, Graziano, & Bonino, 2006; Dutton, Green, Kaltman, Roesch, Zeffiro, & Krause, 2006; Schreck, Stewart, & Fisher, 2006).

The goal of the current dissertation is not to argue an ordering of these factors, but instead to demonstrate that these three risk factors are associated with each other, exhibit overlap, and are likely to occur with at least one of the other risk factors present. Said another way, it is more likely for PTSD and/or abnormal HRR to be present for a person who has experienced a traumatic victimization than for one or both to not be present. Similarly, when PTSD is reported, generally so are trauma and/or abnormal HRR (Kilpatrick et al., 2000). Also, after trauma (such as a victimization event) individuals who experience extended increased HRR immediately after the event are at an increased risk of developing PTSD (Shalev et al., 1998). To an extent a characteristic of each risk factor is an important component or characteristic of one or both of the other two, in that it is difficult to describe only victimization, PTSD, or HRR without discussing some aspect of all of them.

Further, not only are the three risk factors similar but each also can influence and interact with one another. It is important to understand the impact of victimization, PTSD, and HRR as risk factors when all three variables are present in the same model to predict general antisocial behavior and substance use. However, to develop the theoretical framework needed for the final models, each of the preceding sections will build on one another to show how each risk factor has can influence antisocial behavior and how each risk factor is related to one another. As mentioned, in the real world, these three risk factors share a complex and intricate relationship that is not linear. The relationship is better described as a cyclic feedback loop with no defined starting point unless a case by case approach is taken (i.e., not a population based study). For simplicity, the current dissertation will explain the risk factors in a logical ordering, with

a more linear approach explaining trauma, PTSD, and HRR from the victimization event to the onset of PTSD and ANS dysfunction leading to a change in HRR. Again, this is not to argue etiology or ordering, but given the complexity of each risk factor and the abstract relation each risk factor shares, a more linear approach to the literature enables a clearer theoretical scaffolding for the current dissertation.

Consequences & Behavioral Shifts Associated with Victimization

Victimology is an area of interest within the field of criminology that focuses on the victim rather than the offender or crime and has experienced growth in empirical research over the last four decades (Viano, 1990). While much of the early literature concerning victimization dealt primarily with characteristics of victims and victimization events; more recent studies have begun to focus on the consequences of victimization and how victimization events can influence victims' lives and behaviors (Fattah, 1979; Walker, 1983; Hodges & Perry, 1999; Wordes & Nunez, 2002). There are many aspects of victimization events that can have detrimental consequences on victims and their lives (Wordes & Nunez, 2002). For instance, early studies of at-risk populations have shown that individuals who have experienced severe traumatic victimization were more likely to be homeless (Fields, 1981; Simons & Whitbeck, 1991) and that females who experienced sexual abuse were more likely to become addicted to substances, run away from home, and either opt into or be forced into survival sex (Simons & Whitbeck, 1991). Moreover, this research has demonstrated that perceptions of victims' self, well-being, safety, and overall fear of the outside world have changed after being victimized. Changes in perceptions such as seeing the world as disproportionately threatening, or viewing oneself

as having no social value has had negative impacts on mental health and subsequent behavior (Neary & Joseph, 1994; Dull & Wint, 1997; Graham & Juvonen, 1998).

For example, one perception that has been shown to change after victimization is an increased fear of crime. Specifically, an unrealistic or disproportionate fear of crime which has been shown to alter behaviors of individuals attempting to reduce the chances of future victimization by changing routines and or avoiding environments and social interactions that may replicate the original trauma (Dull & Wint, 1997; Stafford, Chandola, & Marmot, 2007). In a longitudinal study of over 10,000 individuals ranging from ages 35 to 55 years old, Stafford and colleagues (2007) found that victimized individuals who reported increased fear of crime also exhibited decreased social interactions. Specifically, these individuals exhibited increased levels of depression, less physical activity (e.g., exercise), and spent less time with friends engaging in social activities (Stafford, Chandola, & Marmot, 2007). Moreover, these detrimental changes occurred after the victimization event, as did the onset increased of fear of crime (Stafford, Chandola, & Marmot, 2007). This effect is unfortunate, as physical activity and prosocial peer support groups have been shown to decrease the negative effects of victimization (Stafford, Chandola, & Marmot, 2007).

Without prosocial interactions, victimized individuals can experience an exacerbation of negative outcomes which can alter day-to-day activities, such as depression, job loss, reduced prosocial relationships (e.g., romantic partners, friends, family, and medical or financial agencies), and substance use (i.e., addiction) (Rigby, 2000; Stafford, Chandola, & Marmot, 2007). These alterations of lifestyle can lead to a host of negative life outcomes through shifts in behavior during the life course from

prosocial to antisocial behaviors (Macmillan, 2001). Further, although seclusion is not needed for negative outcomes and behaviors to occur, it can exacerbate them (Macmillan, 2001). For example, Macmillan (2001) illustrates, in a review of the literature, that individuals who are victimized are at increased risk of experiencing mental stress, exhibiting a lower sense of well-being, and an increased propensity to be involved in antisocial behavior and crime. By experiencing mental and behavioral shifts victims may also experience erosion of their physical and mental health as well as experience legal issues (Koss, Koss, & Woodruff, 1991; Macmillan, 2001). The consequences of victimization can greatly impact individuals' lives and alter behaviors.

Moreover, Koss and colleagues (1991) found that women who had experienced victimization were more likely to experience negative physical health effects that were not present until after the victimization event. In a sample of 413 adult women (194 who had not been victimized and 219 who had experienced a victimization event) using both self-report questionnaires and official medical records, Koss and colleagues (1991) found that as the severity of trauma increased (i.e. noncontact crime, assault, and rape), victims' need for physician visits also increased. Specifically, individuals who had experienced a traumatic victimization event sought medical treatment twice as often than non-victims, explained significantly more daily distress and decreased well-being, and 2.5 times more outpatient costs (Koss, Koss, & Woodruff, 1991). Thus, negative outcomes of victimization have substantial impacts on victims' lives as well as their daily well-being.

Although acute negative health effects are a major concern, research has also shown that the level of trauma experienced during victimization increases the extent of long-term negative effects on victims (Coker et al., 2002). For example, Coker et al.

(2002) studied a sample of 1,152 adult females ranging from the ages of 18 to 65 years old to assess negative outcomes associated with IPV. They used medical histories to establish onset of psychological and physical symptoms and to control for and omit immediate injuries inflicted during the abuse or pre-existing conditions. They found that females who had experienced more severe psychological IPV were significantly more likely to report poor physical and mental health (e.g., depression) following abuse (Coker et al., 2002). Moreover, IPV was also correlated with several negative health outcomes, including chronic back and migraine pain, chronic pelvic pain, stomach ulcers, digestive tract problems, and sexually transmitted infections (Coker et al., 2002). The aforementioned study showed that experiencing trauma of victimization even without physical injury can still have negative physical outcomes based on the severity of the trauma. Often the state of physical health is highly correlated with mental health, overall well-being in life, and behavior (Penedo & Dahn, 2005).

Given the above information, it is unsurprising that often individuals who have experienced traumatic victimization suffer from negative psychological issues which can influence behavioral changes (Kilpatrick et al., 1985). To better understand this dynamic Kilpatrick and colleagues (1985) studied a population of adult females (n = 2,004). They found that victims were more likely to experience mental health problems as well as exhibit negative behavioral shifts (Kilpatrick et al., 1985). Specifically, victimized females without prior mental problems were more likely to exhibit the onset of nervous break downs, suicidal ideation, and suicide attempts significantly more than non-crime victims (Kilpatrick et al., 1985). Moreover, the negative mental effects and behavioral

shifts were more severe in victims who experienced more traumatic victimization events (Kilpatrick et al., 1985).

Victimization has been associated with a plethora of negative outcomes which are associated with changes in routines and lifestyles. Victimized individuals who experience mental health problems associated with traumatic victimization also exhibit a higher propensity to develop psychopathological problems that were not present prior to the victimization event (Kilpatrick et al., 1985). Psychopathologies such as depression, agoraphobia, substance use, addiction, bipolar disorder, eating disorders, and PTSD have been documented as symptoms experienced post-victimization (Kashdan, Morina, & Priebe, 2003; Kauer-Sant'Anna, et al., 2007). Such psychopathologies can involve changes in behaviors and many have had substantial impacts on quality of life and an individual's activities (Kauer-Sant'Anna, et al., 2007). Among the changes associated with victimization events, behavioral shifts that occur alongside the negative effects of victimization are sometimes discussed as coping behaviors (Janoff-Bulman, 1985; Ford, Chapman, Mack, & Pearson, 2006).

A coping behavior is any behavioral change that decreased the negative consequences/effects of victimization (Janoff-Bulman, 1985). Coping behaviors are exhibited on a wide continuum and can be positive behaviors meant to rebuild a victim's life to a more prosocial state (Janoff-Bulman, 1985). However, not all coping behaviors have a positive impact on victims' well-being or life events. Further, it is not uncommon for the behavioral shifts and coping behaviors associated with the consequences of victimization to be manifested as antisocial and criminal behaviors (Macmillan, 2001).

Victimization and Antisocial Behavior

There are several negative aspects concerning the effects victimization has had on victims, some of which are associated with behavioral changes. Specifically, stress associated with traumatic victimization has had a strong impact on victims' lives, such as negative psychological outcomes and increase the risk of antisocial behaviors (Eitle & Turner, 2002; Salston & Figley, 2003). Moreover, victimization, especially violent or sexual victimization, has caused high levels of stress in a short amount of time that can have lasting consequences on physical, psychological, and behavioral characteristics, especially general antisocial behavior and substance use (Aceves & Cookston, 2007). Although experiencing victimization does not mean that individuals will become antisocial or abuse substances, victimization is a risk factor for the onset of such behaviors. As mentioned, one way general antisocial behaviors and/or substance use results from victimization is the manifestation of coping behaviors to deal with trauma (Ford, Chapman, Mack, & Pearson, 2006). Some coping behaviors are characterized by antisocial behavioral shifts including depression, anxiety, isolation, peer rejection, increased conflict in relationships, aggression, and substance use (Ford et al., 2006). Ford and colleagues (2006) argue that many of these negative coping strategies arise as a way for victims to feel they are protecting themselves from subsequent victimization and reducing negative emotions while dealing with the current consequences of past victimization event(s). In addition, some antisocial behavioral shifts can have long-term impacts on victims' lives through substance use turning into addiction, weakening or removal of family relationships, loss of employment, legal issues, and criminal involvement (Widom, 2001; Ford et al., 2006; Cook et al., 2017). Not only can these

initial antisocial behaviors have negative impacts on individuals' lives, but they can also increase the propensity for subsequent antisocial behavior (Ford et al., 2006; Cook et al., 2017). Hence, after a victimization event, the onset of antisocial behavior and deteriorating mental and physical health can have several ongoing negative effects on victims' lives. For example, individuals who are exposed to maltreatment and abuse as children have been shown to be at an increased risk to exhibit general antisocial behavior such aggression, depression, and sexually risky behavior in adulthood and substance use/reliance with drugs and alcohol (Miller & Eisenberg, 1988; Moffitt, 1993).

The negative outcomes of victimization that influence antisocial behavior can appear both quickly or over longer periods of time for different individuals. For example, Schwartz and colleagues (1998) used sociometric interviews with children in grade school approximately 8 to 9 years old (n = 330) to measure acute onset antisocial behavior associated with victimization by child peers. They resampled the same children two years later and found that victimization was associated with an increase in externalizing behaviors and attention dysregulation (Schwartz, McFadyen–Ketchum, Dodge, Pettit, & Bates, 1998). Further, victimized children displayed higher levels of these negative behaviors over time and required more attention from teachers due to a lack of the children's ability to integrate into peer groups (Schwartz et al., 1998). A time span as short as two years showed an increase in the onset of antisocial behaviors after victimization occurred within a sample population of 8 to 9 year old children (Schwartz et al., 1998). This developmental period is important considering the degree of brain development and social behaviors that are occurring (Schwartz et al., 1998). Further, if victimization increases the amount of negative and antisocial behaviors that are exhibited

over time, the behaviors could become more serious as the children age and grow into adulthood.

In that same vein, adolescents who experienced sexual victimization have been shown to exhibit several risky antisocial behaviors and substance use throughout their lives following a victimization event such as binge drinking, illegal drug use, and casual sexual intercourse with multiple partners without contraceptive protection, and engaging in violent behavior (Champion et al., 2004). Similarly, Pollock and colleagues (1990) teased apart the relationship between childhood antisocial environments and victimization events as predictors of adult antisocial behavior. Using a clinical sample of 201 adult men they found that men with alcoholic fathers (n = 131; antisocial childhood environment) were not more likely than men without alcoholic fathers (n = 70) to exhibit antisocial behaviors as adults (Pollock et al., 1990). However, they discovered that men within the same sample who had been physically abused as children, regardless of the presence of alcoholic parents, were much more likely to exhibit general antisocial behaviors and substance use and more likely to act aggressively than men who were not physically abused (Pollock et al., 1990).

Similarly, White and Widom (2003) found that young adults, both male and female, who had been abused as children were more likely to exhibit IPV than a matched control group who had not experienced victimization. The sample consisted of individuals who were abuse or neglected before the age of twelve and were interviewed at age 29 (n = 961; White & Widom, 2003). They found that individuals who had experienced victimization events, abuse as children, were more likely to engage in physical and violent IPV than individuals who had not experienced victimization.

Moreover, individuals who had been abused and engaged in IPV were also more likely to exhibit antisocial personality disorders, hostility and aggression (onset not early aggression measured from ages 12 years and before), and alcohol problems associated with childhood abuse and adult IPV (White & Widom, 2003). Hence, victimization can alter the physical and/or mental health of an individual and act as an antecedent to general antisocial behaviors and substance use that influences the rest of the victim's life. However, victimization does not have to occur during years of development or adolescence to act as a risk factor for antisocial behaviors. Victimization during early adulthood and adulthood can increase the risk of negative outcomes for individuals as well (Burnam et al., 1988).

Using a cross-sectional sample of households in two major Los Angeles communities, Burnam and colleagues (1988) found that victimization events during adulthood can lead to subsequent mental health problems as well as increased risk of antisocial behaviors. They found, like many studies prior, that within their sample individuals who were victimized as children were more likely to develop mental illnesses and antisocial behaviors (Burnam et al., 1988). However, they also found that individuals who were victimized only as adults experienced negative outcomes and onset antisocial behaviors as well (Burnam et al., 1988). In addition, when adults had experienced sexual assault (only as adults), especially if subsequent victimization events occurred they exhibited increased risk for depression, drug addiction, and antisocial personality traits (Burnam et al., 1988).

Moreover, Walker, Archer, and Davies (2005) used media advertising in order to recruit men from the general population who had experienced rape during adulthood.

Male rape victims were interviewed concerning their victimization experiences as well as any effects that had occurred following the victimization event (Walker, Archer, & Davies, 2005). While all victims described some level of psychological disturbance, common antisocial themes also emerged such as increased anger and aggression, decreased emotional attachment, substance use, and self-harming behaviors (Walker, Archer, & Davies, 2005). Thus, traumatic victimization events that occur in adulthood only can act as a risk factor for increased negative psychological effects such as depression and anxiety, and antisocial behaviors. It is important to highlight that mental illness and negative psychological outcomes can exacerbate antisocial behaviors and vice versa (Kilpatrick et al., 1985). Hence, the common outcomes associated with victimization can influence each other and make outcomes increase in intensity.

Understanding the consequence of victimization on general antisocial behaviors and substance use is important given the cumulative impact these consequences can have on one's life trajectory. It is important to keep in mind, however, that victimization and the increased proliferation of antisocial behaviors do not occur in a metaphorical black box. There are both psychological and physiological alterations due to experiencing trauma and stress inherent of victimization that are at play, such as the onset of serious degenerative psychopathologies, most namely PTSD.

Post-Traumatic Stress Disorder

Among the negative consequences that victimization can have on individuals, one of the more serious is PTSD. Post-traumatic stress disorder is generally discussed as having three major components including intrusive thoughts, trigger avoidance, and

hyperarousal/reactivity (Kilpatrick, Ruggiero, Acierno, Saunders, Resnick, & Best, 2003).

Intrusive thoughts are arguably one of the main initial symptoms associated with PTSD that emerge and is considered a central factor from which other major symptoms stem (Kilpatrick et al., 2003; NIH, 2016). Intrusive thoughts can best be described as involuntary memories or dreams of the trauma, or associated images, smells, noises, or any other reminder related or perceived to be in relation to the traumatic event (NIH, 2016). It is important to note that intrusive thoughts are not a product of an individual recounting the traumatic event during therapy or actively trying to think about the event in order to alleviate negative emotions. In fact, it is quite the opposite. Intrusive thoughts can occur while individuals are actively trying not to think about the trauma and/or when a victim is taking part in daily activities that should not remind them of the traumatic event. Individuals who suffer from PTSD attempt to decrease the amount or level of impact that these intrusive thoughts have on themselves and their life (NIH, 2016). Many times, to reduce intrusive thoughts individuals alter their life and daily routines (Pfaltz, Michael, Meyer, & Wilhelm, 2013). Further, and discussed in more detail later in this dissertation, many individuals begin to exhibit behaviors that were not present before the onset of PTSD, such as self-medication to reduce the intrusive thoughts (Shipherd, Stafford, & Tanner, 2005). However, in the early stages of development these coping strategies take on more broad behavioral change (NIH, 2016). For example, individuals may start spending more time at home, drinking alcohol or smoking more often, missing work or school frequently, altering what they wear, ceasing involvement in groups or clubs, and decreasing social interactions (Krause, Kaltman, Goodman, & Dutton, 2008;

NIH, 2016). However, these coping mechanisms meant to relieve the negative aspects of PTSD are also associated with behaviors and symptoms inherent of PTSD (NIH, 2016).

One way individuals with PTSD attempt to reduce the impact or frequency of intrusive thoughts is by attempting to avoid situations, environments, people, objects, or any number of other triggers (Pfaltz et al., 2013). Trigger avoidance is the second major symptom associated with PTSD and can exist on a continuum of behaviors ranging from simple behavioral shifts, such as changing the route to work, to more extreme behaviors such as agoraphobia, where individuals will not leave their home, or restrict movement between as few environments as possible (Pfaltz et al., 2013). Further, trigger avoidance is also associated with individuals changing or reducing the amount of social interactions they have in order to reduce the chances of experiencing anything that could act as a potential trigger through conversation or other forms of social interaction (Şalcıoğlu, Başoğlu, & Livanou, 2007). A tragic example comes from a non-empirical account of a young female who developed later onset PTSD due to being kidnapped and raped by a serial rapist at age ten. She explained that her PTSD symptoms did not become apparent until getting her driving certification. It was at this point of independence that she would often sit in her car for long periods of time assuming her rapist was in the bushes of her parents' home to attack and rape her again. She felt as though there would always be an attacker in her rearview mirror when she looked in it, thus she chose not to drive or leave her home. Home was an area of safety for her and a way to avoid any confrontation of emotions associated with the trauma or triggers of the event. Ultimately, the fear of experiencing a trigger of the event became a new form of stress or revictimization which was increasing the severity of her symptoms. This is a common aspect of PTSD as

avoidance is easier in the short term than confronting the trauma or triggers, and these degenerative avoidance patterns can become habitual if not treated (NIH, 2016).

Hence, avoidance can be a behavioral alteration that changes several aspects of an individual's life. Although the above account was specific avoidant behavior due to a specific victimization event, by and large, avoidant behaviors can exist as changes in peer group composition, size and presence of a peer group and social network, decreased participation in school or work, and a plethora of other negative life outcomes related to decreased prosocial interactions (Shipherd, Stafford, & Tanner, 2005; Şalcıoğlu, Başoğlu, & Livanou, 2007; Pfaltz et al., 2013).

The third component of PTSD associated with both intrusive thoughts and potential anxiety associated with experiencing triggers is hyperarousal reactivity (NIH, 2016). Hyperarousal is generally discussed as the third major symptom of PTSD which includes feeling physically, emotionally, and mentally on edge (Bremner et al., 1996). Many individuals who suffer from PTSD explain the feeling of hyperarousal as being "keyed up", feeling like something bad is always about to happen, or being extremely jumpy and nervous (Bremner et al., 1996; NIH, 2016). Due to the aforementioned feelings associated with anxiety, the second aspect of hyperarousal reactivity results in an increased propensity for overly excitatory responses to stressful stimuli (Bremner et al., 1996). Individuals often explain this sensation as overreacting to events or interactions in a more accelerated aggressive manner compared to before the traumatic event occurred and PTSD symptoms began (Bremner et al., 1996; NIH, 2016). Further, increased aggression is associated with criminal behavior and a propensity toward violence (Dyer et al., 2009). This aspect of aggression with hyperarousal due to PTSD symptoms is

frequently discussed as problematic with maintaining new or existing prosocial relationships (e.g., romantic relationships, family interaction, and employment).

Further, and to better outline the variable of PTSD for the current dissertation, PTSD will be discussed as a measurable construct that captures similar symptoms of individuals' psychological, biological, and behavioral patterns (NIH, 2016). Although the definition and diagnosis of PTSD only began in the 1980s, the symptoms and reality of PTSD for individuals who have experienced stress or trauma is not a modern concept. Throughout recorded human history the effects of trauma and associated alterations to behavior have been discussed in both fictional and non-fictional works. For example, as far back as Homer's *The Iliad* (Butler, 1952) and other literary works concerning the ancient world, the trauma experienced by individuals, namely soldiers, is discussed as having *war sickness* or in some cases *wine sickness* which caused individuals to be aggressive, agoraphobic, and drink too much (Doerries, 2015). In 1761, Josef Leopold, an Austrian physician coined the term *nostalgia* or *soldier's nostalgia* as an over romanticized term specifically associated with soldiers that described PTSD like symptoms such as feeling sad, problems sleeping, and anxiety due to their longing to be back in battle (Gradus, 2014). Leopold's observations led to increased attempts among physicians to solidify a more appropriate medical diagnosis in the late 1800s following the Civil War in the United States and the Franco-Prussian War in Europe (Gradus, 2014). It was around this time that several terms such as *soldier's heart*, *irritable heart*, *railway spine*, and *shell shocked* were used to describe the same (or very similar) traits and behaviors of depression, substance use, trouble sleeping, avoidance, and hyperarousal of the cardiac system in individuals who had experienced trauma (Gradus, 2014).

To illustrate that PTSD was present in real world past populations before the measure of PTSD symptoms was created, Kuch and Cox (1992) conducted a study of holocaust survivors using DSM-III-R diagnostic records looking for explicit accounts fitting current descriptions of PTSD. They found that survivors who faced increased levels of trauma in harsher concentration camps (i.e., Auschwitz) exhibited increased levels of sleep disturbance, recurrent nightmares, and intense distress over reminders (i.e., triggers) of their time in the camps (Kuch & Cox, 1992). Again, although the modern construct of PTSD was not available to clinicians in the 1940s, the aforementioned study shows that severe trauma influences individuals in similar ways. Further, trauma manifests into similar symptoms related to PTSD, be it one of the gravest acts of systematic genocide in known history or trauma experienced by single individuals through any form of severe stress. Traumatic event(s) can influence behavioral shifts based on the severity and duration of the trauma and can have a negative influence on relationships and prosocial networks that could aid in the dulling of symptoms of PTSD (Kuch & Cox, 1992; Silver, Brooks, & Obenchain, 1995).

Silver, Brooks, and Obenchain (1995) gathered self-report and biofeedback data from Vietnam War veterans who were receiving inpatient treatment for PTSD. They found that individuals who suffered from PTSD reported increased anxiety, cardiac issues, anger, depression, isolation, intrusive thoughts, flashbacks, and nightmares (Silver, Brooks, & Obenchain, 1995). Further, these symptoms increased relationship problems and exacerbated symptoms after prosocial relationships deteriorated (Silver, Brooks, & Obenchain, 1995). Although the sample they used were receiving treatment and many of the respondents showed an improvement, some individuals either did not

seek treatment due in part to avoidant behaviors or did not have the socioeconomic means to do so (Goenjian, Walling, Steinberg, Karayan, Najarian, & Pynoos, 2005). Further, past research has suggested that the longer detrimental symptoms go untreated the greater the risk becomes that symptoms associated with PTSD can manifest into increasingly negative outcomes for individuals who have suffered trauma (Goenjian et al., 2005).

Goenjian and colleagues (2005), for example, used a sample of adolescents from the cities of Gumri and Spitak who experienced the Armenian Spitak earthquake in 1988 to better understand the effects of untreated PTSD symptoms. The Spitak earthquake was devastating to both cities and killed around 50,000 people making it a traumatic event for survivors (Goenjian et al., 2005). Goenjian and colleagues (2005) assessed 125 adolescents using the Child Post-traumatic Stress Disorder Reaction Index (CPTSD-RI) at 1.5 and 5 years after the earthquake. Adolescents from the city of Gumri were offered six weeks of psychotherapy geared towards PTSD and depression at the 1.5 year mark but adolescents from Spitak were not able to be given the same treatment and thus were used as a comparison control group (Goenjian et al., 2005). They found that untreated individuals were much more likely to exhibit behaviors above the cutoff for diagnosable PTSD symptoms and were more likely to exhibit comorbid depression (Goenjian et al., 2005). Goenjian and colleagues (2005) argue that if PTSD symptoms remain unchecked it is likely they will not remain static but instead that these symptoms will increase in severity and increase the likelihood of comorbid psychopathologies such as depression and antisocial behavioral outcomes.

Similar to shame associated with victimization, the stigma often associated with PTSD (especially resulting from victimization) can reduce the chances of individuals

seeking professional help, which in turn can exacerbate symptoms (Tangney & Fischer, 1995; Lee, Scragg, & Turner, 2001). In addition, individuals who suffer from PTSD associated with trauma from criminal victimization where they felt powerless and humiliated, namely physical and sexual violence are much more likely to experience shame associated with victimization, not seek help, and exhibit stronger negative symptoms than individuals who seek professional help (Gilbert, 2000). Many times the lack of therapy or other forms of professional help can leave individuals who suffer from PTSD at the mercy of their symptoms and they must rely on themselves to manage the negative effects with makeshift coping strategies that have the possibility of exacerbating the symptoms attempting to be curtailed.

By not seeking help for symptoms of PTSD and related psychopathologies the negative effects can become worse. Further, worsening PTSD symptoms are likely to manifest into behavioral shifts and changes in lifestyles as both a product of and in order to reduce the symptoms of PTSD which can in turn increase antisocial behaviors associated with trauma and symptoms (Kilpatrick et al., 1987; Leeies, Pagura, Sareen, & Bolton, 2010).

PTSD & Antisocial Behavior

Some behavioral shifts due to PTSD have been noted as either antisocial in and of themselves or increase the risk of antisocial behavior to develop (Resnick, Foy, Donahoe, & Miller, 1989). For example, some repeatedly documented behavior changes in individuals who suffer from PTSD are increased substance use, changes in daily routines that reduce social support, increased affiliation with antisocial peers, and increased aggressive behavior (Kilpatrick et al., 1987; Resnick et al., 1989; Kerig, Becker, & Egan,

2010). Specifically, Resnick and colleagues (1989) used data collected from Vietnam War veterans who had sought out psychological help in the Los Angeles area concerning an onset in increased antisocial behaviors. The change in antisocial behavior was defined using the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychological Association, 1980) which measured pre-adult antisocial behaviors and onset antisocial personality disorder (Resnick et al., 1989). They also measured the level of trauma experienced (i.e., amount of combat exposure) and the presence of developed PTSD symptoms (Resnick et al., 1989). They found that both increased combat exposure and pre-adult antisocial behaviors were not significant predictors of onset adult antisocial behaviors when PTSD was present in the model (Resnick et al., 1989). Hence, for this sample population PTSD was a stronger predictor of the development and maintenance of antisocial behaviors than increased trauma (i.e., combat exposure) and pre-adult antisocial behaviors alone (Resnick et al., 1989).

Due to the interrelated and overlapping nature of PTSD symptoms, it is difficult to discuss each symptom of PTSD with associated antisocial behaviors on their own. Generally behavioral shifts are better explained based on one or more of the PTSD symptoms. For example, a common behavioral shift correlated with PTSD symptoms is self-medication through substances such as drugs and/or alcohol (Leeies et al., 2010). However, onset may be due to intrusive thoughts and hyperarousal while the maintenance of substance use can be more associated with avoidant behaviors (Gradus, 2007; Leeies et al., 2010). Considering the prevalence of substance use among individuals with PTSD early researchers wanted to better understand this relationship. For example, Chilcoat and Breslau (1998) sought to understand the order in which substance use and PTSD occurred

relative to the initial traumatic event. At the time their study was conducted many researchers argued that trauma was generally a strong risk factor of substance use which was a risk factor for the development of PTSD. Substance use was thought to be a precursor to PTSD (Chilcoat & Breslau, 1998). However, Chilcoat and Breslau (1998) shifted that paradigm by revealing that substance use resulted as a form of self-medication in which PTSD symptoms formed due to trauma and the purpose of substance use was to dull or remove the negative symptoms. Thus, they showed that it is more likely the antisocial behavior of substance use was an effect of PTSD and not an antecedent (Chilcoat & Breslau, 1998).

Further research has corroborated findings of earlier broad based sample studies, such as Chilcoat and Breslau (1998), but focused on specific groups known to suffer from both substance use/addiction and PTSD. Using a longitudinal sample of 1,006 veterans of the Persian Gulf War, Shiperd, Stafford, and Tanner (2005) found that both drug and alcohol abuse were significantly correlated with all three factors of PTSD (i.e., intrusive thoughts, avoidance, and hyperarousal) for veterans suffering from PTSD six years after being involved in the Persian Gulf War. Moreover, the onset of this antisocial behavior was shown to be more likely after the onset of PTSD symptoms (Shiperd, Stafford, & Tanner, 2005).

Leeies and colleagues (2010) used data from the National Epidemiologic Survey on Alcohol and Related Conditions, which is a nationally representative sample of individuals with mental illness and substance use problems. They found that roughly 20% of individuals suffering from PTSD self-medicated through drugs or alcohol in order to relieve their symptoms. Moreover, a more recent study found that within a sample of

individuals being treated for alcohol abuse disorder (N = 187) that individuals who also suffered from PTSD were more likely to experience major depression, attempted suicide, an earlier peak of drinking problems, increased drinking quantity and withdrawal symptoms, and increased alcohol related blackouts (Neupane, Bramness, & Lien, 2017). Some individuals who suffered from PTSD and turned to self-medication end up exacerbated the already negative symptoms due to aspects of biological levels of addiction and through alienating themselves with prosocial groups (e.g., colleagues, friends, or family) (Holmila, 1995).

Hence, individuals who experience trauma and develop PTSD are at a higher risk of experiencing behavioral changes that increase their risk of antisocial behaviors. Keeping the aforementioned information in mind, substance use is a common antisocial behavior associated with PTSD as well as a well-studied correlate and risk concerning other antisocial and criminal behavior (Cadoret, Troughton, O'Gorman, & Heywood, 1986; Krueger, Hicks, Patrick, Carlson, Iacono, & McGue, 2002; Hussong, Curran, Moffitt, Caspi, & Carrig, 2004). Further, substance use by individuals in an attempt to dull the effects of PTSD symptoms could increase risk factors for exhibiting antisocial behaviors over time (Catalano & Hawkins, 1996; Hussong et al., 2004). Self-medication through substance use is not the only antisocial behavioral shift associated with PTSD. Increased aggression and violent behavior have also been linked to PTSD symptoms (Galovski & Lyons, 2004; Orcutt, King, & King, 2003; Dyer et al., 2009; Elbogen et al., 2010).

In a review of the literature, Galovski and Lyons (2004) explain the increasingly worsening circle of stress and trauma that individuals with PTSD experience and how it

can increase their propensity for antisocial behavior. They argue that PTSD causes stress and deterioration of relationships, especially within the immediate family, which acts as secondary and repeated trauma to the individual with PTSD (Galovski & Lyons, 2004). The repeated nature of family problems exacerbates antisocial behaviors associated with PTSD such as substance use and violent outburst associated with family confrontations (Galovski & Lyons, 2004). For example, Orcutt, King, and King (2003) found that Vietnam veterans who had experienced increased early life stressors, increased exposure to war zone stressors, and exhibited increased PTSD symptoms, namely hyperarousal, were significantly more likely to abuse substances and perpetrate IPV. Further, being involved in instances of IPV and experiencing PTSD symptoms, such as hyperarousal, increased the chances of Vietnam veterans were arrested (Orcutt, King, & King, 2003).

Although family-oriented aspects of PTSD are important, some antisocial behaviors such as anger and aggression are more likely to be inherent of the psychopathology and initial severity of the traumatization (Dyer et al., 2009). Dyer and colleagues (2009) compared two groups of individuals that suffer from PTSD, current common PTSD (e.g., car accident; n = 31) and current complex PTSD (e.g., torture, experiencing IPV, multiple combat exposures, or severe social deprivation–solitary confinement for months; n = 11) to see the differences in manifested outcomes. They found that all individuals who suffer from PTSD exhibit increased levels of anger, aggression, and self-harm, but individuals who suffered from the more severe form of PTSD exhibited significantly higher levels of physical aggression and self-harm than the other individuals with less severe PTSD symptoms (Dyer et al. 2009). These individuals also reported self-loathing and destructive behaviors and a multitude of other less serious

antisocial behaviors such as ill-will towards others, bitterness, and general resentment for people (Dyer et al., 2009).

Moreover, Elbogen and colleagues (2010) interviewed a voluntary sample of 676 veterans of the Iraq and Afghanistan wars who had served from the earliest start date cutoff of September 11, 2001. The interviews consisted of instruments designed to capture psychiatric symptoms, overall physical and mental health, and post-deployment behavioral issues (Elbogen et al., 2010). Elbogen and colleagues (2010) conducted their study to add empirical rigor to the concept of after deployment aggression being a product of stress and trauma associated with PTSD versus pre-existing behaviors and individuals' inherent traits. They found that aggressive impulsivity and urges, difficulty managing anger, and perceived problems managing violent behavior were significantly associated with hyperarousal symptoms of PTSD and were onset after PTSD symptoms began (Elbogen et al., 2010). Further, the other factors associated with PTSD, such as intrusive thoughts and avoidance, were less strongly and less consistently associated with anger and hostility (Elbogen et al., 2010). Hence, while all of the symptoms of PTSD are important concerning antisocial behaviors, hyperarousal seems to be especially associated with behaviors linked to aggression and violent behavior. Hyperarousal is also the component of PTSD that is most closely related to individuals' physiology (NIH, 2016).

The above sections explain the similar effects that the consequences of victimization and the symptoms of PTSD from trauma have on behavior, specifically the increased risk of antisocial behavior. Moreover, due to the intertwined outcomes that victimization and PTSD trauma share, they also manifest into salient negative and antisocial behaviors. Interestingly, there is a physiological component that has been

shown to be associated with victimization, symptoms of PTSD, and antisocial behaviors, namely the functionality of the autonomic nervous system (ANS).

Specifically, studies have shown victimization and PTSD to be associated with dysfunctional ANS that cause a skewed increased HRR to stress (Chrousos, Kino, & Charmandari, 2009). The current dissertation focuses on victimization, PTSD, and HRR (ANS function) as risk factors for general antisocial behaviors and substance use. Considering that the relation victimization and PTSD share with HRR is through the functionality of the ANS, HRR will be discussed as a part of the larger system of the ANS. Herein, the effects of trauma related to ANS dysfunction will be discussed as a risk factor for antisocial behavior, and measured in the current dissertation via HRR.

The Autonomic Nervous System

The autonomic nervous system is a regulatory physiological system involved with both excitatory reactions to stimuli and maintaining long-term homeostasis within the body (Sowers & Mohanty, 1988; Low, 1993). The ANS is a dynamic and complex system, but generally concerning behavior is discussed as two subsystems, the sympathetic and parasympathetic systems (Low, 1993). The sympathetic system acts as the excitatory system and the parasympathetic system exhibits a reactionary calming function to the sympathetic system and maintains long-term homeostasis in the body in lieu of short-term excitatory responses from day to day experiences (Sowers & Mohanty, 1988). Ultimately the evolutionary underpinnings of the sympathetic system serves as a biological reaction to stress or danger, in that the sympathetic system supplies the hormones needed to fight or flight the stressor/danger and the parasympathetic supplies

the hormones to calm the body after the stressful event has ended (Jansen, Van Nguyen, Karpitskiy, Mettenleiter, & Loewy, 1995).

The ANS is part of an integrative set of biological systems that includes interactions between the brain and the body via the hypothalamic pituitary adrenal axis or HPA-axis (Hall, Podawiltz, Mummert, Jones, & Mummert, 2012). Further, the ANS interacts with several expansive biological systems such as the circulatory, endocrine, and neurological systems (McCorry, 2007).[2]

One of the major functions of the ANS is to allow for visceral reactions of the body based on environmental stimuli (McCorry, 2007). An initial reaction from the ANS is likely due to an outside stimulus that is processed within the brain as a perceived stress or threat (Holsen et al., 2012). Specifically, the initial reaction occurs in the amygdala and a message is sent to the hippocampus and prefrontal cortex (Shin et al., 2006). The response of the amygdala is simultaneously processed by the hippocampus and pre-frontal cortex to see if the same or similar stimulus has occurred before, and to filter the information through the logic center to produce the most appropriate initial reaction (Shin et al., 2006; Holsen et al., 2012). Then a message is sent from the brain down the HPA-axis to the adrenal glands where the ANS elicits an excitatory response via the sympathetic system's release of adrenaline and biochemical agents into the bloodstream (Shin et al., 2006; Holsen et al., 2012). When adrenaline and other excitatory biochemical compounds are released into the bloodstream, blood vessels become constricted thereby increasing blood pressure, increasing heart rate, and breathing becomes faster and more

[2] For the sake of the current dissertation the basic interactions of the autonomic nervous system will be discussed in a simple and straightforward description as the behavioral outcomes rather than the biochemistry or biological specifics of the ANS.

shallow (McCorry, 2007). If the stimulus exhibits a threat, then the excitatory response will remain active until the threat is no longer present or the individual is able to remove themselves from proximity of the stimulus (McCorry, 2007). Similarly, in a healthy ANS, if the threat was a misconception by the brain, the excitatory response will not be severe and will have little-to-no effect on the individual's physiological response (McCorry, 2007).

After the excitatory stimulus is over, the body will then release hormones into the brain as a negative feedback system to let the brain know that the body is ready to return to homeostasis (Low, 1993; Hall et al., 2012). The body returns to homeostasis when the parasympathetic system releases the hormone acetylcholine, which causes blood pressure, heart rate, and all other circulatory functions to decrease until homeostasis is reached (Donato et al., 2013). A normal functioning ANS will remain at homeostasis until another excitatory stimulus is present and after the stimulus is over the parasympathetic system will slowly and steadily bring the body back to homeostasis again (Donato et al., 2013). In addition, individuals with normal functioning ANS will exhibit appropriate responses to stressful and excitatory stimuli, as well as a stable return to homeostasis each time stress arises in their environment (Hall et al., 2012).

However, individuals who have experienced high levels of stress can experience a type of biological burnout resulting in a dysfunctional ANS (Chrousos, Kino, & Charmandari, 2009). For example, individuals who have experienced high levels of stress and trauma, especially those who develop PTSD, are at a higher risk of ANS dysfunction and may exhibit an increased propensity to overreact to stimuli and experience an increased vulnerability to stress due to over- or under-activity of either the sympathetic or

parasympathetic systems (Shin et al., 2006; Chrousos, Kino, & Charmandari, 2009; Uy et al., 2013). Further, individuals with PTSD and dysfunctional ANS also exhibit increased vulnerability to stress and hyperactivity on a biological level (Shin et al., 2006; Streeter et al., 2012).

For example, individuals who experience a dysfunctional ANS in the form of an overactive sympathetic system can experience complications in the HPA-axis or within the endocrine system as a whole (Tsigos & Chrousos, 2002; Chrousos, Kino, & Charmandari, 2009). Considering that these individuals' sympathetic systems are always at a higher baseline than a normal functioning ANS these individuals often report feeling on edge and are over responsive to stimuli (Chrousos, Kino, & Charmandari, 2009). Normal day-to-day stressors such as not being able to find keys or being in traffic, while frustrating to all individuals, would have a much stronger negative impact and elicit a much stronger response from individuals with a dysfunctional ANS. Thus, having an overactive sympathetic system can increase vulnerability to stress and increase reactivity to stimuli.

Similar to experiencing an overactive sympathetic system, some individuals experience an underactive parasympathetic system (Chrousos & Gold 1992; Tsigos & Chrousos, 2002; Chrousos, Kino, & Charmandari, 2009). An underactive parasympathetic system reduces the ability of individuals to return to homeostasis after an excitatory reaction occurs. For example, if a stimulus causes a sympathetic system reaction the parasympathetic system either takes longer to reduce the excitatory reaction or does not reduce it enough to achieve homeostasis (Donato et al., 2013). Hence, although a different physiological mechanism occurs the outcome is the same, an

individual who experiences increased hyperarousal for an extended duration of time. Similar to an overactive sympathetic system, individuals with underactive parasympathetic systems are also more vulnerable to stress and exhibit overreactions to stimuli (Chrousos, Kino, & Charmandari, 2009).

The third form of autonomic dysfunction that will be discussed is when either of the subsystems attempts to overcompensate for the dysfunction of the counter subsystem. For example, an individual who experiences acute high sympathetic reactions would experience a very sudden and drastic change in blood pressure, heart rate, and breathing rate and the parasympathetic system may overcompensate with a quick release of a large quantity of acetylcholine (Bracha, 2004). This attempt of the parasympathetic system to overcompensate for the acute and drastic sympathetic reaction will not bring the body slowly and steadily back to homeostasis but instead cause drastic circulatory reactions due to quick concentration changes of adrenaline and acetylcholine in the blood stream within only a few seconds (Bracha, 2004). Again, this form of autonomic dysfunction leads to individuals feeling uneasy and exhibiting behavioral overreactions to stimuli due to a physiologically dysfunctional ANS which can increase general antisocial behaviors and substance use.

The Autonomic Nervous System Dysfunction & Antisocial Behavior

The ANS encompasses several other physiological systems to process information for a physiological response to stimuli (McCorry, 2007). Within the central nervous system, amygdala, hippocampus, and prefrontal cortex dysfunction has been linked to autonomic dysfunction as these brain regions are paramount in assessing stressful or dangerous stimuli in a working neurological union (Shin et al., 2006). Individuals who

suffer from PTSD exhibit an increased likelihood of experiencing brain function problems in these key areas concerning behavior and the biological management of stress. Shin and colleagues (2006) used a functional magnetic resonance imaging system (fMRI) to track the brain function of individuals with PTSD. They found that the amygdala function and responsivity was directly related to the severity of PTSD symptoms such as increased hyperarousal and anxiety related to intrusive thoughts as well as reciprocal avoidance behaviors (Shin et al., 2006). Moreover, individuals who experienced overactive amygdala and exhibited increased severity of PTSD symptoms exhibited decreased functioning of the prefrontal cortex, specifically the medial prefrontal cortex as well as their hippocampus (Shin et al., 2006). These findings are important for understanding PTSD, the ANS, and antisocial behavior considering the function and relationship of each of these brain regions in dealing with stressful stimuli and making logical decisions when acting in concert together. Although in reality, brain regions fire quickly if not almost simultaneously, but for simplicity the brain's reaction to stress will be discussed as a linear set of events starting with the amygdala.

The amygdala sends the initial stimuli response to the rest of the brain and then onto the body. If stress or danger is perceived, the amygdala sends an excitatory response, which in a normal functioning brain would be buffered by the hippocampus and prefrontal cortex to offer the appropriate amount of physiological stress response (Shin et al., 2006). However, as shown by Shin and colleagues (2006) this is likely not the case for individuals suffering from PTSD. In fact, the brain dysfunctions described above are likely to exacerbate the reaction to perceived stress due to the amygdala responses being unchecked by the low functioning hippocampus and prefrontal cortex. To add support to

the concept of individuals with PTSD exhibiting vulnerability to stress, Shin and colleagues (2006) found that over amygdala response as a form of dysfunction was directly associated with more severe PTSD symptoms. Said another way, the amygdala in individuals with PTSD is overactive and is sending stronger and more frequent stress signals to the ANS and the buffering components of the brain (hippocampus and prefrontal cortex) are hypoactive (Shin et al., 2006).

A dysfunctional ANS can exhibit either an increased/decreased sympathetic or parasympathetic response which can influence individuals' behaviors (Tsigos & Chrousos, 2002; Chrousos, Kino, & Charmandari, 2009). Moreover, considering that the ANS is made up of two complimentary subsystems, dysfunction in one system is likely to elicit an over response in the counter system in order to compensate for the prior system's dysfunction. Hence, dysfunction can result from either or both of the two subsystems reacting to or attempting to compensate for the other (Tsigos & Chrousos, 2002; Chrousos, Kino, & Charmandari, 2009). Given the excitatory and calming functions of the ANS any type of dysfunction could be important concerning behavioral reactions to social stimuli and stress. Though the mechanisms and effects of the ANS are complex, one system within the body the ANS can have strong impacts on is the cardiovascular system, namely through resting heart rate (RHR) and HRR (Akselrod, Gordon, Ubel, Shannon, Berger, & Cohen, 1981; Kolloch et al., 2008).

The impact of the ANS on heart rate can be observed through changes to both RHR and the intensity of a HRR (Kolloch et al., 2008). Resting heart rate is the measure of how many times an individual's heart beats during a 60 second time frame when they are at rest and is a consistent measurement that does not change over short amounts of

time (Logan, Reilly, Grant, & Paton, 2000). Conversely, HRR is a cardiovascular response to a stimulus generally manifesting as an acute increase in heart rate (HR) followed by a return to or near the RHR and varies in degree and duration based on the stimuli (Light & Obrist, 1983). Resting heart rate and HRR can be influenced by levels of physical activity, diet, life style, and stress (Dishman, Nakamura, Garcia, Thompson, Dunn, & Blair, 2000). Autonomic nervous system dysfunction concerning RHR and HRR has been shown to be a risk factor for antisocial behaviors, especially aggression and substance use (De Bellis, 2002; Sijtsema, Ojanen, Veenstra, Lindenberg, Hawley, & Little, 2010; Portnoy et al., 2014; Raine, Fung, Portnoy, Choy, & Spring, 2014).

Mezzacappa and colleagues (1997) used a sample of teenage males and starting at the age of 15, tracked their RHR, HRR, anxiety, and antisocial behavior annually for between 4 to 6 years (n = 175). They found a direct relationship between antisocial behavior and low RHR and low HRR (Mezzacappa et al., 1997). Further, they found that high levels of anxiety and stress were associated with high RHR and high HRR (Mezzacappa et al., 1997). Interestingly, within the overall higher HR group that exhibited high levels of anxiety, individuals who exhibited increased antisocial behavior had lower RHR but higher HRR relative to the high HR anxiety group (Mezzacappa et al., 1997). Hence, antisocial behaviors and HR maintain some patterns but anxiety and stress can alter the relationship to an extent.

Moreover, Gottman and colleagues (1995) provided a controlled experimental setting to directly view how social interactions influence HR and how those cardiac changes were associated with antisocial behavior. To do so, they used couples who had past records of domestic disputes where both individuals acted violently and were

identified as an aggressor (Gottman et al., 1995). The males were asked to sit with their eyes closed in a room with their partner present where they were provided with a subject to discuss (Gottman et al, 1995). The subject was selected from topics the couples had provided as problematic issues in their relationship (e.g., how to behave at a party or negative issues surrounding in-laws; Gottman et al., 1995). During the argument (verbal only as physical contact was not allowed), Gottman and colleagues (1995) found that males whose HR lowered (i.e., HRR to the argument) during controlled eye closed verbal argument tests with their partners were more likely to be violent both within and outside their relationship (e.g., accounts of violence concerning friends, strangers, co-workers, and bosses; Gottman et al., 1995). Moreover, males who exhibited an increase in HR (i.e., HRR) during the same tests while still as violent with their partner were not as likely to exhibit antisocial behaviors outside of the relationship (Gottman et al., 1995). In addition, males who experienced a lowering HRR during the tests reported higher levels of overall antisocial behavior, aggression, and belligerency (Gottman et al., 1995).

Ortiz and Raine (2004) conducted a meta-analysis to better understand the association between HR function and antisocial behavior throughout the current literature up to that time. They found that both RHR and HRR (associated with a controlled stressor) were associated with antisocial behavior outcomes. Specifically they used 40 studies encompassing a collective 5,868 children and found that RHR (d = -.44) and HRR (d = -.76) were both significantly associated with antisocial behavior (Ortiz & Raine, 2004). Interestingly, while low RHR was a consistent predictor of antisocial behavior, Ortiz and Raine (2004) discussed that higher levels of RHR and HRR were associated with increased antisocial behavior when stress, anxiety, or PTSD were present.

Thus, antisocial behavior has been shown to be associated with RHR and HRR and aspects of stress and psychopathologies such as PTSD can influence that association (Ortiz & Raine, 2004). In addition, antisocial behavior associated with HR related to ANS dysfunction has been documented among individuals suffering from PTSD (Solomon, 1989). Taken together, the effects of RHR and HRR in relation to ANS dysfunction is a risk factor for antisocial behaviors and has been shown to be prevalent in individuals who have experienced victimization and have exhibited symptoms of PTSD (Solomon, 1989; Scarpa, Haden, & Hurley, 2008; Dyer et al., 2009)

Hence, many of the consequences of victimization and negative behaviors associated with PTSD could be in part based on the physiological alteration to the function of the ANS and how individuals respond to stressful stimuli and the constant perception of potential stress on a daily basis. Individuals who have been victimized can experience an alteration in behaviors and are at a higher risk of suffering from symptoms of PTSD which has been documented along with ANS dysfunction. The literature above argues that individuals who have been victimized, exhibit PTSD, and/or experience ANS dysfunction are at an increased risk of experiencing an increased vulnerability to stressful stimuli. Increased vulnerability to stress causes them to be on edge and exhibit increased levels of antisocial behavior when feeling threatened or overwhelmed (Solomon, 1989; Scarpa et al., 2008; Dyer et al., 2009). Feeling overwhelmed by the stress of everyday tasks or social interactions can influence potential self-medication via substance use to decrease or dull the negative effects (Solomon, 1989; Scarpa et al., 2008; Dyer et al., 2009). Further, several factors can influence the cascade of the risk factors of victimization, PTSD, and ANS dysfunction (abnormal HRR) on antisocial behavior and

substance use. Of the traits that influence these risk factors gender has been shown to impact each risk factor and how they interact.

Gender Differences: Victimization, PTSD, Autonomic Nervous System & Antisocial Behavior

The second aspect to the current dissertation is to better understand how males and females differ based on how the three risk factors of victimization, PTSD, and HRR influence general antisocial behavior and substance use. Gender is an especially interesting factor given that differences have been documented in both the predictor and predicted variables of the proposed dissertation. For example, males and females experience different rates, and types of victimization, intensity of feelings associated with trauma, affects associated with victimization, and rates of antisocial behaviors (NCADV, 2014). Further, in broad terms females have been shown to be more likely to experience PTSD associated with trauma, and have increased HRR to stress (Ordaz & Luna, 2012; NIH, 2016).

To highlight differences between males and females concerning victimization McClellan and colleagues (1997) used a forensic sample of adults and found that females were more likely than males to experience increased levels of maltreatment in their youth and were also more likely to exhibit depression and report substance dependence based on childhood maltreatment experienced. Moreover, Garza and Jovanovic (2017) conducted a review of the literature concerning gender differences and PTSD based on trauma experienced as children and adolescents. They found that a large portion of children and adolescents are exposed to traumatic events and exhibit symptoms related to PTSD, although not at the clinically diagnosable level (Garza & Jovanovic, 2017). They

argue that there are inherent differences among males and females based on how trauma is processed on sociocultural, biological, and psychological levels and that due to these current specifically undefined factors females are twice as likely to develop PTSD and other negative psychopathologies related to trauma (Garza & Jovanovic, 2017).

In addition, when both males and females are exposed to similar types of stress, specifically hyper-traumatic stress such as combat exposure in the military, females still exhibit increased levels of PTSD when compared to males (Luxton, Skopp, & Maguen, 2010). Luxton, Skopp, and Maguen (2010) retrospectively conducted a before and after sample of 516 females and 6,427 males who had actively served in the Afghanistan and Iraq Wars to better understand differences in effects of combat exposure to males and females concerning symptoms associated with PTSD. They found that even though males were exposed to higher levels of combat exposure and had experienced higher amounts of deployments, females still exhibit a higher frequency of PTSD and depression symptoms (Luxton, Skopp, & Maguen, 2010).

There is a possibility that these differences in effects of stress are in some manner influenced by differences in physiological responses between males and females. Kudielka and colleagues (2004) used a sample of 88 individuals to understand differences in gender and physiological responses to stress by comparing their ANS reactions during the Trier Social Stress Test (TSST). They found that within the three major life stages of children, young adulthood, and older-elderly adulthood that females exhibited some form of an increased HRR to stress more so than males regardless of age (Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004). More specifically, girls and young adult females exhibited an increased HRR to stress more so than boys and young adult

males (Kudielka et al., 2004). Further, within older-elderly adults although there were no differences in magnitude of HRR response, males returned to homeostasis much faster than females (Kudielka et al., 2004).[3] Hence, on a physiological level there are differences in how stress and trauma influence individuals based on gender.

More specific to antisocial behavior as an outcome, in a meta-analysis Hubbard and Pratt (2002) point out that many studies focus on factors that increase the propensity for delinquent and antisocial behaviors for males, as males account for a disproportionate amount of delinquency and antisocial behavior compared to females. Further, the well-known risk factors associated with male antisocial behavior is generalized to females such as previous antisocial behaviors in life and antisocial peers (Hubbard & Pratt, 2002). While the same predictors for antisocial behavior in males were also significant in females, they also found that the victimization events of physical or sexual assault were among strong indicators of future antisocial behaviors for females (d = .21; Hubbard & Pratt, 2002).[4] In addition, they argue that according to the studies included in their meta-analysis (n = 97), that victimization had the same level of impact (d = .21) as antisocial personality which both tied for the third strongest predictor of antisocial behavior in their analysis (Hubbard & Pratt, 2002).

Moreover, in a study focused on runaway adolescents McCormack, Janus, and Burgess (1986) found that from 144 respondents, females were approximately twice as likely to have experienced sexual victimization as males. Specifically, out of 89 male runaways 38% of them had experienced sexual victimization, while out of 55 female runaways 73% of them had experienced sexual victimization (McCormack, Janus, &

[3] At this age group HRR is generally more similar between males and females (Kudielka et al., 2004).
[4] Hubbard and Pratt (2002) used mean effect size for their meta-analysis.

Burgess, 1986). In addition, abused individuals, both male and female, were more likely to report anxiety and depression over non-abused individuals (McCormack, Janus, & Burgess, 1986). Interestingly, males exhibited increased fear and mistrust of adult men, while females were more likely to engage in onset delinquent/criminal and antisocial behaviors such as substance use, theft, and assault (McCormack, Janus, & Burgess, 1986). These findings are interesting in that generally males tend to exhibit increased antisocial behaviors and females tend to exhibit increased levels of fear and mistrust of strangers in non-abused populations (Stafford, Chandola, & Marmot, 2007). However, there is evidence presented in the above studies that victimization, PTSD, and HRR differences occur and have been documented to increase the risk of antisocial behavior based on gender.

Purpose of Dissertation

Herein, the purpose of the current dissertation is to examine the relationship between the consequences of victimization, symptoms of PTSD, and ANS dysfunction (measured via HRR) as risk factors for general antisocial behavior and substance use. The inclusion of the three-way interaction between victimization, PTSD, and ANS dysfunction (i.e., increased HRR) will be the model that best shows the relation between increased general antisocial behavior and substance use more so than any combination of a two-way interactions or the three risk factors alone. In addition, given that females have been shown to experience an increased effect associated with all three risk factors to a greater degree than males, the three-way risk factor interaction will be a stronger predictor for antisocial behavior and substance use for females than for males.

Hypotheses

H_1: The primary hypothesis of the current dissertation is that if individuals exhibit increased victimization, increased PTSD, and increased HRR they will also exhibit increased antisocial behavior, soft substance use, and hard substance use for the full sample (see Figure 1 below).

H_2: The second hypothesis is that gender differences exists between victimization, PTSD, and HRR and those risk factors will influence males and females differently concerning antisocial behavior and substance use.

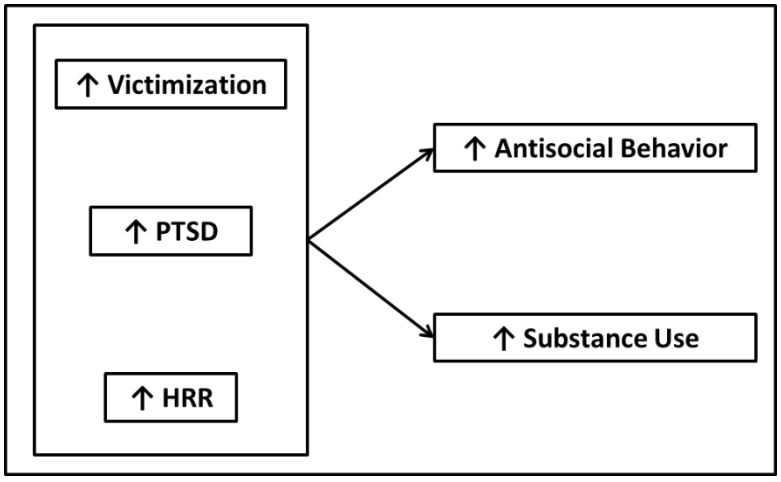

Figure 1. Theoretical figure depicting the predicted effects of the independent variables on the dependent variables.

CHAPTER III

Methods

Sampling Procedure

Data collection for the current dissertation occurred in two stages: in class survey administration followed by a later biological sampling procedure. Both the survey and biological data collection portions of this dissertation were approved by the Institutional Review Board (IRB). The survey administration occurred in criminal just classes consisting of three large classrooms of between 100 to 200 students, and three smaller classrooms of about 30 to 50 students at a southwestern university. Survey data collection occurred between early September and late October during the fall 2016 semester (N = 862). Biological data collection also occurred during that timeframe but due to the time needed with each participant (i.e., approximately 45 minutes), the timeframe was extended through early December (end of fall 2016 semester) to allow for as many students as possible participate (N = 556; 65%).

Upon arrival for the survey data collection, the research team first provided an introduction to the purpose of the project along with a description of the procedures, including information concerning data use, storage, confidentiality, and the voluntary nature of participation in the study. Participants were given time to ask questions concerning voluntary participation, study procedures, and/or data handling and were given contact information of the research team leaders in the event a respondent needed a more in-depth explanation. Contact information of the principal investigator was also given to participants if later questions arose. For those that chose to participate, a written

voluntary consent form was administered and signed.[5] The survey instrument was administered to all voluntary participants and included questions about victimization experiences, past offending behaviors, individual traits, and demographic characteristics. Respondents who opted to take part in the in-class survey were also given instructions and information concerning how to participate in the biological portion of the data collection. More specifically, respondents were shown a map of the building on an overhead projector with specific directions on the location of the biological data collection facilities as well as a brief explanation of what would occur during biological data collection. Each respondent was also given a flier with directions to the location of the biological data collection rooms (see Appendix A) and added to an email list. The respondents were sent an email containing instructions for signing up for an appointment, information regarding extra credit, and instructions concerning participation (see Appendix B). Biological sampling appointments were scheduled for approximately 45 minutes and respondents signed-up directly using doodlepoll.com. Once the respondent arrived to the lab they waited in a waiting room until their scheduled appointment time or the first availability. Walk-ins who were willing to wait were allowed to complete the biological collection if a cancellation occurred or if a respondent did not attend their specified time, thus leaving an opening. While in the waiting area respondents were again briefed on the basics of the biological sampling procedures and told roughly what time they would complete the data collection based on their scheduled participation time.

[5] Professors of the sampled classes offered extra credit to students who participated in both the survey section as well as the biological collection. Further, in order to increase participation for the biological sample we offered free custom koozies that we designed (see Figure 2 below).

After receiving confirmation the respondent was ready to participate, and the biological data collection began.

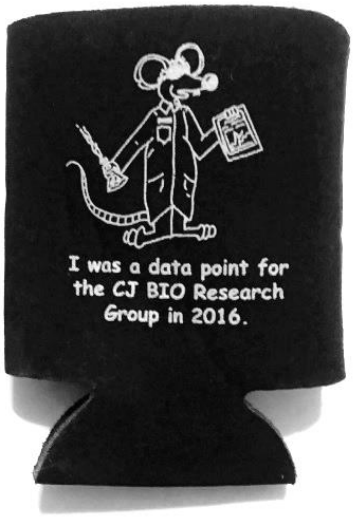

Figure 2. Free custom koozie given to participants after the completion of the biological data collection featuring the mascot of the CJBIO lab, Erv the lab rat.

The biological data collection effort occurred five days a week from 8am to 6pm for the first three weeks of sampling and was then reduced to 10am to 2pm or upon specific request for the duration of the data collection timeframe. This portion of the project occurred in a controlled environment where only the researcher and respondent were present with no loud noises, distractions, or other unplanned stimuli in a room approximately 3.5m x 4.5m. The lab area consisted of two private offices outfitted with collection and storage equipment where physical and biological data were collected from each participant in private. Prior to collection a lab protocol (see Appendix C), one-on-one training, and full lab data collection scenarios were provided and required for each

member of the research team from team leaders who received formal training during prior research collections.[6]

The biological data consisted of measurements of facial symmetry, 2D:4D digit ratio hand scans, comparative 2D:4D hand tracings, and stress reactivity via heart rate, skin conductance, and saliva steroid measurements (cortisol and testosterone), and DNA swabs.[7] The same procedure and protocol was followed for every participant with as little variation as possible regardless of which researcher from the team was present for data collection. The biological data collection protocol is outlined next.

First, participants were asked to sit in a chair behind a divider so they could not see the researcher. At this time the researcher would label a letter sized envelope, the saliva collection tube, and the biological information sheet to be used later with the participants I.D. number so that their name was not attached to any information from this point on. After the labeling was completed the researcher would introduce themselves and again give a briefing on what was about to occur. After the participant agreed to participation the researcher instructed them on how to use the saliva collection tube and asked for at least 2.5 ml of saliva. Upon completion the saliva sample was moved to the pre-stressor sample box where it remained until data collection had ended for the day and it was then moved to a padlocked -6°C deep freezer for storage.[8]

Second, researchers then administered a low-level stress test protocol measuring heart rate (HR) and skin conductance (SC) reactivity using a finger pulse oximeter based

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[6] Research team leaders were either present or on call during the entire collection process if any researchers needed assistance of any kind.

[7] Also respondents filled out a second short survey with information pertaining to general sleep, dietary, and exercise habits.

[8] Samples were transferred to a frozen storage box and not removed until they were being physically analyzed.

on the methodology of Raine and colleagues (2000). The researcher would first attach the HR and SC nodes to the participant's right index and middle finger tips and explained how the equipment worked and that no discomfort or pain would occur due to the measurement equipment. The researcher would then return to the computer behind the partition and inform the participant they needed a moment until the data collection could move forward. During this time, the researcher would watch the live feed of the participant's HR and SC until both stabilized at which point a 30 second baseline measurement was taken. After the baseline measurement was complete, the participant was told the baseline measurement had just been taken. Participants' baseline HR measurements were taken without their knowledge based on trial lab runs that showed if participants knew the measurement was occurring it would cause baseline HR to fluctuate. After the pre-stress HR measurement was taken a mild stressor was induced. To do so, participants were asked to take two minutes to mentally prepare a two minute speech about their faults and weaknesses that would be videotaped.[9] Each researcher read the same prompt to the participant to ensure uniformity of the stressor. After the prompt was read, the participant was allotted two minutes of preparation. After 90 seconds passed, researchers would give a 30 second warning and take a 30 second HR and SC measurement. At the end of the 30 second measurement (and the two minutes allotted for preparation) the researcher would set the digital camera to record and give the participant a silent hand gesture of one thumb up with a closed fist indicating they could begin. Respondents then delivered the speech to the researcher while being videotaped. During the recorded speech, the researcher would first start a stop watch to keep track of the

[9] This video was also collected for future transcription for qualitative analysis.

amount of time since the stressor began and also take a two minute HR and SC measurement. After completing the mild stressor, the researcher would hold up their open hand to the participant to indicate the speech was completed, and at this point the digital camera recording was stopped. The researcher would then take a final post-stressor reading and remove the finger pulse oximeter from the participant (see Figure 3).

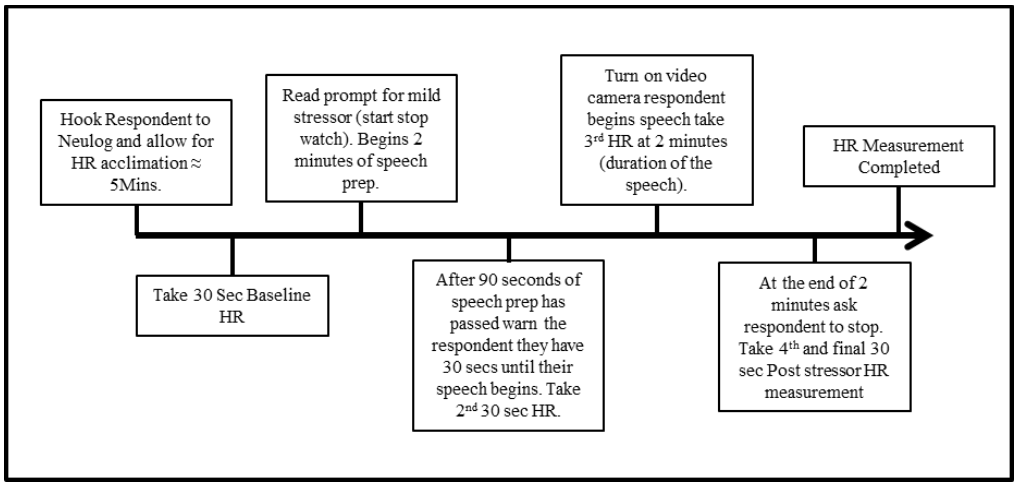

Figure 3. Heart rate reactivity (HRR) measurement data collection steps.

At this point, the researcher would take the letter sized envelope with the participant's I.D. and ask the participant to hold their envelope up near their chest high enough that it was in the camera's field of view but not obstructing their face. The researcher then used the digital camera to take a picture for symmetry measurements ensuring a ruler for reference and the participant's I.D. were visible. During the symmetry picture respondents were asked to remove glasses, headwear, and put their hair behind their ears if possible. The first portion of the biological data collection was now completed and the respondents were then escorted by a researcher to the next lab room.[10]

[10] More than one participant could be present in the second lab as there were no video or heart rate/skin conductance measurements being taken.

In the second lab room, participants' 2D:4D ratios were measured via a hand scan using an office scanner with a ruler on the scanner to use as reference for measurement using the software ImageJ™. Participants were asked to remove any jewelry from their hands and to place their hand palm side down onto the scanner bed. The image was verified that both the participant's hand and the ruler could be seen clearly by the researcher before the image was saved and stored. Also, if the participant had long finger nails they were asked to press the pads of their fingers to the screen, if possible. Both the right and left hands were scanned of each participant and saved to a secure external device with the participants I.D. number. In addition to the actual hand scans, and before any jewelry was put back on, participants were also asked to trace both their right and left hands using a standard number two pencil and 8 x 11 inch printing paper in order to compare the different methodologies scanning versus tracing to measure 2D:4D ratios. Each tracing was labeled matching the participant's I.D. and denoted if the tracing was of the left or right hand.

After the hand tracings were stored the participant was asked to fill out a 2-page information sheet regarding overall past dietary behaviors, sleep, and level of physical activity to act as a potential control for testosterone and cortisol if abnormal results occurred. The primary purpose of the handout was to give insight into factors that could alter testosterone and cortisol levels such as dietary behaviors and sleep patterns. However, other biological control questions were also asked that could correspond to behaviors measured on the survey such as what age the individual underwent puberty. While the participant was filling out the behavior and biological control handout the researcher would check the stop watch time to ensure at least 20 minutes had passed

since the stressor test began. As mentioned above, the researcher who started the application of the mild stressor began the stop watch simultaneously to track the time since the stressor had happened. If more time was needed, participants were offered reading materials or coloring supplies, but were asked not to look at their phones in order to reduce the chances of contact with stressful information (e.g., angry romantic partner or roommate). After at least 20 minutes had passed the post salivary sample was collected, labeled, and stored in the post-sample box. Although clinical studies with small sample sizes generally aim for half an hour or more from the time of the stressor as preferable for salivary cortisol concentrations (Kirschbaum, Pirke, & Hellhammer, 1993) 20 minutes was the most time that could be allotted given the amount of participants and the variety of biological factors collected for the current data collection effort. Further, 20 minutes has been shown to be enough time for a physiological reaction resulting in increased salivary cortisol (Violanti et al., 2006).

At this point the respondent was asked to open the envelope and a buccal swab package and the researcher would then take two buccal swabs of each inner cheek, upper lip, and lower lip. The buccal swabs were returned to the original packaging (a sterile wax paper sleeve) and placed in the open envelope with the participant's I.D. number on it and placed in the padlocked -6°C deep freezer. At the end of the data collection day all storage devices were locked in cabinets and all biological samples were stored in the -6°C deep freezer that was locked via a padlock where the samples remained until analysis.

Lab protocol stated that if any participant displayed adverse or uncomfortable responses to data collection, they were immediately to be asked if they wished to stop taking part in the data collection and/or needed to be referred to the on-campus

counseling center. However, to our knowledge, no participant either during the survey data collection or the biological data collection felt undue stress, asked to stop and be removed from the collection, and/or required the services of the on-campus counseling center. Some respondents asked questions about certain parts of the biological data collection procedures or could not complete the collection task. For example, some were concerned about the hand scans (e.g., "why do you need my hand scan… is this for fingerprints?") or were physically unable to complete the given task (e.g., not being able to produce enough saliva to reach the 2.5 milliliter amount) and for these situations extra help or further explanation was offered to ensure the respondent understood what was being requested and why.

To ensure that data was collected properly and with the most reduced level of risk to the respondents, researchers followed an approved protocol based on previous published research. Specifically, researchers followed the approved protocol by the IRB in which after the completion of all of the biological data, the names of participants were redacted leaving only a unique identifying number connecting the survey and biological data. Thus, any data of the respondents cannot be linked to the name of the respondent but only a numerical identifier. Further, all of the data, both survey and biological is kept under lock and key in a secure office. Specifically, all of the survey data are kept in locked filing cabinets within offices that remained locked when not in use. Further, all electronic data is kept on encrypted storage devices that are also kept under lock and key when not in use. Finally, any physical biological data is stored in a -6°C deep freezer locked via a padlock. Also, all students were offered to contact the researchers if they had

any additional concerns or questions after completing the biological lab portion of the data collection.

Study Population

The current dissertation sampled from a population of undergraduate students at a southwestern university comprised of approximately 20,000 students. The total student population has roughly a 3:2 female to male ratio. Further, the racial/ethnic makeup of the student population is primarily Caucasian (50%), Hispanics (20%), African Americans (17.5%), and other (12.5%). The sampling approach was a convenience sample within the criminal justice department. The total sample taken from the study population was 862 participants with 556 (65%) of those participants having also completed the biological component of the study (see Table 1).

Table 1

Grouping differences between survey only and survey and biological component participation.

Categorical Variable	Survey Only	Survey & Biological Component
Gender		
Male	141	179
Female	162	361
Race/Ethnicity		
African American	59	67
Asian	5	6
Caucasian	124	199
Hispanic	95	203
Hawaiian or Pacific Islander	0	1
American Indian	4	3
Other	10	47

The study sample for the current dissertation was based on voluntary participation from students enrolled in criminal justice classes at a southwestern university. The study sample population exhibited similar demographics to the total population of the university. Specifically, the sample population was made up of about 60% females and 37% males (with 3% who did not identify). The overall ratio of self-reported genders was similar to the overall campus population with a slightly increased number of female respondents. It is important to note that the choices for gender allowed for transgender options. However, only one participant from each category of transgender male and transgender female self-identified as transgender and were subsequently removed from the analyses. The racial make-up of the sample for the current dissertation, while somewhat different than the total population still exhibited similar trends in proportions of racial groups of the total population. The sample population collected had Caucasians as the most represented group (37.5%), Hispanics as the second most represented (34.6%), African Americans are the third most represented (14.6%), and the combination of the remaining under-represented racial groups as Other (8.8%). The primary differences between the total population and the sample population is that Hispanics are represented at a higher frequency almost equal with Caucasians in the sample population and African Americans are slightly under represented in the sample population as compared to the total population.

Outside of the differences between the total population and the sample population there is also two groups within the sample, those who completed the survey only and those who completed both the survey and the biological component. The current dissertation requires biological data on participants, thus only individuals who completed

both aspects of data collection can be used for analyses. To ensure that the subsample of individuals that participated in the biological component of the study versus individuals that only took the survey were not significantly different from one another on key variables, I conducted independent t-tests on the continuous variables and chi-square significance tests on the categorical variables. There were no significant differences between the two groups of respondents for the continuous variables of general antisocial behavior (t = 1.05; p = .30), substance use (t = 1.93; p = .60) victimization (t = -.89; p = .37), post-traumatic stress disorder symptoms (t = -.65; p = .52), age (t = .09; p = .93), delinquent peer behavior (t = 1.89; = .06), and low self-control (t = 1.00; = .38). However, there were significant differences between the two groups for two of the categorical control variables of gender (chi-square = 14.77; *p-value* < .001) and race/ethnicity (chi-square = 2.16; *p-value* = .003) but not for socioeconomic status (chi-square = 4.35; *p-value* = .63).

As mentioned above, there was a significant difference based on gender of which participants completed both the survey and biological portion versus only the survey (*chi-square* = 14.77; *p-value* < .001). Specifically, about 58% of the males in our sample finished both the survey and biological component and about 72% of females completed both the survey and biological portion. As a research group, every opportunity was afforded to respondents and announcements were made repeatedly throughout the semester encouraging participation.[11]

In terms of race/ethnicity, there was a significant difference between individuals who completed both the survey and biological collection portion (65%) and the survey

[11] Although a quota sampling method could have been implemented the goal of the current data collection was to include as many participants as possible and to reach the largest sample size possible.

alone (35%) (chi-square = 20.16; *p-value* = .003) with the race/ethnicity make up of Caucasian (37.2%), Hispanic (38.6%), African American (13.1%), and Other (11.1%) who completed both the biological component and the survey.

Analytical Sample

The analytical sample consisted of 486 participants who had completed both the survey as well as the biological laboratory component of the project. More specifically, individuals could only be included in the analytical sample if respondents had provided responses or measurements for all variables included in the current dissertation. Thus, to ensure that only individuals were included who had also provided the necessary information, I first checked if the respondents had provided the biological variable of interest, HRR. Of the individuals who completed both the survey and biological component and provided the information of HRR only ten respondents were lost from the sample due to lack of demographic information (N = 546). After sorting the subsample by the two main non-biological independent variables of victimization and post-traumatic stress disorder (PTSD) the population was further reduced (victimization: N = 528; PTSD: N = 527). Finally, after sorting and removing individuals from the subsample that did not provide the control variables included in the current dissertation 41 individuals were removed (self-control and delinquent peer behavior: N = 525; race/ethnicity: N = 505; gender: N = 503; socioeconomic status: N = 489; and age: N = 486). Hence, after sorting all of the subsample based on variables included in the current dissertation and removing cases that did not provide the necessary information on all variables of interest the final analytical sample was reached (N = 486). Statistical comparisons between all of the variables for the analytical sample and the total sample and subsample (the portion of

participants that completed the biological portion of the current dissertation) were also conducted. There were no significant differences between any of the variables between the total and analytical sample except for the age of the participants ($p < .001$) where the age of the total sample was slightly younger than the analytical sample. Similar, there were no significant difference between all the variables for the analytical sample and sub sample except for age ($p = .004$) and gender (chi-square = .04) with the analytical sample being slightly older with more females.

Dependent Variables

General Antisocial Behavior

The first dependent variable is a measure of general antisocial behavior, which was taken from the National Youth Survey (NYS, 1987) (26 items; $\alpha = .87$). The questions measure antisocial behavior and are part of a larger original scale that tapped into the construct of antisocial behavior directed from one individual to other individuals or other individuals' property. The original NYS delinquency scale (1987) included 38 items; however, only 26 items were included in the current dissertation (see Appendix D). The decision to exclude 12 of the original items was to ensure the most conservative age-appropriate measure of general antisocial behavior. Items were only included for the current dissertation if two criteria were met: a) the antisocial behavior was a misdemeanor or higher and b) the behavior was likely to be perceived as antisocial by a college age respondent. Some of the items may not have been applicable as antisocial behavior given the age range most participants within the sample currently fall within such as "using or trying to use a credit card(s) without the owner's permission?". This is because given the likelihood of participants' involvement with shared online

memberships with their parents or guardians (e.g., Amazon or Netflix) with access to purchasing options using saved credit card information may have led to inflated responses of antisocial behavior. Items were also excluded if the behavior description could be seen as more benign in nature or be based more on morality rather than purely antisocial such as "failing to return extra change that a cashier gave you by mistake?" where philosophical arguments about victimless crimes could be invoked again possibly inflating perceived antisocial behavior in the population. Beyond perception and interpretation there were other items that had the potential to inflate antisocial behavior measures within the analytical sample, hence were removed. For example, items removed included "avoiding paying for such things as movies, bus, or subway rides, and food?", considering most college age students may be trying to conserve money and opt to attend prosocial gatherings for free movie admission, travel, and food, and "using checks illegally or using phony money to pay for something?" considering perceived lack of check and cash use amongst college age students. Thus, only items measuring behaviors where the respondent was likely to perceive direct harm to an individual or stole or damaged their property was included in the current dissertation. To ensure that the selected 26 items were a valid measure of antisocial behavior a factor analysis was run and the measure of antisocial behavior was verified as reliable with a Kaiser-Meyer-Olkin measure of sampling adequacy of .91 (a minimum of .60 is suggested) and the Bartlett's test of sphericity was significant (chi-square = 14,464.56; $p < .001$). All survey items were prefaced with the question: "How often in the past year were you involved in…?" with response categories ranging from 0 = "Never" to 8 = "2-3 times a day" (see Table 2).

For the current dissertation, increased sums of response values indicate increased levels of antisocial behavior.[12],[13] Further, all responses were summed for an individual and then divided by the total number of items (26). Thus, by averaging the responses across all items in the scale it created an average of antisocial behavior for each individual that could be statistically predicted by the model where the continuous independent variables were treated the same. Moreover, as the average calculated value of antisocial behavior increases for participants so does the amount of antisocial behavior the reported.

Substance Use

The second dependent variable of interest is substance use, which was also derived from the National Youth Survey (NYS, 1987). The scale consists of thirteen items that ask about the frequency of use of a variety of substances in the past year from when the survey was taken. Each of the thirteen items is concerned with a different type/group of substances ranging from nicotine (i.e., "used tobacco?"), alcohol (i.e., "used alcoholic beverages, beer, wine, hard liquor?"), to illegal drugs (e.g., "used heroin? (Horse, H, Skag, Smack, Junk)") ($\alpha = .60$).

Much like general antisocial behavior, increased sums of response values indicate increased levels of substance use. Responses for substance use ranged from 0 = "Never" to 8 = "2 to 3 times a day" (see Appendix E). To better understand the effects of different kinds of substances being used the items were divided into soft substances (alcohol, nicotine, and marijuana) and hard substances (Uppers, Downers, and codeine) similar to

[12] Initially an attempt was made to convert antisocial behavior into Z-scores for ease of explanation of results but due to a need to further alter the data this approach was abandoned.
[13] An attempt was made using a Blom correction to remedy the zero heavy data but again the data would have been further altered, thus the decision was made to not use this correction.

the methods used by Santor, Messervey, and Kusumakar (2000). A factor analysis was conducted and the eigenvalues were for the first component (43.22%) and second component (21.00%) with all of the soft substances loading together and the hard substances loading together. Again, as the calculated continuous sum value increases so did the amount of reported soft and hard substance use. Further, similar to how antisocial behavior was calculated, the sums were then divided by the number of items for soft substance use (n = 3) and hard substance use (n = 3) to result in a continuous score for each type of substance use.

Table 2

Descriptive statistics for controls, independent variables, and dependent variables. Descriptives were calculated for continuous variables by summing responses of participants.

		N	Mean	S.D.	Min	Max
Antisocial Behavior		-	2.27	.18	0	38
Substance Use Total			4.78	.24	0	72
Soft Substance Use						
	Alcohol		2.76	2.12	0	8
	Nicotine		.87	1.96	0	8
	Marijuana		.84	1.65	0	8
Hard Substance Use						
	Uppers		.08	.53	0	8
	Downers		.10	.76	0	8
	Codeine		.09	.51	0	8
Victimization		-	.50	.06	0	10
PTSD		-	15.54	.92	0	128
Low Self-Control		-	35.38	.58	0	72
Delinquent Peers		-	9.50	.29	0	35
HRR		-	1.87	8.35	0	32.77
Age		486	20.29	2.90	17	47
Gender						
	Males	163	-	-	-	-
	Females	323	-	-	-	-
Race						
Caucasian (Non-Hispanic)		186	-	-	-	-

African American	57	-	-	-	-
Hispanic	189	-	-	-	-
Other	54	-	-	-	-
Less than $20,000	45	-	-	-	-
$20,000-$29,000	50	-	-	-	-
$30,000-$39,000	48	-	-	-	-
$40,000-$49,000	68	-	-	-	-
$50,000-$69,000	89	-	-	-	-
$70,000-$99,000	101	-	-	-	-
Over $100,000	85	-	-	-	-

Independent Variables

Victimization

Victimization was measured using the victimization experience questions from the National Youth Survey (NYS, 1987) which includes eleven items concerning events that occurred in the past year prior to the survey. For the current dissertation, however, only questions that involved victimization events that occurred when the victim was present for either property or personal crime were included (9 items; $\alpha = .51$; see Appendix B). This resulted in the following two questions being omitted: "Have things been taken from your car, motorcycle or bike such as hubcaps, books or packages, or clothing ripped up?" and "Have some of your things, such as your jacket, notebooks, or sports equipment been stolen from a public place such as a cafeteria, restaurant or bowling alley?".

The nine victimization questions included in the victimization measure were recorded as a total count of how many times the event had occurred in the past year. An example question is: *"Have you been beaten up or threatened with being beaten up by someone other than your mother or father?"* in which a respondent would record how many times this event had happened in the past year. Originally the respondents were

allowed to supply an open ended numerical answer of how many times they had experienced the victimization event described in each of the nine questions. However, the majority of responses were between 0 to 10 times with very few instances recorded as 10 or higher (1.0%; see Table 3). As such, responses of 10 or higher were recoded as "10" to indicate 10+ (see Table 3). As shown in Table 3, the majority of responses were 0 (79.2%) with the next largest group reported as between 1 to 3 victimization events for the respondents within the past year as 1 victimization event (9.1%) being the most common and, 2 (4.3%) to 3 (4.3%) events making up the next largest portion of responses. Only 1.0% percent reported 10 or more victimization experiences within the past year upon taking the survey. The total sum of values provided for each item was summed to illustrate overall victimization experienced by the participant in the past year from the date of the survey.

Table 3

Total response counts for victimization events excluding reports of zero (n = 385) (9 Items).

Victimization Events	Response Frequency	Response Percentage
0	385	79.2%
1	44	9.1%
2	21	4.3%
3	21	4.3%
4	6	1.2%
5	4	.8%
6	0	0%
7	0	0%
8	0	0%
9	0	0%
10(+)	5	1.0%
Total	101	*100%*

However, there is a theoretical concern treating property and personal victimization the same concerning the effects that the consequences of victimization can have on behavior. Hence, a factor analysis was conducted to see if the 9 victimization questions loaded as property and personal victimization.[14] Although the eigenvalues were relatively low for the first (24.08%) and secondary components (16.10%) the victimization measures did load on two distinct components, property victimization and personal victimization (see Appendix F). Further, considering that the victimization measures loaded as theoretically predicted and it is sensical to treat property and personal victimization differently concerning behavior, each type of victimization was ran independent of the other in each model. Similar to the above independent variables, the answers were summed and divided by the number of items for each type of victimization: property (n = 4) and personal (n = 5) to create the continuous variable where higher values are indicative of more victimization experienced.

PTSD

To measure post-traumatic stress disorder (PTSD), the PTSD checklist civilian version (PCL-C) was used to tap into the general severity of PTSD symptoms present (see Appendix G).[15] The PCL-C is a self-report checklist for civilians and is used to gain insight into symptoms that align with PTSD (Gradus, 2007; Weathers, Litz, Keane, Palmieri, Marx, & Schnurr, 2013). The PCL-C can be modified by number of items, number of possible responses (e.g., 1-3, 1-5, or 0-8), and the directions for the prompt can be changed to fit specific circumstances or time frames (e.g., “in the past month” or

[14] A maximum likelihood approach was attempted but was not able to be used without altering the data, hence a principal component analysis was used.
[15] This is not to be confused with the PCL-5 used in clinical monitoring of PTSD cases.

"in the past year"; Gradus, 2007). Regardless of modification, as the sum of the responses increases so does the severity of PTSD related symptoms present (Gradus, 2007). Hence, a major strength is the versatility of the PCL-C, but interpretation of results should be broad and generalized as per the design of the instrument. Further, the PCL-C is better suited to gain a general understanding of the level but not type of PTSD symptoms present (i.e., low, moderate, or high), and because of the straightforward nature of the PCL-C it can be easily administered to large sample populations with relatively small amounts of guidance or direction (Gradus, 2007). Moreover, the PCL-C is generally not used to tease apart the subcomponents of PTSD (i.e., intrusive thoughts, avoidant behaviors, and hyperarousal) due to the brevity and broad conceptuality of the questions associated with PTSD and is not used for medical diagnosis (Gradus, 2007).[16]

The PCL-C instrument for the current dissertation is a sixteen item scale that consists of questions asking about behaviors in the last twelve months ($\alpha = .95$). An example of questions from the PCL-C that are representative of some of the characteristics inherent of PTSD are as follows: *"Have you been having dreams or nightmares about the trauma?"*; *"Have you felt distant or cut off from others around you?"*; and *"Have you been jumpier, more easily startled (for example, when someone walks up behind you)?"* in which respondents were given the choice between nine response categories ranging from 0 = "Never" to 8 = "2-3 times a day". Although a large portion of participants answered "Never" (62.47%) on many of the 16 questions within

[16] The PCL-C was designed with self-administration in mind for the need of seeking medical help when scoring in the severe category for individuals who fear they are already suffering from PTSD (Gradus, 2007).

the PCL-C there was still a presence of increased levels of PTSD characteristics within the sample as shown on Table 2.

For the current dissertation, considering that increased sums of response values indicate increased presence of PTSD symptoms, all responses were summed for an individual and then divided by the total number of items (16). This allowed for an average PTSD value to be calculated, where higher values indicated greater levels of self-reported PTSD for the participant.

Heart Rate Reactivity

Autonomic nervous system function was measured via heart rate reactivity (HRR). To measure HRR respondents were asked to come to a controlled lab area where they sat on the opposite side of a partition from the researcher and were given a mild stressor. Respondents were then hooked up to the Neulog physiological measurement tools and pre, during, and post heart rates were measured.

For the current dissertation, the heart rate measures used were the pre-stressor HR and the post-stressor HR. These measures were used to calculate non-directional HRR. The current dissertation is focused on non-directional changes in HRR, because the importance is the magnitude in HRR not the direction (i.e., lower or higher HR after the stressor). Heart rate reactivity occurs based on any notable change in HR based on a given stimuli and can be measured as a non-directional absolute value (Gottman et al., 1995). To calculate non-directional HRR the difference between the measurements of post HR and pre (or baseline) HR are taken and then the absolute value of the difference is calculated (Post HR – Pre HR = |HRR|). Thus, again, this measure looks at the magnitude of HRR to the stimuli and does not take into account direction. As such, the

minimum amount of reactivity is 0 and the maximum measurement was 32.77 with a mean of 1.87 and standard deviation of 8.35 (see Table 2). This non-directional measurement of HRR is appropriate for the current dissertation as hyperarousal can influence both excitatory HR and acute drops in HR (due to extreme spikes in HR). Ultimately as the absolute value of HRR increases so did some form of HRR response to the stimulus.

Control Variables

Age

Age was recorded as a self-reported continuous variable by respondents. Given the current dissertation population is a university sample of undergraduate students, the mean age of respondents was 20.29 with a standard deviation of 2.90 (see Table 2). Further, the minimum age was 18 and the maximum age was 47 (see Table 2)

Gender

Participants were asked to self-report their gender as either male, female, transgender male, transgender female, or other. Only one individual responded as transgender male and only one responded as transgender female with no respondents identifying as other. As a result, these two respondents were removed from the analytical sample and only participants identified as either male or female were included in the current analyses (male = 0 and female = 1) (see Table 2).

Race/Ethnicity

Race was based on self-report data whereby respondents indicated whether they identified as African America (= 1), Asian (= 2), Caucasian (= 3), Hawaiian or Pacific Islander (= 4), Hispanic (= 5), American Indian (= 6), or other (= 7). Due to the

dispersion of ethnicity reported within the current dissertation sample, race was recoded as Caucasian (Non-Hispanic) = 0, African American (Non-Hispanic) = 1, Hispanic = 2, and Other = 3 (see Table 2).

Socioeconomic Status

To measure socioeconomic status (SES) participants were asked to provide information concerning the estimated annual income of the household in which they grew up. Specifically, participants were given the choice of seven items based on non-overlapping incremental ranges: 0 = "Less than $20,000", 1 = "$20,000 - $29,000", 2 = "$30,000 – $39,000", 3 = "$40,000 - $49,000", 4 = "$50,000 - $69,000", 5 = "$70,000 – $99,999", and 6 = "Over $100,000" (see Table 2).

Low Self-Control

To measure low self-control the scale created by Grasmick and colleagues (1993) was used in order to tap into characteristics of impulsivity, preferences for simple tasks, favoring physical activities, self-centeredness, and temper (see Appendix H; Gottfredson & Hirschi, 1990). Self-control is a well-studied correlate of antisocial behavior, and is an important characteristic to control for within the current dissertation considering that the six components within the construct of self-control are all risk factors of antisocial behavior (Gottfredson & Hirschi, 1990; Pratt & Cullen, 2000; Cauffman, Steinberg, & Piquero, 2005). The scale is comprised of twenty four items and seeks to tap into the amount of self-control individuals exhibit ($\alpha = .83$). Individuals were asked questions such as: *"When I'm really angry, other people better stay away from me"* in which respondents were given the answer choices ranging from 0 = "Strongly Disagree" to 4 = "Strongly Agree" for each of the twenty-four items (see Table 2). To ensure the

unidimensionality of this scale within the current sample a factor analysis was conducted and showed the first component was 26.79% and the second component as 21.28%. Further, this variable to have high inter-item reliability with a Kaiser Meyer Olkin value of .95 (Chi-square = 10,207.53; $p < .001$). All responses were coded to ensure higher values meant increased levels of low self-control.

For the current dissertation, increased sums of response values indicate increased levels of low self-control. Further, all responses were summed for an individual and then divided by the number of total items (24). Further, higher values are indicative of lower levels of self-control reported by the participants.

Delinquent Peer Behavior

To measure delinquent peer behavior participants were asked to answer questions based on their friends' behaviors for the past year. Respondents were given the following five choices for each of the 14 behaviors inquired about their closest friends' delinquent behaviors: 0 = "None of them", 1 = "Very few of them", 2 = "Some of them", 3 = "Most of them", and 4 = "All of them". All of the behaviors listed were antisocial or delinquent behaviors such as "*Hit or threatened someone for no reason*" or "*Suggested you do something against the law*" (see Appendix I; see Table 2).

For the current dissertation, increased sums of response values indicate increased number of peers who exhibit a greater level of delinquent behaviors. Further, like the other independent variables all responses were summed for an individual and then divided by the total number of items (14). The higher the average score on this measure represents a greater level of delinquent peer association.

Analytical Plan

The primary goal of the current dissertation was to examine the relationship that the three risk factors of victimization, PTSD, and HRR have with general antisocial behavior and substance use. It is important to understand how all three risk factors influence antisocial behavior and substance use given the similarity and comorbidity these risk factors share. As discussed in prior chapters, individuals who are victimized generally experience PTSD symptoms and/or abnormal HRR; PTSD is often associated with trauma (i.e., victimization), and onset physiological changes related to HRR; and victimization followed with an extended time of increased HRR can increase the chances of the development of PTSD. Thus, considering it is likely for more than one of these risk factors to be present in a given individual, it is important to understand how each factor influences the other as well as general antisocial behavior and substance use. Moreover, females have been shown to experience higher levels and stronger effects of all three risk factors more so than males, but males have been shown to exhibit higher levels of overall antisocial behavior than females. Hence, it is also important to understand how gender influences the relationship between the risk factors of victimization, PTSD, and HRR concerning general antisocial behavior and substance use.

The analytical plan for the current dissertation is to first establish the relationships of each of the three risk factors with general antisocial behavior and substance use, and associated control variables through bivariate comparisons. Second, using t-tests and chi-square tests the three risk factors of victimization, PTSD, and HRR were compared based on gender to see if differences are present within the sample (i.e., females exhibiting higher averages for all three risk factors). After associations and gender differences are

established, predictive models made up of control variables and the variables of interest (Victimization, PTSD, and HRR) will be used to better understand how these risk factors influence general antisocial behavior and substance use.

Due to the nature of the data and selected variables, there was a high amount of zeros reported by participants on many of the survey items (e.g., victimization and PTSD symptoms). Considering the current dissertation is using data that has continuous dependent variables with a large amount of reported zeros, a Tobit regression was used in order to compensate for the heavy frequency of zeros while still enabling the use of non-integer values.

The first set of predictive models will focus on the single effects of each of the risk factors on general antisocial behavior and substance use, in that all three risk factors and control variables will be present in the models with no interactions. The second set of predictive models will include the three risk factors, control variables, and the two-way interaction between victimization and PTSD to determine the effects they have on general antisocial behavior and substance use. The third set of predictive models will contain the three risk factors, control variables, and the two-way interaction between victimization and HRR. The fourth set of predictive models will include the three risk factors, control variables, and the two-way interaction between PTSD and HRR. The last set of models will contain the three risk factors, control variables, and a three-way interaction between victimization, PTSD, and HRR. Given the amount of overlap exhibited by the three risk factors of victimization, PTSD, and HRR this final model (allowing all three risk factors to interact) is predicted to best explain the relationship between the risk factors and general antisocial behavior and substance use. Finally, all of the predictive models of the

single effects models, two-way interaction models, and the three-way interaction models will be ran for males and females separately to better understand the impact these risk factors and interactions have concerning general antisocial behavior and substance use.

CHAPTER IV

Results

The primary goal of the current dissertation was to examine whether the interaction of victimization, PTSD, and HRR influences antisocial behavior, soft substance use, and hard substance use.[17] Moreover, the secondary goal was to examine whether these relationships differ based on gender. First, independent t-tests were conducted concerning the outcome variables of antisocial behavior, soft substance use, and hard substance use as well as the independent variables (e.g., property victimization, personal victimization, PTSD, and HRR) to see if the means significantly differed across gender (see Table 4). As shown in Table 4, males exhibited (marginally) significantly higher levels of antisocial behavior ($t = .17$; $p = .09$)[18] and soft substance use ($t = 2.10$; $p = .03$) while females reported significantly higher levels of personal victimization ($t = -2.53$; $p = .01$) and PTSD ($t = -2.92$; $p = .004$; see Table 4). There were no significant differences across gender for measures of hard substance use, property victimization, or HRR. Considering that no significant gender difference was found for the dependent variable of hard substance use, this response variable will not be included in the gender split Tobit models.

[17] Due to the theoretical overlap between the independent variables a test for multicollinearity was conducted and multicollinearity was not found to be an issue.

[18] A 90% confidence interval was used in the current dissertation given the small sample size to capture marginally significance. Throughout the text any commentary regarding statistical significance denotes a 95% CI and marginally significant denotes 90% CI.

Table 4

Independent sample t-test of dependent variables and variables of interest based on differences among gender for the full sample (n = 486).

	Gender							
	Male			**Female**				
	n	Mean	SD	n	Mean	SD	*t*	*p*
Dependent Variables								
Antisocial Behavior	163	2.76	5.12	323	2.02	3.27	1.67	.09†
Soft Substance Use	160	5.07	4.83	323	4.15	3.91	2.09	.03*
Hard Substance Use	162	.24	.93	323	.29	1.75	-.32	.74
Independent Variables								
Property Victimization	155	.58	1.91	307	.33	1.06	1.52	.12
Personal Victimization	161	.13	.41	306	.34	1.32	-2.53	.01**
PTSD	163	12.14	15.84	323	17.26	22.15	-2.92	.004**
HRR	163	5.60	5.16	323	6.49	6.02	-1.61	.10

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

Bivariate Relationships

Bivariate correlations were conducted to establish relationships between the outcome variables and the independent variables for the full sample and split by gender. As seen in Table 5, antisocial behavior was significantly correlated to property victimization (r = .20) and PTSD (r = .18), but was not significantly correlated with personal victimization or HRR. Moreover, soft substance use (r = .16) and hard substance use (r = .03) were significantly related with PTSD, but not with property victimization, personal victimization, or HRR (see Table 5). Finally, property victimization was approaching significance with hard substance use within the full sample (r = .20; see Table 5).

Second, for the gender split sample[19], bivariate analyses revealed for males, antisocial behavior was significantly correlated with property victimization (r = .26), personal victimization (r = .25), and PTSD (r = .23) and soft substance use was significantly correlated with personal victimization (r = .22) (see Table 5). For females, antisocial behavior was marginally correlated (r = .10), significantly correlated with PTSD (r = .19), and soft substance use was significantly correlated to PTSD (r = .20) (see Table 5).

Bivariate correlations were also conducted within the independent variables to see if theoretical relationships from prior research are present within the current dissertation (e.g. increased victimization associated with increased levels of PTSD). For the full

[19] Again, it is important to note that although males and females exhibited significant differences between antisocial behavior and soft substance use concerning victimization, PTSD, and HRR there were no significant differences for any of the independent variables for hard substance use based on gender. Hence, hard substance use was only run for the full sample models where statistical differences existed for the independent variables and was not included within the models split by gender.

sample property and personal victimization were significantly correlated (r = .14), PTSD was significantly correlated to both property (r = .19) and personal victimization (r = .23) while HRR was not significantly correlated to any of the other independent variables (see Table 6). Concerning samples split by genders and similar to the full sample, property victimization was significantly correlated to both personal victimization (r = .17) and PTSD (r = .22), with no other significant correlations between independent variables for males while property victimization was significantly correlated to personal victimization (r = .27) and PTSD was significantly correlated to both property (r = .22) and personal victimization (r = .38) for females (see Table 6).

Table 5

Correlations between dependent variables and independent variables for the full sample (n = 486), males (n = 163), and females (n = 323).

	Antisocial Behavior			**Soft Substance Use**			**Hard Substance Use**		
	Full	*Males*	*Females*	*Full*	*Males*	*Females*	*Full*	*Males*	*Females*
Property Victimization	.20***	.26***	.10†	.08	.08	.07	.02†	-	-
Personal Victimization	.07	.25***	.06	.06	.22*	.06	.04	-	-
PTSD	.18***	.23**	.19***	.16***	.13	.20**	.03***	-	-
HRR	.01	.06	-.01	-.05	.02	.07	-.02	-	-

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

Table 6

Correlations between independent variables of interest for the full sample (n = 486), males (n = 163), and females (n = 323).

	Property Victimization			**Personal Victimization**			**PTSD**			**HRR**		
	Full	*Male*	*Female*	*Full*	*Male*	*Female*	*Full*	*Male*	*Female*	*Full*	*Male*	*Female*
Property Victimization	1.00	1.00	1.00	-	-	-	-	-	-	-	-	-
Personal Victimization	.14**	.27***	.17**	1.00	1.00	1.00	-	-	-	-	-	-
PTSD	.23***	.22**	.22***	.19***	.38***	.21**	1.00	1.00	1.00	-	-	-
HRR	.003	.01	.01	.06	.06	.06	.04	.05	.03	1.00	1.00	1.00

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

Main Effects Tobit Regressions

Tobit regressions were first used to examine the main effects of victimization (both property and personal), PTSD and HRR on levels of antisocial behavior, soft substance use, and hard substance use. Analyses were conducted for the full sample and then split by males and females.[20]

As shown in Table 7, for the full sample, none of the independent variables had a significant main effect on antisocial behavior. However, the control variables of low self-control ($\beta = 9.28$; $p \leq .001$), delinquent peers ($\beta = 48.94$; $p \leq .001$), and SES ($\beta = .08$; $p = .004$) were significantly and positively associated with antisocial behavior for the full sample (see Table 7). Further, similar to antisocial behavior, none of the independent variables had a significant effect on soft substance use, but the control variables of self-control ($\beta = 5.09$; $p \leq .001$), delinquent peers ($\beta = 33.36$; $p \leq .001$), and age ($\beta = .06$; $p \leq .001$), were all significantly and positively related to soft substance use with SES approaching significance ($\beta = .04$; $p = .06$) within the full sample (see Table 7). Finally, PTSD ($\beta = .28$; $p = .003$) had a significant and positive effect on increased hard substance use and the control variables of delinquent peers ($\beta = 47.39$; $p \leq .001$) and age ($\beta = .13$; $p \leq .001$) were also significant and positively associated with hard substance use for the full sample (see Table 7).

[20] Zero being an actual value recorded by the respondents not indicative of missing data.

Table 7

Tobit regression examining the main effects impacts on antisocial behavior, soft substance use, and hard substance use for the full sample (n = 486).

	Antisocial Behavior		**Soft Substance Use**		**Hard Substance Use**	
	B	SE	B	SE	B	SE
Independent Variables						
Property Victimization	.01	.04	-.04	.03	.04	.07
Personal Victimization	.04	.05	.03	.03	.02	.11
PTSD	.05	.04	.01	.03	.28**	.09
HRR	.01	.01	-.001	.01	.002	.02
Control Variables						
Low self-control	9.28***	2.00	5.09***	1.40	-2.83	4.64
Delinquent Peers	48.95***	4.14	33.36***	2.88	47.39***	10.56
Age	.02	.02	.06***	.01	.13***	.04
Gender	-.09	.11	-.02	.08	-.40	.29
SES	.08**	.03	.04†	.02	.10	.08
Race						
African American	.15	.17	-.02	.12	.83	.42
Hispanic	-.09	.12	-.11	.09	.16	.33
Other	.23	.17	-.10	.13	.02	.48

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

For the models split by gender, as seen in Table 8, PTSD (β = .20; p = .04) was the only significant main effect associated with increased antisocial behavior for males and exhibited a positive relationship; however, there were no significant main effects on antisocial behavior for females. In addition, the control variables of self-control (Males: β = 3.92; $p \leq .001$; Females: β = 5.34; p = .002) and delinquent peers (Males: β = 15.32; $p \leq .001$; Females β = 33.70; $p \leq .001$) also exhibited a significant and positive relationship to increased antisocial behavior for both males and females, while only females exhibited a significant relationship between increased SES (β = .10; p = .003) and increased antisocial behavior (see Table 8).

There were no significant main effects for victimization (property or personal), PTSD, or HRR on soft substance use within either the male or female models (see Table 8). However, the control variables of delinquent peers (Males: β = 10.69; $p \leq .001$; Females: β = 21.50; $p \leq .001$) and age (Males: β = .11; $p \leq .001$; Females: β = .04; p = .002) were both significant and positively related to increased soft substance use for both males and females (see Table 8). Further, low self-control (β = 3.77; $p \leq .001$) was also significant and positively related to increased soft substance use, but only for females (see Table 8). Finally, for soft substance use low self-control (β = 1.46; p = .07) was marginally significant for males and SES (β = .04; p = .10) was marginally significant for females (see Table 8)

Table 8

Tobit regression examining the main variable effects on antisocial behavior, soft substance use, and hard substance use for males (n = 163) and females (n = 323).

	Antisocial Behavior				**Soft Substance Use**			
	Males		**Females**		**Males**		**Females**	
	B	SE	B	SE	B	SE	B	SE
Independent Variables								
Property Victimization	.00	(.05)	-.03	(.06)	-.04	(.04)	-.03	(.04)
Personal Victimization	.33	(.23)	.03	(.05)	.07	(.19)	.03	(.03)
PTSD	.20*	(.10)	-.01	(.05)	.07	(.08)	.00	(.03)
HRR	.001	(.18)	.01	(.01)	.01	(.01)	-.01	(.01)
Control Variables								
Low self-control	3.92***	(1.06)	5.34**	(1.70)	1.46†	(.81)	3.77***	(1.14)
Delinquent Peers	15.32***	(2.43)	33.70***	(3.41)	10.69***	(1.86)	21.50***	(2.23)
Age	.01	(.04)	.03	(.02)	.11***	(.03)	.04**	(.01)
SES	.05	(.05)	.10**	(.03)	.04	(.04)	.04†	(.02)
Race								
African American	.18	(.41)	.08	(.19)	.01	(.31)	-.02	(.13)
Hispanic	.16	(.22)	-.22	(.15)	-.14	(.18)	-.10	(.10)
Other	.48	(.30)	.12	(.21)	.19	(.24)	-.24	(.14)

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

Two-way Interaction Tobit Regressions

Tobit regressions were also used for the models including the two-way interactions of property victimization x PTSD; property victimization x HRR; personal victimization x PTSD; personal victimization x HRR; and PTSD x HRR to examine the effects of each of these interactions on antisocial behavior, soft substance use, and hard substance use. Again, analyses are first conducted for the full models and split by gender for antisocial behavior and soft substance use.

Property Victimization x PTSD

First, as shown in Table 9, the interaction of property victimization x PTSD (β = -.003; p = .004) was significant but negatively associated with antisocial behavior. In addition, the control variables of self-control (β = 9.83; $p \leq .001$), delinquent peers (β = 4.78; $p \leq .001$), and SES (β = .08; p = .004) were all significant and showed a positive relationship with increased levels of antisocial behavior for the full sample (see Table 9).

Second, concerning the effects of the two-way interaction of property victimization x PTSD on soft substance use, only the control variables had a significant effect on increased antisocial behavior (see Table 9). Specifically, low self-control (β = 5.32; $p \leq .001$), delinquent peers (β = 33.35; $p \leq .001$), and age (β = .05; $p \leq .001$) were significant and positively associated with increased levels of soft substance use with SES approaching significance (β = .04; p = .06) for the full sample (see Table 9). Finally, the third model concerning hard substance use was approaching significance for the interaction of property victimization x PTSD (β = -.003; p = .09) (see Table 9). However, the control variables of PTSD (β = .34; $p \leq .001$), delinquent peers (β = 46.80; $p \leq .001$), and age (β = .13; $p \leq .001$) were significant and positively related to increased hard

substance use with identifying as African American approaching significance (β = .80; *p* = .06) for the full sample (see Table 9).

Table 9

Tobit regression examining the two-way interaction effect of property victimization x PTSD on antisocial behavior, soft substance use, and hard substance use for the full sample (n = 486).

	Antisocial Behavior		**Soft Substance Use**		**Hard Substance Use**	
	B	SE	B	SE	B	SE
Independent Variables						
Property Victimization	.12*	.05	.01	.04	.18†	.10
Personal Victimization	.03	.05	.02	.03	.01	.11
PTSD	.09	.04	.03	.03	.34***	.10
HRR	.01	.01	-.001	.01	.002	.02
Property V. x PTSD	-.003**	.00	-.001	.001†	-.003†	.002
Control Variables						
Low self-control	9.83***	1.99	5.32***	1.41	-1.71	4.61
Delinquent Peers	4.78***	4.09	33.35***	2.86	46.80***	10.42
Age	.02	.02	.05***	.01	.13***	.04
Gender	-.08	.11	-.01	.08	-.37	.28
SES	.08**	.03	.04†	.02	.10	.08
Race						
African American	.14	.17	-.02	.12	.80†	.41
Hispanic	-.08	.12	-.11	.09	.16	.32
Other	.20	.17	-.12	.13	-.02	.47

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05^{*}$, $p \leq .01^{**}$, and $p \leq .001^{***}$

For the first set of two-way interaction models split by gender, property victimization x PTSD exhibited no significant effects with either of the outcome variables of antisocial behavior, or soft substance use with low self-control (β = 1.44; p = .08) approaching significance for males (see Table 10). However, property victimization x PTSD (β = -.005; $p \leq .001$) was significant but negatively associated with increased levels of antisocial behavior for females (see Table 10). Further, the control variables of low self-control (Male: β = 10.80; $p \leq .001$; Female: β = 5.96; $p \leq .001$) and delinquent peers (Male: β = .11; $p \leq .001$; Female: β = 33.66; $p \leq .001$) were significant and positively related to increased antisocial behavior for both males and females, with SES (β = .10; p = .003) only being significant and positively related to antisocial behavior for females (see Table 10). Similarly, property victimization x PTSD (Females: (β = -.002; $p \leq .001$) was not significant for males but was significant and negatively related to soft substance use for females (see Table 10). Further, only the control variables of delinquent peers (Males: β = 10.80; $p \leq .001$; Females: β = 21.61; $p \leq .001$) and age (Males: β = .11; $p \leq .001$; Females: β = .04; p = .003) were significant and positively associated with increased levels of soft substance use for both males and females (see Table 10). Finally, for females only, both low self-control (β = 4.14; $p \leq .001$) as well as identifying as the racial group Other (β = -.29; p = .04) had a significant impact on soft substance use (see Table 10)

Table 10

Tobit regression examining the two-way interaction effect of property victimization x PTSD on antisocial behavior, soft substance use, and hard substance use for males (n = 163) and females (n = 323).

	Antisocial Behavior				**Soft Substance Use**			
	Males		**Females**		**Males**		**Females**	
	B	SE	B	SE	B	SE	B	SE
Independent Variables								
Property Victimization	-.10	(.09)	.26**	(.09)	-.10	(.07)	.13*	(.06)
Personal Victimization	.38	(.23)	.01	(.05)	.10	(.19)	.02	(.03)
PTSD	.14	(.11)	.05	(.05)	.04	(.09)	.03	(.03)
HRR	.004	(.02)	.02	(.01)	.02	(.01)	-.01	(.01)
Property V. x PTSD	.004	(.002)	-.01***	(.001)	.002	(.002)	-.002***	(.001)
Control Variables								
Low self-control	3.84***	(1.04)	5.96***	(1.66)	1.44†	(.81)	4.14***	(1.13)
Delinquent Peers	15.45***	(2.41)	33.66***	(3.32)	10.80***	(1.86)	21.61***	(2.19)
Age	.01	(.04)	.03	(.02)	.11***	(.03)	.04**	(.01)
SES	.05	(.05)	.10**	(.03)	.04	(.04)	.04†	(.02)
Race								
African American	.21	(.41)	.08	(.18)	.02	(.31)	-.01	(.13)
Hispanic	.18	(.22)	-.21	(.14)	-.13	(.18)	-.09	(.10)
Other	.47	(.30)	.03	(.20)	.19	(.24)	-.29*	(.14)

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

Property Victimization x HRR

The next set of two-way interaction Tobit regression models examined the effects of property victimization x HRR on antisocial behavior and substance use, and hard substance use (see Table 11). First, concerning the full sample, property victimization x HRR (β = -.03; p = .005) had a significant but negative association with antisocial behavior. Moreover, the control variables of low self-control (β = 9.43; $p \leq .001$), delinquent peers (β = 47.43; $p \leq .001$), and SES (β = .08; p = .006) had significant and positive effects on antisocial behavior (see Table 11). Second, property victimization x HRR (β = -.02; $p \leq .001$) also had a significant negative effect on soft substance use (see Table 11). Further, the control variables of low self-control (β = 5.25; $p \leq .001$), delinquent peers (β = 33.41; $p \leq .001$), and age (β = .05; $p \leq .001$) also exhibited a significantly positive relationship with increased soft substance use (see Table 11). Finally, within the full sample, property victimization x HRR (β = -.04; p = .04) showed a significant and negative relationship with increased hard substance use (see Table 11). Further, the independent variable PTSD (β = .28; p = .003) exhibited a significant and positive association with hard substance use (see Table 11). In addition to PTSD, the control variables of delinquent peers (β = 47.99; $p \leq .001$) and age (β = .13; $p \leq .001$) also exhibited a significant and positive effect on hard substance use while identifying as African American was marginally significant (β = .79; p = .06) for the full sample (see Table 11).

Table 11

Tobit regression examining the two-way interaction effect of property victimization x HRR on antisocial behavior, soft substance use, and hard substance use for the full sample (n = 486).

	Antisocial Behavior		**Soft Substance Use**		**Hard Substance Use**	
	B	SE	B	SE	B	SE
Independent Variables						
Property Victimization	.18*	.07	.13*	.05	.29*	.14
Personal Victimization	.03	.05	.02	.03	.02	.10
PTSD	.04	.04	.01	.03	.28**	.09
HRR	.02	.01	.01	.01	.02	.02
Property V. x HRR	-.03**	.01	-.02***	.01	-.04*	.02
Control Variables						
Low self-control	9.43***	1.98	5.25***	1.39	-2.65	4.60
Delinquent Peers	47.29***	4.09	33.41***	2.84	47.99***	10.51
Age	.02	.02	.05***	.01	.13***	.04
Gender	-.07	.11	-.01	.08	-.33	.28
SES	.08**	.03	.03†	.02	.09	.08
Race						
African American	.13	.17	-.04	.12	.79†	.41
Hispanic	-.08	.12	-.11	.09	.20	.32
Other	.20	.17	-.13	.12	.14	.49

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

For the gender split regression models the two-way interaction between property victimization x HRR (β = -.03; p = .02) was both significant and negatively related to increased levels of antisocial behavior for males, but showed no significant effects for females (see Table 12). In addition, the control variables of low self-control (Males: β = 1.02; $p \leq .001$; Females: β = 5.40; p = .002) and delinquent peers (Males: β = 15.01; $p \leq .001$; Females: β = 33.75; $p \leq .001$) were significant and positively related to increased antisocial behavior as in previous models for both males and females, with the exception that personal victimization (β = .37; p = .10) was approaching significance for males only (see Table 12). Also, similar to prior gender sample models, SES (β = .10; p = .003) was significant and positively related to increased antisocial behavior for females but not for males (see Table 12).

Moreover, unlike the above model concerning antisocial behavior, property victimization x HRR (Males:β = -.03; p = .01; Females: β = -.03; p = .02) was significant and negatively related to increased soft substance use for both males and females (see Table 12). Moreover, as shown in Table 12, the control variables of delinquent peers (Males: β = 10.52; $p \leq .001$; Females: β = 21.52; $p \leq .001$) and age (Males: β = .1; $p \leq .001$; Females: β = .04; p = .002) were significantly associated with increased soft substance use for both males and females. Further, low self-control (β = 3.89; $p \leq .001$) was significantly related to increased soft substance use for females only (see Table 12). Finally, low self-control (β = 1.52; p = .06) was approaching significance for males and identifying as Other (β = -.25; p = .08) was approaching significance for females concerning soft substance use (see Table 12).

Table 12

Tobit regression examining the two-way interaction effect of property victimization x HRR on antisocial behavior, soft substance use, and hard substance use for males (n = 163) and females (n = 323).

	Antisocial Behavior				**Soft Substance Use**			
	Males		**Females**		**Males**		**Females**	
	B	SE	B	SE	B	SE	B	SE
Independent Variables								
Property Victimization	.18*	(.09)	.06	(.13)	.12	(.08)	.15†	(.09)
Personal Victimization	.37†	(.22)	.03	(.05)	.11	(.19)	.02	(.03)
PTSD	.14	(.10)	-.001	(.05)	.02	(.08)	.01	(.03)
HRR	.02	(.02)	.02	(.01)	.03†	(.01)	-.001	(.01)
Property V. x HRR	-.03*	(.01)	-.01	(.02)	-.03*	(.01)	-.03*	(.01)
Control Variables								
Low self-control	3.92***	(1.02)	5.40**	(1.71)	1.52†	(.79)	3.89**	(1.13)
Delinquent Peers	15.01***	(2.35)	33.75***	(3.42)	10.52***	(1.82)	21.52***	(2.21)
Age	.002	(.04)	.03	(.02)	.10***	(.03)	.04**	(.01)
SES	.04	(.05)	.10**	(.03)	.03	(.04)	.04†	(.02)
Race								
African American	.10	(.40)	.07	(.19)	-.06	(.30)	-.02	(.13)
Hispanic	.14	(.22)	-.22	(.15)	-.15	(.17)	-.09	(.10)
Other	.39	(.29)	.11	(.21)	.10	(.24)	-.25†	(.14)

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

Personal Victimization x PTSD

The Tobit models that predicted the effects of the two-way interaction of personal victimization x PTSD on antisocial behavior, soft substance use, and hard substance use showed no significant effects for the full sample (see Table 13). However, personal victimization (β = .25; p = .03) was significant and positively related to increased antisocial behavior as a single main effect (see Table 13). Moreover, the control variables of low self-control (β = 8.74; $p \leq .001$), delinquent peers (β = 47.24; $p \leq .001$), and SES (β = .08; p = .005) all showed significant and positive effects on increased antisocial behavior for the full sample. Second, concerning soft substance use, only the control variables of low self-control (β = 4.82; $p \leq .001$), delinquent peers (β = 33.06; $p \leq .001$), and age (β = .05; $p \leq .001$) had significant and positive associations with SES approaching significance (β = .04; p = .07) regarding the full sample (see Table 13). Finally, hard substance use was significant and positively associated with PTSD (β = .31; p = .002) as a main effect and the control variables delinquent peers (β = -.02 $p \leq .001$) and age (β = .13; $p \leq .001$) (see Table 13).

Table 13

Tobit regression examining the two-way interaction effect of Personal Victimization x PTSD on antisocial behavior, soft substance use, and hard substance use for the full sample (n = 486).

	Antisocial Behavior		**Soft Substance Use**		**Hard Substance Use**	
	B	SE	B	SE	B	SE
Independent Variables						
Property Victimization	-.002	.04	-.04	.03	.04	.08
Personal Victimization	.25*	.11	.13	.08	.38	.28
PTSD	.07	.04	.02	.03	.31**	.10
HRR	.01	.01	-.001	.01	.003	.02
Personal V. x PTSD	-.003	.001	-.001	.001	-.01	.01
Control Variables						
Low self-control	8.74***	2.00	4.82***	1.40	-3.17	4.65
Delinquent Peers	47.24***	4.13	33.06***	2.88	45.29***	10.50
Age	.02	.02	.05***	.01	.13***	.04
Gender	-.12	.11	-.03	.08	-.46	.29
SES	.08**	.03	.04†	.02	.10	.08
Race						
African American	.16	.17	-.02	.12	.85	.42
Hispanic	-.08	.12	-.11	.09	.18	.33
Other	.25	.17	-.09	.13	.03	.48

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

The two-way interaction between personal victimization x PTSD split by gender showed no significant relationship with antisocial behavior or soft substance use for either males or females (see Table 14). However, the control variables of low self-control (Males: β = 3.92; $p \leq .001$; Females: β = 4.87; $p = .005$) and delinquent peers (Males: β = 15.32; $p \leq .001$; Females: (β = 33.14; $p \leq .001$) were both significant and positively related to increased levels of antisocial behavior (Females: β = .10; $p = .003$) and SES was significant and positive related to antisocial behavior for females only (see Table 14). In addition, the control variables of delinquent peers (Males: β = 10.69; $p \leq .001$; Females: β = 21.25; $p \leq .001$) and age (Males: β = .11; $p \leq .001$; Females: β = .04; $p = .002$) also both exhibited a significant and positive relationship with soft substance use for both genders. Finally, low self-control was significant and positively related to increased soft substance use for females (β = 3.51; $p = .003$) but not for males (see Table 14).

Table 14

Tobit regression examining the two-way interaction effect of Personal Victimization x PTSD on antisocial behavior, soft substance use, and hard substance use for males (n = 163) and females (n = 323).

	Antisocial Behavior				**Soft Substance Use**			
	Males		**Females**		**Males**		**Females**	
	B	SE	B	SE	B	SE	B	SE
Independent Variables								
Property Victimization	.00	(.05)	-.04	(.06)	-.04	(.04)	-.04	(.04)
Personal Victimization	.33	(.38)	.20†	(.12)	-.11	(.31)	.12	(.08)
PTSD	.20†	(.11)	.01	(.05)	.05	(.03)	.01	(.03)
HRR	.001	(.02)	.02	(.01)	.01	(.01)	-.01	(.01)
Personal V. x PTSD	-.00	(.01)	-.002	(.001)	.01	(.01)	-.00	(.001)
Control Variables								
Low self-control	3.92***	(1.06)	4.87**	(1.72)	1.52	(.81)	3.51**	(1.16)
Delinquent Peers	15.32***	(2.43)	33.14***	(3.41)	10.69***	(1.86)	21.25***	(2.23)
Age	.01	(.04)	.03	(.02)	.11***	(.03)	.04**	(.01)
SES	.05	(.05)	.10**	(.03)	.04	(.04)	.04	(.02)
Race								
African American	.18	(.42)	.10	(.19)	.03	(.31)	-.01	(.13)
Hispanic	.15	(.22)	-.21	(.15)	-.13	(.18)	-.10	(.10)
Other	.48	(.30)	.14	(.21)	.21	(.24)	-.22	(.14)

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

Personal Victimization x HRR

Next, the Tobit model concerning the interaction between personal victimization x HRR (β = -.02; p = .01) was significant and negatively related to increased levels of antisocial behavior but was not significantly related to soft substance use or hard substance use for the full sample (see Table 15). In addition, within the full sample, the control variables of low self-control (β = .63; $p \leq .001$), delinquent peers (β = 45.58; $p \leq .001$), and SES (β = .08; p = .006) were significant and positively associated with antisocial behavior with identifying as Other (β = .10; p = .07) being marginally significant (see Table 15). Second, the control variables of low self-control (β = 4.81; $p \leq .001$), delinquent peers (β = 33.23; $p \leq .001$), and age (β = .05; $p \leq .001$) had a significant effect on increased levels of soft substance use with SES approaching significance (β = .04; p = .07) for the full sample (see Table 15). Third, PTSD (β = .03; p = .003) had a significantly positive association with increased hard substance use (see Table 15). Finally, for the full sample, the control variables of delinquent peers (β = 47.16; $p \leq .001$) and age (β = .13; $p \leq .001$) were also significant and positively associated with increased hard substance use (see Table 15).

Table 15

Tobit regression examining the two-way interaction effect of Personal Victimization x HRR on antisocial behavior, soft substance use, and hard substance use for the full sample (n = 486).

	Antisocial Behavior		**Soft Substance Use**		**Hard Substance Use**	
	B	SE	B	SE	B	SE
Independent Variables						
Property Victimization	-.01	.04	-.04	.03	.04	.07
Personal Victimization	.28**	.10	.14†	.07	.11	.22
PTSD	.05	.04	.01	.03	.28**	.09
HRR	.02†	.01	.001	.01	.01	.02
Personal V. x HRR	-.02*	.01	-.01	.01	-.01	.02
Control Variables						
Low self-control	.63***	1.99	4.81***	1.41	-2.99	4.64
Delinquent Peers	47.58***	4.10	33.23***	2.88	47.16***	10.55
Age	.02	.02	.05***	.01	.13***	.04
Gender	-.12	.11	-.03	.08	-.42	.29
SES	.08**	.03	.04†	.02	.10	.08
Race						
African American	.16	.17	-.02	.12	.83	.42
Hispanic	-.07	.12	-.11	.09	.17	.33
Other	.29†	.17	-.08	.13	.05	.48

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

As shown in Table 16, the Tobit regression including the interaction of personal victimization x HRR showed no significant relationships with either antisocial behavior or soft substance use for males within the gender split model (see Table 16). Further, for males, although the two-way interaction was not significant the main effect of PTSD (β = .20; p = .05) was significant and positively associated with increased antisocial behavior (see Table 16). Moreover, unlike males, personal victimization x HRR (β = -.02; p = .02) was significant and negatively associated with increased antisocial behavior for females (see Table 16). In addition, for both genders, the control variables of low self-control (Males: β = 3.95; $p \leq .001$; Females: β = 4.67; p = .006) and delinquent peers (Males: β = 15.31; $p \leq .001$; Females: β = 33.34; $p \leq .001$) showed a significant and positive relationship with increased antisocial behavior (see Table 16). Again, only the female models exhibited a significant and positive relationship between SES and antisocial behavior (β = .10; p = .004) (see Table 16).

The interaction of personal victimization x HRR was not significant for either males or females concerning soft substance use but the control variables of delinquent peers (Males: β = 10.69; $p \leq .001$; Females: β = 21.42; $p \leq .001$) and age (Males: β = .11; $p \leq .001$; Females: β = .04; p = .002) were significant with a positive relationship with increased soft substance use for both genders (see Table 16). Finally, low self-control was also significant and positively associated with increased soft substance use for females (β = 3.52; p = .002) but not males (see Table 16).

Table 16

Tobit regression examining the two-way interaction effect of Personal Victimization x HRR on antisocial behavior, soft substance use, and hard substance use for males (n = 163) and females (n = 323).

	Antisocial Behavior				**Soft Substance Use**			
	Males		**Females**		**Males**		**Females**	
	B	SE	B	SE	B	SE	B	SE
Independent Variables								
Property Victimization	.00	(.05)	-.06	(.06)	-.04	(.04)	-.04	(.04)
Personal Victimization	.25	(.33)	.29**	(.10)	.14	(.27)	.12	(.07)
PTSD	.20*	(.10)	.001	(.05)	.08	(.08)	.01	(.03)
HRR	-.001	(.02)	.02*	(.01)	.02	(.01)	-.01	(.01)
Personal V. x HRR	.01	(.04)	-.02*	(.01)	-.01	(.03)	-.01	(.004)
Control Variables								
Low self-control	3.95***	(1.06)	4.67**	(1.70)	1.44	(.81)	3.52**	(1.15)
Delinquent Peers	15.31***	(2.43)	33.34***	(3.36)	10.69***	(1.86)	21.42***	(2.22)
Age	.01	(.04)	.03	(.02)	.12***	(.03)	.04***	(.01)
SES	.05	(.05)	.10**	(.03)	.04	(.04)	.04	(.02)
Race								
African American	.20	(.41)	.10	(.18)	-.003	(.31)	-.01	(.13)
Hispanic	.15	(.22)	-.19	(.14)	-.14	(.18)	-.09	(.10)
Other	.48	(.30)	.18	(.21)	.20	(.24)	-.21	(.14)

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

PTSD x HRR

The models concerning the two-way interaction of PTSD x HRR were not significantly related to any of the response variables: antisocial behavior, soft substance use, or hard substance use for the full sample (see Table 17). However, the controls variables of low self-control (β = 9.20; $p \leq .001$), delinquent peers (β = 47.83; $p \leq .001$), and SES (β = .09; $p = .003$) were significant and positive concerning increased levels of antisocial behavior for the full sample (see Table 17). Second, within the full sample, the control variables of low self-control (β = 5.06; $p \leq .001$), delinquent peers (β = 33.27; $p \leq .001$), and age (β = .06; $p \leq .001$) had significant and positive effects on soft substance use with SES (β = .04; $p = .06$) approaching significance. Third, and similar to above models, PTSD (β = .35; $p = .02$), exhibited a significant and positive relationship with hard substance use. Moreover, the control variables of low self-control (β = 47.25; $p \leq .001$) and age (β = .14; $p \leq .001$) which were also significant and positively indicative of increased hard substance use in the full sample (see Table 17).

Table 17

Tobit regression examining the two-way interaction effect of PTSD x HRR on antisocial behavior, soft substance use, and hard substance use for the full sample (n = 486).

	Antisocial Behavior		**Soft Substance Use**		**Hard Substance Use**	
	B	SE	B	SE	B	SE
Independent Variables						
Property Victimization	.003	.04	-.04	.03	.04	.07
Personal Victimization	.05	.05	.04	.03	.03	.19
PTSD	.13	.06	.07	.05	.35**	.15
HRR	.02	.01	.01	.01	.02	.03
PTSD x HRR	-.001	.00	-.00	.00	-.001	.001
Control Variables						
Low self-control	9.20***	1.99	5.06***	1.40	-2.94	4.61
Delinquent Peers	47.83***	4.13	33.27***	2.87	47.25***	10.52
Age	.03	.02	.06***	.01	.14***	.04
Gender	-.10	.11	-.02	.08	-.40	.28
SES	.09**	.03	.04†	.02	.10	.08
Race						
African American	.15	.17	-.02	.12	.82	.42
Hispanic	-.07	.12	-.11	.09	.17	.33
Other	.26	.17	-.08	.13	.05	.48

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

The two-way interaction PTSD x HRR showed no significant relationship with antisocial behavior or soft substance use for either males or females. However, the main effect of HRR (β = .03; p = .02) was significant and positively related to increased antisocial behavior for females (see Table 18). In addition, the control variables of low self-control (Males: β = 3.93; $p \leq .001$; Females: β = 5.22; p = .002) and delinquent peers (Males: β = 15.42; $p \leq .001$; Females: β = 33.83; $p \leq .001$) showed a significant and positive relationship with increased antisocial behavior for both males and females (see Table 18). Further, SES (β = .11; p = .002) was significant and positively associated with increased antisocial behavior for females but not for males (see Table 18). Moreover, the control variables of delinquent peers (Males: β = 10.59; $p \leq .001$; Females: β = 21.54; $p \leq .001$) and age (Males: β = .11; $p \leq .001$; Females: β = .04; $p \leq .001$) showed significant and positive associations with increased soft substance use for both genders (see Table 18). Finally, the control variables of low self-control (β = 3.74; $p \leq .001$) was significant and positively related to increased soft substance use for females only (see Table 18).

Table 18

Tobit regression examining the two-way interaction effect of PTSD x HRR on antisocial behavior, soft substance use, and hard substance use for males (n = 163) and females (n = 323).

	Antisocial Behavior				**Soft Substance Use**			
	Males		**Females**		**Males**		**Females**	
	B	SE	B	SE	B	SE	B	SE
Independent Variables								
Property Victimization	.003	(.05)	-.03	(.06)	-.04	(.04)	-.03	(.04)
Personal Victimization	.31	(.24)	.05	(.05)	.09	(.20)	.03	(.03)
PTSD	.17	(.13)	.10	(.07)	.11	(.11)	.04	(.05)
HRR	-.01	(.02)	.03*	(.01)	.02	(.02)	-.00	(.01)
PTSD x HRR	.00	(.001)	-.001	(.00)	-.00	(.001)	-.00	(.00)
Control Variables								
Low self-control	3.93***	(1.06)	5.22**	(1.70)	1.47	(.81)	3.74***	(1.14)
Delinquent Peers	15.42***	(2.44)	33.83***	(3.40)	10.59***	(1.87)	21.54***	(2.23)
Age	.01	(.04)	.04	(.02)	.12***	(.03)	.04***	(.01)
SES	.05	(.05)	.11**	(.03)	.04	(.04)	.04	(.02)
Race								
African American	.18	(.41)	.08	(.19)	.01	(.31)	-.02	(.13)
Hispanic	.15	(.22)	-.20	(.15)	-.14	(.18)	-.10	(.10)
Other	.49	(.30)	.18	(.21)	.18	(.24)	-.22	(.15)

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

Three-way Interaction Regressions

Tables 19 and 21 reveal that within the full sample the findings of the current dissertation did not support the main hypotheses. Specifically, within the full sample, the three-way interactions of increased victimization (both property and personal), PTSD, and HRR were not indicative of increased levels of antisocial behavior, soft substance use, or hard substance use (see Tables 19 and 21). Specifically, neither the three-way interaction of property victimization x PTSD x HRR or personal victimization x PTSD x HRR had a significant relationship with any of the three response variables of antisocial behavior, soft substance use, or hard substance use within the full sample (see Tables 19 and 21).

Property Victimization x PTSD x HRR

Within the model concerning the three-way interaction of property victimization x PTSD x HRR and antisocial behavior, only the control variables of low self-control (β = 9.17; $p \leq .001$), delinquent peers (β = 47.81; $p \leq .001$), and SES (β = .08; $p = .007$) were significant and positively related for the full sample (see Table 19). In addition, for the full sample, the control variables of low self-control (β = 5.24; $p \leq .001$), delinquent peers (β = 33.03; $p \leq .001$), and age (β = .05; $p \leq .001$) all exhibited significant and positive effects on soft substance use (see Table 19). Finally, as in previous models with two-way interactions present, the main effect of PTSD (β = .41; $p = .009$) was significant and positive concerning hard substance use for the full sample (see Table 19). In addition to PTSD, for the full sample, the control variables of delinquent peers (β = 47.25; $p \leq .001$) and age (β = .14; $p \leq .001$) were also significant and positively associated with hard substance use (see Table 19).

Table 19

Tobit regression including the three-way interaction effects between property victimization, PTSD, and HRR on antisocial behavior and soft substance use for the full sample (n = 486).

	Antisocial Behavior		**Soft Substance Use**		**Hard Substance Use**	
	B	SE	B	SE	B	SE
Independent Variables						
Property Victimization	.12	(.14)	.23*	(.10)	.54†	(.32)
Personal Victimization	.03	(.05)	.03	(.03)	.02	(.10)
PTSD	.11†	(.07)	.07	(.05)	.38*	(.15)
HRR	.02†	(.01)	.01	(.01)	.03	(.03)
Property V. x PTSD	.001	(.003)	-.003	(.002)	-.01	(.01)
Property V. x HRR	-.01	(.02)	-.03**	(.01)	-.04	(.04)
PTSD x HRR	-.00	(.00)	-.00	(.00)	-.00	(.001)
Prop V. x PTSD x HRR	-.001	(.00)	.00	(.00)	-.00	(.001)
Control Variables						
Low self-control	9.78***	(1.96)	5.46***	(1.39)	-1.62	(4.56)
Delinquent Peers	48.08***	(4.05)	33.10***	(2.84)	46.41***	(10.37)
Age	.02	(.02)	.05***	(.01)	.13***	(.04)
Gender	-.06	(.11)	-.01	(.08)	-.30	(.28)
SES	.08**	(.03)	.04*	(.02)	.08	(.07)
Race						
African American	.14	(.17)	-.04	(.12)	.75	(.41)
Hispanic	-.07	(.12)	-.11	(.09)	.18	(.32)
Other	.18	(.17)	-.14	(.13)	-.18	(.49)

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

Concerning the three-way interactions by gender only one of the three-way interaction models was significant. Specifically, the three-way interaction of property victimization x PTSD x HRR ($\beta = .001$; $p = .04$) showed a significant and positive relationship with increased levels of antisocial behavior for males (see Table 20). Similar to other presented models, the control variables of low self-control (Males: $\beta = 4.21$; $p \leq .001$; Females: $\beta = 5.82$; $p \leq .001$) and delinquent peers (Males: $\beta = 14.91$; $p \leq .001$; Females: $\beta = 34.15$; $p \leq .001$) were significant and positively related to increased antisocial behavior for both males and females (see Table 20). Further, SES ($\beta = .10$; $p = .002$) remained significant and positively associated with antisocial behavior, just as in all previous models, concerning the female only sample (see Table 20).

Moreover, the three-way interaction models were not significant concerning soft substance use for males or females (see Table 20). In addition, the control variables of low self-control (Males: $\beta = 1.71$; $p = .03$; Females: $\beta = 4.11$; $p \leq .001$), delinquent peers (Males: $\beta = 10.18$; $p \leq .001$; Females: $\beta = 21.66$; $p \leq .001$), and age (Males: $\beta = .10$; $p \leq .001$; Females: $\beta = .04$; $p = .003$) were significant and positively related to increased soft substance use for both males and females within the three-way interaction model of property victimization x PTSD x HRR (see Table 20). Finally, the variables of SES ($\beta = .04$; $p = .09$) and identifying as Other ($\beta = .04$; $p = .06$) were both marginally associated with soft substance use for females only (see Table 20).

Table 20

Tobit regression including the three-way interaction effects between property victimization, PTSD, and HRR on antisocial behavior and soft substance use for males (n = 163) and females (n = 323).

	Antisocial Behavior				**Soft Substance Use**			
	Males		**Females**		**Males**		**Females**	
	B	SE	B	SE	B	SE	B	SE
Independent Variables								
Property Victimization	.54*	(.24)	-.01	(.17)	.43*	(.20)	.17	(.12)
Personal Victimization	.28	(.23)	.01	(.05)	.07	(.19)	.02	(.03)
PTSD	.12	(.14)	.08	(.07)	.10	(.12)	.04	(.05)
HRR	.02	(.02)	.02	(.01)	.04*	(.02)	-.002	(.01)
Property V. x PTSD	-.01	(.01)	.01	(.01)	-.01†	(.004)	-.001	(.004)
Property V. x HRR	-.08**	(.03)	.05	(.02)	-.06*	(.02)	-.01	(.01)
PTSD x HRR	.00	(.001)	-.00	(.00)	-.001	(.001)	-.00	(.00)
Prop V. x PTSD x HRR	.001*	(.001)	-.001	(.001)	.001	(.001)	-.00	(.00)
Control Variables								
Low self-control	4.21***	(1.01)	5.82***	(1.65)	1.71*	(.80)	4.11***	(1.13)
Delinquent Peers	14.91***	(2.34)	34.15***	(3.29)	10.18***	(1.83)	21.66***	(2.20)
Age	-.001	(.04)	.03	(.02)	.10***	(.03)	.04**	(.01)
SES	.04	(.05)	.10**	(.03)	.02	(.04)	.04†	(.02)
Race								
African American	.04	(.40)	.11	(.18)	-.11	(.30)	-.01	(.12)
Hispanic	.12	(.21)	-.20	(.14)	-.17	(.17)	-.09	(.10)
Other	.47	(.29)	.10	(.20)	.11	(.24)	-.27†	(.14)

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

Personal Victimization x PTSD x HRR

The three-way interaction model for the full sample concerning the relation between personal victimization x PTSD x HRR was not significantly associated with any of the three dependent variables of antisocial behavior, soft substance use, or hard substance use. In addition, the control variables of low self-control (β = 9.21; $p \leq .001$), delinquent peers (β = 47.47; $p \leq .001$), and SES (β = .08; $p \leq .001$) were also significant but positively related to increased levels of antisocial behavior (see Table 21). Further, within the full sample three-way interaction models, the control variables of low self-control (β = 5.11; $p \leq .001$), delinquent peers (β = 33.26; $p \leq .001$), and age (β = .05; $p \leq .001$) were significant and positively related to increased soft substance use with SES (β = .04; $p = .06$) approaching significance (see Table 21). Finally, similar to above models concerning hard substance use, the main effect of PTSD (β = .36; $p \leq .02$) was significant and positively associated with increased hard substance use reported by participants. Finally, the control variables of delinquent peers (β = 44.86; $p \leq .001$), age (β = .01; $p \leq .001$), and being African American (β = .83; $p = .04$) were significant and positively associated with increased hard substance use for the full sample (see Table 21).

Table 21

Tobit regression including the three-way interaction effects between personal victimization, PTSD, and HRR on antisocial behavior and soft substance use for the full sample (n = 486).

	Antisocial Behavior		**Soft Substance Use**		**Hard Substance Use**	
	B	SE	B	SE	B	SE
Independent Variables						
Property Victimization	-.01	.04	-.04	.03	.03	.07
Personal Victimization	.19	.18	.09	.13	-.22	.50
PTSD	.10	.07	.06	.05	.35*	.15
HRR	.02†	.01	.01	.01	.002	.03
Personal V. x PTSD	.001	.003	.00	.002	.01	.01
Personal V. x HRR	-.00	.03	.003	.02	.10	.10
PTSD x HRR	-.00	.00	-.00	.00	-.00	.001
Personal V. x PTSD x HRR	-.00	.00	-.00	.00	-.002	.002
Control Variables						
Low self-control	8.50***	2.00	4.75***	1.42	-3.59	4.66
Delinquent Peers	47.50***	4.11	33.13***	2.87	44.96***	10.42
Age	.02	.02	.06***	.01	.13***	.04
Gender	-.12	.11	-.03	.08	-.48	.29
SES	.08**	.03	.04†	.02	.11	.08
Race						
African American	.16	.17	-.02	.12	.89*	.42
Hispanic	-.06	.12	-.10	.09	.17	.32
Other	.30	.17	-.07	.13	.05	.48

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

The final Tobit models split by gender showed no significance between the three-way interaction term of personal victimization x PTSD x HRR for either of the response variables of antisocial behavior or soft substance use for males or females (see Table 22). In addition, the control variables of low self-control (Males: $\beta = 3.96$; $p \leq .001$; Females: $\beta = 4.55$; $p = .008$) and delinquent peers (Males: $\beta = 15.47$; $p \leq .001$; Females: $\beta = 33.70$; $p \leq .001$) both showed significant and positive relationships with increased antisocial behavior for both males and females (see Table 22). Further, as in previous models, SES ($\beta = .10$; $p = .003$) was also significant and positively associated with increased antisocial behavior for females only (see Table 22). Moreover, the control variables delinquent peers (Males: $\beta = 10.68$; $p \leq .001$; Females: $\beta = 21.40$; $p \leq .001$) and age (Males: $\beta = .11$; $p \leq .001$; Females: $\beta = .04$; $p = .002$) showed both a significant and positive association with increased soft substance use for both genders (see Table 22). Finally, the control variable of low self-control ($\beta = 3.44$; $p = .004$) was significant and positive for soft substance use for females but was only marginally significant for males ($\beta = 1.54$; $p = .06$; see Table 22).

Table 22

Tobit regression including the three-way interaction effects between personal victimization, PTSD, and HRR on antisocial behavior and soft substance use for males (n = 163) and females (n =323).

	Antisocial Behavior				**Soft Substance Use**			
	Males		**Females**		**Males**		**Females**	
	B	SE	B	SE	B	SE	B	SE
Independent Variables								
Property Victimization	.02	(.05)	-.06	(.06)	-.03	(.04)	-.04	(.04)
Personal Victimization	.75	(.60)	.15	(.18)	.19	(.49)	.10	(.13)
PTSD	.24	(.17)	.06	(.08)	.11	(.13)	.03	(.05)
HRR	.00	(.03)	.03*	(.01)	.02	(.02)	-.00	(.01)
Personal V. x PTSD	-.02	(.02)	.003	(.004)	-.00	(.01)	-.003	(.002)
Personal V. x HRR	-.12	(.13)	-.003	(.03)	-.08	(.10)	.001	(.02)
PTSD x HRR	-.001	(.001)	-.001	(.00)	-.001	(.001)	-.00	(.00)
Personal V. x PTSD x HRR	.003	(.003)	-.00	(.00)	.001	(.002)	-.00	(.00)
Control Variables								
Low self-control	3.96***	(1.06)	4.55**	(1.71)	1.54†	(.82)	3.44**	(1.17)
Delinquent Peers	15.47***	(2.46)	33.70***	(3.40)	10.68***	(1.88)	21.40***	(2.24)
Age	.01	(.04)	.03	(.02)	.11***	(.03)	.04**	(.01)
SES	.04	(.05)	.10**	(.03)	.04	(.04)	.04	(.02)
Race								
African American	.13	(.42)	.09	(.18)	.01	(.32)	-.01	(.13)
Hispanic	.16	(.22)	-.19	(.14)	-.12	(.18)	-.09	(.10)
Other	.45	(.31)	.23	(.23)	.19	(.25)	-.20	(.15)

Note: For the above table significance is denoted as $p \leq .1^{\dagger}$, $p \leq .05$*, $p \leq .01$**, and $p \leq .001$***.

Summary of Results

The overall findings of the current dissertation did not support the primary hypothesis that the three-way interaction between the risk factors of victimization (property and personal), PTSD, and HRR increased the propensity for antisocial behavior, soft substance use, or hard substance use. Specifically, no statistically significant effects were found between the three-way interactions on any of the three outcome variables of antisocial behavior, soft substance use, and hard substance use for the full sample. However, the gender split models showed partial support that the three-way interaction of property victimization x PTSD x HRR significantly predicted antisocial behavior in males. Further, when significance between two-way interactions and the dependent variables occurred the interactions were indicative of a decrease, not an increase, in the outcome variables which is the opposite of what was predicted in the current dissertation. Hence, the significant two-way interactions acted as a factor that reduced the predicted outcomes and not as risk factors for the dependent variables of antisocial behavior, soft substance use, and hard substance use. Possible explanations for these findings and relationships will be discussed below.

CHAPTER V

Discussion

The primary goal of the current dissertation was to examine whether the interaction between victimization (both property and personal), PTSD, and HRR had an impact on antisocial behavior, soft substance use, and/or hard substance use. In addition, the secondary goal was to test whether these relationships varied by gender. The overall findings of the current dissertation did not support the major hypotheses and found no significant relationship between the three-way interactions and increased levels of antisocial behavior, soft substance use, and hard substance use. Specifically, the models containing the three-way interactions (e.g., property victimization x PTSD x HRR and personal victimization x PTSD x HRR) showed no significant relationships with any of the three outcome measures of antisocial behavior, soft substance use, and hard substance use within the full sample. However, the findings of the current dissertation did show main effects and two-way interactions exhibiting a significant relationship with all three response variables in the full sample.

In addition, the three-way interaction of property victimization x PTSD x HRR was found to increase antisocial behavior for males within the gender split models. As such, the secondary research question of the current dissertation was only partially supported. Further, given that within gender there were significant differences in antisocial behavior and soft substance use where males perpetrated both behaviors significantly more often than females which are common findings among other studies of antisocial behaviors (Brady & Randall, 1999; Moffitt & Caspi, 2001). It is interesting though that the effects of the risk variables could have compensated for these differences

(e.g. higher rates of antisocial behavior in males) given that, and in alignment with prior research, females experienced significantly more personal victimization and exhibited significantly higher levels of PTSD symptoms than males which were being tested as risk factors for antisocial behavior (Cutter & Nolen-Hoeksema, 1991; Elklit, 2002; Tolin & Foa, 2002). However, the higher levels of these risk factors in females did not account for increased propensity to either antisocial behavior or soft substance use in any of the gender split models.

Main Effects

The primary findings of the main effects for the Tobit models were only in the control variables with no significance among the independent variables of victimization, PTSD, and HRR for the full sample. However, increased PTSD was related to increased antisocial behavior in males for the gender split Tobit models. This is interesting given that males experienced significantly less PTSD than females in the current dissertation sample, but the effects of PTSD on antisocial behavior was still significant for males and not females. This could be in part due to emotional maturity or coping differences between genders of the current sample in that males may be more likely to act out due to stress or the effects of PTSD more so than their female counter parts (Taylor, Klein, Lewis, Gruenewald, Gurung, & Updegraff, 2000). For example, Taylor and colleagues (2000) found that when confronted with stress females have been shown to be more likely to seek support from their social networks whereas males do not and are more likely to exhibit more rash reactionary conflict-based behaviors as coping mechanisms (Taylor, et al., 2000).

Moreover, in general for both the full sample and gender split models the control variables of low self-control, delinquent peers, and SES were associated with increased levels of antisocial behavior. Further, these trends were not only present in the main effect models but maintained in every subsequent model regardless of the addition of two-way or three-way interactions. These findings that low self-control, delinquent peers, and SES are associated with an increase in antisocial behavior are not novel and are common within the field of criminal justice concerning antisocial behavior (Denson, DeWall, & Finkel, 2012; Monahan, Steinberg, & Cauffman, 2009: Tuvblad, Grann, & Lichtenstein, 2006). Hence, these findings do not add to the current literature, but do support the validity of the measures used within the current dissertation.

Also, much like antisocial behavior, increased soft substance use was related to low self-control, delinquent peers, and increased age in the main effects models as well as every subsequent model regardless of the presence of interactions with few differences between the full sample and split by gender sample. Again, although these findings are not novel to the field of criminology these findings are interesting given the sample population (Barnes, Hoffman, Welte, Farrell, & Dintcheff, 2006). The sample for the current dissertation is made up primarily of college age students and age was positively related to soft substance use, meaning that older individuals within the sample reported increased levels of soft substance use. This could have been an effect of individuals who are older generally live off campus and would be above the legal age to buy alcoholic beverages and experience decreased barriers to purchasing and using marijuana. Hence, potentially given the lack of guardianship (i.e. no Resident Assistant and dorm room mate) and/or meeting the legal age to purchase alcohol may have increased the chances of

individuals to be more likely to exhibit soft substance use compared to younger individuals in the college sample.

Interestingly, increased hard substance use was only related to increased levels of PTSD, delinquent peers, and age for the full sample and not the gender split models. These findings, namely the effects of these three variables on hard substance use, are present in every model including the interaction models (e.g. two-way and three-way). The positive relationship between PTSD, delinquent peers, and age on hard substance use makes sense given previous research (Kilpatrick, Acierno, Saunders, Resnick, Best, & Schnurr, 2000). Given that PTSD can become more serious if not professionally treated over time potentially older individuals may have been experiencing increasing severity of symptoms simply due to the cumulative amount of time they have been experiencing their PTSD symptoms (Back et al., 2014). In that same vein, individuals experiencing worsening symptoms may have increased propensity towards hard substance use as a coping mechanism (Clark, Masson, Delucchi, Hall, & Sees, 2001; Back et al., 2014). Further, it may be that older individuals have greater access to hard substances perhaps through their delinquency peer networks rather than younger individuals making hard substance use possible. Also, older individuals who have been exhibiting symptoms of PTSD and self-medicating for longer periods of time may have found that hard substance use is a more effective form of self-medication over soft substance use (Najavits, Weiss, & Shaw, 1997). This scenario is viable given that past research has established a link between the compounding negative effects of PTSD and substance use disorder being associated with the use of stronger or harder substances (Najavits, Weiss, & Shaw, 1997). Moreover, it may also be that individuals who already had delinquent peers could have

increased hard substance use simply through allowing access for hard substances considering these substances are more likely to be difficult to obtain (e.g. getting beer versus getting heroine). However, another etiological pathway is that older individuals who have experienced PTSD for long amounts of time and needed harder substances to cope and experienced a shift in peer groups from prosocial to more delinquent groups based on a shift towards more delinquent behavior and substance use (Feiring, Miller-Johnson, & Cleland, 2007). Hence, these factors of PTSD, delinquent peers, and age could all be factors that are related with each other, the onset, and maintenance of hard substance use, but given that this data is cross-sectional these relationships cannot be teased apart but future research should seek to better understand these relationships.

Two-way Interactions

The two-way interaction models showed significant effects on both antisocial behavior and soft substance use within both the full and the gender split samples. However, all of the significant two-way interactions exhibited a negative relationship with the outcome variables. Both the primary and secondary hypotheses for the current dissertation predicted that the relationship between the interactions and response variables would be positive based on prior literature. Although these results did not match the predictions made in the current dissertation the findings concerning the two-way interactions may provide additional insight into how these risk factors may be operating. Below these findings will be discussed explaining possible scenarios as to why the negative relationship between the two-way interactions and the response variables were in the opposite direction of the predictions made in the current dissertation.

First, the interaction between property victimization and PTSD was associated with decreased levels of antisocial behavior for the full sample. Again, although these findings do not align with the predictions made, this relationship could be due to factors inherent of experiencing property victimization and exhibiting PTSD. For example, the presence of property victimization and exhibiting increased levels of PTSD could possibly be indicative of individuals living in areas of increased crime, thus being at greater exposure to experience these risk factors (Stafford & Galle, 1984; Finkelhor, Turner, Ormrod, &, Hamby, 2009). Further, prior research has found a relationship between living in high crime areas and experiencing increased levels of victimization and PTSD (Bisson & Shepherd, 1995; Berman, Kurtines, Silverman, & Serafini, 1996). Said another way, areas where property crime happen may also be areas of increased overall crime making the risk of victimization and the onset of PTSD higher for everyone in the area. Experiencing both property victimization and PTSD could alter the routine activities of these individuals by increasing their propensity towards seclusion and/or avoidance behaviors. Thus, through seclusion and avoidance they would decrease interactions with other individuals which would decrease respondents' chances to exhibit antisocial behaviors (Kirkpatrick & Heller, 2014; Thompson & Waltz, 2010). Basically, due to living in an area of increased crime, individuals within the sample who experienced increased property victimization and PTSD may reduce the time they spend in public as a safety precaution in turn decreasing their ability to exhibit antisocial behavior as measured in the current sample (i.e. antisocial behavior that requires physical interactions with individuals or their property). It is important to note that the data used in the current dissertation did not have neighborhood level information such as crime rates or

qualitative information indicative of individuals' experiences of crime throughout the life course, but this information would be important for future research endeavors to include.

Both components of the interaction of property victimization and PTSD have been shown to increase seclusion and avoidant behaviors (Borooah & Carach, 1997; Kirkpatrick & Heller, 2014). For example, individuals who exhibit increased levels of fear of crime have been shown to minimalize their time away from their property in order to decrease exposure to victimization which would also decrease the availability of circumstances in which they could exhibit antisocial behavior (Borooah & Carach, 1997). In addition, the second portion of the interaction, PTSD, has also been shown to decrease the amount of interactions individuals have through the avoidance of triggers (Foa, Steketee, & Rothbaum, 1989). Hence, by experiencing increased property crime and exhibiting increased levels of PTSD these individuals may decrease their ability to commit antisocial behaviors due to decreased interactions with other individuals. Further, even if their property victimization and PTSD are unrelated etiologically, both of these factors being present could be compounding the effects of avoidance or lack of interaction, thus decreasing the propensity and level of antisocial behaviors. As mentioned above, regardless of the scenario, the interaction of property victimization and PTSD are likely to be altering the routine activities of the individuals within the sample which in turn decreases their overall interactions with individuals thus reducing antisocial behavior (Kirkpatrick & Heller, 2014).

For the gender split models only females exhibited a negative relationship between property victimization and PTSD concerning antisocial behavior with similar trends to the full sample models. Specifically, staying with the reasons given for the

negative relationship between the two-way interaction (property victimization x PTSD) and antisocial behavior for the full sample model above, the gender split model showed that this relationship was particularly important for females. These findings are interesting given that past research has found that women who spend increased amounts of time away from home (or living space) experience a significant increase in the likelihood of property victimization (Franklin, Franklin, Nobles, & Kercher, 2012). Moreover, women, more so than men, who have experienced property damage (e.g. been burglarized) have been shown to develop an increased fear of crime and are more likely to alter their routine activities by increasing reclusive behaviors in order to reduce exposure to possible subsequent victimization (Braungart, Braungart, & Hoyer, 1980). Also, females who experience stress and trauma have been shown to exhibit increased symptoms of PTSD about roughly twice as much as their male counterparts (Elklit, 2002). Hence, these findings align with the proposed explanation of decreased contact with others due to either an increased fear of crime or attempting to decrease subsequent victimization by decreasing exposure. Thus, given past literature and the findings of the current dissertation there is support that females who experience both property victimization and PTSD may be more likely than males to alter their routine activities and exhibit seclusion and avoidant behaviors (Braungart, Braungart, & Hoyer, 1980). It is also possible that, this interaction could be exhibiting a compounding effect of seclusion due to a fear of crime and avoidance behaviors associated with PTSD which could greatly reduce social interactions and the chances available to individuals to exhibit antisocial behavior.

In addition to the two-way interaction of property victimization and PTSD having a negative effect on antisocial behavior for females, this interaction also exhibited a decreased effect on soft-substance use for females within the gender split models. Again, this finding does not align with the predictions of the current dissertation or prior research in that increased victimization and PTSD normally is related to increases in soft substance use. However, the best explanation could be related to the scenario above where if women are beginning to alter their routine activities and exhibit less social interactions which may reduce the amount of soft substance use as many times these behaviors are exhibited in social settings (e.g., drinking alcohol or smoking marijuana with peers). Also, soft substance use may be lower for these females if they do not get out often to buy the substances, at either the store or from a drug dealer, due to fear of crime and avoidance behaviors.

Second, the interaction between property victimization and HRR showed a negative relationship with all three measures of antisocial behavior, soft substance use, and hard substance use for the full sample. These findings concerning HRR are also not what was predicted in the current dissertation but may be due to another related area of literature concerning the relationship between heart rate and antisocial behavior (Portnoy & Farrington, 2015). In the current dissertation, it was predicted that individuals who exhibited increased HRR in juncture with either trauma or PTSD would be more reactionary and exhibit a higher propensity for antisocial behavior (Ortiz & Raine, 2004). However, low resting heart rate (highly correlated with low heart rate reactivity) has also been linked to increased antisocial behavior (Portnoy & Farrington, 2015). Hence, the relationship with HR and antisocial behavior may have been problematic in the current

sample considering that increased HRR is more likely to increase antisocial behavior with the presence of trauma and/or PTSD (Ortiz & Raine, 2004). It may be that the majority of individuals with higher HRR levels did not also exhibit trauma or PTSD and more individuals exhibited both antisocial behavior and LRHR. Hence, the effect of HRR requires more in depth study to unpack the effects that HRR and PTSD have on antisocial behavior.

Concerning property victimization and increased HRR with decreased antisocial behavior the reasons could have been the same as the above interactions, in that individuals exhibiting property victimization and increased HRR altered their routine activities to reduce exposure to subsequent victimization due to potentially living in a higher crime area and attempting to avoid interactions as a means of safety. Similar to the effects of PTSD, increased HRR could also contribute as a means of reducing overall interactions with other individuals, thus reducing antisocial behavior, due to increased levels of anxiety or general fear or worry (Gorman & Sloan, 2000).

Moreover, the relationship between the interaction of property victimization and increased HRR and decreased substance use could have been due to the scenarios of routine activity given above as well as the age of the current sample. Experiencing both property crime and increased HRR could be associated with less substance use because the respondents may have access to other forms of therapy or legitimate medication. In that same vein, it may be that the effects of property victimization and increased HRR have not had time to negatively influence the participants' mental health in order to start abusing substances as a means to cope.

Specifically, the gender split models showed that males exhibited a significant relationship between property victimization x increased HRR and a decrease in antisocial behavior and soft substance use. Again, this may be due to a lack of exposure to social interactions by altering routine activities due to fear of crime and anxiety causing an overall decrease of social interactions (Gorman & Sloan, 2000). However, respondents suffering from fear of exposure and anxiety may be medicated due to parental support while in college and thus are able to afford legitimate medication, thus decreasing the need for substance use. Another confounding factor could be that use of anxiety medication both prescribed and non-prescribed has been on the rise in college campuses reducing the need for soft substances as a coping mechanisms (McCabe, West, Teter, & Boyd, 2014). Ideally the use of a non-prescribed medication should have been reported by respondents' within the substance use portion of the survey. However, many college students have been found to purchase over the counter controlled anxiety related medications from friends and feel little stigma associated with this being the same as buying illegal drugs (DeSantis, Webb, & Noar, 2008). Thus, there is the chance that even though their actions were illegal and would be defined as substance use that respondents saw this behavior as more benign and due to having the anxiety drug did not abuse other substances and did not view their acquisition of a non-prescribed drug as abuse.

Finally, and similar to the above interactions, the two-way interaction of personal victimization and increased HRR decreased antisocial behaviors for the full sample and for males in the gender split models. Again, this is not the effect predicted within the current dissertation and the examples given above may also apply here. In addition, individuals within this age group may not be in a stage of brain development where they

can fully process the effects of a personal victimization and understand the symptoms associated with increased HRR (i.e. just assuming they are stressed out due to tests or grades, etc.). This is not to suggest that their experiences are trivial in any way, but that they could still be in denial or be young enough that they are not experiencing the negative affects to a strong enough extent to be at risk of increased propensity for antisocial behavior. Further, research has suggested that current college students are experiencing increased levels of anxiety associated with their college experience compared to prior cohorts of college students (Beiter et al., 2015). This means there is also the possibility that they could overlook the consequences of their victimization and high HRR assuming that everyone is experiencing the same levels of stress after discussing their anxiety with peers.

The relationships presented in the above two-way interactions are interesting but need future studies aimed at elucidating these relationships. Although the scenarios described above are both logical and rooted in the literature these descriptions cannot be verified within the current dissertation, but can still act as a foundation for future researchers to better understand these inter-relationships.

Three-way Interaction

Only the gender split three-way interaction between property victimization x PTSD x HRR had a significant effect on increased levels of antisocial behavior for males. This relationship was predicted based on males exhibiting higher levels of property victimization, PTSD, and increased HRR all being risk factors for increased antisocial behavior. These findings are interesting given that the two-way interactions of property victimization with increased PTSD and increased HRR had the opposite effects on the outcome variables. Thus, these findings show the importance that all three risk factors being present, trauma (victimization), PTSD, and HRR to increase the propensity of antisocial behavior to be propagated in males. Said another way, with only two risk factors present a reduction in antisocial and substance use behaviors may occur , and that for males the presence of all three risk factors increase antisocial behavior.

The findings of the current dissertation suggest that three risk factors of property victimization x increased PTSD x increased HRR may increase males' propensity for antisocial behavior. These findings align with the original predictions made in previous chapters considering that each of the variables of victimization, increased PTSD, and increased HRR have been shown to be risk factors for increased antisocial behavior (Gottman et al., 1995; Sullivan, Farrell, & Kliewer, 2006; Jakupcak et al., 2007). Hence, within the current sample, individuals who have experienced property victimization, exhibit increased PTSD symptoms, and increased HRR have been shown to have an increased propensity towards exhibiting antisocial behaviors if they are male.

Moreover, as previously discussed, males are more likely to exhibit antisocial behavior and reactionary behaviors when confronted with or experiencing stress and

anxiety (Taylor et al., 2000). However, unlike the two-way interactions, for males who experienced property victimization, exhibit increased PTSD, and increased HRR (with both PTSD and increased HRR being correlated and indicative of increased stress and anxiety; Ortiz & Raine, 2009; Kirkpatrick & Heller, 2014) may increase males' likelihood of participation in antisocial behavior instead of trying to avoid it like their female counter parts. Further, by exhibiting the predisposition to exhibit negative and/or exhibit reactionary behaviors (in an environment that is not lacking stimuli) could be acting as a compounding factor between the three risk factors of property victimization x increased PTSD x increased HRR for increasing antisocial behavior for males. These findings suggest a need to better understand how these risk factors influence individuals especially based on gender. Also, potentially with a non-college sample the effect of these risk factors would be more pronounced and occur within females as well.

Limitations

No studies are without limitations and the current dissertation is no exception. Although measures were taken to reduce foreseeable problems, limitations, primarily based on temporal restrictions, were still present. First, although the research group was made up of several trained individuals, funding and space only allotted for one to two respondents' data to be collected at a time in the two lab rooms.[21] Specifically, the data collection had to be done in a linear fashion where each respondent started and ended at the same data collection point to be able to process the highest number of participants per day as efficiently as possible. Thus, data collection took a long time and the work was intense. Further, the number of researchers only mattered in reducing burn out as only

[21] Although the data collection setup consisted of two rooms, each room collected different types of data from respondents.

one researcher could work in one of the two rooms at a time. Each individual spent several hours a week in one of the two rooms in addition to their own work/class schedule collecting data. In that same vein, given that eight different individuals were collecting data, and although each individual was trained the group all double checked each other's work as human error/researcher variability could have impacted the data and current dissertation.[22]

Second, another limitation of the current dissertation is that the data collected and used was cross-sectional. Cross-sectional data has many advantages considering data collection logistics, and was the only form of data collection available to our research group at the onset of this project. However, many of the variables present within the current dissertation are better suited for a longitudinal approach. For example, the main independent variables of the current dissertation were the three risk factors of victimization, PTSD, and HRR and were measured by asking respondents to report how many times they had experienced different types of victimization events and experienced PTSD symptoms in the past year, while HRR was measured in a single sitting via a stress reactivity test. Thus, the data was collected at a single time and variation or changes for individuals' measures could not be captured. As discussed in previous chapters these three risk factors share a strong theoretical link to one another which makes teasing them apart challenging. The limitation is that the data used in the current dissertation cannot make etiological or causal links between these risk factors as the data was cross-sectional. Thus, given the current data it is impossible to know if our measure of PTSD is directly connected to victimization or vise a versa. This is not to say that victimization and PTSD

[22] It is important to note that only minor errors were found with coding which were corrected by the multiple checks conducted by lab members who systematically checked each other's work.

are completely unrelated in the current sample, but increased reported levels of PTSD could be indicative of other stressful experiences occurring prior to their victimization. In that same vein, increased HRR could be due to the presence of increased levels of PTSD/trauma, or could be due to unhealthy lifestyles or inherited biological factors. Hence, although this limitation does not necessarily alter the importance or strength of the current study, it does restrict what can be deduced from the findings.

Third, the data was collected from a southwestern state university. Limitations inherent of a college sample could potentially be present such as the age of individuals and the homogeneity of the sample population. Hence, the data used in the current dissertation may not be generalizable to a broader non-college sample or the general population. As discussed above, the age of participants from this and many other college samples could potentially be a limitation due to individuals' relatively young age and that many of them have been under the supervision and protection of guardians for most of their lives. Moreover, given the fairly homogeneous population of individuals who attend college the primary limitation of a college sample is the relative low level of variation in antisocial behaviors and substance use when compared to other possible samples such as clinical or incarcerated populations.

Future Research

Future research concerning the interactions between victimization (both property and personal), PTSD, and HRR is needed to better unpack the relationship that these factors have with each other and how these variables influence antisocial behavior, soft substance use, and hard substance use. First, many of the studies discussed in the first two chapters of the current dissertation used forensic samples. Hence, future research endeavors could use comparisons between forensic and non-forensic samples to better understand how these risk factors influence antisocial and substance use outcomes. Further, such comparative studies could also help to better understand the findings of the current dissertation concerning two-way interactions acting more as factors that reduce antisocial and substance use behaviors rather than increasing the risks of these behaviors in undergraduate students.

Second, future cross-sectional data collections, similar to the current dissertation should include questions that retroactively focus on trauma throughout the life course and the onset of PTSD (or PTSD like) symptoms that include a severity rating of each traumatic event, any symptoms that followed and recording HRR to stress upon time of survey. This approach with the addition of the HR measure could be implemented similar to life history calendars (Harris & Parisi, 2007). Although this approach has limitations given that memory recall can be less reliable in earlier stages of life, it has been shown to be a valuable and reliable option when longitudinal data collection is not an option (Harris & Parisi, 2007). Although the PTSD measure used, the PCL-C, is cited as an extremely reliable instrument (Weathers, Litz, Herman, Huska, & Keane, 1993) other than having an official diagnosis by psychiatrists, having other measures to capture a

wider array of symptoms and how each individual rate the severity of those symptoms would give more insight to researchers about what respondents are experiencing. This would allow researchers to better understand not only at what stage of development that the traumatic event occurred but also the perceived severity by the participant and any potential negative symptoms that emerged around the time of the events. In addition, this would allow the separation of victimization and non-victimization trauma to see if the severity of these life events is perceived differently and if different symptoms are associated with different traumas and if HRR is impacted differently. To enable comparability, in lieu of only open ended questions, an extensive list of general traumatic events could be supplied to participants. These traumatic events could range from common events such as the loss of an elderly family member to more severe events such as physical victimization. The respondents could allocate any of the events that they experienced into a table divided up into common life course stages (e.g. adolescence, teens, young adult, and adult) where each event from the trauma list could be recorded as many times needed in each section along with the number of times each event occurred and the self-perceived severity rating. This approach would be similar to the Revised Conflict Tactics Scale (CTS-2) but with a broader range for the severity scale instead of only the two categories of *minor* or *severe* (Straus, Hamby, Boney-McCoy & Sugarman, 1996). To broaden the response of severity the approach by Nylund and colleagues (2007) could be altered where a Likert scale was implemented to understand the probability that individuals felt they were at risk of certain types of victimization. However, the scale would be adjusted to reflect individuals' perception of severity rather than possibility of experiencing a certain type of victimization. Similarly, an extensive

list of negative symptoms could be included to be recorded in each life course stage as well similar to the traumatic events.

Overall, a longitudinal data collection approach with focused specific measures concerning trauma/victimization, PTSD, and HRR would be the most beneficial to better understanding the relationship between these three risk factors, antisocial behavior, and substance use. This approach removes the black box of etiological unknowns to each of the risk factors and would enable researchers to better understand how each risk factor influences each other as well as the antisocial outcomes. Further, by utilizing a longitudinal approach temporal aspects of the onset of different forms of antisocial behaviors could be better understood. For example, a longitudinal approach for several years can enable researchers to understand if general antisocial behaviors (e.g. acting out, aggression, or law breaking behaviors) emerge first or if these behaviors are linked to the onset of self-medicating through substance use.

In summary, the findings of the current dissertation suggest that the relationship between trauma, PTSD, and HRR with antisocial behavior and substance use appear to be more complicated than theoretically proposed in previous chapters. Thus, these findings suggest the need for further study to unpack how these risk factors influence antisocial behavior and substance use and etiological ordering of these relationships. Further, considering that there are differences between how the risk factors of victimization, PTSD, and HRR increased the propensity for antisocial behavior for males in the current dissertation but not females shows that gender differences need further attention as well. Specifically, the findings of the current dissertation showed that the presence of only two risk factors were associated with a reduction in antisocial behavior and substance use for

both males and females, but the inclusion of all three risk factors increased antisocial behavior for males only. Although these findings are counter intuitive and unexpected, unless these effects are restricted to the current data set, they show the diverse effects that trauma can have on individuals, and that gender differences are important. Thus, the understanding of how trauma influences individuals is important considering that the findings of the current dissertation suggest that the presence or absence of certain risk factors can decrease or increase the propensity for antisocial outcomes. In addition, the current dissertation also suggest that gender differences could be an important factor in how certain risk factors play a role in the propensity for antisocial behaviors.

In conclusion, the findings of the current dissertation support the need for a better understanding of the relationships between trauma (personal and property victimization), PTSD, and increased HRR and the possible link they share to the onset of antisocial and criminal behavior within the field of criminology. Understanding the effects and consequences of trauma are paramount for both the treatment and curtailment of associated negative symptoms and behaviors. Further, by understanding the consequences of trauma and associated risk factors that increase individuals' propensity for antisocial behavior it is possible to reduce the antecedent behaviors that could be related to an increased propensity for crime and criminal behavior.

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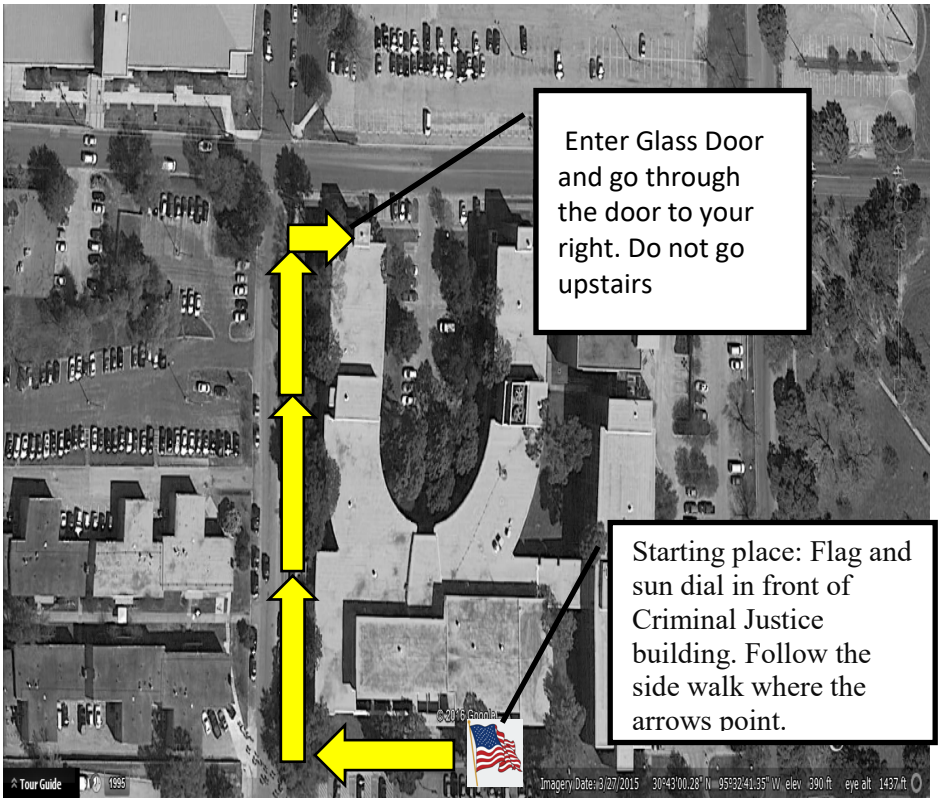
APPENDIX A


Enter Glass Door and go through the door to your right. Do not go upstairs
Starting place: Flag and sun dial in front of Criminal Justice building. Follow the side walk where the arrows point.
Tour Guide
Imagery Date: 3/27/2015

APPENDIX B

Email sent to all participants (areas highlighted to ensure participants would know this was the biological portion and they needed to sign up for it).

Hello, and thank you for agreeing to participate in the CJ Bio research project! Your involvement will help enable us to improve criminological research on how biology and the environment influence behavior.

As a reminder, here are the links to sign up for a time slot in our CJ Bio Lab, which is the 2nd part of the project (now that you've completed the survey in class). The lab will be open from 9 am to 12 noon Tuesday through Friday.

Please click on one of the links below and choose a time that works for you:
http://www.signupgenius.com/go/4090c44a9a82fabfd0-cjbiosocial8

We have just a few things we'd like to inform/remind you of:

1. Once you complete the lab, we will notify your instructor that your participation in the project is complete and extra credit will be awarded. Participation, however, is completely voluntary and if you at any point decide you would like to stop participating you are free to do so. Your instructor may offer you an alternative assignment, please contact him/her directly.
2. As mentioned, at the lab, you will also receive a koozie to show our appreciation!
3. The lab rooms are located in the basement below the faculty offices in rooms CL-26 and CL-35. There are signs posted throughout the CJ building to help you find the rooms but if you're having trouble, come see me in my office A208 and I (or one of the administrative assistants) will show you where to go.
4. You should expect the time you spend in the lab to take around 25-45 minutes.
5. If possible, please avoid consuming food, drink (water is okay), or tobacco 30 minutes before your scheduled lab time since this could affect the results.

Thank you so much! If you have any questions pertaining to your involvement in this research project, please do not hesitate to contact us.

From the CJ Bio Research Group!

APPENDIX C

- Students meet us in CL-26
  - Pull confirmation sheet and give them a clip board (Excel- Google Sheets)
  - Do not have them fill out yet (Fill out survey later in CL 35)
    - Number survey and envelope with their ID number from sheet
    - Record time on sheet
- Enter CL-26
  - Welcome and seat participant
  - Initial Saliva Sample – Offer a piece of gum for saliva sample
    - With gloves on, assemble and label tube with ID # (underline ID #)
    - Have them complete saliva sample at gray desk (until full or 3 minutes has passed)
    - Put in Pre-Stress Box
  - Neulog Sensors – make sure these a snug and on the pads of their fingers
    - Clean sensors
    - Clean participant skin
    - Heart Rate (right hand, pinky finger)
    - Skin Conductance (right hand, index and middle fingers)
  - Wait 3-5 minutes (Chat or don't - make them as comfortable as possible- use your judgement)
  - Hit record on computer (let run for 30 seconds) stop recording
  - Read stressor prompt (but don't tell them it is a stress inducer)
    - Start stop watch – This watch will continue until this student is completely done.
    - Remember 1:30 prompt (this is when they are about to start talking)
  - Take second HR and SC reading
    - Hit record, let run for 30 seconds, stop
  - At 2 minutes, start video recording with them holding their envelope, ID # facing out – (HR, SC, then camera)
    - Have them speak for 3 minutes (until camera timer reads 17:00)
  - Take final HR and SC (record, 30 sec, stop)
    - Save files as ID_pre, ID_test, and ID_post
  - Take symmetry photo – with envelope – remove anything from their head or face
    - Try to get their face as straight as possible

CL-26 Quick guide

1. Clip board
2. Saliva 1
3. Hook up and wait 3-5 minute
4. HR/SC1
5. Stress
6. HR/SC 2
7. Video
8. HR/SC 3
9. Picture

- Go to CL-35
  - DNA swab
    - Wear gloves
    - Write ID from envelope on swab sleeve
    - Swab cheek (x2)
    - Put sleeve in envelope and store
  - Hand scanner
    - Right and left
    - Rename file as ID#_L and ID#_R (ex. 34_R)
  - Have them trace their hands (write ID # on paper) - WASTE TIME
  - Tell them there is one more thing and encourage them to wait as long as they can or till stop watch reads 25:00, whichever comes first
  - Have them complete Health Survey
  - Begin saliva sample #2
    - With gloves on, assemble and label tube
    - Have them complete saliva sample at gray desk
    - Record time ended- when sample is done
    - Record stop watch time – AS CLOSE TO ABOVE TIME AS POSSIBLE
    - Put in Post-Stress Box

CL-35 Quick Guide

1. Swab
2. Right hand scan
3. Left hand scan
4. Right hand trace
5. Left hand trace
6. Saliva 2

APPENDIX D

Antisocial Behavior

How often, in the past year, were you involved in…

1. Purposely damaging or destroying property belonging to your parents or other family members?
2. Purposely damaging or destroying property belonging to your employer?
3. Purposely damaging or destroying other property that did not belong to you, not counting family, or work property?
4. Stealing or trying to steal a motor vehicle such as a car or motorcycle?
5. Stealing or trying to steal something worth more than $50?
6. Knowingly buying, selling or holding stolen goods or tried to do any of these things?
7. Purposely setting fire to a building, a car, or other property or trying to do so?
8. Carrying a hidden weapon other than a plain pocket knife?
9. Stealing or trying to steal things worth $5 or less?
10. Attacking someone with the idea of seriously hurting or killing that person?
11. Gang fights?
12. Selling marijuana or hashish? ("POT", "GRASS", "HASH")
13. Stealing money or other things from your parents or other members of your family?
14. Stealing money, goods, or property from the place where you work?
15. Having or trying to have sexual relations with someone against their will?
16. Hitting or threatening to hit one of your parents?
17. Hitting or threatening to hit your supervisor or other employee?
18. Hitting or threatening to hit anyone else (other than parents, persons at work)?
19. Selling hard drugs such as heroin, cocaine, and LSD?
20. Trying to cheat someone by selling them something that was worthless or not what you said it was?
21. Buying or providing liquor for a minor?
22. Using force or strongarm methods to get money or things from people?
23. Being drunk in a public place?
24. Stealing or trying to steal things worth between $5 and $50?
25. Breaking or trying to break into a building or vehicle to steal something or just to look around?
26. Snatching someone's purse or wallet or picked someone's pocket?

APPENDIX E

Substance use

How often, in the past year, have you…

1. Used alcoholic beverages, beer, wine, hard liquor?
2. Used tobacco?
3. Used marijuana or hashish? (GRASS, POT, HASH)
4. Used hallucinogens, LSD, Acid, peyote, mescaline, Psilocybin? (PSYCHEDELICS)
5. Used tranquilizers such as Valium, Librium, Thorazine, Miltown, Equanil, Meprobamate, etc.?
6. Used amphetamines, uppers, ups, speed, pep pills or bennies? (DEXEDRINE, BENZEDRINE, WHITES, DIET PILLS, DEXIES, DEXAMYL, STP)
7. Used barbiturates, downers, reds, yellows, blues? (RAINBOWS, GOOF BALLS, PHENOBARBITAL, PRESCRIPTION SLEEPING PILLS, SECONALS, YELLOW JACKETS OR NEMBUTAL)
8. Used codeine?
9. Used heroin? (HORSE, H, SKAG, SMACK, JUNK)
10. Used crack?
11. Used cocaine, or coke, other than crack?
12. Used inhalants glue, paint, nail polish, or aerosol sprays?
13. Used angel dust or PCP? (PHENCYCLIDINE, SERNYLAN, CRYSTAL, PEACE HILL, HOG, SHEETS)

APPENDIX F

Victimization

How many times in the past year:

Property Victimization Items:

1. Has something been taken directly from you or an attempt made to do so by force or threatening to hurt you?
2. Has your car, motorcycle or bicycle been stolen or an attempt made to do so?
3. Have any of your things been damaged on purpose, such as car or bike tires slashed or books and clothing ripped up?
4. Has your pocket been picked or your purse or wallet snatched or an attempt made to do so?

Personal Victimization Items:

1. Has someone such as a date or friend pressured or pushed you to do more sexually than you wanted to do?
2. Have you been sexually attacked or raped or an attempt made to do so?
3. Have you been beaten up by your mother, stepmother, father or stepfather?
4. Have you been beaten up or threatened with being beaten up by someone other than your mother or father?
5. Have you been attacked with a weapon, such as a gun, knife, bottle or chair by someone other than your mother or father?

APPENDIX G

Post-traumatic Stress Disorder

In the past year have you ever felt…

1. Have you had upsetting thoughts or images about the trauma that came into your head when you didn't want them to?
2. Have you been having bad dreams or nightmares about the trauma?
3. Have you had the experience of reliving the trauma, acting or feeling as if it were happening again?
4. Have you been very EMOTIONALLY upset when reminded of the trauma (includes becoming very scared, angry, sad, etc.)?
5. Have you been having PHYSICAL reactions (for example, break out in a sweat, heart beats fast) when reminded of the trauma?
6. Have you been trying not to think about or have feelings associated with the trauma?
7. Are there any important parts about the trauma that you still cannot remember?
8. Have you found that you are not interested in things you used to enjoy doing?
9. Have you felt distant or cut off from others around you?
10. Have you felt emotionally numb (for example, feel sad but can't cry, unable to have loving feelings)?
11. Have you felt that any future plans or hopes have changed because of the trauma (for example, will have no career, marriage, children, or long life)? DO NOT INCLUDE MOVING.
12. Have you been having problems falling or staying asleep?
13. Have you been more irritable or having outbursts of anger?
14. Have you been having more difficulty concentrating (for example, drift in and out of conversations, lose track of story on television, difficulty in remembering what you have read)?
15. Have you been overly alert (for example, checking to see who is around you, uncomfortable with your back to a door, etc)?
16. Have you been jumpier, more easily startled (for example, when someone walks up behind you)?

APPENDIX H

Self-Control

1. I often act on the spur of the moment without stopping to think
2. I don't devote much thought and effort to preparing for the future
3. I often do whatever brings me pleasure here and now, even at the cost of some distant goal
4. I'm more concerned with what happens to me in the short run than in the long run
5. I frequently try to avoid projects that I know will be difficult
6. When things get complicated, I tend to quit or withdraw
7. The things in life that are easiest to do bring me the most pleasure
8. I dislike really hard tasks that stretch my abilities to the limit
9. I like to test myself every now and then by doing something a little risky
10. Sometimes I will take a risk just for the fun of it
11. I sometimes find it exciting to do things for which I might get in trouble
12. Excitement and adventure are more important to me than security
13. If I had a choice, I would almost always rather do something physical than something mental
14. I almost always feel better when I am on the move than when I am sitting and thinking
15. I like to get out and do things more than I like to read or contemplate ideas
16. I seem to have more energy and a greater need for activity than most other people my age
17. I try to look out for myself first, even if it means making things difficult or other people
18. I'm not very sympathetic to other people when they are having problems.
19. If things I do upset people, it's their problem not mine
20. I will try to get the things I want even when I know it's causing problems for other people
21. I lose my temper pretty easily
22. Often, when I'm angry at people I feel more like hurting them than talking to them about why I am angry
23. When I'm really angry, other people better stay away from me
24. When I have a serious disagreement with someone, it's usually hard for me to talk calmly about it without getting upset

APPENDIX I

Delinquent Peer Behavior

Think about your friends. In the past year how many of your friends have…

1. Cheated on their income tax
2. Purposely damaged or destroyed property
3. Used marijuana or hashish
4. Stole something (less than $5)
5. Hit or threatened to hit someone without any reason
6. Used Alcohol
7. Broke into a vehicle
8. Sold hard drugs (heroin, cocaine or LSD)
9. Stolen something (more than $50)
10. Suggested you do something that's against the law
11. Gotten drunk once in a while
12. Used prescription drugs when there was no medical need
13. Sold or given alcohol to kids under 18

VITA

RICHARD H. LEWIS

EDUCATION

Ph.D. Criminal Justice, expected May 2019
Sam Houston State University
Huntsville, TX, 77341
Dissertation: *The effects of victimization and post-traumatic stress disorder on antisocial behavior*
Chair: Dr. Danielle Boisvert

M.S. Biology, 2012
Sam Houston State University
Huntsville, TX, 77341
Thesis: *The effects of fluctuating asymmetry on female choice and sperm quality in the Largespring Gambusia, (Gambusia geiseri).*
Chairs: Dr. Raelynn Deaton; Dr. Anne Gaillard

B.S. Biology and Minor in Statistics, 2008
Sam Houston State University
Huntsville, TX, 77341

PEER REVIEWED PUBLICATIONS

Lewis, R. H., Connolly, E. J., Boisvert, D. L., & Boutwell, B. B. (2019) A Behavioral Genetic Analysis of the Co-Occurrence of Psychopathy and Criminal Behavior. (MS ID: 022019-102) for consideration for publication in the *Journal of Contemporary Criminal Justice* special issue on biosocial criminology.

Kavish, N., Boisvert, D., Wells, J., Lewis, R., Cooke, E., Woeckener, M., & Armstrong, T. (2019). On the associations between indicators of resting arousal levels, physiological reactivity, sensation seeking, and psychopathic traits. *Personality and Individual Differences*, *141*, 218-225.

Woeckener, M., Boisvert, D. L., Cooke, E. M., Kavish, N., Lewis, R. H., Wells, J., ... & Harper, J. M. (2018). Parental rejection and antisocial behavior: the moderating role of testosterone. *Journal of Criminal Psychology*, *8*(4), 302-313.

Cooke, E. M., Armstrong, T., Boisvert, D., Wells, J., Lewis, R. H., Hughes-Stamm, S., & Gangitano, D. (2018). The relationship between the MAOA-uVNTR polymorphism, delinquent peer affiliation, and antisocial behavior with a consideration of sex differences. *Psychiatric Quarterly*, 1-13.

Connolly, E. J., Lewis, R. H., & Boisvert, D. (2017). The effect of socioeconomic status on delinquency across urban and rural contexts: Using a genetically informed design to identify environmental risk. *Criminal Justice Review,* 0734016817724200.

Boisvert, D., Wells, J., Armstrong, T. A., & Lewis, R. H. (2017). Serotonin and self-control: A genetically moderated stress sensitization effect. *Journal of Criminal Justice*.

Wells, J., Armstrong, T., Boisvert, D., Lewis, R. H., Gangitano, D., & Hughes-Stamm, S. (2017). Stress, genes, and generalizability across gender: Effects of MAOA and stress sensitivity on crime and delinquency. *Criminology*, 55, 548–574.

Boisvert, D., Wells, J., Armstrong, T., Lewis, R. H., Woeckener, M., & Nobles, M. R. (2017). Low resting heart rate and stalking perpetration. *Journal of Interpersonal Violence*, 0886260517698823.

Boutwell, B. B., Nedelec, J. L., Lewis, R. H., Barnes, J. C., & Beaver, K. M. (2015). A behavioral genetic test of the evolutionary taxonomy. *Evolutionary Psychological Science*, *1*(4), 241-250.

Boutwell, B. B., Franklin, T. W., Barnes, J. C., Beaver, K. M., Deaton, R., Lewis, R. H., ... & Petkovsek, M. A. (2013). County-level IQ and fertility rates: A partial test of differential-K theory. *Personality and Individual Differences*, *55*(5), 547-552.

Sanchez, J. L., Boutwell, B. B., Hamontree, S. T., Garrett, G. P., Lewis, R. H., Ragan, A. N., ... & Haynes, R. D. (2014). Reproductive characteristics of two Gambusia congeners in west Texas. *The Southwestern Naturalist*, *59*(3), 438-441.

Sanchez, J. L., Stoops, S. B., Allan, N. L., Cureton, J. C., Garrett, G. P., Kroll, C. W., ... & Deaton, R. (2013). Current Distribution of the Introduced Largespring Gambusia, Gambusia geiseri, In Texas. *The Southwestern Naturalist*, *58*(4), 497-502.

Lewis, R. H., Allan, N. L., Stoops, S. B., Garrett, G. P., Kroll, C. W., West, J., & Deaton, R. (2013). Status of the endangered Pecos gambusia (Gambusia nobilis) and Comanche Springs pupfish (Cyprinodon elegans) in Phantom Lake Spring, Texas. *The Southwestern Naturalist*, *58*(2), 234-238.

Cureton II, J. C., Martin, R. E., Lewis, R. H (L – typo in publication)., Stoops, S. B., & Deaton, R. (2011). Effects of a trematode infestation on body condition, reproduction and mating behaviors in a livebearing fish. *Behaviour*, *148*(8), 967-984.

BOOK CHAPTERS

Boutwell, B. B. & Lewis, R. H. (2014) Some Kind of Madness: The Biosocial Origins of Intimate Partner Violence. In Beaver, K. M., Barnes, J. C., & Boutwell, B. B. (Eds.). *The Nurture Versus Biosocial Debate in Criminology: On the Origins of Criminal Behavior and Criminality*. (pp. 269-282) SAGE Publications.

PAPERS UNDER REVIEW

Armstrong, T. A., Boisvert, D., Wells, J., Lewis, R. H., Woeckener, M., & Cooke, E. "Are Self-Control and Psychopathy the Same Thing? Conceptualization and Measurement with the Grasmick Self-Control Scale and Levenson Self-Report Psychopathy Scale" Under review at *The Journal of Quantitative Criminology.*

Deaton-Haynes, R., Raven, A. J., Felder, C. D., Gaides, L. A., Lewis, R. H., Martin, R. E., & Rosado, S. K. "Female age-specific mate preference for a male's potential for resources" Under review at *Evolution, Mind, and Behavior.*

CURRENT RESEARCH

Lewis, R. H., Boisvert D., Franklin, D., & Gangitano, D. "A Proposed Psychophysiological Model of "The Relationship Between Victimization & Post-traumatic Stress Disorder: The Influence Of Acetylcholinesterase".

Lewis, R. H., Franklin, T. W., Boutwell, B. B., Barnes, J. C., & Beaver, K. M. "Attractiveness, perception, and the criminal justice system".

Lewis, R.H., & Pyrooz, D. "The effects of intelligence on gang membership and position within gang".

Cooke, E., Lewis, R. H., Boisvert, D., & Armstrong, T. "The relationship between psychopathy, proactive, and reactive aggression on rape myth acceptance across gender".

CONFERENCE PRESENTATIONS

Lewis, R. H., Boisvert, D., Boutwell, B. B., Barnes, J. C., & Beaver, K. M. (2016). "A Behavioral Genetic Analysis of the Co-Occurrence of Psychopathy and Criminal Behavior". Paper presented at the annual meeting of the American Society of Criminology, New Orleans, LA.

Lewis, R. H., Armstrong, T. A., Wells J., & Boisvert, D. (2015). "A Test of the Genetic Substrate of Psychopathy". Paper presented at the annual meeting of the American Society of Criminology, Washington, D.C.

Lewis, R. H., Boutwell, B. B., Franklin, C., Barnes, J. C. & Beaver, K. M. (2014). "The Relationship Between Genetic Risk Factors for Victimization & Criminal Justice Processing". Paper presented at the annual meeting of the American Society of Criminology, San Francisco, CA.

Lewis, R. H., Franklin, T. W., & Boutwell, B. B. (2013). "Eye of the Beholder: An Evolutionary Study Concerning the Role of Attractiveness in Criminal Justice Processing". Paper presented at the annual meeting of the American Society of Criminology, Atlanta, GA.

AWARDS AND HONORS

Recipient of "Who's Who Among Students", Sam Houston State University - Spring 2015

Recipient of Sam Houston State University Excellence in Writing Award, Sam Houston State University – Spring 2015

Recipient of the Summer Research Fellowship, Sam Houston State University - 2015

Recipient of the Research Assistantship, College of Criminal Justice Sam Houston State University - 2014-2015

Recipient of 1st place Oral Presentation Award (Best Presentation), Graduate Student Research Exchange Symposium, Sam Houston State University - Spring 2014

Recipient of the Summer Research Fellowship, Sam Houston State University - 2013

Recipient of the Research Assistantship, College of Criminal Justice, Sam Houston State University - 2012-2013

Recipient of 1st Place Poster Award, Department of Biological Sciences, Graduate Student Symposium, Sam Houston State University - Spring 2011

Recipient of 1st Place Oral Presentation Award, Graduate Student Exchange Symposium, Sam Houston State University - Spring 2010

Recipient of 1st Place Oral Presentation Award, Graduate Student Exchange Symposium, Sam Houston State University - Spring 2009

Recipient of 2nd Place Poster Award, Tri Beta Biological Honor Society, Sam Houston State University - Spring 2008

COURSES TAUGHT

Terrorism (Residential & Online) – Spring 2019

Graduate Level Social Statistics (Residential) – Fall 2018

Introduction to Criminal Justice (Residential) – Fall 2018

Film & Society (Residential) – Spring 2018

Criminology (Two Sections) (Residential) – Fall 2017

Understanding Human Behavior (Online) – Summer II 2017

Understanding Human Behavior (Residential) – Fall 2016

Gender and Crime (Online) – Summer II 2016

Introduction to Research Methods (Residential) – Spring 2016

Criminology (Residential) – Fall 2015

Criminology (Online) – Summer 2014

INVITED PRESENTATIONS

Guest Lecture for the Honors Class -Film and Society- where we discussed the bad science concerning psychopathy, criminal behavior, and profiling concerning the film *Copycat* - Fall 2017

Guest Lecture for the Honors Class -Film and Society- where we discussed sociological and human behavioral factors concerning the cold war, terrorism, and 9/11 with the film *Red Dawn (2010 remake)* – Spring2017

Guest Lecture for Criminology, Sam Houston State University, Overview of Psychopathy and Biosocial Science - Spring 2016

Guest Lecture for the Honors Class -Film and Society- where we discussed the bad science of biosocial science, and ethics of policing and law concerning the film *Minority Report* - Fall 2016

Guest Lecture for the Honors Class -Film and Society- where we discussed the bad science of psychopathy, criminal behavior, and intelligence concerning the film *Silence of the Lambs* - Fall 2015

Guest Lecture for Criminology, Sam Houston State University, Overview of Psychopathy and Biosocial Science - Spring 2015

Guest Lecture for Methodology, Biosocial Methodology, Sam Houston State University - Spring 2015

Guest Lecture for Criminology, Overview of Biosocial Science, Sam Houston State University - Spring 2015

Guest Lecture for Criminology, Introduction to Evolutionary Psychology and Biosocial Methods, Sam Houston State University - Spring 2015

Guest Lecture for Methods, Introduction to Evolutionary Psychology and Biosocial Methods, Sam Houston State University - Summer 2014

Guest Lecture for Woodville Elementary School, TX, Sun Fish Parasitology, Ecology, and Physiology - Fall 2013

PROFESSIONAL EXPERIENCE

Collection and processing of cortisol and testosterone from saliva
Entering coded data from surveys
Collection of cheek swab DNA samples
DNA extraction of cheek swab samples
DNA real time PCR of extracted DNA samples
Genotyping of DNA samples
Basic lab cleaning and care techniques
Conducting wet lab based classes as a Lab Teaching Assistant
Ordering lab equipment for a large scale data collection
Managing biological laboratory data collection
Co-Lead on large scale survey and biological data collection

PUBLICATIONS IN NON-CRIMINOLOGY AND CRIMINAL JUSTICE FIELDS

Sanchez, J. L., Boutwell, B. B., Hamontree, S. T., Garrett, G. P., Lewis, R. H., Ragan, A. N., ... & Haynes, R. D. (2014). Reproductive characteristics of two Gambusia congeners in west Texas. *The Southwestern Naturalist*, *59*(3), 438-441.

Sanchez, J. L., Stoops, S. B., Allan, N. L., Cureton, J. C., Garrett, G. P., Kroll, C. W., ... & Deaton, R. (2013). Current Distribution of the Introduced Largespring Gambusia, Gambusia geiseri, In Texas. *The Southwestern Naturalist*, *58*(4), 497-502.

Lewis, R. H., Allan, N. L., Stoops, S. B., Garrett, G. P., Kroll, C. W., West, J., & Deaton, R. (2013). Status of the endangered Pecos gambusia (Gambusia nobilis) and

Comanche Springs pupfish (Cyprinodon elegans) in Phantom Lake Spring, Texas. *The Southwestern Naturalist*, *58*(2), 234-238.

Cureton II, J. C., Martin, R. E., Lewis, R. H (L – typo in publication)., Stoops, S. B., & Deaton, R. (2011). Effects of a trematode infestation on body condition, reproduction and mating behaviors in a livebearing fish. *Behaviour*, *148*(8), 967-984.

PROFESSIONAL SERVICES

Graduate Peer Mentor, Department of Criminal Justice and Criminology, SHSU - Spring and Fall 2016

CAT exam grading for a grant that evaluates the critical thinking of students before and after specific lecture classes, SHSU – Fall 2010 to Spring 2017

Meeting with Dean of Graduate Studies applicants, SHSU - Spring 2016

Leading College of Criminal Justice and campus wide tours for visiting program auditors - Spring 2016

Student Member of the Beto Lecture Series Committee, College of Criminal Justice, SHSU - Fall 2015

Chair of the committee on Organizational Standing, CJ Graduate Student Organization, College of Criminal Justice, SHSU - Fall 2013 and Spring 2014

RESEARCH AND TEACHING INTERESTS

Gender and Crime
Victimology
Criminological Theory
Life Course Criminology
Research Methods
Statistics
Understanding Human Behavior
Biosocial Criminology
Behavioral Genetics
Gene-Environment Interactions
Genetic/Biological Correlates of Antisocial Behavior
Co-Evolution between Human and Canines in Response to Stress
Psychopathy
Post-Traumatic Stress Disorder