The Roles of Respiratory Sinus Arrhythmia Reactivity and Intimate Partner Violence in Childhood as Predictors of Adolescent Risky Behavior

Charlotte Heleniak

A thesis

submitted in partial fulfillment of the

requirements for the degree of

Master of Science

University of Washington 2013

Committee:

Lynn Fainsilber Katz

Wendy Stone

Program Authorized to Offer Degree:

Psychology

University of Washington

Abstract

The Roles of Respiratory Sinus Arrhythmia Reactivity and Intimate Partner Violence in Childhood as Predictors of Adolescent Risky Behavior

Charlotte Heleniak

Chair of the Supervisory Committee:
Research Professor Lynn Fainsilber Katz, PhD
Psychology Department

Adolescence is a time of experimentation, associated with heightened engagement in risky behaviors such as alcohol and drug use and unsafe sexual behavior. Recent literature suggests that individual differences in physiological stress reactivity and exposure to intimate partner violence (IPV) in childhood may predict problem behavior in adolescence (Silk et al. 2003; Evans et al. 2008). This study aims to examine whether respiratory sinus arrhythmia reactivity (RSAR) in middle childhood predicts risky behaviors in adolescence, and to determine whether the presence of intimate partner violence in childhood moderates this relation. Forty-three mother and child dyads were recruited as part of a longitudinal study of children at risk for behavior problems. Children's RSAR was assessed during an interpersonal stressor task when they were 9 years old on average. At this time point, mothers were assessed for the experience of intimate partner violence. When these children reached age 16, they completed questionnaires about their risky behaviors. Results indicated that RSA augmentation to interpersonal stress in childhood predicted adolescent risky behaviors. Intimate partner violence was not significantly related to a composite measure of adolescent risky behaviors, but did significantly strengthen the relation between childhood RSA

augmentation to interpersonal stress and problems from alcohol use during adolescence. These results suggest that RSA augmentation to interpersonal stress may be an individual differences factor that sets some children at risk for problems with alcohol use in adolescence. The presence of intimate partner violence in their homes compounds this risk. Future research should investigate interventions that target emotion regulation skills in children exposed to IPV to prevent these children from exhibiting risky behavior in adolescence.

The greatest threats to the wellbeing of young people in industrialized countries come from preventable and often self-inflicted causes, including automobile and other accidents as well as drug and alcohol use, and sexual risk-taking (Blum & Nelson-Mmari 2004). Focused public policy and healthcare in the last two decades have made tremendous strides in the treatment of serious and chronic disease in this age group. However, equal progress has not been achieved in the reduction of the risky behaviors that endanger young people the most (Centers for Disease Control and Prevention, 2010). A recent national survey on youth risk behaviors revealed that approximately 40% of high school students drank alcohol within the last month, 40% had tried illegal drugs in their lifetime, and 12% had not used a condom the last time they engaged in sexual intercourse (Eaton et al., 2012). While these behaviors vary by gender, overall problem behaviors are prevalent in both genders during adolescence (MacArthur et al., 2012).

Youth who engage in risky behaviors in adolescence begin a trajectory that leads to a wide range of negative consequences. Young people who engage in risky behaviors in adolescence are more likely to become delinquent in later adolescence, commit suicide, and have an early pregnancy (Bonomo et al. 2004; Sher & Zalsman 2005). Furthermore, this subset of adolescents is more likely to develop adult psychopathology such as substance use disorders, major depressive disorder, and antisocial personality disorder (McGue & Iacono 2005). If children vulnerable to later risky behavior are identified early, it may be possible to alter their path along the teen problem behavior trajectory associated with harm and suffering during adolescence and into adulthood. For this reason it is important to uncover risk factors that set children up to initiate risk behavior as adolescents.

To date, a range of characteristics common to the adolescents using substances and engaging in sexual risk behavior have been examined. Biological factors from genetic influences to neurodevelopmental processes underlying deficits in the ability to effectively regulate behavior, emotion, and physiology, have been implicated in risky behavior in adolescence (Luna et al., 2013; McGue, 1999; Romer et al., 2011; Silk, Steinberg, & Morris, 2003). Stressful environmental circumstances also appear to play a role in the development of adolescent problem behavior, from the experience of poverty and racial discrimination to low parental monitoring and exposure to interparental violence and aggression (Beauchaine et al., 2008; Dishion & McMahon 1998; Mccubbin et al., 1985; Jessor, 1993; Vasilev et al. 2009). These findings suggest that, when living in an intensely stressful environment, a child with compromised capacity for emotion regulation may be at particularly high risk for engaging in problem behavior in adolescence. In order to investigate this assumption, we will first examine the role of emotions and a biological index of their regulation before elaborating on the role of exposure to intimate partner violence as childhood vulnerability factors for adolescent risky behavior.

Emotions, Emotion Regulation and Adolescent Problem Behavior

Adolescence is an important time to regulate emotions because it is a developmental period characterized by heightened emotionality. Pubertal onset is accompanied by an increase of hormones that contribute to the strength of adolescent emotions (Dahl, 2004). Peers provide new social contexts which have been shown to increase an array of emotions for adolescents, including jealousy, competition, excitement and worry (Larson & Asmussen 1991). The parts of the brain most sensitive

to emotional reactivity – the subcortical limbic regions such as the nucleus accumbens and amygdala – develop at a faster rate in adolescence than the part of the brain responsible for inhibitory control, the prefrontal cortex (Casey et al., 2008; Casey & Jones 2010; Steinberg, 2010)). As a result of these changes in level and intensity of emotions, youth may be making decisions based on feelings rather than logic (Gardner & Steinberg, 2005; Reyna & Farley 2006). Together, these findings emphasize the greater need for effective regulation of emotions during adolescence.

The adolescents who are less able to effectively regulate their emotions are more likely to endorse substance use disorders and sexual risk behavior (Cooper, Frone et al., 1995; Tapert et al., 2001; Wilens et al., 2013; Wills et al., 2011). While the stability of emotion regulation across the lifespan is not yet understood, recent evidence indicates that individual differences in emotion regulation in childhood are predictive of later emotion regulation abilities (Beauchaine et al., 2007; Gottman & Katz, 2002; Gross, 1998). Effective emotion regulation in childhood supports the development of later interpersonal and intrapersonal skills essential to navigating adolescence and adulthood (Southam-Gerow & Kendall 2002). However, the inability to manage affective reactions to stressful situations in childhood increases the likelihood of emotional and behavior problems later in life (Cole et al. 1996; Garber et al. 1995; Hilt et al. 2011; Kring & Sloan 2010; McLaughlin et al. 2011; Rydell et al. 2003; Silk et al. 2003; Spinrad et al. 2006). Children who exhibit deficits in emotion regulation abilities may become the adolescents whose emotional reactions to stress contribute to their risk behavior.

One way emotion regulation has been indexed is with respiratory sinus arrhythmia (RSA), a measure of the deployment of the parasympathetic nervous system

(Katona & Jih 1975)**.** Considerable evidence suggests that the ability to flexibly engage and retract the parasympathetic nervous system in response to stressful situations is an index of the ability to effectively regulate emotions (Porges 1995, 1997, 1998, 2001, 2003). Baseline RSA is a direct measurement of the parasympathetic nervous system activation, and is thought to index the general ability to regulate emotions. Children who have high baseline RSA are more likely to be socially-engaged, express empathy, and be buffered from risk of psychopathology associated with stressful family environments (Fox & Field 1989; Fabes et al. 1993; Katz & Gottman 1997). While baseline RSA indicates general emotion regulation ability, RSA reactivity is thought to reflect the regulation of emotion in response to a specific situation. A decrease in RSA (i.e., RSA withdrawal) is characteristic of the fight or flight response, where resources are freed up so one can focus on the threat at hand. Recent research suggests that RSA withdrawal is thought to be an adaptive response in situations where interpersonal threat may necessitate an immediate response to the environment (Leary & Katz, 2004). An increase in RSA (i.e., RSA augmentation) is characteristic of the dampening of physiological and emotional arousal. During intellectual challenge in which hyperarousal may distract from the task at hand, RSA augmentation may be an adaptive response (Obradovic et al. 2011). RSA withdrawal during stress in middle childhood has been identified as an early predictor of later emotion regulation, and a buffer against the development of internalizing and externalizing behaviors (Calkins, 1997; Calkins, Graziano, & Keane, 2007; Gentzler et al., 2009; Gottman & Katz, 2002). Yet despite the links between RSA reactivity and externalizing behaviors and emotion dysregulation, factors highly correlated with adolescent risky behaviors, no research has been published to date

examining the role of RSA reactivity in middle childhood as a predictor of adolescent risky behavior. This is the first study to evaluate RSA reactivity during stress in middle childhood and its role in the prediction of risky behaviors in adolescence.

Intimate Partner Violence and Adolescent Problem Behavior

One particular challenge to a child's ability to effectively regulate his or her behavior and emotions is exposure to intense environmental stressors. Of the many environmental stressors that children are exposed to, we chose to examine IPV because of its negative effect on children and its high incidence rate in the US. Children whose mothers are subjected to IPV are more likely to demonstrate drinking problems, behavioral problems, delinquency and aggression in adolescence (Jaffe et al., 1986; Miller, 1989; Sternberg et al., 2006). This is a similar constellation of problems exhibited by children with deficits in emotion regulation (Beauchaine et al., 2007). In fact, research studies have observed that the functioning of the autonomic nervous system, which serves a prominent role in emotion regulation, is more likely to be compromised in in children exposed to IPV (Margolin & Vickerman 2011).

It is possible that children who both exhibit limited emotion regulation capabilities and who are exposed to IPV may be particularly likely to go on to engage in risky behavior in adolescence. Estimates suggest that between 3 and 17.8 million children are exposed to intimate partner violence each year (Carlson, 1984; Straus, 1992; Holden et al., 1998). It is important to identify which of these children go on to engage in risky behavior given that IPV is present in many homes across the country, with some estimates suggesting that approximately 22% of women have been physically assaulted by a current or former partner (Tjaden & Thoennes 2000).

The Current Study

Early identification of children who are predisposed to engaging in risky behaviors as adolescents could inform targeted prevention strategies. Evidence suggests that children who are unable to flexibly regulate their physiological reactions to stressful situations may have poor emotion-regulation abilities, and are at high risk for the development of internalizing disorders, externalizing disorders, and poor self-regulation in childhood and adolescence (Hinnant & El-Sheikh, 2009; Beauchaine et al., 2007; Calkins, Graziano, & Keane, 2007). If these children are already living in a stressful home environment, risk for later problem behavior may be heightened (Evans et al., 2008). This study aims to discern which children are at risk for developing problem behaviors in adolescence based on a physiological index of emotion regulation, and whether exposure to IPV enhances the relationship between poor emotion regulation and risky behaviors. Our first hypothesis is that high reactivity to stress in middle childhood will predict risky behaviors in adolescence. In particular, we predict that children who show high augmentation to interpersonal stress, when RSA withdrawal would be expected, will be display emotion regulation issues that predispose them to problems in adolescence. Our second hypothesis is that this relation will be strengthened by exposure in childhood to IPV.

Methods

Participants

Participants were originally recruited as part of a longitudinal study designed to evaluate the relation between family functioning and conduct problems in children. Preschool-aged children (Mean = 4.43, *SD* = .51, range = 4-6 years) and their families were recruited for participation through preschools, newspaper announcements, and offices of pediatricians and pediatric dentists. Recruitment brochures and advertisements targeted two-parent families who have a 4-6 year old child, and families were offered $150 for their time. Inclusion into the study was determined by children's scores on a telephone version of the Eyberg Child Behavior Inventory (ECBI) administered to mothers (Robinson et al., 1980). Following established cutoffs, children who received a score of 11 or higher on the ECBI were assigned to a conduct-problem group (CP) and children who received a score of seven or lower were assigned to the control group. Groups were case-matched for age, socioeconomic status, and neighborhood. With the exception of one child, groups were also case-matched for gender. Sixty-five families had children who met criteria for the CP group (41 male, 24 female), and 65 families had children who met criteria for the control group (40 males, 25 females). Sixty-two percent of the sample was comprised of boys (N = 81), which is generally consistent with the higher prevalence rate of conduct problems in boys. The majority of the sample (87.7 percent) identified themselves as White, with the remainder of the sample identifying themselves as African-American (5.4 percent), Asian (4.6 percent), Hispanic/Latino (1.5 percent), and biracial or multiracial (.8 percent).

Families were recontacted four years later when children were on average 9.03 years old (SD = .44, range = 8–10 years). Of the original 130 families, 12 (nine percent) were not eligible to participate because of the age of the children overlapping with the

target age for the next assessment point in the study. Of the remaining 118 eligible families, 65 percent participated in the Time 2 procedures and had complete data on measures used for the current investigation (N = 82). Fifty-four (66.85%) of the children in this subset were boys, and the average age was 9.03 years (*SD* = .44, range = 8-10 years). The majority of the sample (89.9 percent) identified themselves as White, 5.1 percent were biracial or multiracial, and 5.1 percent were African-American.

Families were once again contacted seven years later when children were on average 16.15 **years old** (*SD* = .63*,* range = 14-17 years). Sixty-eight percent of the original sample of families participated in Time 3 (n = 88, 65% boys). Ethnicity was largely White (91.9%) with 3.5% of the sample African-American and 4.6% biracial or multiracial. Average household income of the final sample was approximately $79,000 and ranged from under $10,000 to over $100,000**.** The majority of parents, 81 percent of mothers and 74 percent of fathers, had completed an undergraduate college education.

Participants were compared across time points on key demographic variables to test whether there were systematic effects of participant attrition. A series of t-tests were conducted to assess whether families who dropped out of the study differed significantly from the families that remained in the study at Time 2 and 3. These analyses found that families who participated did not significantly differ from families that dropped out in their level of household income ($t(1, 50) = .35$, p = NS), ethnic background ($t(1,120) = .57$, p = NS), child age ($t(1, 127) = -.41$, p = NS), conduct group assignment ($t(1, 127) = .64$, p = NS), mother's depressive symptoms ($t(1,126) = -.71$, p = NS), and child total behavior problems ($t(1, 126) = -.914$, p = NS). Child gender was the only construct that

was significantly affected by attrition ($t(1, 127) = -1.014$, $p = .04$), with girls dropping 65% from 49 to 17 and boys dropping 57% from 81 to 35.

Because the current study aims to examine the relation between emotion regulation in middle childhood and behavior problems in adolescence, only data from families who participated at Times 2 and 3, when children were on average ages 9 and 16 respectively, were included. The final sample comprised 43 mother-child dyads, with 51% of the sample in the control group and 49% in the at risk group. The families that participated in both of these time points were 82% Caucasian, 6% African American, 6% Multiracial with 2% identifying as Latino. The procedures and measures used at each of these time points are described below.

Time 2

Procedures.

At Time 2, mothers and their children participated in a lab visit in which mothers completed questionnaires and children participated in a baseline and peer provocation paradigm while RSA was measured.

Baseline Condition.

Baseline RSA was obtained over a 2-minute period. Electrodes were placed on the child at the beginning of this lab visit. After making a light abrasion with Omni-prep solution (Omni International, Marietta, GA) to ensure a clear signal, five Beckman silver chloride electrodes (Beckman Coulter, Fullerton, CA) were placed on the children's chest to measure cardiac interbeat interval. Beckman electrolyte (Beckman Coulter, Fullerton, CA) was used to facilitate conductivity of electrical signals. Electrocardiogram data were

collected using Coulbourn bioamplifiers (Coulbourn Instruments, Allentown, PA). The electrocardiogram waveform was digitized at 128 Hz using a MetraByte A-D converter (Keithley Instruments, Cleveland, OH) in conjunction with ASYST software (Asyst Technologies, 1992) that averaged inter- beat intervals into 1-s intervals. To obtain an assessment of baseline RSA, children's heart rate was monitored while they listened to a two-minute neutral story about an elephant.

The child's cardiac interbeat interval (IBI) was assessed continuously by measuring the time between successive R-waves of the electrocardiogram (EKG). Physiological data were recorded continuously using a Coulbourn Instruments (Allentown, PA) system with a multipurpose high gain bioamplifier (S75-01), with the low pass filter set at 40 Hz and the high pass filter set at 0.1 Hz. Miniature Beckman silver-silver chloride electrodes were applied to either side of the child's chest after lightly abrading the area with Omni-prep solution. Beckman electrolyte was used to facilitate conductivity of electrical signals. The physiological data were recorded using an analog-to-digital board (Metrabyte) and a custom program (ASYST) in a PC environment. IBI was averaged into 1-second intervals to allow synchronization with behavior for other analyses. The EKG waveform was digitized at a sampling rate of 128 Hz and stored using ASYST programs during the session. Subsequently, an ASYST data reduction program was used that read and displayed the EKG waveform data, detected and flagged R-spikes, and allowed manual verification of R-spike locations by the operator. This program produced the IBI series data file. The IBI series data file was then cleaned with another custom program which detected and split long IBI's which resulted from missed R-spikes.

Peer Provocation Paradigm. To assess RSA reactivity, heart rate was also obtained while children participated in a peer provocation paradigm. This consisted of a 10-minute session in which they played a computer game with what they thought was a same-sex, same-age peer. However, this peer was a child actor paid to be a confederate in the study. The computer game was also rigged so that the participating child experienced a delay on his controller, and lost 75% of the time. After the participating child lost a game, the confederate peer would make provocative statements such as "Why do you keep losing? Don't you want to win that prize?" and "I'm master of this game, and you don't have a chance." The session was videotaped, and on average, actors made 12.32 comments per session.

After the game, the actor completed a questionnaire and participated in an interview to assess their experience. All children were then debriefed about the true nature of the game. Before leaving the laboratory, children had the chance to play the game again with the actor child, either with or without the delay on the actor's keys. Mothers were telephoned within two days after the session for a follow-up on the family's reactions to the laboratory visit. There were no significant reports of distress at the telephone follow-up.

Measures

Respiratory Sinus Arrhythmia. RSA was calculated by measuring the time among successive R-waves of the electrocardiogram. The amount of variance in the interbeat interval spectrum that was within the child's respiratory sinus frequency band was examined using spectral time-series analysis. The sum of the power densities in the interbeat interval spectrum within the 0.33- to 0.42-Hz band over the total amount of

power across all frequency bands was used as the measure of RSA (Behrman and Kliegman 2002). This method of calculating RSA has been well validated in previous studies of child adjustment and has been found to relate to children's ER abilities, child adjustment, and family processes (Gottman and Katz 1989; Katz and Gottman 1997; Leary and Katz 2005). It is also highly correlated with output from Porges's MXEDIT program (r = .96; Gottman et al., 1997). The program SPEC from the Gottman–Williams computer program time-series package was used to conduct spectral analyses (Williams & Gottman 1981).

Baseline RSA was calculated as the average RSA during the 2-minute baseline period immediately prior to the children playing the video game, after the children have had an opportunity to adjust to the lab setting. RSA in the peer provocation paradigm was calculated as the mean RSA across the total 10 min of play during the peer provocation paradigm. RSA reactivity was then calculated as the change score in RSA from baseline to the peer provocation paradigm.

Intimate Partner Violence. Mothers completed the Physical Aggression Subscale of the Conflict Tactics Scale (CTS) (Straus, 1979). The Conflict Tactics Scale is the most widely used measure of physical aggression between spouses. The Physical Aggression subscale has adequate reliability with alphas ranging from .82 to .88, and adequate construct validity (Straus, 1979). Mothers reported the frequency of their partners' aggressive acts over the past 12 months answering items including "threw something that hit the other one"; "pushed, grabbed, or shoved the other one"; "kicked, bit, or hit with a fist"; and "threatened partner with a knife or a gun". Severity of violence in the items was low, as expected in a community sample. The most frequently endorsed items

included pushing the partner, throwing something at the partner, or blocking a partner from leaving the room. The total violence score represented each mother's report of total aggression perpetrated by her partner. Because the distribution of responses was kurtotic (kurtosis = 4.94), as expected in a sample not recruited specifically for IPV exposure, this violence score was dichotomized to reflect presence of any violence versus no presence of intimate partner violence.

Time 3

Procedures. At Time 3, adolescents were mailed questionnaires assessing their use of alcohol and drugs, and their risky sexual behavior. Questionnaires were mailed back to the lab for analysis.

Measures.

Adolescent Drinking. Adolescents completed the Alcohol Use Questionnaire adapted from the Drinking Calendar (Cahalan et al., 1969), to assess their alcohol use over the past year. Age at first drink was assessed with the question "How old were you when you first had more than a sip of beer, wine or liquor?" Typical drinking was assessed by summing the number of alcoholic beverages consumed in a typical week in the last month.

Problems from Alcohol Use. Adolescents also completed the Rutgers Alcohol Problem Index (RAPI) (White & Labouvie 1989), an 18-item questionnaire that taps the frequency that adolescents report experiencing negative consequences due to their alcohol use during the last three years (e.g., how many times have you not been able to do

your homework or study for a test because of your drinking). Reponses were rated on a Likert scale (0 = never, 1 = 1-2 times, 2 = 3-5 times, 3 = 6-10 times, and 4 = more than 10 times). The RAPI has been found to have good internal consistency and validity in previous studies with adolescents (White & Labouvie 1989).

Marijuana Use. Adolescents reported on their marijuana use with select items from the American Drug and Alcohol Survey (ADAS; Edwards, 1993), which has been widely used and administered to students in middle school and high school. It consists of 57 questions and has excellent reliability and validity (Farrow et al., 1993). Marijuana use was assessed with a single item, "Have you ever tried marijuana?" Responses were rated on a binary scale (0 = no, 1 = yes). Marijuana use in combination with alcohol use was assessed with the question "Have you ever used alcohol and marijuana together?"

Sexual Risk Taking. Adolescents completed a Sexual Behavior Questionnaire (SBQ) that assessed lifetime sexual activity, and types and amount of sexual activity in the last year. Questions were based on items developed and utilized in previous studies with similar aged adolescents, with high reliability and validity (Beadnell et al., 2005). For the current study, two items were used. "In the last year, how many people have you had sexual intercourse with?" was used to assess recent degree of sexual risk as indexed by number of sexual partners, and "How often do you use a condom when engaged in sexual intercourse" was used to assess frequency of condom use. Responses were rated on a Likert scale (0=never, 1=sometimes, 2 = often, 3 = always) and reverse coded.

Results

Effects of RSA reactivity on risky behaviors

Difference scores were calculated to determine RSA reactivity to interpersonal stressor task by averaging children's RSA across the task, and then subtracting it from their average baseline RSA. Inspection of the data indicated that all children showed RSA augmentation from baseline to stressor ($t(1, 43) = -11.814$, $p < .01$, Cohen's d = 2.52). To assess for adolescent risky behaviors, a composite measure was created using items from several well-validated measures: the Drinking Calendar (Cahalan et al., 1969), the Rutgers Alcohol Problem Index (White & Labouvie 1989), the American Drug and Alcohol Survey (Edwards, 1993), and the Sexual Behavior Questionnaire (Beadnell et al., 2005). The composite was designed to assess problems from drinking, alcohol use (alcohol use frequency, largest binge drinking episode in past year) and drug use (marijuana use, marijuana and alcohol use at same time, total number of illicit drugs tried, planned future drug use). Risky sexual behavior was omitted from the final composite variable because only eight out of 43 adolescents completed this measure. After running an exploratory factor analysis and ensuring these drug and alcohol items loaded onto one factor, we ran Cronbach's alpha to ensure internal consistency. The index demonstrates acceptable reliability with an alpha of .75. In linear regression analyses, RSA reactivity was predictive of this composite of risk behavior, supporting our first hypothesis ($R(42) = .38$ $p < .05$).

Moderation of the Effects of RSA reactivity on risky behaviors

Using total IPV scores, children were placed into groups based on the presence or absence of IPV in the home. Initial analyses using an ordinary least squares regression revealed that the interaction of RSA augmentation in an interpersonal stressor task in childhood and the presence of IPV at home predicted adolescent risky behavior (R^2 = 0.46, $F(6, 35) = 4.06$, $p = .005$) when controlling for gender, ethnicity, and age (see Table 1 and Figure 1). However, when we conducted relative model fit comparisons comparing the ordinary least squares regression models against several zero-inflated modeling strategies, zero-inflated negative binomial (ZINB) indicated the best model fit (AIC = 121.818, BIC = 137.669). This two-part regression approach was optimal because the outcome measure, the adolescent risky behavior composite variable, was highly kurtotic (kurtosis = 2.77) and skewed (skew = 1.87). Using the zero-inflated negative binomial regression model allowed us to determine first the likelihood of an adolescent reporting *any* risky behaviors as well as the level of these problems.

A new composite measure was created using only count variables as is required for ZINB regressions. This index was comprised of age the adolescent first had more than a sip of alcohol, problems from drinking, and total drugs tried. No associations were found between RSA augmentation and this new composite of adolescent risk behaviors. In post hoc analyses using the zero-inflated negative binomial regression model, we found that the association between RSA augmentation to interpersonal stress in childhood and the level of alcohol problems in adolescence was conditioned by an interaction with intimate partner violence in childhood (β=.31, $p<.01$). Figure 2 illustrates this effect. Specifically, at high levels of intimate partner violence, RSA augmentation was

positively related to problems from drinking; however for low levels of IPV the opposite was true.

Discussion

Adolescents who engage in risk behavior, from alcohol and drug abuse to risky sex, comprise a subset of youth who will be increasingly likely to endorse internalizing and externalizing psychopathology in adolescence and into adulthood (Bonomo et al., 2004; McGue & Iacono 2005; Sher & Zalsman 2005). Although a variety of environmental stressors and biological vulnerabilities proffer risk of engaging in problem behavior during adolescence, a consensus has not yet been reached on which biological underpinnings of adolescent risk taking can be identified as early as middle childhood. This study is the first to identify high RSA augmentation in response to interpersonal stress as one such susceptibility factor in middle childhood. When this biological marker of emotion dysregulation is compounded by an environmental stressor, in this case a home in which IPV is present, the relation between RSA reactivity in childhood and adolescent problems from alcohol use is strengthened. These findings highlight how one combination of biological and environmental risk factors increases the risk of problems from alcohol use in youth.

We first hypothesized that high RSA augmentation to childhood interpersonal stress would identify youth particularly at risk for exhibiting a range of risky behavior in adolescence. While we corroborated this hypothesis using ordinary least squares regression, these findings were not replicated using the better model fit of a zero-inflated negative binomial regression. In a post-hoc analysis using ZINB, high RSA

augmentation was found to significantly predict one component of adolescent problem behavior: problems from alcohol use. This result complements previous research findings suggesting that children who are unable to effectively regulate their emotions are more likely to engage in substance use and sexual risk behavior as adolescents, while children skilled in self-regulation are buffered against alcohol-related problems and other risky behavior in adolescence (Hull & Slone 2004; Quinn & Fromme 2010; Silk et al., 2003; Wiederman, 2004).

In the outset of this study we hypothesized that high augmentation, in particular, to peer stress in childhood would predict adolescent risk behavior. This prediction arose out of recent literature suggesting that the context of stress determines what is an appropriate direction of RSA change, with RSA augmentation thought of as appropriate for intellectual challenge and RSA withdrawal appropriate for interpersonal challenge (Leary & Katz, 2004; Obradovic et al., 2011). A continuum of RSA reactivity was expected in response to the peer provocation paradigm, from high withdrawal to high augmentation. Surprisingly, all children showed RSA augmentation. This reaction indicates that the peer provocation paradigm was indeed highly stressful. Although RSA withdrawal was expected as an appropriate response to interpersonal stress, it is possible that low augmentation was equally appropriate in this particular situation. The children who displayed low augmentation may have been regulating their stress reaction in order to enhance attention to the video game that framed the peer provocation. These children did not go on to endorse risky behaviors in adolescence, suggesting that low levels of RSA augmentation to interpersonal stress may not serve as a vulnerability factor in middle childhood. We found that only the children who displayed high levels of

augmentation to the peer provocation paradigm were the ones who later reported problems from alcohol use. These findings imply that not only the direction of RSA change but also the level of change should be considered when identifying early biomarkers of later risk behaviors.

One mechanism that may be responsible for this connection between RSA reactivity in childhood and problems from alcohol use in adolescence is the employment of ineffective emotion regulation strategies, such as disengagement coping, for regulating negative emotions during stressful situations (Wills et al., 2001; Connor-Smith et al., 2000). Perhaps children with high RSA augmentation to peer provocation are the ones most strongly disengaging from the distress caused by the harsh remarks of their peer and instead focusing on the videogame. These children may then be developing pathways of disengagement coping which sets the stage for use of alcohol to disconnect from stress in adolescence (Connor-Smith et al. 2000).

While it is important to study early biological markers of adolescent risky behaviors, it may be of equal importance to examine how stressful environments may interact with early biomarkers. A secondary hypothesis of this study was that the presence of an environmental stressor would strengthen the predictive power of high RSA augmentation in childhood. The impact of IPV was examined because existing research studies suggest that IPV is linked with child emotion dysregulation and behavior problems (Maughan & Cicchetti 2002). Furthermore, previous literature has indicated that exposure to violence is associated with alcohol use, aggression, and antisocial behavior in early and middle adolescence (Schwab-Stone et al., 1995). Living in an environment where the threat of violence is present has been shown to further impair

emotion regulation capabilities in children as well as negatively impact a child's long-term functioning (Evans et al., 2008; Grych et al., 2000; Wolfe et al., 2003). The current results support that the presence of IPV strengthens the relation between emotion dysregulation in an interpersonally stressful task in childhood and problems from alcohol use in adolescence.

The presence of IPV may serve as a catalyst for children who are more emotionally dysregulated in childhood to go on to endorse an outcome highly correlated with alcohol dependence – problems from alcohol use (Dick et al., 2011). This result carries some serious implications as adolescents who are most problematic in their use of alcohol are the ones who may be putting themselves and others in danger when drinking (Bonomo et al. 2001; Blum, 1987; Millstein et al., 1992;). The combination of a biological risk factor like physiological reactivity and an environmental stressor such as IPV produces behavior that is more concerning than the more typical adolescent experimental behaviors like trying small amounts of alcohol or marijuana. In fact, low levels of alcohol and marijuana use are considered by some researchers to be normative (Shedler & Block, 1990). Unlike occasional drink and drug use in adolescence, problems from drinking are more likely to predict later problem drinking and alcoholism (Dick et al., 2011).

While IPV is a stressor for any child, those children who are able to successfully regulate their RSA in response to this stress may be buffered from its deleterious effects. Katz & Gottman (1995) demonstrated that children who could flexibly regulate their emotions as measured by their physiological responses were buffered from the stress of their parents' marital conflict. Similarly, our study found no association between the

presence of IPV and problems from alcohol use in children with low RSA augmentation in response to peer provocation. This finding suggests that only children who are highly reactive to interpersonal stress may be vulnerable to negative effects of IPV.

This study had many strengths, including a prospective longitudinal design, and multiple methods of assessment from questionnaires to an in-vivo stressor task. However, there are also several limitations. While we found support for the hypothesis that RSA reactivity in childhood in combination with the presence of IPV predicted one of the adolescent risk outcomes - increased problems from alcohol use - the same pattern of findings for other risky behaviors such as drug and alcohol use was not observed. It is possible that with the small sample size in this study there was not enough power to detect the role of IPV in moderating the relation between RSA reactivity and adolescent alcohol and drug use. Furthermore because we assessed for IPV and RSA at the same time point, we are unable to identify whether the biological vulnerability of RSA augmentation to stress or the environmental stressor of IPV has primacy in predicting adolescent problems from alcohol use. This puts our interpretation of the findings – that children who are physiologically dysregulated are placed at greater risk for problem behavior in adolescence by a stressful environment – into question. It is equally possible that a stressful environment is what compromises a child's ability to physiologically regulate in the first place. In fact, consistent with our finding that IPV and RSA were highly correlated, there is considerable evidence that exposure to chronic stress appears to disrupt the regulation of autonomic arousal (Tsigos & Chrousos 2002). Future large-scale longitudinal studies with high risk populations will need to evaluate children's RSA

reactivity before they are exposed to IPV to determine the direction of effects in the development of these early risk factors for adolescent problem behavior.

Several additional limitations also must be acknowledged. Adolescent risk behavior was measured by the youths' responses to self-report questionnaires. It is possible that adolescents underreported their drug and alcohol use for fear of legal consequences despite assurances of study participant confidentiality. Multiple informants of adolescent risk behavior, from parents to siblings to teachers, would have strengthened the internal validity of our outcome measure. Another noteworthy limitation of this study is that IPV and adolescent risk behavior have very low base rates in the community and this sample is no different. With only two of the teenagers who endorsed alcohol problems also endorsing any levels of intimate partner violence in their homes these results are not generalizable. New prospective studies should investigate both biological and behavioral measures of emotion regulation in children recruited from homes in which IPV is present and compare them with children from non-violent homes to better differentiate the mechanism of susceptibility to adolescent risk behavior.

This study exemplifies why it is essential that prevention researchers evaluate the effects of biological risk and biology by environment interactions in predicting psychopathology. This study contributes to adolescent risk prevention research by identifying the predictive power of the biological risk factor of RSA augmentation to interpersonal stress and its interaction with the environmental stressor of IPV. These findings suggest that interventions directed at enhancing emotion regulation skills in childhood may reduce problems from alcohol use in adolescence and its associated economic and health costs (Eaton et al., 2010). Furthermore, these results indicate that an

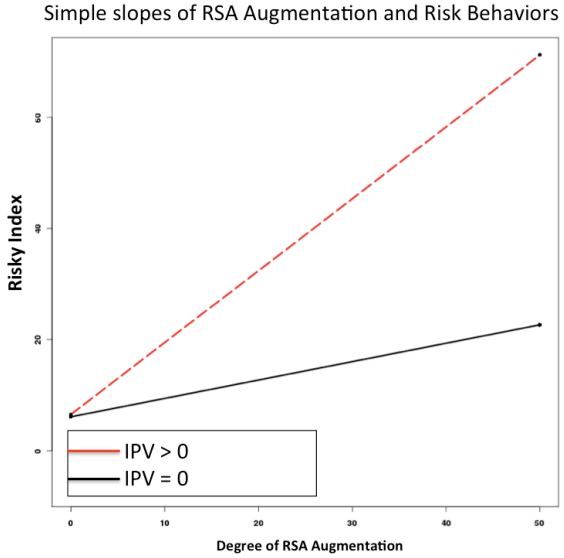
important population for whom to target these interventions would be children who have lived in a home where their parents are violent with one another.

Figures

Figure 1. Ordinary Least Squares Regression:
IPV moderates the relation between RSA augmentation in childhood and risky behavior in adolescence.

Figure 2: Zero-inflated Negative Binomial Regression:
The presence of IPV strengthens the relation between RSA augmentation and adolescent alcohol problems.

Figure 1. Ordinary Least Squares Regression:
IPV moderates the relation between RSA augmentation in childhood and risky behavior in adolescence.



B = .36, $p < .05$
$F^2 = .15$

Figure 2: Zero-inflated Negative Binomial Regression:
The presence of IPV strengthens the relation between RSA augmentation and adolescent alcohol problems.

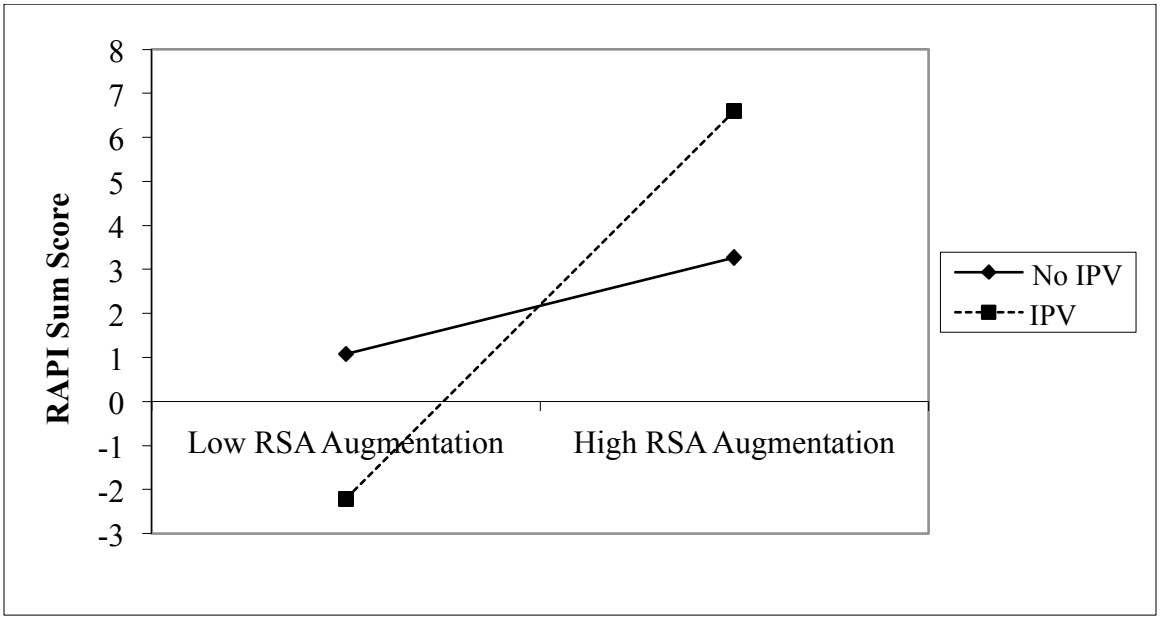


Table 1. Multiple Regression Predicting Risk Index			
Variable	**B (SE)**	**β**	**p**
Gender	**-.67 (2.61)**	**-.04**	**.800**
Ethnicity	**1.50 (3.37)**	**.06**	**.660**
Age	**4.97 (2.35)**	**.31**	**.043**
RSA Reactivity	**.35 (.12)**	**.43**	**.005**
IPV	**-.22 (2.38)**	**-.16**	**.927**
RSA Reactivity * IPV	**.36 (.17)**	**.37**	**.044**

References

Asyst Technologies. (1992). Asyst (Computer software). Fremont, CA: Author

Aiken, L. S. and S. G. West (1991). Multiple regression: Testing and interpreting interactions, Sage.

Beadnell, B., D. M. Morrison, et al. (2005). "Condom use, frequency of sex, and number of partners: Multidimensional characterization of adolescent sexual risk−taking." Journal of Sex Research **42**(3): 192-202.

Beauchaine, T. P., L. Gatzke-Kopp, et al. (2007). "Polyvagal theory and developmental psychopathology: Emotion dysregulation and conduct problems from preschool to adolescence." Biological psychology **74**(2): 174-184.

Beauchaine, T. P., E. Neuhaus, et al. (2008). "Ten good reasons to consider biological processes in prevention and intervention research." Dev Psychopathol **20**(3): 745-774.

Behrman, R. E. and R. M. Kliegman (2002). Nelson essentials of pediatrics (4th ed.). Philadelphia, W.B. Saunders.

Blum, R. W. (1987). "Adolescent substance abuse: diagnostic and treatment issues." Pediatric Clinics of North America **34**(2): 523.

Blum, R. W. and K. Nelson-Mmari (2004). "The health of young people in a global context." J Adolesc Health **35**(5): 402-418.

Bonomo, Y., C. Coffey, et al. (2001). "Adverse outcomes of alcohol use in adolescents." Addiction **96**(10): 1485-1496.

Bonomo, Y. A., G. Bowes, et al. (2004). "Teenage drinking and the onset of alcohol dependence: a cohort study over seven years." Addiction **99**(12): 1520-1528.

Bonomo, Y. A., G. Bowes, et al. (2004). "Teenage drinking and the onset of alcohol dependence: a cohort study over seven years." Addiction **99**(12): 1520-1528.

Brook, D. W. and J. S. Brook (1990). The etiology and consequences of adolescent drug use. Drug and alcohol abuse prevention, Springer**:** 339-362.

Cahalan, D., I. H. Cisin, et al. (1969). "American drinking practices: A national study of drinking behavior and attitudes." Monographs of the Rutgers Center of Alcohol Studies.

Carlson, B. E. (1984). "Causes and maintenance of domestic violence: An ecological analysis." The Social Service Review: 569-587.

Casey, B. and R. M. Jones (2010). "Neurobiology of the adolescent brain and behavior: implications for substance use disorders." Journal of the American Academy of Child & Adolescent Psychiatry **49**(12): 1189-1201.

Casey, B., R. M. Jones, et al. (2008). "The adolescent brain." Annals of the New York Academy of Sciences **1124**(1): 111-126.

Cicchetti, D. (2007). "Gene-environment interaction." Dev Psychopathol **19**(4): 957-959.

Cole, P. M., C. Zahn-Waxler, et al. (1996). "Individual differences in emotion regulation and behavior problems in preschool children." Journal of Abnormal Psychology **105**(4): 518.

Connor-Smith, J. K., B. E. Compas, et al. (2000). "Responses to stress in adolescence: measurement of coping and involuntary stress responses." Journal of consulting and clinical psychology **68**(6): 976.

Crowell, S. E., T. P. Beauchaine, et al. (2005). "Psychological, autonomic, and serotonergic correlates of parasuicide among adolescent girls." Development and Psychopathology **17**(4): 1105.

Dahl, R. E. (2004). "Adolescent brain development: a period of vulnerabilities and opportunities. Keynote address." Annals of the New York Academy of Sciences **1021**(1): 1-22.

Dawes, R. M. and P. E. Meehl (1966). "Mixed group validation: A method for determining the validity of diagnostic signs without using criterion groups." Psychological Bulletin **66**(2): 63-67.

Dick, D. M., F. Aliev, et al. (2011). "Rutgers alcohol problem index scores at age 18 predict alcohol dependence diagnoses 7 years later." Alcoholism: Clinical and Experimental Research **35**(5): 1011-1014.

Eaton, D. K., L. Kann, et al. (2010). "Youth risk behavior surveillance—United States, 2009." MMWR Surveill Summ **59**(5): 1-142.

Edwards, R. W. (1993). "Drug use among 8th grade students is increasing." Substance Use & Misuse **28**(14): 1621-1623.

Evans, S. E., C. Davies, et al. (2008). "Exposure to domestic violence: A meta-analysis of child and adolescent outcomes." Aggression and Violent Behavior **13**(2): 131-140.

Fabes, R. A., N. Eisenberg, et al. (1993). "Behavioral and Physiological Correlates of Childrens Reactions to Others in Distress." Dev Psychol **29**(4): 655-663.

Farrow, J. A., W. R. Smith, et al. (1993). Adolescent drug and alcohol assessment instruments in current use: A critical comparison, The Division.

Fox, N. A. and T. M. Field (1989). "Individual differences in preschool entry behavior." Journal of Applied Developmental Psychology **10**: 527-540.

Galván, A. (2013). "The Teenage Brain Sensitivity to Rewards." Current directions in psychological science **22**(2): 88-93.

Garber, J., N. Braafladt, et al. (1995). "Affect regulation in depressed and nondepressed children and young adolescents." Development and Psychopathology **7**(01): 93-115.

Gottman, J. M. and L. F. Katz (1989). "Effects of marital discord on young children's peer interaction and health." Developmental psychology **25**(3): 373.

Gottman, J. M., L. F. Katz, et al. (1997). Meta-emotion: How families communicate emotionally, Psychology Press.

Grych, J. H., E. N. Jouriles, et al. (2000). "Patterns of adjustment among children of battered women." Journal of consulting and clinical psychology **68**(1): 84.

Hall, G. S. (1904). "Adolescence (Vols. 1 & 2)." New York: Appleton.

Hilt, L. M., J. L. Hanson, et al. (2011). Emotion Dysregulation. Encyclopedia of Adolescence. S. T. Hauser and M. J. Prinstein, Academic Press. **3**.

Holden, G. W., R. E. Geffner, et al. (1998). Children exposed to marital violence: Theory, research, and applied issues, American Psychological Association.

Hull, J. G. and L. B. Slone (2004). "Alcohol and self-regulation." Handbook of self-regulation: Research, theory, and applications: 466-491.

Jaffe, P., D. Wolfe, et al. (1986). "Similarities in behavioral and social maladjustment among child victims and witnesses to family violence." American Journal of Orthopsychiatry **56**(1): 142-146.

Katona, P. G. and F. Jih (1975). "Respiratory sinus arrhythmia: noninvasive measure of parasympathetic cardiac control." J Appl Physiol **39**(5): 801-805.

Katz, L. F. and J. M. Gottman (1995). "Vagal tone protects children from marital conflict." Development and Psychopathology **7**: 83-83.

Katz, L. F. and J. M. Gottman (1997). "Buffering children from marital conflict and dissolution." J Clin Child Psychol **26**(2): 157-171.

Katz, L. F. and J. M. Gottman (1997). "Buffering children from marital conflict and dissolution." Journal of clinical child psychology **26**(2): 157-171.

Kring, A. and D. Sloan (2010). Emotion Regulation and Psychopathology: A Transdiagnostic Approach to Etiology and Treatment. New York, Guilford Press.

Larson, R. and L. Asmussen (1991). "Anger, worry, and hurt in early adolescence: An enlarging world of negative emotions." Adolescent stress: Causes and consequences: 21-41.

Leary, A. and L. F. Katz (2005). "Observations of aggressive children during peer provocation and with a best friend." Developmental psychology **41**(1): 124.

Luna, B., D. J. Paulsen, et al. (2013). "The Teenage Brain Cognitive Control and Motivation." Current directions in psychological science **22**(2): 94-100.

Margolin, G. and K. A. Vickerman (2011). "Posttraumatic stress in children and adolescents exposed to family violence: I. Overview and issues."

Maughan, A. and D. Cicchetti (2002). "Impact of child maltreatment and interadult violence on children's emotion regulation abilities and socioemotional adjustment." Child Dev **73**(5): 1525-1542.

McGue, M. and W. G. Iacono (2005). "The association of early adolescent problem behavior with adult psychopathology." American Journal of Psychiatry **162**(6): 1118-1124.

McGue, M. and W. G. Iacono (2005). "The association of early adolescent problem behavior with adult psychopathology." Am J Psychiatry **162**(6): 1118-1124.

McLaughlin, K. A., M. L. Hatzenbuehler, et al. (2011). "Emotion dysregulation and adolescent psychopathology: A prospective study." Behaviour research and therapy **49**(9): 544-554.

Miller, G. (1989). "Violence by and against America's children." Journal of Juvenile Justice Digest, XVII **12**(6).

Miller, T. (2004). The social costs of adolescent problem behavior. New York, Guilford Press.

Millstein, S. G., C. E. Irwin, et al. (1992). "Health-risk behaviors and health concerns among young adolescents." Pediatrics **89**(3): 422-428.

Muthen, B. and L. Muthen (2009). Mplus User's Guide. Seventh Edition. Los Angeles, CA, Muthen & Muthen.

Obradovic, J., N. R. Bush, et al. (2011). "The interactive effect of marital conflict and stress reactivity on externalizing and internalizing symptoms: The role of laboratory stressors." Development and Psychopathology **23**(1): 101.

Porges, S. W. (1995). "Cardiac vagal tone: a physiological index of stress." Neurosci Biobehav Rev **19**(2): 225-233.

Porges, S. W. (1995). "Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A polyvagal theory." Psychophysiology **32**(4): 301-318.

Porges, S. W. (1997). "Emotion: an evolutionary by-product of the neural regulation of the autonomic nervous system." Ann N Y Acad Sci **807**: 62-77.

Porges, S. W. (1998). "Love: an emergent property of the mammalian autonomic nervous system." Psychoneuroendocrinology **23**(8): 837-861.

Porges, S. W. (2001). "The polyvagal theory: phylogenetic substrates of a social nervous system." Int J Psychophysiol **42**(2): 123-146.

Porges, S. W. (2003). "The Polyvagal Theory: phylogenetic contributions to social behavior." Physiol Behav **79**(3): 503-513.

Pulkkinen, L. and T. Pitkänen (1994). "A prospective study of the precursors to problem drinking in young adulthood." Journal of Studies on Alcohol and Drugs **55**(5): 578.

Quinn, P. D. and K. Fromme (2010). "Self-regulation as a protective factor against risky drinking and sexual behavior." Psychology of Addictive Behaviors **24**(3): 376.

Reyna, V. F. and F. Farley (2006). "Risk and rationality in adolescent decision making implications for theory, practice, and public policy." Psychological science in the public interest **7**(1): 1-44.

Robinson, E. A., S. M. Eyberg, et al. (1980). "The standardization of an inventory of child conduct problem behaviors." Journal of Clinical Child & Adolescent Psychology **9**(1): 22-28.

Romer, D., L. M. Betancourt, et al. (2011). "Does adolescent risk taking imply weak executive function? A prospective study of relations between working memory performance, impulsivity, and risk taking in early adolescence." Developmental science **14**(5): 1119-1133.

Rydell, A.-M., L. Berlin, et al. (2003). "Emotionality, emotion regulation, and adaptation among 5-to 8-year-old children." Emotion **3**(1): 30.

Santucci, A. K., J. S. Silk, et al. (2008). "Vagal tone and temperament as predictors of emotion regulation strategies in young children." Developmental psychobiology **50**(3): 205-216.

Schwab-Stone, M. E., T. S. Ayers, et al. (1995). "No safe haven: A study of violence exposure in an urban community." Journal of the American Academy of Child & Adolescent Psychiatry **34**(10): 1343-1352.

Shedler, J. and J. Block (1990). "Adolescent drug use and psychological health: A longitudinal inquiry." American Psychologist **45**(5): 612.

Sher, L. and G. Zalsman (2005). "Alcohol and adolescent suicide." Int J Adolesc Med Health **17**(3): 197-203.

Sher, L. and G. Zalsman (2005). "Alcohol and adolescent suicide." International Journal of Adolescent Medicine and Health **17**(3): 197-204.

Silk, J. S., L. Steinberg, et al. (2003). "Adolescents' emotion regulation in daily life: links to depressive symptoms and problem behavior." Child Dev **74**(6): 1869-1880.

Southam-Gerow, M. A. and P. C. Kendall (2002). "Emotion regulation and understanding: Implications for child psychopathology and therapy." Clinical psychology review **22**(2): 189-222.

Spinrad, T. L., N. Eisenberg, et al. (2006). "Relation of emotion-related regulation to children's social competence: A longitudinal study." Emotion **6**(3): 498.

Steinberg, L. (2004). "Risk taking in adolescence: what changes, and why?" Annals of the New York Academy of Sciences **1021**(1): 51-58.

Steinberg, L. (2010). "A dual systems model of adolescent risk−taking." Developmental psychobiology **52**(3): 216-224.

Sternberg, K. J., M. E. Lamb, et al. (2006). "Effects of early and later family violence on children's behavior problems and depression: A longitudinal, multi-informant perspective." Child abuse & neglect **30**(3): 283-306.

Straus, M. A. (1979). "Measuring intrafamily conflict and violence: The conflict tactics (CT) scales." Journal of Marriage and the Family: 75-88.

Straus, M. A. (1992). Sociological research and social policy: The case of family violence. Sociological Forum, Springer.

Tjaden, P. and N. Thoennes (2000). "Full report of the prevalence, incidence, and consequences of violence against women."

Tsigos, C. and G. P. Chrousos (2002). "Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress." Journal of psychosomatic research **53**(4): 865-871.

White, H. R. and E. W. Labouvie (1989). "Towards the assessment of adolescent problem drinking." Journal of Studies on Alcohol and Drugs **50**(01): 30.

Wiederman, M. W. (2004). "Self-control and sexual behavior." Handbook of self-regulation: Research, theory, and applications: 525-536.

Wilens, T. E., M. Martelon, et al. (2013). "Difficulties in emotional regulation and substance use disorders: A controlled family study of bipolar adolescents." Drug and alcohol dependence.

Williams, E. and J. M. Gottman (1981). A user's guide to the Gottman-Williams time-series programs. New York, Cambridge University Press.

Wolfe, D. A., C. V. Crooks, et al. (2003). "The effects of children's exposure to domestic violence: A meta-analysis and critique." Clinical child and family psychology review **6**(3): 171-187.