

ABSTRACT

Title of Dissertation: EXPERIMENTALLY TESTING THE EFFECT OF PARENT ADOLESCENT CONFLICT ON HIV RISK, AND INVESTIGATION OF A NEUROBIOLOGICAL MODERATOR OF THIS EFFECT

Sarah Ann Thomas, Doctor of Philosophy, 2016

Dissertation directed by: Andres De Los Reyes
Associate Professor, Department of Psychology

Human immunodeficiency virus (HIV) is a condition in which immune cells become destroyed such that the body may become unable to fight off infections. Engaging in risk-taking behaviors (e.g., substance use) puts people at heightened risk for HIV infection, with mid-to-late adolescents at increasing risk (Leigh & Stall, 1993). Environmental and neurological reasons have been suggested for increased risk-taking among adolescents. First, family-level precursors such as parent-adolescent conflict have been significantly associated with and may pose risk for engaging in substance use and risk-taking (Duncan, Duncan, Biglan, & Ary, 1998). Thus, parent-adolescent conflict may be an important proximal influence on HIV risk behaviors (Lester et al., 2010; Rowe, Wang, Greenbaum, & Liddle, 2008). Yet, the temporal relation between parent-adolescent conflict and adolescent HIV risk-taking behaviors is still unknown. Second, at-risk adolescents may carry a neurobiological predisposition for engaging in trait-like expressions of

disinhibited behavior and other risk-taking behaviors (Iacono, Malone, & McGue, 2008). When exposed to interpersonally stressful situations, their likelihood of engagement in HIV risk behaviors may increase. To investigate the role of parent-adolescent conflict in adolescent HIV risk-taking behaviors, 49 adolescents ages 14-17 and their parent were randomly assigned to complete a standardized discussion task to discuss a control topic or a conflict topic. Immediately after the discussion, adolescents completed a laboratory risk-taking measure. In a follow-up visit, eligible adolescents underwent electrophysiological (EEG) recording while completing a task designed to assess the presence of a neurobiological marker for behavioral disinhibition which I hypothesized would moderate the links between conflict and risk-taking. First, findings indicated that during the discussion task, adolescents in the conflict condition evidenced a significantly greater psychophysiological stress response relative to adolescents in the control condition. Second, a neurobiological marker of behavioral disinhibition moderated the relation between discussion condition and adolescent risk-taking, such that adolescents evidencing relatively high levels of a neurobiological marker related to sensation-seeking evidenced greater levels of risk-taking following the conflict condition, relative to the control condition. Lastly, I observed no significant relation between parent-adolescent conflict, the neurobiological marker of behavioral disinhibition and adolescent engagement in real-world risk-taking behavior.

EXPERIMENTALLY TESTING THE EFFECT OF PARENT ADOLESCENT
CONFLICT ON HIV RISK, AND INVESTIGATION OF A NEUROBIOLOGICAL
MODERATOR OF THIS EFFECT

by

Sarah Ann Thomas

Dissertation submitted to the Faculty of the Graduate School of the
University of Maryland, College Park, in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
2016

Advisory Committee:
Associate Professor Andres De Los Reyes, Chair
Assistant Professor Edward Bernat
Professor Natasha Cabrera
Associate Professor Lea Dougherty
Professor Carl Lejuez

© Copyright by
Sarah Ann Thomas
2016

Table of Contents

Table of Contents	ii
List of Tables	v
List of Figures	vii
Chapter 1: Introduction	1
HIV Risk in Adolescence.....	1
Adolescent Risk-Taking.....	2
Theoretical and Conceptual Framework	4
HIV Risk Behaviors and Family Environment	7
Potential Neurobiological Moderator of Link between Parent-Adolescent Conflict and HIV Risk Behaviors	9
Methodological Factors Impacting the Study of Family-Level and Neurobiological Characteristics of HIV Risk Behaviors	12
Summary and Overall Significance	14
Primary Aims and Hypotheses:	15
Aim 1	15
Aim 2	15
Aim 3	15
Chapter 2: Method.....	17
Overall Design	17
Aims 1 and 2: Behavioral Visit.....	17
Participants and recruitment.....	17
Procedures.	20
Measures.....	21
<i>Demographics</i>	21
<i>Adolescent psychophysiological reactivity</i>	21
<i>Parent-adolescent conflict topics</i>	22

<i>Conflict discussion task.</i>	23
<i>Coding of associated conflict behaviors.</i>	24
<i>Adolescent analog risk-taking.</i>	27
<i>Assessing condition equivalency on and correlates of real-world risk-taking.</i>	27
<i>Real-world risk-taking behaviors.</i>	28
<i>HIV risk behaviors</i>	29
<i>Assessing condition equivalency on Parent-adolescent relationship characteristics.</i>	29
<i>Parenting quality.</i>	29
<i>Adolescent disclosure.</i>	30
Aim 3: EEG Visit.	30
Participants and recruitment.	31
Procedures.	32
Measures.	32
<i>Neurobiological risk for substance use.</i>	32
<i>Oddball task.</i>	32
<i>EEG data acquisition and processing.</i>	33
Data Analysis	35
Overview.	35
Aim 1.	35
Aim 2.	35
Aim 3.	36
Condition equivalency on real-world risk-taking, and exploratory analyses.	37
Chapter 3: Results	38
Preliminary Analyses	38
Aim 1	40
Aim 2	41
Aim 3	42

Exploratory Aim	44
Chapter 4: Discussion	45
Main Findings	45
Interpretation of Results.....	46
Limitations	51
Future Directions	53
Conclusion	55
Appendix A.....	57
Table	57
Appendix B	72
Appendix C	73
Bibliography	74

List of Tables

Table A1 Comparison of Baseline Demographic Characteristics Between Discussion Conditions	57
Table A2 Means (M), Standard Deviations (SD), and Internal Consistency (α) Estimates of Survey Measures of TTI and Comparison Between Discussion Conditions	58
Table A3 Means (M), and Standard Deviations (SD), and Reliability Estimates of Discussion Task Observed Behavior and Comparison Between Discussion Conditions	59
Table A4 Means (M), Standard Deviations (SD), and Internal Consistency (α) Estimates of Survey and Performance-based Measures of Risk taking and Comparison Between Discussion Conditions	60
Table A5 Means (M), Standard Deviations (SD), and Internal Consistency (α) Estimates of Survey Measures of Parent-adolescent Relationship Characteristics and Comparison Between Discussion Conditions	61
Table A6 Correlations among Measures of Parenting, Observed Behavior, Psychophysiology, and Risk-behavior	62
Table A7 Means (M) and Standard Deviations (SD) of Adolescent Heart Rate and P3, and Comparison Between Discussion Conditions	63
Table A8 Generalized Estimating Equations Predicting Adolescent Performance-based Risk-taking (BART-Y) as a Function of Time (Pre vs Post) and Discussion Condition	

Status(control vs. conflict; n = 49)	64
Table A9 Generalized Estimating Equations Predicting Adolescent Performance-based Risk-taking (BART-Y) as a Function of Time (Pre vs Post), Discussion Condition Status (control vs. conflict; n = 12), and P3	65
Table A10 Generalized Estimating Equations Post-hoc Tests of Moderation Probing the Interaction of Discussion Condition and Low P3	67
Table A11 Generalized Estimating Equations Post-hoc Tests of Moderation Probing the Interaction of Discussion Condition and High P3	68
Table A12 Hierarchical Linear Regression Analyses Summary for Real-World Risk-taking (YRBS Risk), Discussion Condition, and P3 (N = 12)	69
Table A13 Hierarchical Regression Analyses Summary for Real-World HIV Risk (YRBS HIV), Discussion Condition, and P3 (N = 12)	70

List of Figures

Figure 1. Model of factors impacting HIV risk status	6
Figure 2. Hypothesized model of adolescent HIV risk in proposed project	6
Figure 3. Behavioral Visit participant enrollment table	19
Figure 4. Behavioral task session timeline	21
Figure 5. Grand average waveform for P3 Target stimuli	34
Figure 6. Interaction effect between Discussion Condition, neurobiological marker for behavioral disinhibition (P3 mean amplitude), and Balloon Analogue Risk Task-Youth Version	43

Chapter 1: Introduction

HIV Risk in Adolescence

Human immunodeficiency virus (HIV), spread via contact with certain bodily fluids (e.g., blood, vaginal fluid, rectal fluid, pre-seminal fluid, semen or breast milk), is a condition in which certain immune cells can become destroyed. Destruction to these immune cells makes the body more susceptible to the effects of disease to the point that the body can no longer fight infection, thereby progressing to acquired immunodeficiency syndrome (AIDS; CDC, 2014). Although there are treatments that can halt the progression of HIV to AIDS, there is no cure (CDC, 2014). HIV treatment represents a large cost to the United States, with estimates indicating that lifetime treatment for all HIV cases diagnosed in 2009 alone will cost \$16.3 billion dollars (CDC, 2013). HIV infection rates have increased in the United States to epidemic levels, and many of these new infections occur in young people aged 13-29 years (CDC, 2012). Specifically, although the number of new HIV diagnoses remained stable from 2007-2010, there was a 10% increase in new HIV diagnoses among individuals between the ages of 15-19, and a 33% increase among individuals aged 20-24 (CDC, 2012). These data suggest that understanding the factors that lead to HIV infection is vital in order to inform prevention efforts.

In adolescence, risk-taking includes behaviors such as substance use and unprotected sex, which are harmful behaviors in their own right, and also pose risk for HIV infection (Leigh & Stall, 1993; Lowry, Holtzman, Truman, & Kann, 1994). In particular, substance use and sexual risk behaviors are tightly linked in that substance use

may increase disinhibited behaviors that facilitate unsafe sexual practices (Lowry et al., 1994). Thus, HIV risk behaviors include those behaviors that either directly (e.g., injecting drug use, unprotected sex) or indirectly (e.g., substance use leading to impaired decision-making) increase the likelihood of contracting HIV (Earnshaw, Bogart, Dovidio, & Williams, 2013). Prior research supports the link between the use of substances and co-occurring engagement in HIV risk behaviors. For example, relative to no use, alcohol use results in a roughly seven-fold decreased likelihood to use a condom during sexual intercourse; methamphetamine use results in a nearly 16-fold decreased likelihood (Baskin-Sommers & Sommers, 2006). Marijuana use is also associated with reduced condom use and increased incidence of sexually transmitted infections (STIs) among adolescents (Hendershot, Magnan, & Bryan, 2010). Many explanations for why adolescents engage in HIV risk behaviors have been proposed, including family-level factors (e.g., parent-adolescent conflict) and differential maturation rates for brain regions responsible for affective versus regulatory responses (Steinberg, 2007). However, still unknown are the causal pathways by which these mechanisms confer risk for the development and expression of adolescent engagement in HIV risk behaviors (Donenberg & Pao, 2005). Consequently, a key aim of my dissertation was to examine family-level and neurobiological factors that may increase risk for adolescents engaging in HIV risk behaviors.

Adolescent Risk-Taking

Heightened risk-taking during the adolescent developmental period occurs in both humans and animals, suggesting it serves an evolutionarily adaptive role in development

(Spear, 2011). Adolescents, relative to adults, have more hyperactive brain responses to reward stimuli (e.g., monetary incentives for performance on response inhibition tasks; Geier, Terwilliger, Teslovich, Velanova, & Luna, 2010), and to uncertain reward conditions (e.g., a series of 3 incrementally presented images that could result in a monetary reward if presented in the correct combination; Van Leijenhorst et al., 2010). Collectively, this work indicates that adolescents are relatively more likely than adults to willingly try new experiences, yet at the same time this poses challenges for them to consider the implications of their actions during these experiences. However, adolescents do not reason about risk differently than adults. That is, adolescents do not believe they are more or less invulnerable to the effects of risk taking, relative to adults (Beyth-Marom et al., 1993; Quadrel, Fischhoff, & Davis, 1993). Thus, reasoning about risk does not appear to explain adolescents' heightened levels of risk-taking.

Recent work focuses on neurological antecedents of risk-taking behavior among adolescence. In particular, not only is the adolescent brain different from the child brain and adult brain, but also brain regions of adolescents develop at different rates (Steinberg, 2005). Specifically, the limbic regions (e.g., the amygdala) develop first, enabling adolescents, relative to younger children, to engage in more intense emotional arousal, whereas the brain regions that regulate this emotional arousal (e.g. prefrontal cortex) develop later in adolescence (Steinberg, 2005). Importantly, brain maturation cannot be the only explanation for adolescent engagement in HIV risk behaviors. Indeed, adolescents' brains normatively mature in this way, and thus if maturation processes wholly accounted for adolescent risk-taking, then most or all adolescents would be risk-takers. In fact, excessive risk-taking behaviors themselves are not normative (Steinberg,

2007). Thus, identifying the environmental and neurological mechanisms underlying risk-taking may increase understanding of the etiology of this risk. In turn, this increased understanding may inform the development of targeted HIV prevention programs and refinement of existing programs.

Theoretical and Conceptual Framework

Theory and research demonstrate that family characteristics, such as quality of relationships and amount or type of conflict, in conjunction with individual factors (e.g., neurobiological characteristics) influence the development of HIV risk behaviors (Brook, Brook, Richter, & Whiteman, 2006; Nation & Heflinger, 2006; Tinsley, Lee, & Sumartojo, 2004). It has been hypothesized that negative family interactions affect adolescents' ability to cope, leading them to seek substances or become associated with peer groups who engage in substance use and risky sexual practices (Simons & Robertson, 1989). Prior work testing a social learning model of adolescent substance use supports this hypothesis: rejecting-parenting directly and indirectly relates to an increased probability of adolescents' association with deviant peer groups, avoidant coping style, and use of substances (Simons & Robertson, 1989). On a more specific level, stress can impair pre-frontal cortical functioning (Arnsten, 1999; Qin, Hermans, van Marle, Luo, & Fernández, 2009): a brain region implicated in self-regulation. Thus, interpersonal stress between parents and adolescents may affect adolescents' decision-making.

The conceptual model (Tinsley et al., 2004) for my dissertation originated from a larger model of factors influencing adolescent HIV risk (**see Figure 1**). These theoretical models typically involve many factors that directly or indirectly relate to adolescent HIV

risk behaviors (e.g., Brook et al., 2006; Nation & Heflinger, 2006), such as parents' personality and substance use, and adolescents' associations with deviant peers. Further, macro-level factors such as stigma originating from socioeconomic disadvantage contribute to engaging in HIV risk behaviors (Prado, Lightfoot, & Brown, 2013). However, the focus of this project was on specific portions of this larger model (Tinsley et al., 2004): (a) family factors (e.g., parent-adolescent conflict) and (b) adolescent individual factors (e.g., neurobiological characteristics). In line with a robust line of work on the influence of situational factors on expressions of behaviors reflective of dispositional states (Mischel & Shoda, 1995), in this project I investigated a family-level situational factor on HIV risk-taking behavior, and tested a personality-related neurobiological factor as a moderator of this relation (**see Figure 2**). Specifically, in this model the interactive combination of high parent-adolescent conflict and relatively high levels among adolescents of a neurobiological marker for behavioral disinhibition is expected to increase risk for adolescent engagement in HIV risk behaviors.

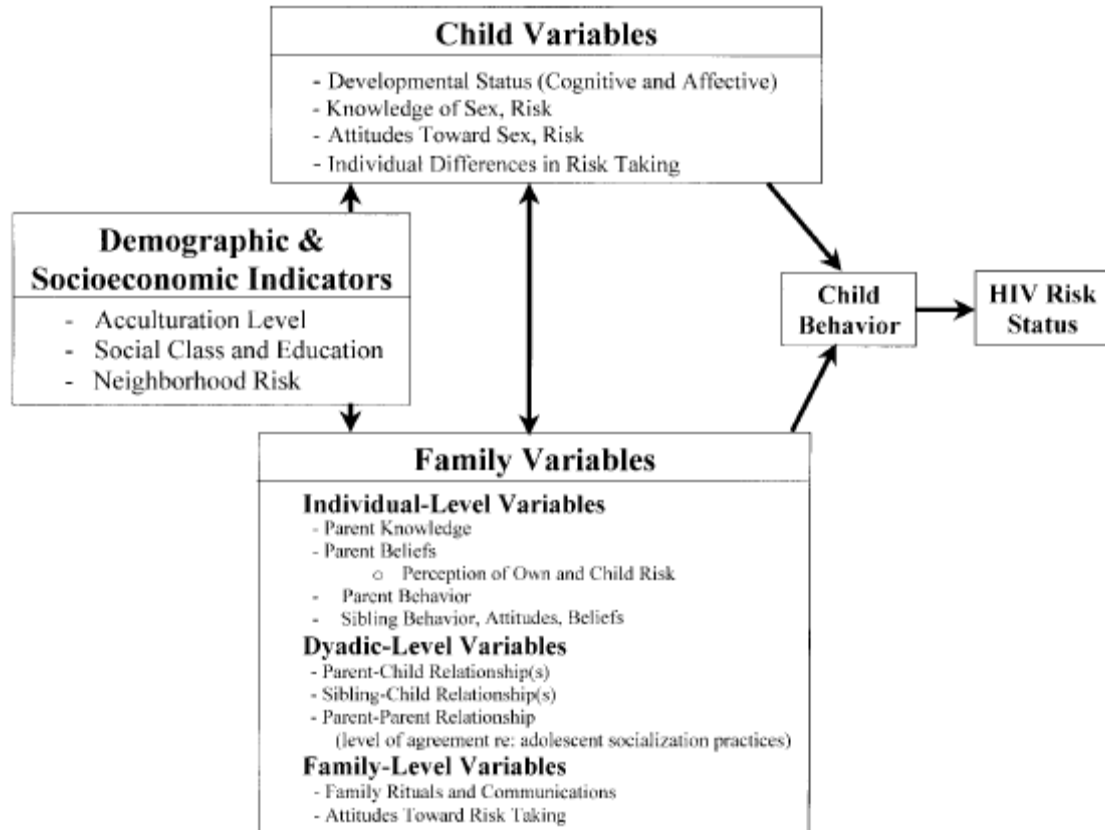


Figure 1. Individual and family processes affecting child HIV risk status.

Figure 1. Model of factors impacting HIV risk status (Tinsley, Lee, & Sumartojo, 2004, pg. 210)

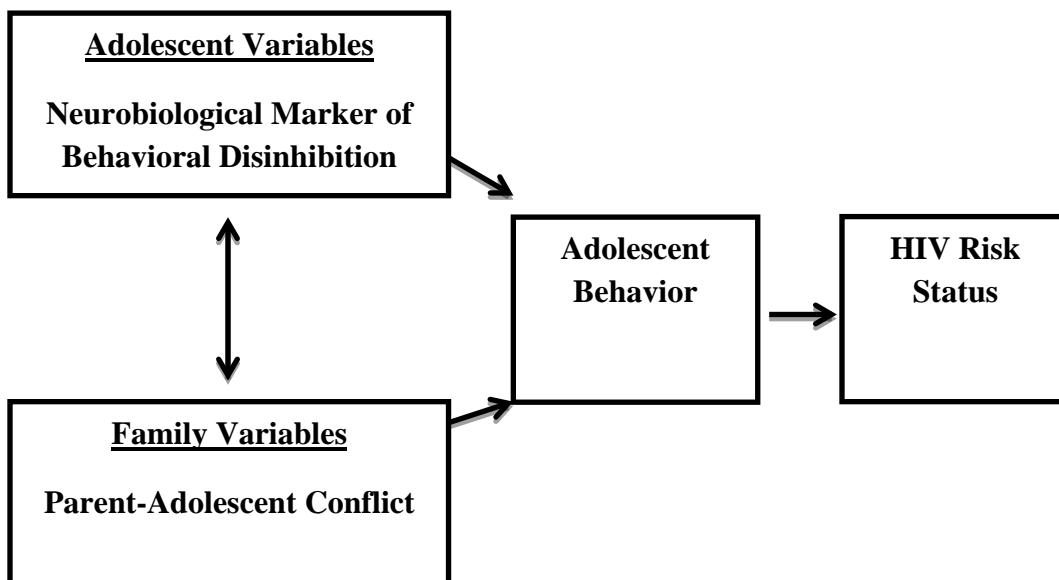


Figure 2. Hypothesized model of adolescent HIV risk in proposed project

HIV Risk Behaviors and Family Environment

The family environment provides an important context for understanding the development of youth's mental and physical health (Fisher, & Feldman, 1998; Repetti, Taylor, & Seeman, 2002; Tinsley et al., 2004). Despite the increase of peer influences as adolescents mature, parental influences nonetheless have a strong impact on adolescent development (Ary, Duncan, Duncan, & Hops, 1999; Brook, Whiteman, Gordon, and Brook, 1985; Krosnick & Judd, 1982). Family-level precursors have been significantly associated with risk-taking behavior. Specifically, parent-adolescent conflict (Ary, Duncan, Biglan, Metzler, Noell, & Smolkowski, 1999; Duncan et al., 1998) may place adolescents at risk for engaging in substance use, delinquency, and risk-taking, while the quality of the parent-adolescent relationship may protect against adolescent engagement in these behaviors (Anderson & Henry, 1994; Ryan, Jorm, & Lubman, 2010; Williams, & Steinberg, 2011). Moreover, parent-adolescent conflict is one of the most robust predictors of poor psychosocial outcomes in adolescence (Dmitrieva, Chen, Greenberger, & Gil-Rivas, 2004; Van Doorn, Branje, & Meeus, 2008), and thus it may be a proximal influence of HIV risk behaviors (Lam et al., 2007; Lester et al., 2010; Rowe et al., 2008). In fact, increases in parent-adolescent conflict from one time point to the next result in an increased rate over time of behaviors that pose risk for HIV, such as substance use (Duncan et al., 1998). However, some exposure to parent-adolescent conflict may be developmentally adaptive for adolescents. That is, conflict with parents may facilitate the development of adolescents' skills to manage conflict with others outside the family unit (e.g., peers and coworkers) (Laursen & Hafen, 2010). Further, although parents and adolescents experience regular disagreements together, only about 25% of parent-

adolescent dyads engage in maladaptive conflict (Rutter, Graham, Chadwick, & Yule, 1976). Taken together, the evidence points to a subgroup of adolescents for whom conflict increases likelihood of HIV risk behaviors. Still unknown is the mechanism by which parent-adolescent conflict is related to HIV risk behaviors—it is possible there is a causal relationship or that it is a correlate of another causal factor.

One possible mechanism by which increased parent-adolescent conflict relates to increased HIV risk behaviors is the influence of social stress on behavior. Indeed, social stress has been extensively studied in research on social support (e.g., Uchino, Cacioppo, & Kiecolt-Glaser, 1996), particularly in the marital conflict literature (Gottman & Levenson, 1992; Kiecolt-Glaser, Fisher, Ogrocki, & Stout, 1987). In particular, social stress impacts brain functioning (Arnsten, 1999; Qin et al., 2009). Specifically, increased stress relates to decreased prefrontal cortex activity (Arnsten, 1999), an area implicated in adolescent risk-taking due to its role in regulatory processes and executive functioning (Steinberg, 2007). For instance, during a stress induction administered within a functional neuroimaging task, researchers found that social stress (i.e., viewing aversive human images) dysregulates functioning in the dorsolateral prefrontal cortex (Qin et al., 2009). Further, a recent study found that increased heart rate reactivity (indexing a stress response) during a parent-adolescent conflict task was related to increased adolescent alcohol use (Chaplin et al., 2012).

The role of stress induction on adolescent risk-taking was also experimentally tested in a sample of older adolescents (Johnson, Dariotis, & Wang, 2012). Both the control and experimental groups completed baseline and follow-up risk assessments using the Balloon Analog Risk Task (BART; Lejuez et al., 2002), and only the

experimental condition experienced the stress induction, consisting of a modified stressful public speaking task. As expected, the stress-induction group demonstrated more follow-up risk-taking behavior. Further, adolescents' baseline BART risk-taking scores were used to create three categories of adolescents based on their risk-taking behaviors: Conservative, Calculated, and Impulsive. The Conservative category included low risk-takers (i.e., very few balloon pumps); the Calculated category included adolescents who exhibited high accuracy, high planning, and high risk-taking without cognitive impulsivity; and the Impulsive category included adolescents who exhibited poor accuracy, poor planning, high risk-taking, and high cognitive impulsivity. The follow-up BART risk-taking behavior was significantly related to adolescents' risk categorization. In comparing pre- to post-risk taking in each of the experimental conditions within the risk category, only the adolescents in the Impulsive category had a significant relation between the stressed and non-stressed condition. That is, Impulsive adolescents in the stress-induction condition exhibited significantly greater risk-taking behaviors (e.g., less planful and accurate) relative to the Impulsive adolescents in the no-stress condition. These results strongly suggest that 1) context matters with regard to how one expresses personality traits (Mischel & Shoda, 1995), 2) social stress is significantly temporally related to risk-taking behaviors in adolescents, and 3) impulsive adolescents are at even greater risk for engaging in risk-taking behavior. Importantly, impulsivity, a feature of externalizing disorders, is one of the most robust correlates of youth engagement in HIV risk behaviors (Donenberg & Pao, 2005; Mustanski et al., 2013).

Potential Neurobiological Moderator of Link between Parent-Adolescent Conflict

and HIV Risk Behaviors

Parent-adolescent conflict, externalizing disorders (e.g., Attention-Deficit Hyperactivity Disorder, Conduct Disorder, and Oppositional Defiant Disorder), and substance use disorders (SUDs) are related because they appear to share an underlying genetic vulnerability to expressing behavioral disinhibition (Iacono et al., 2008), with the presence of parent-adolescent conflict increasing risk for youth externalizing disorders (Burt, Krueger, McGue, & Iacono, 2003). Further, the relation is bidirectional, in that adolescent externalizing behavior may influence conflict, and conflict may worsen this behavior (Burt, McGue, Krueger, & Iacono, 2005). However, adoption studies have highlighted the importance of the shared environment in parent-adolescent conflict. For instance, genetic correlates of externalizing behaviors, while having explanatory value, nonetheless do not account completely for the relation between conflict and externalizing behaviors (Klahr, Rueter, McGue, Iacono, & Burt, 2011). Further, a recent review on impulsivity (Sharma, Markon, & Clark, 2014) concluded that dispositional trait behaviors alone do not entirely explain risky behavior, suggesting that the environment also plays an important role in negative outcomes (e.g., HIV risk behavior). Indeed, parenting behaviors (e.g., negativity, low warmth) have accounted for the link between genetic vulnerability and conduct problems in adolescents (Feinberg, Button, Neiderhiser, Reiss, & Hetherington, 2007).

One of the most robust predictors of the onset of externalizing behaviors is behavioral disinhibition, “an inability to constrain impulses to behave in socially undesirable ways” (Iacono et al., 2008, pg. 328). Similarly, the personality construct of behavioral undercontrol relates to early-onset adolescent drinking (McGue, Iacono,

Legrand, Malone, & Elkins, 2001). Along these lines, an inability to constrain one's behavior relates to increased risk for substance use disorders (SUDs) in late adolescence and early adulthood, and also relates to early SUDs onset (Elkins, King, McGue, & Iacono, 2006). Thus, behavioral disinhibition may reflect an inability to keep in mind long term goals to inhibit immediate, possibly harmful, behavioral responses impeding those goals (Patterson & Newman, 1993).

This research suggests that a broad factor related to psychopathology and personality, with genetic underpinnings for substance abuse risk, may be an important individual differences factor impacting engagement in HIV risk behaviors like substance abuse. A possible neurobiological marker for behavioral disinhibition¹ is an electrophysiological brain activity component referred to as P3. P3 is a positive-going brain potential thought to represent working memory updating and neuroinhibition (Polich, 2007), essential components of executive functioning. P3 amplitude is typically assessed while participants complete a task requiring sustained attention to stimuli presented on a computer screen (i.e., continuous performance task [CPT] or "oddball task"). A CPT consists of exposing a participant to frequent non-target stimuli and rare target stimuli, with instructions to the participant to enact a response when presented with rare stimuli (Polich, 2012). The P3 wave is elicited in response to rare stimuli (Polich, 2003). Participants evidencing a reduced P3 amplitude wave (P3AR) tend to also be those participants at risk for developing SUDs, and this risk occurs even in the absence of a diagnosis of an externalizing disorder (Iacono, Carlson, Malone, & McGue, 2002).

¹ This neurobiological marker has also been referred to as a neurobiological marker for externalizing proneness (Nelson, Patrick, & Bernat, 2011) and substance use problems (Yoon, Iacono, Malone, & McGue, 2006).

Consequently, P3AR has been proposed to be a neurobiological marker for behavioral disinhibition (Iacono, Carlson, & Malone, 2000). Importantly, P3 amplitude is stable across multiple measurements across time (Carlson & Iacono, 2006; Sinha, Bernardy, & Parsons, 1992). Further, P3AR is a robust neurobiological marker, indexing genetic risk for the development of later substance use problems (Hill, Steinhauer, Locke-Wellman, & Ulrich, 2009) and yet its measurement is not impacted by prior substance use (i.e., its presence is not due to an individual already having engaged in substance use; Perlman Johnson, & Iacono, 2009). Therefore, in my dissertation I examined whether levels of P3 moderate the relation between parent-adolescent conflict and adolescent risk-taking.

Methodological Factors Impacting the Study of Family-Level and Neurobiological Characteristics of HIV Risk Behaviors

Although the link between parent-adolescent conflict and HIV risk behaviors has been previously identified (Ary et al., 1999; Burt et al., 2003), the field has not yet moved toward understanding this link in terms of both assessment methods that approximate real-world circumstances and determining mechanisms of this relation. One barrier to understanding these mechanisms may be how researchers have assessed conflict. First, previous research on the role of parent-adolescent conflict in adolescent problem behaviors has used subjective survey methods to assess conflict; this may be problematic because adolescents and parents often have different definitions of what constitutes “conflict” (i.e., resulting in uncertain conflict ratings; De Los Reyes et al., 2012a). Second, researchers often assess conflict by constructing laboratory tasks to observe parents and adolescents discussing topics that tend to elicit conflict in the home. Yet,

recent work indicates these paradigms do not successfully elicit reliable stress responses from adolescents (Gunnar, Talge, & Herrera, 2009). Without an ecologically valid conflict task paradigm, it is difficult to investigate why parent-adolescent conflict robustly predicts negative outcomes.

A recent study used a structured interview that was designed to elicit reports from parents and adolescents on topics about which they engage in behavioral manifestations of conflict (i.e., arguing or fighting about chores), as well as reports on topics about which they hold different subjective impressions (i.e., hold different beliefs about whether adolescents should complete chores). In this study, the To(may)to-To(mah)to Interview (TTI; De Los Reyes et al., 2012a; De Los Reyes, & Suarez, 2009), was used to examine whether a laboratory conflict task based on reports from this interview could successfully elicit stress responses from adolescents (Thomas, et al., 2014). Pilot data from this conflict task indicate that the task reliably elicits a stress response among adolescents. Specifically, in an uncontrolled pre-post study, 32 parents and adolescents aged 14-17 discussed for six minutes each two conflict topics identified with the TTI. In a “control” discussion administered before the conflict discussion, the same parents and adolescents planned a vacation. Heart rate variability (HRV) measurements during all discussions indexed adolescents’ stress responses. Lower HRV levels (i.e., a marker for greater stress responses; Allen, Chambers, & Towers, 2007; Berntson, Cacioppo, & Grossman, 2007) were observed for adolescents during the conflict task, relative to the control task. These findings indicate that this conflict task paradigm should be able to reliably produce a physiological stress response for adolescents.

Further, using performance-based measures like this conflict paradigm would

greatly enhance the assessment of parent-adolescent conflict and HIV risk behaviors. To date no prior studies have addressed whether parent-adolescent conflict is a correlate of or temporally related to HIV risk behaviors; therefore, a paradigm designed to address the question of temporality using performance-based measures is essential in determining the direction of the effect and ruling out other causes (e.g., Odgers et al., 2008).

Performance-based measures include an analogue risk-taking task that relates to real-world substance use (BART-Y; Lejuez, Aklin, Zvolensky, & Pedulla, 2003; Lejuez et al., 2007), as well as a performance-based task that measures behavioral disinhibition while recording electrophysiological brain activity. Thus, modifying the methods used to study factors impacting adolescent HIV risk behaviors may enable greater understanding of the mechanisms underlying these factors.

Summary and Overall Significance

Overall, parent-adolescent conflict plays an important role in adolescents' engagement in HIV risk behaviors (Lester et al., 2010; Rowe et al., 2008). Further, dispositional traits, such as behavioral disinhibition and impulsivity, pose risk for adolescent HIV risk behaviors (McGue et al., 2001). In fact, the broad externalizing trait is one of the most robust factors influencing adolescent engagement in HIV risk behaviors (Donenberg & Pao, 2005; Mustanski et al., 2013). It is imperative not only to understand which factors contribute to adolescents' greater propensity for engagement in HIV risk behaviors, but also to investigate the mechanisms by which these factors exert their influence. To date, studies have not investigated the causal link between parent-adolescent conflict and HIV behaviors using performance-based and real-world measures

of risk taking and conflict in adolescents. In this research project, I examined the temporal relation between parent-adolescent conflict and HIV risk behaviors, and measured the extent to which a neurobiological marker moderates the relation between family factors and HIV risk behaviors.

Primary Aims and Hypotheses:

Aim 1. Determine that the discussion task conditions (vacation vs. conflict) differ on adolescent psychophysiology and observed behavior as a manipulation check that the conflict discussion condition elicits a greater stress response than the vacation discussion condition.

- Adolescents in the conflict discussion condition will exhibit greater psychophysiological reactivity (i.e., heart rate) during the conflict task, relative to the vacation discussion task.
- Adolescents in the conflict discussion condition will exhibit greater hostility behavior and reduced attachment behavior during the conflict task, relative to the vacation discussion task.

Aim 2. Determine if there is a temporal link between parent-adolescent conflict and HIV risk behaviors.

- Adolescents in the conflict discussion condition with their parent will evidence significantly more post-task risk-taking on a performance-based measure of risk relative to adolescents in the control condition.

Aim 3. Investigate the extent to which P3AR moderates the relation between parent-adolescent conflict and HIV risk behaviors.

- Adolescents who evidence P3AR will have a stronger relation between parent-adolescent conflict and adolescent risk-taking than adolescents who do not evidence P3AR.

Chapter 2: Method

Overall Design

My study involved two visits: The Behavioral Visit used a between-subjects design to examine the effect of parent-adolescent discussion condition (control versus conflict) on adolescent HIV risk behavior. The independent variable was the conflict discussion condition (between subjects; control topic vs. conflict topic). The dependent variable was HIV risk behavior, measured using a performance-based laboratory task assessing risk (BART-Y; Lejuez et al., 2007). The EEG Visit assessed a neurobiological marker of behavioral disinhibition (P3AR) as a moderator for the relation between parent-adolescent conflict and engagement in HIV risk behavior. All study protocol and procedures were approved by the University of Maryland Institutional Review Board (IRB).

Aims 1 and 2: Behavioral Visit

This portion of my dissertation study is based on a pre-doctoral National Research Service Award training grant from the National Institute on Drug Abuse (NIDA; 1F31DA033913-01; Sponsor: Andres De Los Reyes, Co-Sponsor: Carl Lejuez) with funding dates from 7/15/2012-7/15/2015. Forty-nine parent and adolescent dyads completed this visit.

Participants and recruitment. Randomized participants were 51 adolescents between the ages of 14-17, and their parent. One adolescent was excluded during the study due to his parent acknowledging he did not meet the age criteria, despite previously

endorsing his age as within the study limit. A memo was submitted to the IRB to report this incident and a multi-step gating process was put in place to ensure this did not occur again. The parent of a second adolescent declined to consent to the study before they participated but after they were randomized, resulting in a final sample of 49 parent-adolescent dyads (see Figure 3 for enrollment flow diagram). Participants initially were recruited with their parent from an ongoing multiwave longitudinal study (PI: Dr. Lejuez) which focuses on the development of risk taking in youths (“parent study”). Adolescents participating in the parent study were recruited from media and community outreach; the two main inclusionary criteria were that they were in the targeted age range and spoke English. At the time of recruitment, the larger study’s sample was approximately 50% African American and 54% male. Participants were asked by research personnel affiliated with the parent study if they were interested in participating in the proposed study. Those participants who expressed interest left contact information for research personnel affiliated with the proposed study to contact them and describe study procedures. Research personnel scheduled with interested participants their visit to the laboratory.

In order to be eligible for participation in the present study, families had to speak English, understand the consent/assent process, have an adolescent living at home who did not have a reported history of substantial developmental, learning, or psychotic disorders, and be available to come in for an assessment with both parent and adolescent. Forty-nine parent-adolescent dyads completed the Behavioral Visit assessment. Adolescents had been enrolled in the parent study for an average of 3.57 years ($SD = 2.51$; range 1-8). Demographic characteristics of the Behavioral Visit sample, divided by discussion condition, are provided in Table A1. Adolescents ranged in age from 14-17

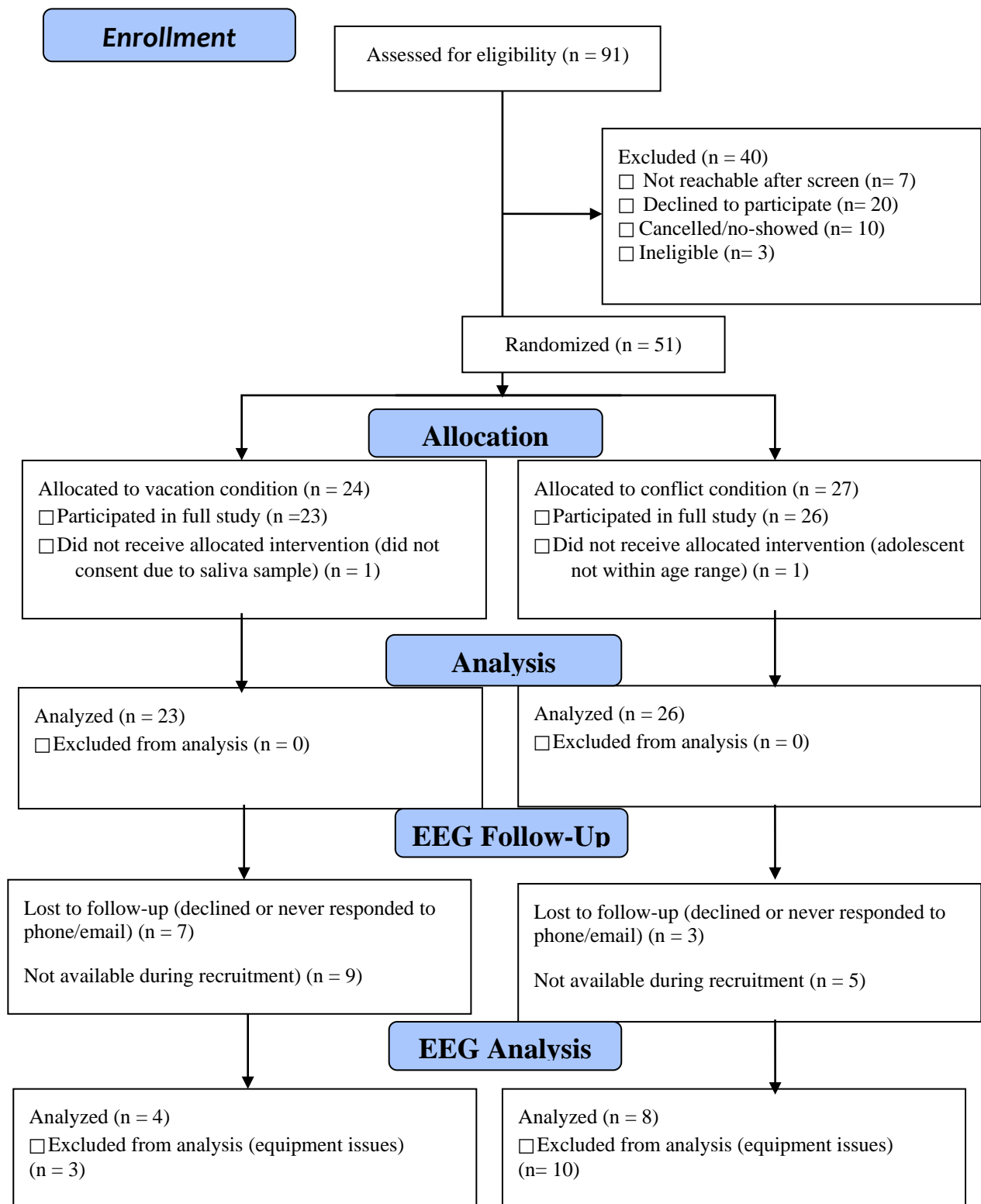


Figure 3. Behavioral Visit participant enrollment table.

(24 males and 25 females; M age = 14.96 [SD = 1.02]). Parents reported the adolescent's ethnicity/race as African American or Black (65.3%); White, Caucasian American, or European (36.7%); Hispanic or Latino/a (2.0%); or Asian American (2.0%). The composition of family ethnicity/race totals above 100% because there was overlap among the ethnic/racial categories. Participants were able to select more than one ethnic/racial category. Adults were: 9 biological fathers (18.36%), 38 biological mothers, (77.55%), one adoptive mother (2%), and one grandmother (2%). All caregivers reported completing at least a high-school education. According to parent-report, 20.4% of the families had a weekly household income of \$500 or less; 36.7% had a weekly income between \$501 and \$900; and 42.9% earned \$901 or more per week.

Procedures. Once a family was identified for participation, study personnel explained via telephone the purpose and procedures of the study and answered all questions before scheduling a visit. At the start of their visit, families provided informed consent/assent and were assigned a participant number that was listed on all data forms. I ensured that participants understood all facets of the consent/assent forms and the study. Video and audio recordings of assessments using Noldus Observer XT were taken for later behavioral coding. Two families declined to be video recorded; consequently, there is no observational behavioral data for these families during the discussion task. Adolescents completed a pre-task BART-Y and a survey measure at the same time as their parent completed a battery of survey measures (see **Figure 4**). Parents and adolescents separately took part in a structured interview (TTI) to identify conflict topics, and then were randomly assigned to complete together a 5-minute discussion task of either a conflict topic or a “control” topic (plan their dream vacation). Immediately after

the discussion task, adolescents completed a post-task BART-Y. Participants wore heart rate (HR) monitors throughout their discussion task. The Behavioral Visit characterized the relation between parent-adolescent conflict and HIV risk-taking. Participants were reimbursed for their time commitment (\$30 each to parents and adolescents), and adolescents won a small, medium, or large prize at the completion of each BART-Y, for a total of two prizes.

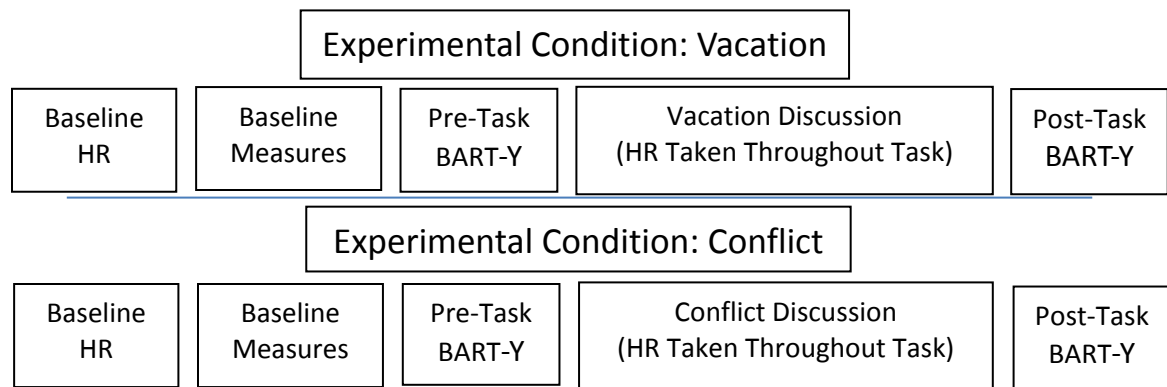


Figure 4. Behavioral task session timeline; HR = heart rate; BART-Y = Balloon Analogue Risk Task.

Measures. Assessments were focused on five domains: **(1)** demographics, **(2)** adolescent psychophysiological regulation, **(3)** parent-adolescent conflict, **(4)** adolescent analog and real-world risk behaviors, and **(5)** parent-adolescent relationship characteristics.

Demographics. This parent-completed survey assessed demographics including age, gender, race/ethnicity, education level, marital and employment status, and household income (see Table A1).

Adolescent psychophysiological reactivity. As a manipulation check on whether the conflict condition elicited a greater stress response than the control condition, I took

peripheral measures of adolescents' psychophysiology (De Los Reyes et al., 2012b) throughout the tasks. Adolescents wore monitors to track their heart rates (i.e., Polar Electro RS800CX; Anderson & Hope, 2009). The equipment involves a watch worn on the wrist of their non-dominant hand, and an elastic band around their ribcage that is worn under their clothes. After the consent/assent process, adolescents met with a research assistant individually to learn how to apply the heart rate monitor. Participants were provided with a secure changing area within the laboratory in which to apply their heart rate monitors. Heart rate was measured during a 5-minute baseline period during which adolescents sat in a chair alone in a room and were instructed to rest. During the discussion task, I measured adolescents' heart rate as an index of psychophysiological stress response. Specifically, from the measurements of adolescent HR, I created data points consisting of the average HR during the 5-minute segments for both the conflict conditions and the vacation conditions, similar to other methods used in parent-adolescent conflict tasks (Chaplin et al., 2012).

Parent-adolescent conflict topics. The To(may)to-To(mah)to Interview (TTI; De Los Reyes et al., 2012a; De Los Reyes, & Suarez, 2009) reliably and validly assesses 1) behavioral conflict about daily life topics, and 2) parent and child perceptions of whether discrepancies exist between their beliefs about these topics. This structured interview included youth and parent versions that gather informants' reports of parent-child conflict and parent-child discrepant beliefs on 16 daily life topics (e.g., youth's computer time, spending time with the family). The 16 topics were derived from research in the adolescent development literature on topics of parent-adolescent disagreement (e.g., youth's computer time, spending time with the family, quality of grades; whether parents

like youth's friends; Darling, Cumsille, Caldwell, & Dowdy, 2006). After the introduction, the interviewer solicited informants' responses for items on scales of "0" (Value labels represent the quantity "None"), "1" (Value labels represent the quantity "Some"), and "2" (Value labels represent the quantity "A lot"). The scale labels were worded for their relevance to separate items for each topic that assess self-perceptions of the current status of topics (e.g., How often does your child get to listen to music at home?), parent-child conflict (e.g., How often do you and your child argue or fight about your child getting to listen to music at home?), and perceptions of whether parents and their children have different beliefs on how things should be (e.g., Do you think that you and your child have different beliefs about how often children your child's age should get to listen to music at home?). Two interviewers were available for each parent-child dyad so that both were administered the interview simultaneously. Table A2 presents the means, standard deviations, and internal consistency estimates for parent and adolescent behavioral conflict and discrepant beliefs. Coefficient alpha estimates ranged between .72 and .86, demonstrating good internal consistency.

Conflict discussion task. Parents and adolescents engaged in a widely used 5-minute conflict discussion task (Gottman, 1979), with the goal of coming to a resolution on the topic. As part of the experimental testing of parent-adolescent conflict on HIV risk behaviors, in the between-subjects design, families in the Control Condition ($n = 23$) were randomly assigned the discussion topic of planning a vacation; families in the Conflict Condition ($n = 26$) were randomly assigned the discussion of a conflict topic, based on previously identified topics from the TTI (see Appendix B for frequencies of conflict topics). At the conclusion of the TTI, for families randomized to the conflict

condition, the conflict discussion topic was chosen immediately from the adolescent's TTI responses. The topic selection was prioritized along a continuum according to the level of behavioral conflict present: that is, a topic about which an adolescent reported s/he and the parent argue or fight was prioritized first for selection, with the least desirable topic for parents and adolescents to discuss being one in which they do not argue or fight about and do not have different beliefs. This is because I expected topics about which adolescents report that they and their parents argue or fight to elicit the greatest adolescent stress response. Once the topic was identified (i.e., dream vacation or a specific TTI-identified conflict topic), a research staff member introduced the topic to the parent and adolescent, elicited a brief description of how this topic comes up for each of them, and then explained that they would like the parent and adolescent to come to a resolution on the details of the topic for 5 minutes. The research staff member left the room during the discussion task and returned when the 5 minutes had elapsed.

Coding of associated conflict behaviors. As an additional manipulation check, parents' and adolescents' behaviors during the discussion task were classified using observational coding methods. During the discussion task, two video cameras in the room were focused on the adolescent and the parent, respectively, resulting in two video files that enabled separate coding of parent and adolescent behavior. The two families who declined video-taping were not included in the coding of conflict behaviors. Additionally, video-recording difficulties resulted in 1 parent video being excluded. Research staff used observational methods to code for attachment related behaviors as an indicator of parent-adolescent relationship characteristics. I focused on two types of behaviors: hostility behaviors, as an index for negative interactions during conflict discussions, and secure-

base behavior, as an index of parent-adolescent validation and support of one another's perspective on the discussion topic (e.g., Ehrlich, Dykas, & Cassidy, 2012). The Hostile Conflict Scale and Secure Base Use/Provision Scale of the Conflict Task Coding System, a macro coding system, was used by four trained coders (Ziv, Cassidy, & Ramos-Marcuse, 2002). Behaviors are rated on a 7-point anchored scale (i.e., 1 to 7), with higher scores indicating more of that behavior. Behaviors coded as hostile include nonverbal behaviors such as negative facial expressions, as well as verbal behaviors like yelling, mocking, interrupting, and rejecting what the other person is saying. High scores (e.g., 5 to 7) are reserved for intense hostile behavior which includes character assassinations, globalizations (e.g., "you *never* care about what I say"; "you *always* expect other people to clean up after you"), or persistent negative hostile interactions throughout the 5 minute task. Behaviors coded as secure base (Allen et al., 2003) differ according to adolescent and parent. For adolescents, secure base use means that they express their emotions to their parent during the task, accept their parent's support, seek advice and emotional comfort from their parent, and demonstrate that they are comfortable sharing their experiences and feelings with their parent while using their parent as a source of comfort and support. Nonverbal secure base use includes eye contact, smiling, and nodding. For parents, secure base provision is demonstrated by active listening to their adolescent, providing emotional and instrumental support, validating the adolescent's perspective and emotions even when there is disagreement (e.g., "I understand it's frustrating to you that you have to clean when you get home from school but we all have to pitch around the house"), and nonverbal actions such as smiling and nodding.

Four research assistants served as the coders: one coder each was trained on

coding parent behavior or adolescent behavior, and two coders were trained on coding both parent and adolescent behaviors. First, coders were trained to agreement on the behavioral coding system using archived videos from a similar laboratory coding task. All coders had to have average scores with less than 1 point difference from the “true” scores from the training videos before they could progress to the coding of the videos in the current project. Coders who rated both parent and adolescent had to reach this training threshold for each coding scheme (parent and adolescent). From the current coding videos, approximately 42% of adolescent videos and 39% of parent videos were randomly selected as reliability cases (i.e., assigned to two coders) in order to check that coders were rating behaviors similar to one another, consistent with their training. Coders were blind to which cases would be used for reliability prior to coding them. I held weekly reliability meetings (one meeting for parent coding and one meeting for adolescent coding) where reliability cases were discussed and viewed. Coders provided justification for their ratings if they were discrepant from one another. For these reliability cases, I made the final decision on ratings when necessary (e.g., coders could not agree or it was clear both coders had not rated the behavior accurately). Final ratings for the reliability cases were what were used in the statistical analyses for the aims of the study. For reliability cases, interrater reliability was calculated using intraclass correlation coefficients (ICCs) for “average measures”. Means, standard deviation, and interrater reliability estimates are presented in Table A3. For adolescent hostility, the ICC was .39, which is quite low and unexpected. The average mean rating for both ratings for adolescent hostility was 1.5, and conducting a paired t-test showed no significant differences in how the videos were rated. One case had hostility ratings of 1 and 4, and

by excluding that one reliability case, the ICC rose from .39 to .73, suggesting that it was strongly influenced by one discrepant case.

Adolescent analog risk-taking. At the Behavioral Visit, I assessed adolescent risk-taking using the BART-Y (Lejuez et al., 2007), a performance-based measure of adolescent risk-taking behavior. Participants inflated 30 computer-generated balloons, one at a time. Each pump is worth one point, but if the balloon is pumped past its explosion point, then all points accrued for that balloon are lost. The probability that any particular balloon will explode is 1/128 for the first pump, 1/127 for the second pump, and so on until the 128th pump at which point the probability is 1/1. Thus, explosion values form a normal distribution around 64 pumps (Lejuez et al., 2002). During the task, participants had the opportunity to stop pumping the balloon at any time prior to an explosion and allocate the accrued points to a permanent prize meter. After a balloon exploded or points were allocated to the permanent prize meter, a new balloon appeared. After completing 30 balloon trials, the position of the prize meter determined the final prize. Adolescents completed this task at baseline (“pre-task”), and then immediately after engaging in a discussion task with their parent (“post-task”). BART-Y risk-taking was quantified as adjusted number of pumps across balloons (Lejuez et al., 2002) to account for some trials ending earlier than others (i.e., balloon explosions). Higher scores index greater risk-taking.

Assessing condition equivalency on and correlates of real-world risk-taking.

The following survey was used a) to assess condition equivalency on real-world risk-taking, and b) to conduct exploratory analyses of links between real-world risk-taking and observed risk-taking as assessed on the BART-Y.

Real-world risk-taking behaviors. To examine prevalence of real world risk behaviors, including adolescent substance use, I used a modified shortened version of the Youth Risk Behavior Surveillance System (YRBS; Centers for Disease Control and Prevention, 2002). This data was collected as part of the adolescents' most recent visit to the parent study prior to the Behavioral Visit. A composite score including both risk behaviors and substance use has been previously published (Aklin, Lejuez, Zvolensky, Kahler, & Gwadz, 2005; Lejuez et al., 2007; MacPherson et al., 2010). In total, the shortened measure assessed risk taking behaviors across the domains of risky behavior, drug and alcohol use, and risky sexual behavior. Items were rated on a scale from "0" to "5", with the range of frequency of engagement in behavior including "zero", "once", "a few times", "1-3 times per month", "1-3 times per week", and "almost every day or more". Because participants in this sample endorsed risk items at a low base rate, the responses were dichotomized: "0", indicating never engaged in that behavior, and "1", indicating they had engaged in that behavior, to any degree. For behaviors for which participants endorsed engaging in them at a relatively higher rate, the scores were dichotomized using a median split to create as equal as two groups as possible, with 0 indicating "low" engagement in that behavior, and 1 indicating "high" engagement in that behavior. Consistent with prior research (Aklin et al., 2005; Lejuez et al., 2007; MacPherson et al., 2010), scores were then summed to create a composite score, which will be referred to as YRBS Risk Score. Table A4 contains the means, standard deviations, and reliability for risk-taking behavior. Because the YRBS Risk score violated distributional assumptions, I used a square root transformation whenever it was used as a predictor variable. Appendix C includes the list of behaviors and how they were

dichotomized.

HIV risk behaviors. To measure behaviors that increase adolescents' risk for HIV infection, responses on the YRBS were used to create a composite score of HIV Risk. Only one behavior overlapped with the YRBS score described above ("past year, intercourse with no condom"). Behaviors included sexual behavior and substance use behavior that puts one at risk for contracting HIV. A full list of the behaviors included in this score is available in Appendix C. Similar to calculating the YRBS Risk Score, adolescent responses were dichotomized according to whether they had never engaged in the behavior ("0"), or ever engaged in the behavior ("1"). Internal consistency estimates for this composite measure, referred to as YRBS HIV Risk Score, and the YRBS Risk Score were good—see Table A4. Because the YRBS HIV Risk score violated distributional assumptions, I used a square root transformation whenever it was used as a predictor variable. After transformation, skewness and kurtosis were within acceptable range (1.45, 1.00, respectively).

Assessing condition equivalency on Parent-adolescent relationship characteristics.

The following surveys were used to assess condition equivalency on parent-adolescent relationship characteristics.

Parenting quality. To examine parenting behavior, I used two scales from the parent report of the Alabama Parenting Questionnaire (APQ; Frick, 1991): the Total Positive Parenting Score and Total Negative Parenting Score. The APQ is used to assess parenting behaviors related to involvement, monitoring, positive attention, and punishment. Specifically, the Total Positive Parenting Score includes items such as "You

let your child know when he/she is doing a good job with something.”, and “You ask your child what his/her plans are for the coming day.”. The Total Negative Parenting Score includes items such as “You threaten to punish your child and then do not actually punish him/her.”, and “Your child stays out in the evening past the time he/she is supposed to be home.”. Scores range from “1” to “5”, representing “Never” to “Always”. Means, standard deviations, and reliability are presented in Table A5.

Adolescent disclosure. To index adolescent relationship quality, I used the subscale Adolescent Disclosure (i.e., adolescent’s report) from a measure of parental monitoring completed by adolescents and parents (Kerr & Stattin, 2000). Adolescent Disclosure is the extent to which adolescents tell their parents about their activities and includes items such as “Do you talk at home with your caregiver about how you are doing in the different subjects in school” and “If you are out at night, when you get home, do you tell what you have done that evening?”. Scores range from “1” to “5”, representing a range of frequency such as “Most of the Time” to “Almost Never”. Two items in the Adolescent Disclosure Scale were reverse coded so that all items were scored in the same direction. Specifically, the Adolescent Disclosure scale has been shown to longitudinally predict not only parental knowledge of adolescents’ activities but also changes in delinquency over time, such that as disclosure decreases, delinquency increases (Kerr, Stattin, & Burk, 2010). Means, standard deviations, and reliability are presented in Table A5.

Aim 3: EEG Visit

This portion of my dissertation study is based on a pre-doctoral National Research

Service Award training grant from the National Institute on Drug Abuse (NIDA; 1F31DA033913-01; Sponsor: Andres De Los Reyes, Co-Sponsor: Carl Lejuez) with funding dates from 7/15/2012-7/15/2015; a Psi Chi Graduate Research Grant, with funding dates from 2/15/2013-8/30/2014; and an Elizabeth Munsterberg Koppitz Child Psychology Graduate Student Fellowship, with funding dates from 3/24/2014-5/29/2015. Twenty-five adolescents completed this visit.

Participants and recruitment. In a visit occurring after the Behavioral Visit, adolescents completed a CPT task while EEG recordings were taken. Adolescents were recruited from Aim 1. Adolescents were eligible to participate in the EEG Visit if: 1) both adolescent and parent indicated on the consent/assent forms completed in the Behavioral Visit that they were interested in being contacted for an optional follow-up study; 2) they had no history of a major head injury; and 3) they were available during the recruitment period (June 2014 to September 2014). Because some adolescents were 17 years old when they completed the Behavioral Visit and had since turned 18, the age range for the EEG Visit was expanded to 14-18. Once an adolescent was identified for participation, study personnel contacted the parent by telephone to explain the purpose and procedures of the study and answered all questions before scheduling a visit. If the parent was interested in the adolescent participating in the visit, study personnel also spoke with the adolescent to explain the procedures and to obtain confirmation that they were interested in completing these activities. Participants were recruited at a rate of approximately 2 EEG sessions per week. Participants were no longer recruited starting in October 2014 due to EEG equipment issues that necessitated the return of the equipment for servicing to BrainVision LLC. At the start of the EEG visit, families provided informed

consent/assent and used the same participant number from the Behavioral Visit that was listed on all data forms. I ensured that participants understand all facets of the consent/assent forms and the study.

Procedures. Adolescents completed a visual oddball task (CPT; described below) while electrical brain activity was recorded. Adolescents sat in a chair 100 cm from a video monitor on which stimuli from the oddball task (Begleiter, Porjesz, Bihari, & Kissin, 1984; Nelson, Patrick, & Bernat, 2011) was presented visually using E-prime Version 2.0. Adolescents first completed a practice task to attain 80% or greater accuracy on responding to the rotated heads stimuli. Capping the adolescents took between 20-30 minutes and the visual oddball task took 18 minutes. Adolescents were compensated \$40 for their time commitment, \$5 for a prize, and parents were compensated \$10 for transportation costs.

Measures.

Neurobiological risk for substance use.

Oddball task. The task adolescents completed while undergoing EEG recordings is a visual oddball task (“rotated heads paradigm”; e.g., Begleiter et al., 1984; Nelson et al., 2011). This 3-stimulus task consists of 240 trials of rapidly-presented frequent non-target stimuli (70%), rare target stimuli (15%), and novel non-target stimuli (15%). Stimuli were presented for 100 ms, and the intertrial interval between stimuli was between 1-2 s. Frequent non-target stimuli consisted of an oval. The rare stimuli consisted of a head with a nose and an ear in which the participant indicates with a Psychology Software Tools button box which side of the head the ear is on during a 1.5 s response period. The novel non-target stimuli are affective pictures that consisted of

pleasant pictures and unpleasant pictures, the majority of which were chosen from the International Affective Picture System (Center for Study of Emotion and Attention, 1999). Pleasant pictures were from the following categories: action (e.g., skydiving), nurturing (e.g., grandparents and grandchildren), and erotic (e.g., clothed couples and male/female individuals in undergarments). Unpleasant stimuli were threatening (e.g., snake poised to strike, gun pointed at camera). For the purpose of these analyses, only non-target and rare target stimuli were used in calculations.

EEG data acquisition and processing. Electrical brain activity was recorded using an EEG system (BrainVision LLC) and amplified/digitized using actiCHamp amplifiers (BrainProducts, Germany). EEG was recorded using actiCAP active electrodes and Pycorder recording software (BrainVision LLC). Data was processed and analyzed using BrainVision Analyzer 2 software, Matlab scripts, and EEGLAB software. EEG activity was recorded from 64 Ag/AgCl scalp electrodes. One electrode each was placed above and below participants' left eye, and on the outer edges of both eyes, to record eye blink muscle activity. During recording, scalp electrode Cz was used as the reference. After recording, electrodes were then re-referenced offline to the linked mastoids (electrodes TP9 and TP10). EEG recording settings were as follows: low frequency filter settings were .01 hz; high frequency filter settings were 200 hz; and sampling rate was 1000 hz. P3 was calculated from 3 parietal electrodes: PZ, P1, and P2. Epochs of three seconds were then taken from 1000 milliseconds (ms) pre-stimulus to 2000ms post-stimulus with a 150ms pre-stimulus baseline, and were averaged according to stimulus type (target or non-target). Two methods of data cleaning were then used. In the first method, trials were rejected if activity at electrodes F3 or F4 exceeded $\pm 100 \mu V$ in either the pre-stimulus

period of -1000 to -1ms, or the post stimulus period of 1 to 2000ms. Within-trial individual electrodes were rejected if activity exceeded $\pm 100 \mu\text{V}$ during the same pre- and post-stimulus time periods. Consistent with prior research (Gilmore, Malone, Bernat, & Iacono, 2010; Yoon et al., 2006), P3 amplitude was extracted from the different averaged stimulus types (target and non-target) and calculated using the peak-in-window approach, using the time frame of approximately 280ms to 625ms post-stimulus, where the window included the non-target and target P3 peak. P3 was quantified using mean voltage (Luck, 2005) and calculated by subtracting non-target P3 amplitude from target P3 amplitude and amplitude. Blink artifacts were corrected using regression (Gratton, Coles, & Donchin, 1983). Adolescents with improbable or excessively “noisy” data (e.g., P3 occurring at unexpected time) were not included in analyses. Because of equipment difficulties that introduced substantial noise into the recordings, 13 adolescents had to be excluded from data analyses. Figure 5 presents the grand-averaged P3 waveform for Targets.

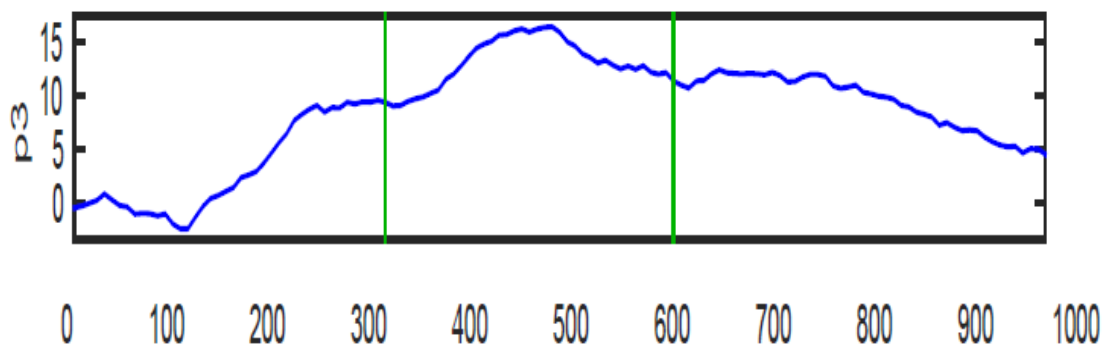


Figure 5. Grand average waveform for P3 Target stimuli across participants from EEG Visit. P3 window is identified between green lines. Time is in milliseconds on the x-axis. Amplitude is in μV is on the y-axis.

Data Analysis

Overview. Data was entered and analyzed using SPSS. When completing questionnaires during the Behavioral Visit, participants responded using Qualtrics online surveys, which enables data to be exported in SPSS format. Participants were required to answer a question before advancing to the next question. All data was double-entered, compared, cleaned, and verified. Descriptive statistics were performed to check distributional assumptions and outliers. For continuous independent variables that deviated from absolute values of approximately 1 for skewness and kurtosis, a data transformation was performed (Tabachnick & Fidell, 1996). Based on prior work, I included age and gender as between-subjects demographic covariates (De Los Reyes et al., 2012a).

Aim 1. I tested the hypothesis that adolescents in the conflict discussion condition would exhibit a greater stress response relative to adolescents in the vacation discussion condition. I expected adolescents assigned to the conflict discussion condition with their parent to evidence significantly greater psychophysiological reactivity, greater hostility behavior, and reduced secure base use, relative to the vacation discussion task. Thus, I conducted separate independent samples *t*-tests with discussion condition as the independent variable and mean HR, Hostility, and Secure Base Use as the dependent variables.

Aim 2. I tested the hypothesis that there is a temporal relation between parent-adolescent conflict and risk-taking behaviors. I expected adolescents assigned to the conflict discussion condition with their parent to evidence greater risk-taking post-discussion compared to adolescents assigned to the vacation condition. Thus, I used

generalized estimating equations (GEE), an extension of the general linear model that assumes correlated observations of dependent and/or independent variables (Hanley, Negassa, Edwardes, & Forrester, 2003) to statistically model the two BART-Y assessments as a repeated-measures (i.e., one measure at pre-task and another post-task) dependent variable varying as a function of: (a) a within-subjects factor of Time (i.e., pre- vs. post-BART-Y risk-taking), (b) Condition (i.e., vacation discussion vs. conflict discussion), and (c) Time \times Condition interaction. I expected to observe 2 X 2 interaction effects: there will be significant differences between pre- and post-task risk taking, but only for adolescents in the Conflict Topic condition.

Aim 3. I tested the hypothesis that a neurobiological marker for behavioral disinhibition moderates the association between parent-adolescent conflict and HIV risk behaviors. The model for Aim 3 was run separately from Aim 2 because the number of participants differed. I expected adolescents in the conflict condition to show greater changes in risk than adolescents in the control condition, but those changes would largely be accounted for by the adolescents with P3AR. I tested this hypothesis using GEE to statistically model the two BART-Y assessments as a repeated-measures (i.e., one measure at pre-task and another post-task) dependent variable, varying as a function of seven factors: (a) a within-subjects factor of Time (i.e., pre- vs. post-BART-Y), (b) Condition (i.e., vacation discussion vs. conflict discussion), (c) P3 (i.e., mean amplitude in voltage), (d) Time \times Condition interaction, (e) Time \times P3 interaction, (f) Condition \times P3 interaction, and (g) Time \times Condition \times P3 interaction. I tested main and interaction effects. Because P3 is a continuous variable, I probed the interaction effects by using one standard deviation above and below the means to approximate high and low groups, with

the low group representing P3AR (Holmbeck, 2002).

Condition equivalency on real-world risk-taking, and exploratory analyses.

First, using the measures of real-world risk behaviors, I demonstrated that the two conditions of the discussion task were equivalent on baseline performance-based risk taking. Second, as exploratory analyses to determine whether these hypothesized mechanisms applied to real-world risk behaviors in which the participants engage, I conducted separate hierarchical linear regression analyses with these measures of real-world risk taking serving as criterion variables (i.e., YRBS Risk; YRBS HIV Risk). In the first step of each analysis, I entered P3 and a dichotomous variable representing the discussion condition as independent variables (conflict coded “1”; vacation coded “0”). In the second step, I entered the interaction term for the discussion condition and P3.

Chapter 3: Results

Preliminary Analyses

I conducted preliminary analyses to determine whether any variables deviated from normality. Any variable with a skewness or kurtosis greater than approximately 1 (absolute value) was transformed. Transformed variables are noted in the text below and in the tables. When presented in tables, the untransformed means and standard deviations are presented, and in the notes section, the transformed values are presented. In statistical analyses, the transformed variables were used. Correlations among continuous variables are presented in Table A6.

It was important first to establish that the participants, randomized to experimental condition, did not differ on baseline characteristics in order to determine whether any effects of the discussion task were due solely to that task and not to systemic differences between groups. To compare the discussion condition on demographics, psychophysiology, observed behavior, adolescent risk-taking, and parent-adolescent relationship quality, I conducted independent samples *t*-tests for continuous variables and chi-squared analyses for categorical variables. The demographics results are presented in Table A1, and the results for other variables are presented in their respective tables. The groups did not significantly differ on any baseline characteristics.

The following analyses are exploratory; therefore, I used Bonferroni corrections when presenting the *p*-value for any significant effect. Because I ran 7 tests, I used the corrected *p*-value = .007 ($p = .05/7$) as the threshold for significance. In order to determine whether real-world risk-taking, as measured by the YRBS Risk score, is a

predictor of performance-based risk-taking, as measured by the BART-Y post-discussion task, I conducted a linear regression with YRBS Risk score as the independent variable and BART-Y post-task as the dependent variable. YRBS Risk was not a significant predictor of post-discussion task BART-Y [$F(1, 47) = 0.61, p = .438$].

Parental behavior could differ according to gender. In order to determine whether parent gender was related to observed hostility behavior during the discussion task, I ran an independent samples *t*-test for parent hostility, with gender coded as 0 for male and 1 for female. Parent hostility violated the distributional assumptions; therefore, I used a square root transformation whenever it was used as a predictor variable and untransformed values when displaying means and standard deviations. There were non-significant differences for scores of parental hostility ($t(44) = -1.47, p = .148$) between female caregivers ($M = 2.48, SD = 1.80$) and male caregivers ($M = 1.55, SD = 0.72$), indicating that female caregivers exhibited non-significantly greater hostility than male caregivers during the discussion task.

A subset of adolescents from the Behavioral Visit participated in the EEG Visit ($n = 25$), and only a portion of that data was usable ($n = 12$). In order to determine whether the EEG sample was different from the Behavioral Visit sample on BART-Y performance at the Behavioral Visit, I ran independent samples *t*-tests. First, I compared adolescents who attended the EEG Visit with adolescents who did not attend the EEG Visit on post-discussion task BART-Y performance. There were non-significant differences for post-task BART-Y ($t(47) = 1.62, p = .112$) between adolescents who did not attend the EEG Visit ($M = 41.68, SD = 12.60$) and adolescents who did attend the EEG Visit ($M = 35.19, SD = 15.29$), indicating that adolescents who did not attend the

EEG Visit had non-significantly greater post-discussion task risk-taking than adolescents who did attend the EEG Visit. Next, I compared adolescents with usable EEG data from the EEG Visit with adolescents who did not attend the EEG Visit or had unusable data on post-discussion task BART-Y performance. There were non-significant differences for post-task BART-Y ($t(47) = -1.64, p = .106$) between adolescents with no EEG data ($M = 40.25, SD = 13.11$) and adolescents with EEG data ($M = 32.57, SD = 16.67$), indicating that adolescents with no EEG data exhibited non-significantly greater post-discussion task risk-taking than adolescents with EEG data.

Lastly, I ran several analyses related to the adolescents' BART-Y performance. Because adolescents in this sample had completed the BART-Y at previous visits to the Parent Study, which may have impacted their performance on the BART-Y (e.g., lack of novelty), I conducted a linear regression to test whether years in the parent study predicted BART-Y post-task performance, the results of which were not significant [$F(1, 47) = 1.68, p = .20$]. Additionally, adolescent gender and age were not significantly related to BART-Y performance. Males ($M = 40.80, SD = 13.04$) and females ($M = 36.03, SD = 15.26$) did not significantly differ on post-task BART-Y ($t(47) = 1.17, p = .247$). Adolescents' age and post-task BART-Y were also not significantly related ($r = .07, p = .603$).

Aim 1

First, I conducted a “manipulation check” to test whether the control and conflict conditions differed on psychophysiology and behavior during the discussion task. Using independent samples t-tests, I compared the control condition (coded “0”) with the

conflict condition (coded “1”) on mean HR and adolescent behavior. Results for adolescent observed behavior are presented in Table A3. Adolescent hostility violated the distributional assumptions; therefore, I used a square root transformation whenever it was used as a predictor variable. After transformation, skewness and kurtosis were within acceptable range (1.51, 1.76, respectively). Although adolescents in the conflict condition had higher scores on secure base use and hostility than adolescents in the control condition, these differences were not significant. Results for adolescent mean HR are presented in Table A7. Adolescents did not significantly differ on mean HR during the 5-minute baseline period. However, during the discussion task, there were significant differences for mean HR ($t(38) = -2.34, p = .025, d = -0.76$), such that adolescents in the conflict discussion task exhibited greater cardiovascular reactivity than the adolescents in the vacation discussion task.

Aim 2

To test whether adolescents in the conflict discussion condition exhibited greater post-task risk-taking as measured by the BART-Y, relative to adolescents in the control discussion condition, I ran a GEE to statistically model the two BART-Y assessments as a repeated-measures (i.e., one measure at pre-task and another post-task) dependent variable varying as a function of: (a) a within-subjects factor of Time (i.e., pre- vs. post-BART-Y risk-taking), (b) Condition (i.e., vacation discussion vs. conflict discussion), and (c) Time \times Condition interaction. Table A8 displays the results of the GEE model. There were no significant relations among the variables.

I also tested whether adolescents scoring in the top 25% for change in scores from

pre- to post-BART-Y significantly differed from the rest of the sample on hostility and mean HR. First, a difference score was created by subtracting the post-BART-Y score from the pre-BART-Y score. Next, adolescents were categorized as to whether they scored in the top 25% of BART-Y change, which were difference scores of 3.98 and greater, coded “1”. All other scores were coded 0. There were non-significant differences for scores of adolescent hostility ($t(45) = -0.42, p = .677$) between adolescent scores in the top 25% ($M = 1.36, SD = 0.37$) and the remaining sample ($M = 1.30, SD = 0.45$), indicating that adolescents scoring in the top 25% of BART-Y change exhibited non-significantly greater hostility than adolescents who did not score in the top 25% of BART-Y change. There were non-significant differences for scores of adolescent mean HR ($t(38) = -1.26, p = .215$) between adolescent scores in the top 25% ($M = 81.04, SD = 8.67$) and the remaining sample ($M = 76.16, SD = 11.13$), indicating that adolescents scoring in the top 25% of BART-Y change exhibited non-significantly greater mean HR than adolescents who did not score in the top 25% of BART-Y change.

Aim 3

A separate GEE statistical model was conducted to test whether P3 was a moderator of the relation between parent-adolescent conflict and adolescent performance on the BART-Y. Only the 12 participants with usable EEG data were included in the model. As presented in Table A9, there was a significant two-way interaction between discussion condition and P3. There were also significant main effects of time (pre- vs. post-task BART-Y) and P3 that will not be interpreted due to the interaction effect. The interaction between discussion condition and P3 was probed using the method described

in Holmbeck (2002). I created Low P3 and High P3 scores by adding and subtracting one standard deviation from the centered P3. As a reminder, Low P3 would approximate P3AR. To test the simple slopes of the interaction, I ran two additional GEE models: one model with the low P3 score as a predictor, condition, and the interaction of low P3 and condition, and another model with high P3 score as a predictor, condition, and the interaction of high P3 and condition. Results from post-hoc probing are presented in Tables A10 and A11. Contrary to my hypothesis, probing the interaction illustrated that for adolescents with High P3, there was a significant and strong relation between discussion condition and BART-Y performance. Figure 6 depicts the interaction effect.

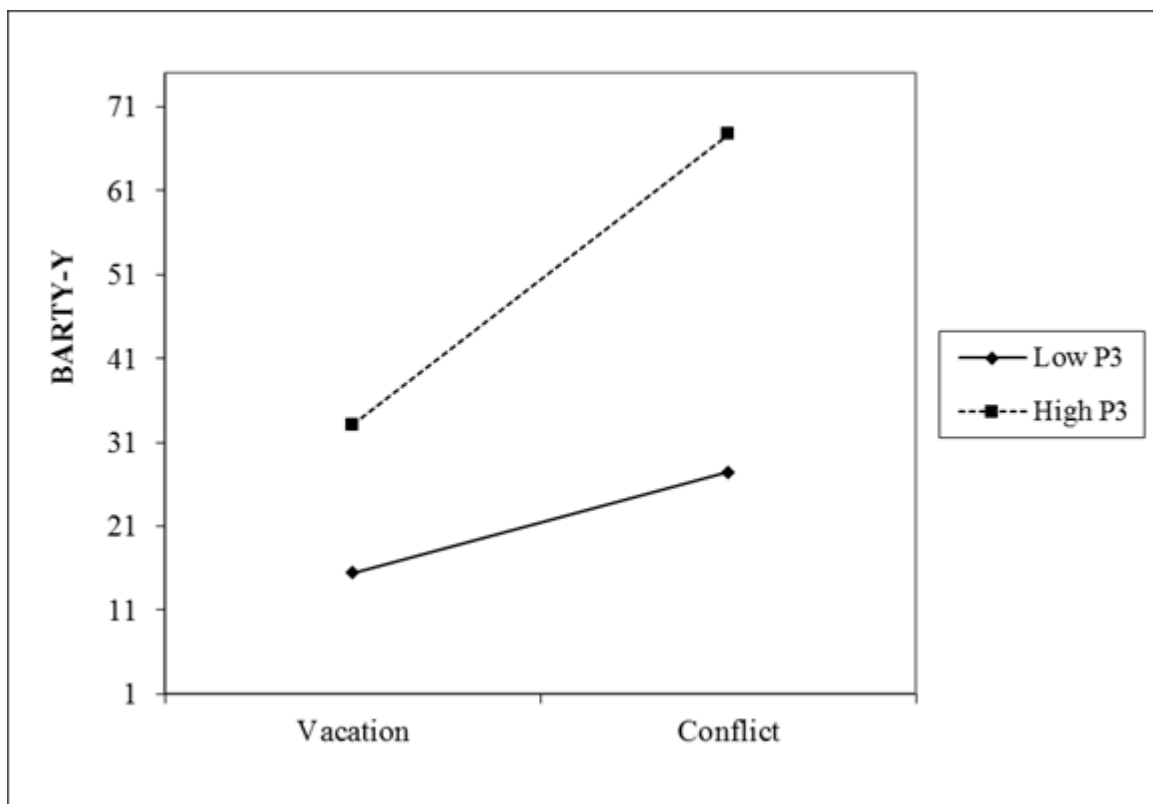


Figure 6. Interaction effect between Discussion Condition and neurobiological marker for behavioral disinhibition (P3 mean amplitude), positively relating to Balloon Analogue

Risk Task-Youth Version (BART-Y). Post-hoc probing analyses indicated that adolescents with High P3 experienced positive relations between Discussion Condition and BART-Y adjusted average pumps (see Table A11).

Exploratory Aim

Lastly, in the exploratory aim I tested whether groups differed on real world risk behaviors and whether P3 moderated the relation between discussion condition and adolescent real world risk-behavior. As shown in Table A4, there were no significant differences between discussion groups on baseline scores of YRBS Risk and YRBS HIV Risk. Table A12 presents the results of the hierarchical linear regression in which discussion condition, P3, and the interaction between discussion condition and P3 are used to predict real-world risk behaviors (YRBS Risk) for the 12 participants with usable EEG data. In Step 1, adolescent age and gender were entered. In Step 2, discussion condition and P3 were entered. In Step 3, the interaction between discussion condition and P3 were entered. There were no significant main or interaction effects in this model. A similar model was run for YRBS HIV Risk as the dependent variable, as shown in Table A13. There were no significant main or interaction effects in this model.

Chapter 4: Discussion

Main Findings

I had 3 main aims in my study. In the first aim, I hypothesized that adolescents randomized to the conflict discussion task with their parent would exhibit a greater stress response, as measured by psychophysiology and observed behavior, relative to adolescents randomized to the vacation discussion task. This hypothesis was partially supported. Adolescents in the conflict discussion task did exhibit significantly higher cardiovascular reactivity, as measured by average heart rate over 5 minutes, compared to adolescents in the vacation task. However, there were no significant differences in observed behavior. Specifically, adolescents in the conflict discussion task did not exhibit more hostile behavior or less secure base use than adolescents in the vacation discussion task, contrary to expectations.

In the second aim, I expected that adolescents randomized to the conflict discussion task would exhibit greater risk-taking on the performance-based measure of risk-taking than adolescents in the vacation discussion task, controlling for baseline BART-Y performance. I observed no significant effect of experimental condition (vacation or conflict discussion) on post-discussion BART-Y performance.

In my third aim, I tested whether a neurobiological marker of impulsivity, mean P3 amplitude, moderated the relation between conflict condition and risk-taking on the BART-Y. I hypothesized that low P3 amplitude in particular would be related to riskier behavior, given the association with reduced P3 amplitude and behavioral disinhibition (Iacono et al., 2002). P3 amplitude significantly moderated the relation between

discussion condition and risk taking, albeit in an unexpected direction. Specifically, adolescents exhibiting relatively high P3 amplitude while completing a visual oddball task had a positive significant relation with discussion condition and performance-based risk-taking behavior, whereas adolescents exhibiting relatively low P3 amplitude evidenced a statistically non-significant positive relation with discussion condition and performance-based risk-taking behavior.

Lastly, none of the exploratory analyses testing whether parent-adolescent conflict and P3 amplitude were significantly related to real-world risk-taking behaviors were significant.

Interpretation of Results

My goal of this study was to gain a greater understanding of why parent-adolescent conflict is related to risk-taking behaviors, and particularly HIV risk behaviors. While this study did provide some insights into the relation and some future directions for study design, ultimately this is still an open question. Despite the lack of significant results on some of the aims, I gained several important insights into both parent-adolescent conflict and its link with adolescent risk-taking, and the measurement of these constructs.

First, although I tried to create a control discussion condition in order to have an absence of conflict so that I could isolate the effect of conflict preceding risk-taking, it is likely not the case that parent-adolescent conflict only occurs when “induced”. Adolescents in the conflict discussion group did exhibit a significantly greater psychophysiological stress response, indicating that the discussion manipulation worked

with regard to inducing greater stress in the conflict condition relative to the vacation condition. However, there were no significant differences in observed behavior between the two groups. I believe this illustrates that parent-adolescent conflict can be induced but it also exists in a “trait” type way of a dyadic relationship. In other words, some parent-child relationships may be characterized by conflictual ways of interacting with one another (e.g., increased hostility and invalidation), and even in “benign” situations, those behaviors will still be exhibited because they are a function of the dyad’s interactions across social contexts. Consequently, this made it more difficult to detect an effect of conflict predicting risk-taking because some adolescents in both conditions may have been experiencing aversive interactions with parents across my discussion task conditions.

Along these lines, despite the fact that there was not a significant difference between the two discussion groups on BART-Y risk-taking after the discussion task, the averages of the two groups did show a non-significant difference in the expected direction. Adolescents in the conflict discussion had non-significantly higher average adjusted pumps on the BART-Y than the adolescents in the vacation discussion.

Second and related, it is interesting to consider what role the conflict topic plays in parent-adolescent conflict interactions. Conflict topics were chosen according to the topic the adolescent had indicated had the highest rating for arguing/fighting on the TTI. However, it is possible the cause of stress is less about the conflict topic and more about the content and process of a discussion between parents and adolescents. Even if adolescents report that there is a topic about which they fight with their parents frequently, if they both “fight fair”, this may not be stressful. Indeed, prior work indicates

that only approximately 25% of parent-adolescent conflict result in maladaptive outcomes (e.g., adolescent psychosocial dysfunction; Rutter et al., 1976). It is likely that discussion situations in which adolescents perceive their parents to be invalidating, rejecting, and not accepting of them as a person may be more salient than the actual topic, although certainly choosing a topic with a reported high frequency of arguing is more likely to induce those behaviors. If parents behave in a way towards adolescents that makes the adolescent feel that their negative behaviors are generalized to everything they do, their emotions are not validated, or they are not accepted as a person, this could potentially suggest a mechanism for increased adolescent stress and subsequent need for coping behaviors (e.g., substance use, risk behaviors outside the home). The frequency of these aversive parental behaviors was not measured in this study, and the sample size likely would have limited any analyses, but future research would benefit from investigating whether the content and process of discussion tasks, such as specific aversive hostile behaviors, is directly related to adolescent risk-taking performance.

Third, as has been alluded to, the findings leave unanswered whether parent-adolescent conflict is a cause or a correlate of risk-taking behavior. If it were a correlate of risk-taking behavior, that would possibly suggest that there is an underlying vulnerability that influences both the parent-adolescent conflict and risk-taking behavior. Previous research has found that underlying genetic vulnerabilities moderate the relation between parenting and adolescent conduct problems (Feinberg et al., 2007; Hicks, South, DiRago, Iacono, McGue, 2009). Importantly, this link has not been found in children (Burt & Klump, 2014), suggesting adolescence is the ideal time to investigate the link between environmental factors and risk behaviors. Research on executive functioning,

ADHD, and impulsivity provides support for neurobiological characteristics influencing multiple domains of functioning. In situations in which there are executive functioning deficits that impact behavior, we know that these deficits often relate to behavior across development that may pose risk for negative parent-child interactions through an adolescents' development (Edwards, Barkley, Laneri, Fletcher, & Metevia, 2001). In fact, according to coercion theory (Patterson, 1982), there is a pathway by which a history of these coercive interactions between parents and children results in parental disengagement from the adolescent, reductions in parental monitoring, and subsequent increase in adolescent risky behavior. If this behavior is truly an individual differences factor like impulsivity, then adolescents who are behaviorally disinhibited with their parents are likely to be behaviorally disinhibited when exposed to the opportunity to engage in risky behaviors. To "responsibly" engage in a conflict discussion and resist risky behaviors requires planning and judgement in a brief amount of time to make a behavioral choice, and for those adolescents who are neurobiologically vulnerable to impulsivity, inhibiting their response to these behaviors may prove too difficult. The fact that there was an interaction between adolescents' neurobiology and discussion condition that was related to greater risk-taking on the BART-Y, albeit in an unexpected direction, provides some tenuous support for the importance of understanding how underlying neurobiology contributes to adolescent risky behavior.

Fourth and related, as hypothesized, P3 amplitude did moderate the relation between discussion condition and increased performance-based risk-taking. However, this relation was moderated by high P3, and not low P3 as had been expected given the link between reduced P3 amplitude and behavioral disinhibition (Iacono et al., 2000,

2002). Again, given the low sample size, caution must be taken in interpreting these results. As will be explained below, this relation, although unexpected, does provide support for the hypothesis that for some adolescents, neurobiological predispositions toward impulsivity, coupled with environmental context (e.g., conflict with parents) is related to greater risk-taking.

The majority of prior research on P3 has investigated reduced P3 amplitude as a neurobiological marker for impulsivity, yet there is some evidence to suggest that *high* P3 also has a separate but related link to impulsive behavior, particularly with regard to the personality construct of sensation-seeking. Sensation-seeking has been described as a component of impulsivity related to the personality characteristics of extroversion/positive emotion (Sharma et al., 2014) as well as arousal regulation (Zuckerman, Bone, Neary, Mangelsdorff, & Brustman, 1972). In contrast, behavioral disinhibition, also a component of impulsivity, is characterized by lack of constraint and planning (Iacono et al., 2008; Sharma et al., 2014). In several studies, particularly in non-clinical samples as opposed to samples recruited specifically for elevated levels of or risk for impulsive behavior, high P3 was related to greater endorsement of reward-seeking behavior (Hansenne, 1999; Nijs, Franken, & Smulders, 2007; Pierson, le Houezec, Fossaert, Dubal, & Jouvent, 1999). For example, in a study primarily comprised of undergraduate students, researchers found a significant relation between P3 recorded during a two-stimulus oddball task and responses on a questionnaire assessing the Behavioral Approach System (BAS; Nijs, Franken, & Smulders, 2007). Specifically, P3 was significantly positively correlated with BAS Total score and the Reward and Fun subscales. Furthermore, P3 was not related to the Behavioral Inhibition System Scale.

Researchers concluded that P3 indexed reward seeking behavior in their sample. In another study of healthy participants, researchers found a significant positive relation between P3 and the personality construct novelty seeking, measured via questionnaire (Hansenne, 1999). In the present study, the significant positive relation between P3 and BART-Y indicates that adolescents with high P3 engaged in more reward-seeking behavior than adolescents without high P3. Indeed, earning more points on the BART-Y does result in winning a larger prize. Additionally, the significant interaction between discussion condition and P3 indicated that adolescents with high P3 in the conflict discussion condition demonstrated greater reward-seeking behavior on the BART-Y than adolescents without high P3. One possible interpretation of this finding is that when adolescents with high P3 engage in uncomfortable and/or conflictual interactions with their parents, their arousal is depleted, for which they subsequently compensate by engaging in greater reward-seeking behavior to increase their arousal, relative to adolescents who do not have high P3 scores.

Limitations

There were several limitations to the current study. First, the sample from which I recruited adolescents for the Behavioral Visit, and subsequent EEG Visit, was part of an on-going longitudinal HIV risk study. As part of their annual visit to the parent study, adolescents completed the BART-Y. Because the average number of years they had been in the parent study was 3.5 and they completed the BART-Y each year, this task may have been no longer novel for them and may not have been able to accurately index stress-induced risk-taking. Additionally, I included in the current study design a pre-

discussion task BART-Y in order to be able to control for floor or ceiling effects on adolescents' risk-taking performance, which may also have reduced the novelty and thus my ability to assess changes in risk-taking due to the experimental manipulation of conflict. In the future, I would implement a task to which study participants had not yet previously been exposed, such as the another probabilistic gambling task (e.g., Burnett, Bault, Coricelli, & Blakemore, 2010) and a Go/Nogo task (Casey et al., 2001).

Second, although I was interested in studying predictors and correlates of adolescent risk-taking behavior, the current sample evidenced low-base rates of risk-taking behavior in that they endorsed an average of only 3 risky behaviors (see Table A4). It may be that recruitment strategies implemented in my study resulted in a final sample of participants unlikely to exhibit high rates of risk-taking behavior. Specifically, demands of the current study may have influenced the level of risk-taking exhibited by the sample, including requiring 2 hours of the parents' and adolescents' time, requiring a 15 minute telephone screen, and commuting to a college campus. In future research, I would consider pre-screening adolescents in order to make sure the sample adequately captures a range of risky behaviors.

Third, findings from this study are limited by low sample size, particularly with regard to the fact that there were several between and within subject variables that reduced power to detect statistical effects. Over the span of 15 months, I recruited 51 families, two of which had to be excluded for reasons discussed earlier. Because the study included several assessments that were included in order to account for hypothesized measurement difficulties in prior studies (e.g., using an in-person structured interview to assess conflict, facilitating an actual discussion between parents and

adolescents), the study visit required several research assistants. Coordinating the schedules of both participants and staff meant that only 2-3 visits could be run per week. Therefore, a longer recruitment period would be needed to increase the sample size, which was not feasible for this project length. Additionally, the sample was further reduced with the EEG Visit. Although 25 adolescents did participate in that visit, due to equipment issues over half of the data was not usable. Thus, although there were some trends in the data, a larger sample size, particularly for the EEG portion of the study, is needed in order to have enough power to detect effects, given that there were several conditions (i.e., discussion, time) in the design and analyses.

Future Directions

There are several future directions that would improve this line of research that I have not yet mentioned. First, it will be important to sample specifically for adolescents who meet threshold for behavioral disinhibition, to make sure that that potential vulnerability is adequately included within the sample, given its hypothesized role in both conflict and risk-taking. Second, it would benefit understanding the mechanism of parent-adolescent conflict and risk-taking if the function of adolescents' risk-taking behavior was known. For example, for adolescents who engage in risk-taking as sensation-seeking, their link between conflict with parents and risk-taking may be a correlate, whereas adolescents who report they engage in risk-taking to cope with stress may experience parent-adolescent conflict that causes risk-taking through stress (consistent with a model of negative reinforcement). Third, it would be helpful in measuring the neurobiological marker of impulsivity to not only increase the sample size for power but to also constrain

analyses by age, given that substantial brain development is occurring during this time. Because inhibitory control increases across development (Steinberg, 2008), analyses may provide more definitive conclusions if the recruitment age range was shortened (e.g., 14-15 year olds or 16-17 year olds; Lewis, Lamm, Segalowitz, Stieben, & Zelazo, 2006). Fourth, knowing what role the parent plays in the adolescents' life will be helpful in conceptualizing conflict. It may be the case that some adolescents in the current study came in with a parent who was not the "enforcer" of the rules at home, and this may have resulted in little to no conflict between the dyad.

Additionally, the low base-rate engagement in HIV risk behaviors in the present sample precluded my ability to assess the relation between parent-adolescent conflict and adolescent HIV risk. This relation requires further study to understand why HIV risk in particular is related to parent-adolescent conflict, in addition to the reasons discussed above, so that we can better understand how to modify the link between these two factors. Compared to other risky behaviors that could be conducted alone (e.g., speeding while driving, smoking marijuana, stealing from a store), the vast majority of HIV transmission opportunity requires interpersonal contact (e.g., sexual behavior, sharing needles). Further, it is well-established that HIV infection is not uniformly distributed—it disproportionately affects regions and neighborhoods where people live who are already lacking resources and experiencing poverty (Pellowski, Kalichman, Matthews, & Adler, 2013), both of which also are related to greater executive functioning deficits (Farah et al., 2006; Hackman & Farah, 2009; Hicks, South, DiRago, Iacono, McGue, 2009). There is then a great opportunity for adolescents to contract HIV if they live in a neighborhood typified by high HIV prevalence, but they may also be seeking social support outside

their family if they experience greater parent-adolescent conflict. If an adolescent does not perceive support at home, they may seek that support elsewhere through friends and community social connections. That may then provide greater opportunities to engage in behaviors that pose risk for HIV infection, which is further exacerbated by adolescents' propensity to have difficulty making reasoned decisions in the heat of the moment (Steinberg, 2008), despite being able to judge the riskiness of a behavior similar to adults (Beyth-Marom et al., 1993; Quadrel, Fischhoff, & Davis, 1993). In fact, prior research has established the significant influence of peers on increased adolescent risky decision making (Cavalca et al., 2013; Smith, Chein, & Steinberg, 2014). Thus, it is possible that the "social deficit" related to increased parent-adolescent conflict may influence adolescents to seek that social support outside the home, and when they live in neighborhoods with higher HIV prevalence, that provides greater opportunity for normative adolescent self-control deficits to amplify with environmental risk, potentially leading to HIV infection. This hypothesized model merits further study.

Conclusion

Using an experimental design, the current study investigated the link between parent-adolescent conflict and HIV risk-behaviors. Although there was no significant relation between parent-adolescent conflict and risk-taking behavior, adolescents did exhibit a greater cardiovascular stress response in the discussion condition, relative to the vacation condition. Further, within the subset of adolescents with usable EEG data, adolescent neurobiological function (i.e., P3) moderated the relation between discussion condition and risk-taking. In sum, there are likely several pathways by which parent-

adolescent conflict relates to HIV risk-taking, which the current study was not able to test due to sample size restrictions. For some adolescents, parent-adolescent conflict is a correlate of HIV risk because the source is the same—an underlying genetic vulnerability that is exhibited as impulsivity/behavioral disinhibition. For other adolescents, this developmental period of their life consisting of increased emotional vulnerability, decreased regulatory control, greater independence, and greater discrepancy between their own goals and those of their parents may strengthen the link between their perceived stress and maladaptive coping behaviors. There is also likely a third group for which conflict is both a correlate and cause of HIV risk behavior due to their genetic predisposition to behavioral disinhibition, and the addition of stress in the form of parent-adolescent interactions, which leads to increased risk-taking behaviors. Future research that takes these multiple pathways into account may be better equipped to identify mechanisms between parent-adolescent conflict and HIV risk behaviors, which may inform effective prevention efforts.

Appendix A

Table A1

Comparison of Baseline Demographic Characteristics Between Discussion Conditions

Demographic Characteristic	Vacation (<i>n</i> = 23)				Conflict (<i>n</i> = 26)				<i>p</i>
	<i>M</i>	<i>SD</i>	#	%	<i>M</i>	<i>SD</i>	#	%	
Age	14.87	1.01			15.04	1.04			<i>p</i> = .57
Adolescent Gender (% female)			13	56.5			12	46.2	<i>p</i> = .47
Ethnicity									<i>p</i> = .15
African American or Black			18	78.3			14	53.8	
Asian American or Asian				NA			1	3.8	
Hispanic or Latino/a (Spanish)			1	4.3				NA	
White, Caucasian American, or European			6	26.1			12	46.2	
Parent Marital Status									<i>p</i> = .88
Never married			6	26.1			6	23.1	
Married/living together			13	56.5			14	53.8	
Separated/divorced			4	17.4			6	23	
Family Weekly Earnings									
\$500 or less			7	30.4			3	11.5	<i>p</i> = .08
\$501 and \$900			5	21.7			13	50	
\$901 or more per week			11	47.8			10	38.5	

Table A2

Means (M), Standard Deviations (SD), and Internal Consistency (α) Estimates of Survey Measures of TTI and Comparison Between Discussion Conditions

Variable	<i>M</i>	<i>SD</i>	α	<i>t</i> -Test	<i>p</i>
Adolescent TTI Argue/Fight Total	6.22	3.67	.72	1.00	$p = .32$
Vacation Condition	6.78	3.61			
Conflict Condition	5.73	3.72			
Adolescent TTI Different Beliefs Total	8.24	5.27	.79	1.56	$p = .12$
Vacation Condition	9.48	5.24			
Conflict Condition	7.15	5.16			
Parent TTI Argue/Fight Total	7.30	4.53	.80	-0.25	$p = .80$
Vacation Condition	7.13	3.85			
Conflict Condition	7.46	5.14			
Parent TTI Different Beliefs Total	11.24	6.81	.86	0.05	$p = .95$
Vacation Condition	11.30	6.02			
Conflict Condition	11.19	7.57			

Note. TTI = To(may)to-To(mah)to Interview. Vacation condition ($n = 23$). Conflict Condition ($n = 26$).

Table A3

Means (M), and Standard Deviations (SD), and Reliability Estimates of Discussion Task Observed Behavior and Comparison Between Discussion Conditions

Variable	<i>M</i>	<i>SD</i>	ICC	<i>t</i> -Test	<i>p</i>
Adolescent Secure Base Use	5.00	1.63	.81	0.71	<i>p</i> = .48
Vacation Condition	5.18	1.56			
Conflict Condition	4.84	1.70			
Adolescent Hostility and Rejection	1.93	1.43	.39	-0.74	<i>p</i> = .46
Vacation Condition	1.72	0.98			
Conflict Condition	2.12	1.74			
Parent Secure Base Provision	5.34	1.55	.95	-0.50	<i>p</i> = .62
Vacation Condition	5.22	1.54			
Conflict Condition	5.46	1.58			
Parent Hostility and Rejection	2.30	1.68	.98	-0.52	<i>p</i> = .60
Vacation Condition	2.13	1.39			
Conflict Condition	2.46	1.93			

Note. Parent hostility and adolescent hostility variables violated distributional assumptions. Means and standard deviations displayed in the table are untransformed. Parent hostility was transformed using a square root transformation (transformed *M* = 1.43, *SD* = 0.50; vacation: *M* = 1.39, *SD* = 0.44; conflict: *M* = 1.47, *SD* = 0.55). Adolescent hostility was transformed using a square root transformation (transformed *M* = 1.32, *SD* = 0.43; vacation: *M* = 1.27, *SD* = 0.33; conflict: *M* = 1.37, *SD* = 0.51). *t*-tests were conducted with transformed variables. ICC = intraclass correlation coefficient.

Table A4

Means (M), Standard Deviations (SD), and Internal Consistency (α) Estimates of Survey and Performance-based Measures of Risk taking and Comparison Between Discussion Conditions

Variable	<i>M</i>	<i>SD</i>	α	<i>t</i> -Test	<i>p</i>
YRBS Risk Score	3.26	2.92	.79	0.04	$p = .96$
Vacation Condition	3.17	2.40			
Conflict Condition	3.34	3.35			
YRBS HIV Risk Score	0.57	1.17	.76	-0.42	$p = .67$
Vacation Condition	0.48	0.94			
Conflict Condition	0.65	1.35			
Pre-BART-Y adjusted average pumps	37.37	13.77	n/a	0.03	$p = .97$
Vacation Condition	37.45	13.61			
Conflict Condition	37.31	14.17			
Post- BART-Y adjusted average pumps	38.37	14.27	n/a	-0.47	$p = .63$
Vacation Condition	37.33	13.51			
Conflict Condition	39.29	15.12			

Note. YRBS = Youth Risk Behavior Surveillance System, BART-Y = Balloon Analog Risk Task Youth Version. YRBS Risk scores violated distributional assumptions. Means and standard deviations displayed in the table are untransformed. YRBS Risk score was transformed using a square root transformation (transformed $M = 1.57$, $SD = 0.90$; vacation: $M = 1.58$, $SD = 0.84$; conflict: $M = 1.57$, $SD = 0.96$). YRBS HIV Risk score was transformed using a square root transformation (transformed $M = 0.38$, $SD = 0.65$; vacation: $M = 0.34$, $SD = 0.61$; conflict: $M = 0.42$, $SD = 0.70$). *t*-tests were conducted with transformed variables.

Table A5

Means (M), Standard Deviations (SD), and Internal Consistency (α) Estimates of Survey Measures of Parent-adolescent Relationship

Characteristics and Comparison Between Discussion Conditions

Variable	<i>M</i>	<i>SD</i>	α	<i>t</i> -Test	<i>p</i>
Parent APQ Positive Parenting	62.04	7.17	.82	-0.75	$p = .45$
Vacation Condition	61.21	7.98			
Conflict Condition	62.77	6.45			
Parent APQ Negative Parenting	35.34	7.10	.74	0.48	$p = .63$
Vacation Condition	35.87	6.09			
Conflict Condition	34.88	7.98			
Adolescent disclosure	10.22	3.57	.79	-0.65	$p = .52$
Vacation Condition	9.87	3.25			
Conflict Condition	10.54	3.87			

Note. APQ = Alabama Parenting Questionnaire.

Table A6

Correlations among Measures of Parenting, Observed Behavior, Psychophysiology, and Risk-behavior

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13
1 Pos Parenting-P													
2 Neg Parenting-P	-.40**												
3 Adolescent Disclosure	-.39**	.55***											
4 Baseline HR	-.02	-.33*	-.06										
5 Discussion HR	-.02	-.45**	-.13	.88***									
6 TTI Argue/Fight	-.09	.42**	.28*	-.25	-.30								
7 TTI Diff. Beliefs	-.06	.31*	.30*	-.28	-.38*	.62***							
8 Hostility	-.28	.03	-.09	-.12	-.11	.02	-.11						
9 Secure Base Use	.16	-.18	-.13	.23	.16	-.28	-.09	-					
								.61***					
10 YRBS Risk	-.01	.22	.38**	-.30	-.30	.35*	.31*	-.04	-.14				
11 YRBS HIV Risk	-.26	.17	.26	-.14	-.14	.28	.21	-.01	-.02	.67***			
12 BART-Y Pre	-.24	.27	.11	-.20	-.19	.13	.02	.19	-.04	.04	.04		
13 BART-Y Post	-.25	.16	.04	-.09	-.04	.13	-.04	.17	.02	-.11	.05	.92***	

Note. Pos Parenting-P = parent-report on Alabama Parenting Questionnaire (APQ) Total Positive Parenting Score. Neg Parenting-P = parent-report on Alabama Parenting Questionnaire (APQ) Total Negative Parenting Score. HR = mean heart rate. TTI = To(may)to-To(mah)to Interview. YRBS = Youth Risk Behavior Surveillance System. BART-Y = Balloon Analog Risk Task Youth Version. Transformed versions of variables that violated distributional assumptions were used (adolescent hostility, YRBS Risk, YRBS HIV Risk).

* $p < .05$; ** $p < .01$, *** $p < .001$.

Table A7

Means (M) and Standard Deviations (SD) of Adolescent Heart Rate and P3, and Comparison Between Discussion Conditions

Variable	<i>M</i>	<i>SD</i>	<i>t</i> -Test	<i>p</i>
Adolescent Baseline HR (<i>n</i> = 42)	78.28	11.98	-1.75	<i>p</i> = .08
Vacation Condition	74.80	10.56		
Conflict Condition	81.16	12.55		
Adolescent Discussion HR (<i>n</i> = 40)	77.38	10.68	-2.34	<i>p</i> = .02
Vacation Condition	73.45	9.65		
Conflict Condition	80.94	10.52		
Adolescent P3 (<i>n</i> = 12)	10.62	4.83	-1.39	<i>p</i> = .19
Vacation Condition (<i>n</i> = 4)	7.99	3.57		
Conflict Condition (<i>n</i> = 8)	11.93	5.03		

Note. HR = mean heart rate. P3 = Mean amplitude of Target P3 minus Non-Target P3.

Table A8

Generalized Estimating Equations Predicting Adolescent Performance-based Risk-taking (BART-Y) as a Function of Time (Pre vs Post) and Discussion Condition Status(control vs. conflict; n = 49)

Factor	<i>B</i>	<i>SE</i>	<i>R</i> ²	<i>Wald</i> 95% <i>CI</i>	<i>p</i>
Main and Interaction Effects					
Adolescent Age (centered)	0.84	1.79	.06	[-2.68, 4.36]	.64
Adolescent Gender	-5.12	3.96	.44	[-12.88, 2.64]	.19
Discussion Condition	-0.81	3.90	.01	[-8.46, 6.84]	.95
Time	-0.12	1.18	.00	[-2.43, 2.19]	.23
Discussion condition × Time	2.10	1.56	.48	[-0.96, 5.16]	.18

Note. BART-Y = Balloon Analog Risk Task Youth Version; *B* = Unstandardized beta; *SE* = Standard error; 95% *CI* = 95% Wald confidence interval.

Factor contrasts based on comparisons in descending order, with the Time factor coded Pre-task = “0” and Post-task = “1”. Discussion Condition is coded Vacation = “0” and Conflict = “1”. Adolescent Gender is coded Male = “0” and Female = “1”. *p*-values are reported from the Tests of Model effects. Coefficients are reported from Parameter Estimates.

Table A9

Generalized Estimating Equations Predicting Adolescent Performance-based Risk-taking (BART-Y) as a Function of Time (Pre vs Post), Discussion Condition Status (control vs. conflict; n = 12), and P3

Factor	<i>B</i>	<i>SE</i>	<i>R</i> ²	Wald 95% <i>CI</i>	<i>p</i>
Main and Interaction Effects					
Adolescent Age (centered)	12.57	3.71	.27	[5.28, 19.85]	.001**
Adolescent Gender	15.85	6.72	.13	[2.67, 29.03]	.02*
P3 (centered)	1.42	0.41	.28	[-.61, 2.22]	.000***
Time	-1.44	1.15	.03	[-3.70, 0.81]	.49
Discussion Condition	23.69	8.56	.18	[6.91, 40.47]	.003**
Discussion condition × Time	0.75	3.11	.00	[-5.35, 6.86]	.81
Time x P3	-0.05	0.20	.00	[-0.44, 0.33]	.85
Discussion condition × P3	2.38	1.26	.08	[-0.09, 4.84]	.04*
Discussion condition × Time × P3	0.19	0.46	.00	[-0.72, 1.11]	.68

Note. *B* = Unstandardized beta; *SE* = Standard error; 95% *CI* = 95% Wald confidence interval. P3 = Mean amplitude of Target P3 minus Non-Target P3. Factor contrasts based on comparisons in ascending order, with the Time factor coded Pre-task = “0” and Post-task = “1”. Discussion Condition is coded Vacation = “0” and Conflict = “1”. Adolescent Gender is coded Male = “0” and Female = “1”. For statistical tests of main and interaction effects, *p* values and 95% *CI*s reported reflect significance tests for the reported unstandardized betas. *p*-values are reported from the Tests of Model effects. Coefficients are reported from Parameter Estimates.

* $p < .05$; ** $p < .01$, *** $p < .001$.

Table A10

Generalized Estimating Equations Post-hoc Tests of Moderation Probing the Interaction of Discussion Condition and Low P3

Factor	<i>B</i>	<i>SE</i>	<i>R</i> ²	Wald 95% CI	<i>p</i>
Main and Interaction Effects					
Adolescent Age (centered)	12.57	3.71	.28	[5.28, 19.85]	.001**
Adolescent Gender	15.85	6.72	.13	[2.67, 29.03]	.02*
P3-Low	1.39	0.37	.35	[-.67, 2.11]	.000***
Time	-1.36	0.91	.05	[-3.15, 0.42]	.13
Discussion Condition	12.11	6.83	.07	[-1.28, 25.49]	.07
Discussion condition × P3-Low	2.47	1.20	.10	[0.11, 4.84]	.04

Note. *B* = Unstandardized beta; *SE* = Standard error; 95% CI = 95% Wald confidence interval. P3 = Mean amplitude of Target P3 minus Non-Target P3.

Factor contrasts based on comparisons in ascending order, with the Time factor coded Pre-task = “0” and Post-task = “1”. Discussion Condition is coded Vacation = “0” and Conflict = “1”. Adolescent Gender is coded Male = “0” and Female = “1”. For statistical tests of main and interaction effects, *p* values and 95% CIs reported reflect significance tests for the reported unstandardized betas. *p*-values are reported from the Tests of Model effects.

Coefficients are reported from Parameter Estimates.

p* < .05; *p* < .01, ****p* < .001.

Table A11

Generalized Estimating Equations Post-hoc Tests of Moderation Probing the Interaction of Discussion Condition and High P3

Factor	<i>B</i>	<i>SE</i>	<i>R</i> ²	Wald 95% CI	<i>p</i>
Main and Interaction Effects					
Adolescent Age (centered)	12.57	3.71	.25	[5.28, 19.85]	.001**
Adolescent Gender	15.85	6.72	.12	[2.67, 29.03]	.02*
P3-High	1.39	0.37	.31	[-.67, 2.11]	.000***
Time	-1.36	0.91	.05	[-3.15, 0.42]	.13
Discussion Condition	36.03	12.53	.18	[11.47, 60.60]	.004**
Discussion condition × P3-High	2.47	1.20	.09	[0.11, 4.84]	.04

Note. *B* = Unstandardized beta; *SE* = Standard error; 95% CI = 95% Wald confidence interval. P3 = Mean amplitude of Target P3 minus Non-Target P3.

Factor contrasts based on comparisons in ascending order, with the Time factor coded Pre-task = “0” and Post-task = “1”. Discussion Condition is coded Vacation = “0” and Conflict = “1”. Adolescent Gender is coded Male = “0” and Female = “1”. For statistical tests of main and interaction effects, *p* values and 95% CIs reported reflect significance tests for the reported unstandardized betas. *p*-values are reported from the Tests of Model effects.

Coefficients are reported from Parameter Estimates.

p* < .05; *p* < .01, ****p* < .001.

Table A12

Hierarchical Linear Regression Analyses Summary for Real-World Risk-taking (YRBS Risk), Discussion Condition, and P3 (N = 12)

Variable	<i>B</i>	<i>SeB</i>	β	R^2	ΔR^2	<i>p</i>
Step 1				.09	.09	.64
Adolescent age (centered)	-0.29	0.73	-0.13			.69
Adolescent gender	1.17	1.41	0.26			.43
Step 2				.21	.12	.60
Discussion condition	-2.10	2.05	-0.45			.34
P3 (centered)	0.10	0.20	0.21			.64
Step 3				.22	.00	.82
Discussion condition \times P3	-0.12	0.52	-0.21			.82

Note. *B* = Unstandardized beta; *SeB* = Standard error; β = Standardized beta. P3 = Mean amplitude of Target P3 minus Non-Target P3. YRBS = Youth Risk Behavior Surveillance System. ΔR^2 statistics for each step were based on variables entered in that step. Discussion Condition is coded Vacation = “0” and Conflict = “1”. Adolescent Gender is coded Male = “0” and Female = “1”.

Table A13

Hierarchical Regression Analyses Summary for Real-World HIV Risk (YRBS HIV), Discussion Condition, and P3 (N = 12)

Variable	<i>B</i>	<i>SeB</i>	β	R^2	ΔR^2	<i>p</i>
Step 1				.05	.05	.78
Adolescent age (centered)	0.13	0.22	0.19			.57
Adolescent gender	-0.14	0.42	-0.11			.74
Step 2				.34	.29	.27
Discussion condition	-0.01	0.55	0.00			.99
P3 (centered)	-0.09	0.05	-0.68			.13
Step 3				.34	.00	.95
Discussion condition \times P3	0.00	0.14	0.05			.95

Note. *B* = Unstandardized beta; *SeB* = Standard error; β = Standardized beta. P3 = Mean amplitude of Target minus non-target P3 voltage. YRBS = Youth Risk Behavior Surveillance System. ΔR^2 statistics for each step were based on variables entered in that step. Discussion Condition is coded Vacation = “0” and Conflict = “1”. Adolescent Gender is coded Male = “0” and Female = “1”.

Appendix B

Frequencies of Assigned Topics During Discussion Condition

Discussion Topic	Frequency	Percent
Vacation	23	46.94
Doing your chores	7	14.28
Spending time with the rest of the family	3	6.12
Getting to go fun places with friends	3	6.12
Getting good grades	2	4.08
Doing your homework	2	4.08
Getting to do things that you want to do on the weekend	2	4.08
Getting to spend time outside of the house with friends	2	4.08
Getting to hang out with friends that you like	1	2.04
Getting to do fun things after school	1	2.04
Getting to do what you want after dinner	1	2.04
Getting to go on the computer or talk on the phone with friends	1	2.04
Getting to do fun things on the weekend*	1	2.04

Note. *Research assistant said the wrong wording for the assigned topic “getting to do things that you want to do on the weekend”.

Appendix C

Division of YRBS Items into Two Scales

Item	YRBS Risk	YRBS HIV Risk
During your life, how many times have you:		
used marijuana?	X	
taken steroid pills or shots without a doctor's prescription?	X	
In the past year, how many times have you:		
been in a car without wearing a seatbelt? ±	X	
ridden a bike without wearing a helmet? ±	X	
been in a physical fight?	X	
carried a weapon (gun, club, or knife) outside your home?	X	
stolen something from store?	X	
gambled money in person?	X	
had a drink of alcohol?	X	
smoked a cigarette (even a puff)?	X	
used cocaine or crack?	X	
used heroin?	X	
used methamphetamines, including speed or crystal meth?	X	
used hallucinogens, including PCP?	X	
sniffed glue, breathed aerosol spray cans, inhaled paints or sprays to get high?	X	
used ecstasy (MDMA)?	X	
used derbisol (dirt, durb, db)?	X	
used a needle to inject any drugs above?		X
re-use a needle from someone else) even if you cleaned it?)		X
given or received oral sex?		X
had intercourse with no condom?	X	X
Have you ever had sexual intercourse?		X
Did you drink alcohol or use drugs before you had sex the last time?		X
±		

Note. ± Indicates this item was dichotomized using a median split. All other variables dichotomized according to “0” Never engaged in behavior, “1” Engaged in behavior. YRBS = Youth Risk Behavior Surveillance System.

Bibliography

- Aklin, W. M., Lejuez, C. W., Zvolensky, M. J., Kahler, C. W., & Gwadz, M. (2005). Evaluation of behavioral measures of risk taking propensity with inner city adolescents. *Behaviour Research and Therapy*, 43(2), 215-228.
doi:10.1016/j.brat.2003.12.007
- Allen, J. J. B., Chambers, A. S., & Towers, D. N. (2007). The many metrics of cardiac chronotropy: A pragmatic primer and a brief comparison of metrics. *Biological Psychology*, 74(2), 243-262. doi: 10.1016/j.biopsycho.2006.08.005
- Allen, J. P., McElhaney, K. B., Land, D. J., Kuperminc, G. P., Moore, C. W., O'Beirne-Kelly, H., & Kilmer, S. L. (2003). A secure base in adolescence: Markers of attachment security in the mother-adolescent relationship. *Child Development*, 74(1), 292-307.
- Anderson, A. R., & Henry, C. S. (1994). Family system characteristics and parental behaviors as predictors of adolescent substance use. *Adolescence*, 29(114), 405-420.
- Anderson, E. R., & Hope, D. A. (2009). The relationship among social phobia, objective and perceived physiological reactivity, and anxiety sensitivity in an adolescent population. *Journal of Anxiety Disorders*, 23(1), 18-26. doi: 10.1016/j.janxdis.2008.03.011
- Arnsten, A. T. (1999). Development of the cerebral cortex: XIV. Stress impairs prefrontal cortical function. *Journal of The American Academy Of Child & Adolescent Psychiatry*, 38(2), 220-222. doi:10.1097/00004583-199902000-00024

- Ary, D. V., Duncan, T. E., Biglan, A., Metzler, C. W., Noell, J. W., & Smolkowski, K. (1999). Development of adolescent problem behavior. *Journal of Abnormal Child Psychology: An official publication of the International Society for Research in Child and Adolescent Psychopathology*, 27(2), 141-150.
- Ary, D. V., Duncan, T. E., Duncan, S. C., & Hops, H. (1999). Adolescent problem behavior: The influence of parents and peers. *Behaviour Research and Therapy*, 37(3), 217-230.
- Baskin-Sommersa, A., & Sommers, I. (2006). The co-occurrence of substance use and high-risk behaviors. *Journal of Adolescent Health*, 38(5), 609-611.
- Begleiter, H., Porjesz, B., Bihari, B., & Kissin, B. (1984). Event-related brain potentials in boys at risk for alcoholism. *Science*, 225(4669), 1493-1496.
doi:10.1126/science.6474187
- Berntson, G. G., Cacioppo, J. T., & Grossman, P. (2007). Whither vagal tone. *Biological Psychology*, 74(2), 295-300. doi: 10.1016/j.biopsycho.2006.08.006
- Beyth-Marom, R., Austin, L., Fischhoff, B., Palmgren, C., & Jacobs-Quadrel, M. (1993). Perceived consequences of risky behaviors: Adults and adolescents. *Developmental Psychology*, 29(3), 549-563.
- Brook, J., Brook, D., Richter, L., & Whiteman, M. (2006). Risk and protective factors of adolescent drug use: Implications for prevention programs. In W. J. Bukoski (Ed.), *Handbook of drug abuse prevention* (pp. 265-287). US: Springer.
- Brook, J. S., Whiteman, M., Gordon, A. S., & Brook, D. W. (1985). Father's influence on his daughter's marijuana use viewed in a mother and peer context. *Advances in*

Alcohol & Substance Abuse, 4(3-4), 165-190.

Burnett, S., Bault, N., Coricelli, G., & Blakemore, S. J. (2010). Adolescents' heightened risk-seeking in a probabilistic gambling task. *Cognitive Development*, 25(2), 183-196. doi:10.1016/j.cogdev.2009.11.003

Burt, S. A., & Klump, K. L. (2014). Parent-child conflict as an etiological moderator of childhood conduct problems: An example of a 'bioecological' gene-environment interaction. *Psychological Medicine*, 44(5), 1065-1076.
doi:10.1017/S0033291713001190

Burt, S., Krueger, R. F., McGue, M., & Iacono, W. (2003). Parent-child conflict and the comorbidity among childhood externalizing disorders. *Archives of General Psychiatry*, 60(5), 505-513. doi:10.1001/archpsyc.60.5.505

Burt, S., McGue, M., Krueger, R. F., & Iacono, W. G. (2005). How are parent-child conflict and childhood externalizing symptoms related over time? Results from a genetically informative cross-lagged study. *Development and Psychopathology*, 17(1), 145-165. doi:10.1017/S095457940505008X

Carlson, S. R., & Iacono, W. G. (2006). Heritability of P300 amplitude development from adolescence to adulthood. *Psychophysiology*, 43(5), 470-480.
doi:10.1111/j.1469-8986.2006.00450.x

Casey, B.J., Forman, S.D., Franzen, P., Berkowitz, A., Braver, T.S., Nystrom, L.E., et al. (2001). Sensitivity of prefrontal cortex to changes in target probability: A functional MRI study. *Human Brain Mapping*, 13 (1), 26- 33. doi: 10.1002/hbm.1022

Cavalca, E., Kong, G., Liss, T., Reynolds, E. K., Schepis, T. S., Lejuez, C. W., &

- Krishnan-Sarin, S. (2013). A preliminary experimental investigation of peer influence on risk-taking among adolescent smokers and non-smokers. *Drug and Alcohol Dependence*, 129(1-2), 163-166. doi:10.1016/j.drugalcdep.2012.09.020
- Centers for Disease Control and Prevention. (2014). *About HIV/AIDS*. Atlanta: Author. Retrieved from <http://www.cdc.gov/hiv/basics/whatishiv.html>
- Centers for Disease Control and Prevention. (2013). *HIV Cost-effectiveness*. Atlanta: Author. Retrieved from <http://www.cdc.gov/hiv/prevention/ongoing/costeffectiveness/>
- Centers for Disease Control and Prevention (2012). *HIV Surveillance Report, 2010: vol. 22: Diagnoses of HIV infection and AIDS in the United States and Dependent Areas*. <http://www.cdc.gov/hiv/topics/surveillance/resources/reports/>. Published March 2012. Accessed 4/6/2014.
- Centers for Disease Control and Prevention. (2002). Youth risk behavior surveillance—United States, 2001. *Morbidity and Mortality Weekly Report*, 51, 1–64.
- Center for the Study of Emotion and Attention [CSEA-NIMH] (1999). *The international affective picture system: Digitized photographs*. Gainesville, FL: Author.
- Chaplin, T. M., Sinha, R., Simmons, J. A., Healy, S. M., Mayes, L. C., Hommer, R. E., & Crowley, M. J. (2012). Parent–adolescent conflict interactions and adolescent alcohol use. *Addictive Behaviors*, 37(5), 605-612. doi:10.1016/j.addbeh.2012.01.004
- Darling, N., Cumsille, P., Caldwell, L. L., & Dowdy, B. (2006). Predictors of adolescents' disclosure to parents and perceived parental knowledge: Between-

- and within-person differences. *Journal of Youth and Adolescence*, 35(4), 667-678.
- De Los Reyes, A., Aldao, A., Thomas, S.A., Daruwala, S.E., Swan, A.J., Van Wie, M., Goepel, K.A., & Lechner, W. (2012b). Adolescent self-reports of social anxiety: Can they disagree with objective psychophysiological measures and still be valid? *Journal of Psychopathology and Behavioral Assessment*, 34, 308-322. doi: 10.1007/s10862-012-9289-2
- De Los Reyes, A., Lerner, M.D., Thomas, S.A., Daruwala, S.E., & Goepel, K.A. (2013). Discrepancies between parent and adolescent beliefs about daily life topics and performance on an emotion recognition task. *Journal of Abnormal Child Psychology*, 41, 971-982. doi: 10.1007/s10802-013-9733-0
- De Los Reyes, A., Salas, S., Menzer, M.M., & Daruwala, S.E. (2013). Criterion validity of interpreting scores from multi-informant statistical interactions as measures of informant discrepancies in psychological assessments of children and adolescents. *Psychological Assessment*, 25, 509-519. doi: 10.1037/a0032081
- De Los Reyes, A., & Suarez, L. (2009). *Manual for the To(may)to-To(mah)to Interview-caregiver and youth versions*. Unpublished manuals. University of Maryland at College Park.
- De Los Reyes, A., Thomas, S.A., Swan, A.J., Ehrlich, K.B., Reynolds, E.K., Suarez, L., Dougherty, L.R., MacPherson, L., & Pabón, S.C. (2012a). “It depends on what you mean by ‘disagree’”: Differences between parent and child perceptions of parent-child conflict. *Journal of Psychopathology and Behavioral Assessment*, 34, 293-307. doi: 10.1007/s10862-012-9288-3.
- Dmitrieva, J., Chen, C., Greenberger, E., & Gil-Rivas, V. (2004). Family relationships

- and adolescent psychosocial outcomes: Converging findings from Eastern and Western cultures. *Journal of Research on Adolescence*, 14(4), 425-447.
- Donenberg, G. R., & Pao, M. (2005). Youths and HIV/AIDS: Psychiatry's role in a changing epidemic. *Journal of The American Academy Of Child & Adolescent Psychiatry*, 44(8), 728-747. doi:10.1097/01.chi.0000166381.68392.02
- Duncan, S. C., Duncan, T. E., Biglan, A., & Ary, D. (1998). Contributions of the social context to the development of adolescent substance use: A multivariate latent growth modeling approach. *Drug and Alcohol Dependence*, 50(1), 57-71.
- Earnshaw, V. A., Bogart, L. M., Dovidio, J. F., & Williams, D. R. (2013). Stigma and racial/ethnic HIV disparities: Moving toward resilience. *American Psychologist*, 68(4), 225-236. doi:10.1037/a0032705
- Edwards, G., Barkley, R. A., Laneri, M., Fletcher, K., & Metevia, L. (2001). Parent–adolescent conflict in teenagers with ADHD and ODD. *Journal of Abnormal Child Psychology*, 29(6), 557-572.
- Ehrlich, K. B., Dykas, M. J., & Cassidy, J. (2012). Tipping points in adolescent adjustment: Predicting social functioning from adolescents' conflict with parents and friends. *Journal of Family Psychology*, 26(5), 776-783. doi:10.1037/a0029868
- Elkins, I. J., King, S. M., McGue, M., & Iacono, W. G. (2006). Personality traits and the development of nicotine, alcohol, and illicit drug disorders: Prospective links from adolescence to young adulthood. *Journal of Abnormal Psychology*, 115(1), 26-39. doi:10.1037/0021-843X.115.1.26
- Farah, M. J., Shera, D. M., Savage, J. H., Betancourt, L., Giannetta, J. M., Brodsky, N.

- L., ... & Hurt, H. (2006). Childhood poverty: Specific associations with neurocognitive development. *Brain Research, 1110*(1), 166-174.
doi:10.1016/j.brainres.2006.06.072
- Feinberg, M. E., Button, T. M., Neiderhiser, J. M., Reiss, D., & Hetherington, E. M. (2007). Parenting and adolescent antisocial behavior and depression: Evidence of genotype \times parenting environment interaction. *Archives of General Psychiatry, 64*(4), 457-465. doi:10.1001/archpsyc.64.4.457
- Fisher, L., & Feldman, S. S. (1998). Familial antecedents of young adult health risk behavior: A longitudinal study. *Journal of Family Psychology, 12*(1), 66-80.
- Frick, P. J. (1991). *The Alabama Parenting Questionnaire*. Unpublished rating scale, University of Alabama.
- Geier, C. F., Terwilliger, R., Teslovich, T., Velanova, K., & Luna, B. (2010). Immaturities in reward processing and its influence on inhibitory control in adolescence. *Cerebral Cortex, 20*(7), 1613-1629.
- Gilmore, C. S., Malone, S. M., Bernat, E. M., & Iacono, W. G. (2010). Relationship between the P3 event-related potential, its associated time-frequency components, and externalizing psychopathology. *Psychophysiology, 47*(1), 123-132.
doi:10.1111/j.1469-8986.2009.00876.x
- Gottman, J. M. (1979). *Marital interaction: Experimental investigations*. New York: Academic Press.
- Gottman, J. M., & Levenson, R. W. (1992). Marital processes predictive of later dissolution: Behavior, physiology, and health. *Journal of Personality and Social*

- Psychology*, 63(2), 221-233.
- Gratton, G., Coles, M. G., & Donchin, E. (1983). A new method for offline removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology*, 55, 468–484.
- Gunlicks-Stoessel, M. L., & Powers, S. I. (2008). Adolescents' emotional experiences of mother-adolescent conflict predict internalizing and externalizing symptoms. *Journal of Research on Adolescence*, 18(4), 621-642.
- Gunnar, M. R., Talge, N. M., & Herrera, A. (2009). Stressor paradigms in developmental studies: What does and does not work to produce mean increases in salivary cortisol. *Psychoneuroendocrinology*, 34(7), 953-967.
- Hackman, D. A., & Farah, M. J. (2009). Socioeconomic status and the developing brain. *Trends in Cognitive Sciences*, 13(2), 65-73. doi:10.1016/j.tics.2008.11.003
- Hanley, J.A., Negassa, A., Edwardes, D.B., & Forrester, J.E. (2003). Statistical analysis of correlated data using generalized estimating equations: An orientation. *American Journal of Epidemiology*, 157, 364-375.
- Hansenne, M. (1999). P300 and personality: An investigation with the Cloninger's model. *Biological Psychology*, 50(2), 143-155. doi:10.1016/S0301-0511(99)00008-3
- Hendershot, C. S., Magnan, R. E., & Bryan, A. D. (2010). Associations of marijuana use and sex-related marijuana expectancies with HIV/STD risk behavior in high-risk adolescents. *Psychology of Addictive Behaviors*, 24(3), 404-414.
- Hicks, B. M., South, S. C., DiRago, A. C., Iacono, W. G., & McGue, M. (2009). Environmental adversity and increasing genetic risk for externalizing disorders.

Archives of General Psychiatry, 66(6), 640-648.

doi:10.1001/archgenpsychiatry.2008.554

Hill, S. Y., Steinhauer, S. R., Locke-Wellman, J., & Ulrich, R. (2009). Childhood risk factors for young adult substance dependence outcome in offspring from multiplex alcohol dependence families: A prospective study. *Biological Psychiatry*, 66(8), 750-757. doi:10.1016/j.biopsych.2009.05.030

Holmbeck, G. N. (2002). Post-hoc probing of significant moderational and mediational effects in studies of pediatric populations. *Journal of Pediatric Psychology*, 27(1), 87-96.

Iacono, W. G., Carlson, S. R., & Malone, S. M. (2000). Identifying a multivariate endophenotype for substance use disorders using psychophysiological measures. *International Journal of Psychophysiology*, 38(1), 81-96. doi:10.1016/S0167-8760(00)00132-X

Iacono, W. G., Carlson, S. R., Malone, S. M., & McGue, M. (2002). P3 event-related potential amplitude and the risk for disinhibitory disorders in adolescent boys. *Archives of General Psychiatry*, 59(8), 750-757. doi:10.1001/archpsyc.59.8.750

Iacono, W. G., Malone, S. M., & McGue, M. (2008). Behavioral disinhibition and the development of early-onset addiction: Common and specific influences. *Annual Review of Clinical Psychology*, 4, 325-348. doi:10.1146/annurev.clinpsy.4.022007.141157

Jackson, J., Kuppens, P., Sheeber, L. B., & Allen, N. B. (2011). Expression of anger in depressed adolescents: The role of the family environment. *Journal of Abnormal Child Psychology: An official publication of the International Society for*

Research in Child and Adolescent Psychopathology, 39(3), 463-474.

Johnson, S. B., Dariotis, J. K., & Wang, C. (2012). Adolescent risk taking under stressed and nonstressed conditions: Conservative, calculating, and impulsive types.

Journal of Adolescent Health, 51(2, Suppl), S34-S40.

doi:10.1016/j.jadohealth.2012.04.021

Kerr, M., & Stattin, H. (2000). What parents know, how they know it, and several forms of adolescent adjustment: Further support for a reinterpretation of monitoring.

Developmental Psychology, 36, 366-380. doi: 10.1037/0012-1649.36.3.366

Kerr, M., Stattin, H., & Burk, W. J. (2010). A reinterpretation of parental monitoring in longitudinal perspective. *Journal of Research on Adolescence*, 20(1), 39-64.

doi:10.1111/j.1532-7795.2009.00623.x

Kiecolt-Glaser, J. K., Fisher, L. D., Ogrocki, P., & Stout, J. C. (1987). Marital quality, marital disruption, and immune function. *Psychosomatic Medicine*, 49(1), 13-34.

Klahr, A. M., Rueter, M. A., McGue, M., Iacono, W. G., & Alexandra Burt, S. S. (2011).

The relationship between parent-child conflict and adolescent antisocial behavior:

Confirming shared environmental mediation. *Journal of Abnormal Child*

Psychology, 39(5), 683-694. doi:10.1007/s10802-011-9505-7

Krosnick, J. A., & Judd, C. M. (1982). Transitions in social influence at adolescence:

Who induces cigarette smoking? *Developmental Psychology*, 18(3), 359-368.

Lam, W. K. K., Cance, J. D., Eke, A. N., Fishbein, D. H., Hawkins, S. R., & Williams, J.

C. (2007). Children of African-American mothers who use crack cocaine:

Parenting influences on youth substance use. *Journal of Pediatric Psychology*,

32(8), 877-887.

Laursen, B., Coy, K. C., & Collins, W. A. (1998). Reconsidering changes in parent–child conflict across adolescence: A meta-analysis. *Child Development*, 69(3), 817-832.

Laursen, B., & Hafen, C. A. (2010). Future directions in the study of close relationships: Conflict is bad (Except when it's not). *Social Development*, 19(4), 858-872.

Leigh, B. C., & Stall, R. (1993). Substance use and risky sexual behavior for exposure to HIV: Issues in methodology, interpretation, and prevention. *The American Psychologist*, 48(10), 1035.

Lejuez, C. W., Aklin, W., Daughters, S., Zvolensky, M., Kahler, C., & Gwadz, M. (2007). Reliability and validity of the youth version of the Balloon Analogue Risk Task (BART-Y) in the assessment of risk-taking behavior among inner-city adolescents. *Journal of Clinical Child and Adolescent Psychology*, 36(1), 106-111.

Lejuez, C. W., Aklin, W. M., Zvolensky, M. J., & Pedulla, C. M. (2003). Evaluation of the Balloon Analogue Risk Task (BART) as a predictor of adolescent real-world risk-taking behaviours. *Journal of Adolescence*, 26(4), 475-479.
doi:10.1016/S0140-1971(03)00036-8

Lejuez, C. W., Read, J. P., Kahler, C. W., Richards, J. B., Ramsey, S. E., Stuart, G. L., . . . Brown, R. A. (2002). Evaluation of a behavioral measure of risk taking: The Balloon Analogue Risk Task (BART). *Journal of Experimental Psychology: Applied*, 8(2), 75-84.

Lester, P., Stein, J., Bursch, B., Rice, E., Green, S., Penniman, T., & Rotheram-Borus, M. (2010). Family-based processes associated with adolescent distress, substance use

- and risky sexual behavior in families affected by maternal HIV. *Journal of Clinical Child & Adolescent Psychology*, 39(3), 328-340.
- Lewis, M. D., Lamm, C., Segalowitz, S. J., Stieben, J., & Zelazo, P. D. (2006). Neurophysiological correlates of emotion regulation in children and adolescents. *Journal of Cognitive Neuroscience*, 18(3), 430-443.
doi:10.1162/jocn.2006.18.3.430
- Lowry, R., Holtzman, D., Truman, B. I., & Kann, L. (1994). Substance use and HIV-related sexual behaviors among US high school students: Are they related?. *American Journal of Public Health*, 84(7), 1116-1120.
doi:10.2105/AJPH.84.7.1116
- Luck S. J. (2005). An introduction to the event-related potential technique. Cambridge, MA: MIT Press.
- MacPherson, L., Reynolds, E. K., Daughters, S. B., Wang, F., Cassidy, J., Mayes, L. C., & Lejuez, C. W. (2010). Positive and negative reinforcement underlying risk behavior in early adolescents. *Prevention Science*, 11(3), 331-342.
doi:10.1007/s11121-010-0172-7
- McGue, M., Iacono, W. G., Legrand, L. N., Malone, S., & Elkins, I. (2001). Origins and consequences of age at first drink: I. Associations with substance-use disorders, disinhibitory behavior and psychopathology, and P3 amplitude. *Alcoholism: Clinical and Experimental Research*, 25(8), 1156-1165. doi:10.1111/j.1530-0277.2001.tb02330.x
- Mischel, W., & Shoda, Y. (1995). A cognitive-affective system theory of personality: Reconceptualizing situations, dispositions, dynamics, and invariance in

- personality structure. *Psychological Review*, 102(2), 246-268. doi:10.1037/0033-295X.102.2.246
- Mustanski, B., Byck, G. R., Dymnicki, A., Sterrett, E., Henry, D., & Bolland, J. (2013). Trajectories of multiple adolescent health risk behaviors in a low-income African American population. *Development and Psychopathology*, 25(4), 1155-1169. doi:10.1017/S0954579413000436
- Nation, M., & Heflinger, C. (2006). Risk factors for serious alcohol and drug use: The role of psychosocial variables in predicting the frequency of substance use among adolescents. *The American Journal of Drug and Alcohol Abuse*, 32(3), 415-433.
- Nelson, L. D., Patrick, C. J., & Bernat, E. M. (2011). Operationalizing proneness to externalizing psychopathology as a multivariate psychophysiological phenotype. *Psychophysiology*, 48(1), 64-72. doi:10.1111/j.1469-8986.2010.01047.x
- Nijs, I. M., Franken, I. H., & Smulders, F. T. (2007). BIS/BAS sensitivity and the p300 event-related brain potential. *Journal of Psychophysiology*, 21(2), 83-90. doi:10.1027/0269-8803.21.2.83
- Odgers, C. L., Caspi, A., Nagin, D. S., Piquero, A. R., Slutske, W. S., Milne, B. J., & ... Moffitt, T. E. (2008). Is it important to prevent early exposure to drugs and alcohol among adolescents?. *Psychological Science*, 19(10), 1037-1044. doi:10.1111/j.1467-9280.2008.02196.x
- Patterson, G. R. (1982). Coercive family process (Vol. 3). Castalia Publishing Company.
- Patterson, C., & Newman, J. P. (1993). Reflectivity and learning from aversive events: Toward a psychological mechanism for the syndromes of disinhibition.

- Psychological Review*, 100(4), 716-736. doi:10.1037/0033-295X.100.4.716
- Pellowski, J. A., Kalichman, S. C., Matthews, K. A., & Adler, N. (2013). A pandemic of the poor: Social disadvantage and the U.S. HIV epidemic. *American Psychologist*, 68(4), 197-209. doi:10.1037/a0032694
- Perlman, G., Johnson, W., & Iacono, W. G. (2009). The heritability of P300 amplitude in 18-year-olds is robust to adolescent alcohol use. *Psychophysiology*, 46(5), 962-969. doi:10.1111/j.1469-8986.2009.00850.x
- Pierson, A., le Houezec, J., Fossaert, A., Dubal, S., & Jouvent, R. (1999). Frontal reactivity and sensation seeking an ERP study in skydivers. *Progress In Neuro-Psychopharmacology & Biological Psychiatry*, 23(3), 447-463. doi:10.1016/S0278-5846(99)00008-1
- Polich, J. (2003). Theoretical overview of P3a and P3b. In J. Polich (Ed.), *Detection of change: Event-related potential and fMRI findings*. (pp. 83-98). Dordrecht Netherlands: Kluwer Academic Publishers.
- Polich, J. (2007). Updating p300: An integrative theory of P3a and P3b. *Clinical Neurophysiology*, 118(10), 2128-2148. doi:10.1016/j.clinph.2007.04.019
- Polich, J. (2012). Neuropsychology of P300. In Luck, S. J., & Kappenman, E. S. (Eds.), *The Oxford Handbook of Event-Related Potential Components*. (pp. 159-188). New York, NY US: Oxford University Press.
- Prado, G., Lightfoot, M., & Brown, C. (2013). Macro-level approaches to HIV prevention among ethnic minority youth: State of the science, opportunities, and challenges. *American Psychologist*, 68(4), 286-299. doi:10.1037/a0032917

- Prinz, R. J., Foster, S. L., Kent, R. N., & O'Leary, K. D. (1979). Multivariate assessment of conflict in distressed and nondistressed mother–adolescent dyads. *Journal of Applied Behavior Analysis*, 12(4), 691-700.
- Qin, S., Hermans, E. J., van Marle, H. F., Luo, J., & Fernández, G. (2009). Acute psychological stress reduces working memory-related activity in the dorsolateral prefrontal cortex. *Biological Psychiatry*, 66(1), 25-32.
doi:10.1016/j.biopsych.2009.03.006
- Quadrel, M. J., Fischhoff, B., & Davis, W. (1993). Adolescent (in)vulnerability. *American Psychologist*, 48(2), 102-116.
- Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin*, 128(2), 330-366.
- Rowe, C. L., Wang, W., Greenbaum, P., & Liddle, H. A. (2008). Predicting HIV/STD risk level and substance use disorders among incarcerated adolescents. *Journal of Psychoactive Drugs*, 40(4), 503-512. doi:10.1080/02791072.2008.10400656
- Rutter, M., Graham, P., Chadwick, O. F., & Yule, W. (1976). Adolescent turmoil: Fact or fiction? *Journal of Child Psychology and Psychiatry*, 17(1), 35-56.
- Ryan, S. M., Jorm, A. F., & Lubman, D. I. (2010). Parenting factors associated with reduced adolescent alcohol use: A systematic review of longitudinal studies. *Australian and New Zealand Journal of Psychiatry*, 44(9), 774-783.
- Sharma, L., Markon, K. E., & Clark, L. (2014). Toward a theory of distinct types of “impulsive” behaviors: A meta-analysis of self-report and behavioral measures.

Psychological Bulletin, 140(2), 374-408. doi:10.1037/a0034418

Simons, R. L., & Robertson, J. F. (1989). The impact of parenting factors, deviant peers, and coping style upon adolescent drug use. *Family Relations*, 38(3), 273-281.

Sinha, R., Bernardy, N., & Parsons, O. A. (1992). Long-term test-retest reliability of event-related potentials in normals and alcoholics. *Biological Psychiatry*, 32(11), 992-1003. doi:10.1016/0006-3223(92)90060-D

Smith, A. R., Chein, J., & Steinberg, L. (2014). Peers increase adolescent risk taking even when the probabilities of negative outcomes are known. *Developmental Psychology*, 50(5), 1564-1568. doi:10.1037/a0035696

Spear, L. (2011). Rewards, aversions and affect in adolescence: Emerging convergences across laboratory animal and human data. *Developmental Cognitive Neuroscience*, 1(4), 390-403. doi:10.1016/j.dcn.2011.08.001

Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in Cognitive Sciences*, 9(2), 69-74.

Steinberg, L. (2007). Risk taking in adolescence: New perspectives from brain and behavioral science. *Current Directions in Psychological Science*, 16(2), 55-59.

Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review*, 28(1), 78-106. doi:10.1016/j.dr.2007.08.002

Tabachnick, B. G., & Fidell, L. S. (1996). *Using Multivariate Statistics*. (3rd Edition). New York: HarperCollins College Publishers.

Thomas, S.A., Ehrlich, K.B., Augenstein, T. M., Kline, K., Giron, S., Greco, M.,

McFadden, C., Jain, A., & De Los Reyes, A. (2014). *A parent-adolescent conflict*

task derived from the To(may)to-To(mah)to Interview: Behavioral psychophysiological, and survey support. Manuscript in preparation.

- Tinsley, B. J., Lees, N. B., & Sumartojo, E. (2004). Child and adolescent HIV risk: Familial and cultural perspectives. *Journal of Family Psychology, 18*(1), 208-224.
- Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin, 119*(3), 488-531. doi:10.1037/0033-2909.119.3.488
- Van Doorn, M. D., Branje, S. J. T., & Meeus, W. H. J. (2008). Conflict resolution in parent-adolescent relationships and adolescent delinquency. *The Journal of Early Adolescence, 28*(4), 503-527.
- Van Leijenhorst, L., Zanolie, K., Van Meel, C. S., Westenberg, P. M., Rombouts, S. A. R. B., & Crone, E. A. (2010). What motivates the adolescent? Brain regions mediating reward sensitivity across adolescence. *Cerebral Cortex, 20*(1), 61.
- Williams, L. R., & Steinberg, L. (2011). Reciprocal relations between parenting and adjustment in a sample of juvenile offenders. *Child Development, 82*(2), 633-645.
- Yoon, H. H., Iacono, W. G., Malone, S. M., & McGue, M. (2006). Using the brain P300 response to identify novel phenotypes reflecting genetic vulnerability for adolescent substance misuse. *Addictive Behaviors, 31*(6), 1067-1087. doi:10.1016/j.addbeh.2006.03.036
- Ziv, Y., Cassidy, J., & Ramos-Marcuse, F. (2002). *The conflict task coding system*. Unpublished manuscript. University of Maryland, College Park, Maryland

Zuckerman, M., Bone, R. N., Neary, R., Mangelsdorff, D., & Brustman, B. (1972). What is the sensation seeker? Personality trait and experience correlates of the Sensation-Seeking Scales. *Journal of Consulting and Clinical Psychology*, 39(2), 308-321.