THE RELATIONSHIP BETWEEN RESPONSE INHIBITION DEFICITS AND INTIMATE PARTNER VIOLENCE PERPETRATION

by

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ABSTRACT

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Victims of intimate partner violence (IPV) perpetration suffer from lasting deleterious impacts of trauma. Although various risk factors of IPV have been explored, fewer studies have focused on executive functioning as potential risk factors for IPV perpetration. Response inhibition is a form of cognitive control which serves to stop the initiation of a maladaptive action/response. Within the context of IPV, adaptive response inhibition may inhibit utilizing aggression as a conflict strategy. Therefore, exploring the relationship between response inhibition deficits and IPV may further our understanding of IPV perpetration.

This study sought to explore the relationship between response inhibition and IPV in young LGB+ adults (N=207) while controlling for related IPV covariates. More specifically, how deficits in response inhibition correlate to specific types of IPV and if response inhibition deficits predict future IPV perpetration. The results showed that response inhibition was not significantly related to IPV perpetration, however, future research is needed to further explore how response inhibition may affect other aspects of IPV perpetration.

Keywords: Intimate Partner Violence; Response Inhibition; Stop Signal Task

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The Relationship between Response Inhibition Deficits and Intimate Partner Violence Perpetration

Intimate partner violence (IPV) causes detrimental impacts on the wellbeing of IPV victims which extrapolate to harm families, communities, and society as a whole. In fact, the estimated lifetime cost associated with personal and societal loss to IPV victimization is around 3.6 trillion dollars (CDC, 2021). The National Intimate Partner and Sexual Violence Survey reports around a third of men and women experience physical violence (e.g., scratching, choking, punching), around 35 % of men and 45 % of women experience psychological aggression (name-calling, threats of violence, and limiting access to money), and around 40% of women and 25% of men experiences sexual violence (e.g., unwanted sexual contact, verbal sexual harassment, and rape) within their lifetime (Smith et al., 2018). Recent evidence also suggests that intimate partner violence peaks during young adulthood with a quarter of young adults reporting the perpetration of IPV (Halpern et al., 2009). Additionally, a large IPV national survey study suggests that young adults from 20 to 25 years of age experience the most instances of IPV compared to adolescents and older adults (Johnson et al., 2009). Furthermore, research has indicated that risk of IPV may be higher in LGB+ relationships with some studies reporting instances of IPV as high as 97% when considering instances of a wide range of physical, psychological, and sexual IPV (Hequembourg et al., 2008). Therefore, further research is needed to understand contributing factors to IPV in high-risk populations.

Victims of IPV perpetration suffer from lasting physiological and psychological consequences due to the trauma of IPV (Mazza et al., 2020). IPV victims are at higher risk for mental health disorders including post-traumatic stress disorder (PTSD), substance use disorder,

mood and anxiety disorders, and eating disorders (El-Serag & Thurston, 2020). A wide range of maladaptive behaviors also manifest due to IPV victimization including self-harm, suicidal ideation, emotional regulation deficits, increased avoidance, increased use of emotional suppression, and expression of negative emotion to stress including IPV perpetration (Gruhn and Compas, 2020). Additionally, victims are also at higher risk for adverse physical conditions such as cardiovascular disorders, chronic pain, poor sleep, sexually transmitted diseases, and traumatic brain injuries (El-Serag & Thurston, 2020).

IPV does not occur within a vacuum and numerous risk factors are thought to contribute to the perpetration of violence between partners. Contextual characteristics of partners (e.g., demographic, community factors), relationship factors (e.g., relationship satisfaction, partner discord), developmental characteristics (e.g., family influence such as childhood maltreatment, witnessing IPV in childhood; McMahon et al., 2018; Roberts et al., 2010) and partner psychological and behavioral characteristics (e.g., personality disorders, cognitive factors) coalesce to influence the perpetration of IPV (Capaldi et al., 2012). Due to the wide range of risk factors, it is imperative to find modifiable correlates of IPV perpetration that can be targeted for intervention.

Researchers have investigated various psychological and behavioral characteristics which may predispose individuals to initiate interpersonal violence. A plethora of these factors have often been cited within the literature which contribute to the perpetration of IPV. Some of the more prominent covariates of IPV include trait impulsivity (Shorey et al., 2011), emotional dysregulation (Gratz & Roemer, 2004; Jakupcak et al. 2005), alcohol use (Eckhardt et al., 2015; McKinney et al., 2010), post-traumatic stress disorder symptoms (Bell & Orcutt, 2009; Orcutt et al., 2003; Taft et al., 2007b), and minority stress (Martin-Storey & Fromme, 2017; Edwards &

Sylaska, 2013). Although extensive research has been conducted to understand the risk factors which impel aggressive behaviors, less research has been conducted on the underlying cognitive deficits that may be associated with the inhibition of IPV.

Response inhibition is a potential cognitive factor associated with IPV due to its vital importance in successful cognitive and behavioral control (Chambers et al., 2008). Response inhibition is a form of executive functioning which requires voluntary control, or suppression, of goal-irrelevant stimuli, cognitions, and behaviors during goal-driven behavior (Mostofsky & Simmonds, 2008). This form of cognitive control is required to withhold preplanned responses, interrupt ongoing actions, delay responses and is essential for behavioral and social adaption (Tamm et al., 2002). Within IPV, the ability to inhibit aggressive thoughts and behaviors is theoretically crucial to reduce the perpetration of violence. However, there has been a paucity of research looking into the association between response inhibition deficits and IPV. Therefore, it is crucial to investigate how response inhibition deficits, and related covariates, contribute to the perpetration of IPV.

Executive Functioning Deficits and IPV

A range of executive function (EF) cognitive deficits have been shown in individuals who perpetuate IPV (Ali & Naylor, 2013; Aupperle et al., 2016; Corvo et al., 2006; Edalati et al., 2018; Pinto et al. 2010; Romero-Martínez & Moya-Albiol, 2013; Rosenbaum et al. 1989). Indeed, IPV perpetrators have been shown to have lower abilities in controlling impulsive behavior, cognitive flexibility, and sustaining attention (Romero-Martínez & Moya-Albiol, 2013). Additionally, adverse events (e.g., traumatic brain injury), and maladaptive behaviors leading to behavioral disinhibition (e.g., habitual alcohol consumption) (Romero-Martínez &

Moya-Albiol, 2013; McKinney et al., 2010), may affect executive dysfunction which is associated with increased IPV perpetration. Indeed, in some samples, one quarter of spouses report an increase of verbal abuse and physical violence from their partner who had experienced a traumatic brain injury, and report that these aggressive behaviors become progressively worse over a five year period (Mauss-Clum & Ryan, 1981; Brooks et al., 1986) Within several studies, half of individuals who have perpetuated IPV were found to have a history of a traumatic brain injury (TBI) with most damage occurring within the frontal lobe (Pinto et al., 2010; Rosenbaum et al., 1994). Due to self-reparative process of the brain (i.e., brain plasticity), recent TBIs are often more predictive of violence (Stoddard et al., 2011). Young adults are most at risk for TBIs and thus may also be at a higher risk of perpetrating violence due to the impact of TBI on brain areas responsible for inhibition (CDC, 1999; Williams et al., 2018). Further evidence connecting brain abnormalities to maladaptive social behavior is shown through increased aggressive and antisocial behavior in individuals with frontal lobe damage (Brower & Price, 2001). It is important to note that brain areas important for response inhibition (e.g., dorsolateral prefrontal cortex, ventrolateral prefrontal cortex) are housed within this common site of injury within IPV individuals (Blasi et al., 2006).

Evidence also suggests that individuals with higher magnitudes of EF deficits perpetrate more severe forms of IPV (Corvo et al., 2006). The influence of EF deficits on IPV perpetration have also been shown to go beyond the effects of habitual use of alcohol and head injury, which may indicate predisposition to poor EF may contribute to intensity of IPV (Horne et al., 2020; Pinto et al., 2010). What factors of EF contribute most directly to IPV remain unclear, which has created a call for further research delineating specific executive functioning deficits, such as deficits in response inhibition, that contribute to IPV (Horne et al., 2020). Very little is known

about how cognitive deficits in response inhibition are associated with IPV perpetuation beyond self-reported data of related personality traits and behavioral tendencies (e.g., impulsivity). Thus, it is crucial to explore the association between perpetration of IPV and response inhibition deficits assessed by well-established cognitive task paradigms such as the stop-signal task (Verbruggen & Logan, 2008; Verbruggen & Logan, 2009; Li et al., 2008).

Covariates of IPV

To evaluate the association between response inhibition deficits and the perpetration of IPV, it is important to take into consideration relevant covariates of IPV. Trait impulsivity (Shorey et al., 2011), emotional dysregulation (Gratz & Roemer, 2004; Jakupcak et al. 2005), problematic alcohol use (Eckhardt et al., 2015; McKinney et al., 2010), post-traumatic stress disorder (PTSD) symptoms (Bell & Orcutt, 2009; Orcutt et al., 2003; Taft et al., 2007b), and minority stress (Martin-Storey & Fromme, 2017; Edwards & Sylaska, 2013) have been shown as risk factors for IPV perpetration. Controlling for these covariates will allow us to examine how response inhibition increases the incremental validity of the model when predicting IPV perpetration.

Trait Impulsivity

Impulsivity is a stable personality factor shown to increase risk for aggressive behaviors (Derefinko et al., 2011). Impulsivity is characterized by rapid decision making and lack of planning that can lead to careless actions (Magid et al., 2007). IPV perpetrators are more likely to have personality disorders such as conduct disorder, antisocial personality disorder, and/or borderline personality disorder (Yu et al., 2012) that can be characterized by trait impulsivity and

aggression. When an intimate partner has a personality with higher trait aggression, having a tendency towards impulsive behavior will increase the likelihood of this aggression may go unchecked and lead to IPV perpetration. In fact, individuals with marked impulsivity are more likely to perpetuate psychological (Shorey et al., 2010) and physical IPV (Cunradi et al., 2009). Furthermore, individuals who perform impulsive aggressive acts have been shown to commit more moderate and severe forms of IPV compared to less severe IPV (Cascardi et al., 2018). In this sense, impulsive individuals will show greater difficulty in withholding rapid and poorly planned conflict tactics leading to physical and emotional outbursts that result in IPV. A key component of impulsivity is the inability to inhibit responses while performing goal directed behavior (Shen et al., 2014). Therefore, trait impulsivity is important to explore as a covariate in examining the association between response inhibition deficits and IPV.

Emotional Dysregulation

Emotional regulation is defined as a goal driven process to modify the intensity and duration of emotional experience and expression (Thompson, 1991). This can come in the form of emotional suppression or cognitive reappraisal (e.g., realigning your perspective on the cause and the consequences of a conflict) (Gross, 1998b). In contrast, emotional dysregulation is characterized by difficulties engaging in goal-directed behavior and impulse control, lack of emotional awareness and clarity, and limited access to affective regulation strategies (Gratz & Roemer, 2004).

When an intimate partner feels intense negative emotions related to a relationship conflict, without proper emotional regulation violence may become a tool to resolve the conflict and abate the flood of negative affect (Gratz & Roemer, 2004; Jakupcak et al. 2005). Indeed, the

ability to regulate negative emotions is associated with less IPV perpetration and emotional regulation has been shown to be a moderator between proximal negative affect and physical aggression (McNulty & Hellmuth, 2008; Shorey et al., 2015). Furthermore, emotional dysregulation has been shown as a mediator between a history of childhood maltreatment and intimate partner physical aggression in men (Gratz et al., 2009). In this sense, emotional dysregulation decreases the ability to effectively inhibit negative affect (e.g., anger) during conflicts with a partner and increases the perpetuation of learned violent behavior as a conflict resolution technique.

Alcohol Use

Within several studies it has been shown around 50 percent of individuals who perpetuate IPV have received treatment for substance abuse disorders (Murphy & O'Farrell, 1994; O'Farrell & Murphy, 1995). Although substance use is a risk factor for IPV, both perpetration and victimization, there are mixed findings for specific illicit substances that increase IPV perpetration (Kraanen et al., 2014; Cafferky et al., 2018). However, factors of alcohol use have been repeatedly shown as a predictor of IPV perpetration (e.g., Chase et al., 2003, Fals-Stewart et al., 2003, Pan et al., 1994, Schumacher et al., 2003; Thompson & Kingree, 2006). Alcohol is a substance known to cause individuals to become disinhibited and to ignore cues from their environment (Källmén & Gustafson, 1998; Steele et al., 1990). Within the context of IPV, this disinhibition can cause difficulties regulating emotions and inhibiting maladaptive conflict resolution tactics (Acheson et al., 2011; La Berre, 2019).

Alcohol is the substance most prominently linked to violence perpetration and proximal use has been shown to increase IPV severity (e.g., Langenderfer, 2013; McKinney et al. 2010).

Indeed, the likelihood of perpetuating physical and psychological IPV are increased on days when individuals consume alcohol relative to non-drinking days (Moore et al., 2011). When a partner within a violent couple has been drinking, research shows that the IPV is more frequent, severe, and more likely to lead to reciprocal violence between both partners (McKinney et al., 2010). Furthermore, alcohol use has been shown as a partial mediator between emotional dysregulation and intimate partner violence, and a mediating factor between impulsivity and violence (Grigorian et al., 2020; Field et al., 2004).

A meta-analysis of alcohol's impact on IPV perpetration have shown that problematic drinking (e.g., problematic drinking behaviors, abuse, dependence) may be more significantly associated with IPV perpetration than consumption (e.g., frequency, amount) itself (Cafferky et al., 2018). This suggests that alongside the disinhibiting effects of alcohol, poor impulse control may also lead to maladaptive behaviors surrounding alcohol consumption.

Post-Traumatic Stress Symptoms

The association between post-traumatic stress disorder (PTSD) and the perpetration of aggression has been well-established within the literature (Taft et al., 2007; Mcfall et al., 1999; Jakupcak et al., 2007). Lifetime prevalence of PTSD predicts IPV perpetration after controlling for other covariates of IPV such as substance use and major depressive disorder (MDD) (Hahn et al., 2015). Furthermore, individuals who perpetrate IPV were found to be twice as likely to have PTSD (Rosenbaum & Leisring, 2003).

Individuals with PTSD struggle with intrusive symptoms (e.g., physical reactivity after exposure to traumatic reminders), avoidance symptoms (e.g., avoiding trauma related thoughts), alterations in cognitions and mood (e.g., overly negative thoughts about self or others), and

alterations in arousal/reactivity (e.g., aggression, destructive behavior) (Hoge et al, 2014). Research has also shown veterans with elevated PTSD symptomology are more likely to report anger, hostility, aggression with subsequent IPV perpetration (Bell & Orcutt, 2009).

Increased levels of reactivity are associated with hyperarousal, which occurs when individuals have heightened reactivity to perceived threats in their environment (Chemtob et al., 1997). Indeed, research has shown an overactive amygdala (producing fear) and an underactive prefrontal cortex (inhibition of fear) is associated with hyperarousal symptoms common to individuals with PTSD (Wolf & Herringa, 2016). Taft et al. (2007a) demonstrated a relationship between hyperarousal and the initiation of aggressive behaviors (e.g., physical violence, verbal abuse). Individuals with PTSD have been shown to have higher levels of anger and emotional reactivity which may lead to increased perpetration of IPV when not successfully inhibited (Taft et al., 2007b). After controlling for a history of dysfunctional family origin, previous antisocial behavior, and the severity of trauma exposure, hyperarousal still had a significant positive association with IPV perpetuation (Orcutt et al., 2003). Thus, this suggests the combination of heightened emotional lability and weakened inhibition may lead to more instances of IPV.

Minority Stress

The minority stress model (Meyer, 2003) posits that chronic expectations of social rejection, stress, and internalized sexual minority stress associated with sexual orientation, creates deleterious mental health consequences. Indeed, LGB+ individuals have shown an increased likelihood of developing anxiety and mood disorders throughout their lifetime compared to heterosexual individuals (Institute of Medicine, 2011). More specifically, experiences of discrimination towards an individual's sexual orientation have been shown to be

associated with negative mental health outcomes (Mays, 2001). In addition to poor mental health outcomes, minority stress in LGB+ individuals has shown to be associated with increased perpetration of physical and psychological IPV (Martin-Storey & Fromme, 2017; Edwards & Sylaska, 2013).

The strength model of self-control posits that self-regulation is a limited resource which can dwindle with repeated use. Research suggests that individuals who repeatedly face stressful situations such as minority stress exhaust self-regulatory resources (Baumeister & Vohs, 2003). This can also occur when needing to suppress negative affect related to concerns with identity. Matheson and Cole (2004) found that individuals who experience identity threats results in self-regulatory expressive suppression which may have consequences on executive functions such as response inhibition. Indeed, Johns, Inzlicht, and Schmader (2008) found that victims of stereotype threat engage in more expressive suppression effortfully, which has been shown to cause poorer performance on executive functioning tasks. Therefore, with a need to perform expressive suppression in the face of internalized or externalized minority stress consuming vital self-regulatory resources, instances of IPV perpetration may be more difficult to inhibit.

Current Study and Rationale

Executive function deficits have been shown in individuals who perpetuate IPV (Horne et al., 2020). Although various risk factors have been explored within the IPV literature, few studies have focused on cognitive deficits associated with IPV perpetration risk. Within the context of IPV, response inhibition deficits may lead perpetrators to fail to inhibit aggressive behaviors as a conflict resolution strategy. Additionally, further understanding of the link between response inhibition and IPV perpetration will possibly pave the way for new clinical

approaches to IPV prevention. Therefore, it is crucial to further explore the relationship between response inhibition deficits and how these deficits relate to IPV perpetration.

The current study examined the relationship between response inhibition and IPV. More specifically, how deficits in response inhibition, alongside related covariates (i.e., trait impulsivity, alcohol use, emotional dysregulation, PTSD symptom severity, and minority stress), correlate to specific types of IPV (e.g., physical, psychological, sexual) and explored if response inhibition deficits predict future IPV perpetration assessed approximately five months later.

Study Aims and Hypotheses

Aim 1: To examine the relationship between response inhibition and IPV perpetration while taking into account related covariates.

> **Hypothesis 1**: Deficits in response inhibition will significantly explain the perpetration of physical, sexual, and psychological IPV after controlling for the influence of trait impulsivity, emotion dysregulation, alcohol use, PTSD symptoms, and minority stress.

Aim 2: To examine how response inhibition at Time 1 predict subsequent IPV perpetration at Time 2 (= 5 month follow up)

> **Hypothesis 2:** Response inhibition deficits will predict significantly physical, sexual, and psychological IPV perpetration at Time 2 while controlling for trait impulsivity, emotion dysregulation, alcohol use, PTSD symptoms, and minority stress as well as the level of IPV at Time 1.

Methods

Study Participants

The current study used pre-existing data derived from a larger project that sought to examine temporal and prospective relations between alcohol and IPV perpetration, and victimization among LGB+ young adults. This study was comprised of young adults (18-25 years old), recruited from a population of students and community members at the University of Wisconsin - Milwaukee, the University of Tennessee - Knoxville, and from across Wisconsin and Tennessee. Inclusion criteria included the following: (1) Identify as lesbian, gay, bisexual, queer, pansexual, asexual, same-gender loving or another non-heterosexual orientation (LGBQ+), (2) Be 18-25 years old, (3) Be in a dating relationship that has lasted at least one month with an individual who is 18+ years old, (4) Have a minimum of 2 contact days weekly with their partner, (5) Not be fearful of their partner, (6) Have consumed alcohol in the past month. Some studies have shown that IPV rates are higher within LGBQ+ couples compared to opposite sex community (Cannon & Buttell, 2016). This population was chosen for the previous study to understand more about unique factors that contribute to IPV perpetration and victimization within the LGBQ+ community. The final sample included in the current study consisted of 207 individuals who were predominantly white (80%), non-Hispanic (89%), and female (78%) for sex assigned at birth with an average age of 21.5 years old (SD = 2.02). A large majority reported as Bi+ as their sexual orientation, which is comprised of queer, pansexual, asexual, same-gender loving, and alternate-self indications including queer/asexual. Additionally, the majority of participant's identified their gender as woman (see Table 1).

Recruitment and Evaluation

Participants were recruited from a population of students at the University of Wisconsin - Milwaukee and the University of Tennessee - Knoxville enrolled in psychology or other courses around each campus. The original study also recruited community members across the states of Wisconsin and Tennessee. This study was advertised using flyers around campus and in the surrounding communities, via email serv lists to departments across campus, within psychology course class forums, using social media platforms (e.g., Facebook, Craigslist), in person at events and festivals, and within local newspapers/magazines.

Participants were required to sign a consent form before completing screening questions for the study. After providing their consent, participants were screened using Qualtrics for the previously mentioned inclusion/exclusion criteria. If they met these criteria, they were then contacted via email or phone to complete the baseline assessment for the study. The baseline assessment took approximately 2 hours and consisted of multiple sets of questions (e.g., measures, demographics) and behavioral tasks (e.g., stop signal task). This pre-existing data set also gathered 60 days of daily diary reports that will not be used in this study. Follow up surveys were conducted 3 months following the daily diary portion of the study. The baseline and follow up assessments were either collected online utilizing Qualtrics or in person at each university's lab.

Table 1

Demographics (n = 207)		Mean	SD
Age		21.53 years	2.02
Sex Assigned at Birth	Male	22.5 %	-
	Female	77.5 %	-
Race	American Indian/ Alaskan Native	2.16 %	-
	Asian	5.19 %	-
	Black/African American	4.33 %	-

	White/Caucasian	80.09 %	-
	Multiracial	5.19 %	-
	Other	2.16 %	-
	Prefer Not to Say	0.87 %	-
Gender[a]	Man	20.9 %	
	Women	58.2 %	
	Trans Man	8.7 %	
	Trans Women	.6 %	
	Gender Queer/Non-Conforming	17 %	
	Alternative Identity	8 %	
Ethnicity			-
	Hispanic or Latino	9.7 %	-
	Not Hispanic or Latino	89.3 %	-
	Prefer Not to Say	1 %	-
Sexual Orientation	Gay	15 %	-
	Lesbian	15 %	-
	Bi+	70 %	-
Relationship Length		18.5 (months)	18

[a] *Participants were allowed to choose multiple genders to allow the most representative description of their gender identity. Several alternative gender identities were provided including non-binary, demi-girl, demi-man, agender, trans-masculine, trans-feminine, and agender*

Measures

Self-Report Measures

Demographics. The demographics form assessed age, race, ethnicity, sex assigned at birth, gender identity, academic status, employment status, relationship status and history, and parental education history.

Intimate Partner Violence. The Conflict in Adolescent Dating Relationships Inventory (CADRI; Wolfe et al., 2001) is a 50-item measure of physical (e.g., I kicked, hit, or punched my partner), verbal (e.g., I insulted my partner with put downs), and sexual (e.g., I forced my partner to have sex when my partner didn't want to) abuse, as well as relational abuse and threatening behavior relating to dating partners. We plan to only assess for physical, psychological, and sexual violence within this measure. The 50-items were divided into two sets of questions (25-items each) related to self-perpetration and partner-perpetration of IPV. Respondents completed

the instrument only in reference to conflict with a current or recent ex-dating partner. If the respondent had multiple partners, they were asked to answer each question combining the frequency of IPV from all of their partners. Participants are asked to rate the number of times a specific conflict tactic was used by both the participant and their partner in the past year. Participants were given a 8 point Likert scale ranging from 0-7 with the following options: This never happened (0), Once in the past 12 months (1), Twice in the past 12 months (2), 3-5 times in the past 12 months (3), 6-10 times in the past 12 months (4), 11-20 times in the past 12 months (5), More than 20 times in the past 12 months (6), or Not in the past 12 months, but it did happen before (7). A common and effective way to score this measure is utilizing frequency scoring (Shorey et al., 2012). Frequency scoring uses the midpoint within selected responses for each item. For example, if a participant selected a response of "3" (i.e., 3-5 times in the past 12 months) for an item, the frequency would be calculated as 4. However, responses without a range are scored as is (e.g., twice in the past 12 months is a frequency of 2). Frequency scoring was used to gauge levels of IPV perpetration within this study.

Trait Impulsivity. The UPPS-P Short Version (Cyders et al., 2014). The UPPS-P Short Version is a 20-item questionnaire that assesses impulsivity. It is separated into five empirically-supported facets of impulsivity: positive urgency (tendency to act impulsively in times of positive affect), negative urgency (tendency to act impulsively in times of negative affect), lack of perseverance (tendency to give up in the face of boredom, fatigue or frustration), lack of premeditation (tendency to act without consideration of the potential consequences of behavior), and sensation seeking (tendency to pursue activities that are exciting and novel).

Emotion Dysregulation. The Difficulties in Emotion Regulation Scale - 18 (DERS-18; Victor & Klonsky, 2016) is a short-form of a self-report measure that assess difficulties in

emotion regulation (i.e., emotion dysregulation). In particular, the DERS-18 assesses an individual's acceptance, awareness, and understanding of their emotion. Items are rated from 1 ("almost never") to 5 ("almost always") with some items being reverse-coded such that higher scores reflection greater amounts of emotional dysregulation. This scale consists of six subscales measuring different forms of difficulties in emotion regulation. The DERS-18 has exhibited good reliability, internal consistency, and convergent validity. Scores on the DERS-18 also demonstrate good convergent validity with the original DERS (Victor & Klonsky, 2016). Only total scores were used within the current study.

Alcohol Use. The Alcohol Use Disorder Identification Test (AUDIT; Barbor et al., 2001) is a 10 item self-report measure that assesses alcohol consumption, drinking behavior, and alcohol-related problems. Participants indicate their answers by choosing the response that best describes their drinking behavior for each question based on quantity and frequency of alcohol use. Participants completed this measure twice: once in reference to their own alcohol use and once in reference to their partner's alcohol use.

PTSD Symptoms. The PTSD Checklist for DSM-5 (PCL-5; Weathers et al., 2013) was used to measure participants' PTSD symptoms. It contains 20 items rated using a 5-point Likert scale (from 0 - 4; not at all -- extremely). The main measure of PTSD severity will be the total PCL-5 score, but we will also examine each symptom cluster provided by the scale: intrusion, avoidance, negative alterations in cognition and mood, and hyperarousal. The PCL-5 has good test-retest reliability ($r = 0.82$-0.84), good internal consistency ($\alpha = 0.94$-0.96), and good convergent and discriminant validity.

Minority Stress. The Internalized Homophobia Scale (IHS; Theodore et al., 2013) was used to measure participant internalized sexual minority stress. Considering the nature of the

current study sample, IHS is a relevant measure to assess the experience of minority stress. The IHS is a 12-item self-report measure which asks participants to indicate their level of agreement with items related to self-assurance and worth, public appearance of sexual orientation, and extreme or maladaptive measures to change sexual orientation.

Response Inhibition Task

Stop Signal Task. We assessed participant response inhibition ability using the Stop Signal Task (SST; Verbruggen et al., 2008). This task lasts around 15 minutes consisting of a practice block (i.e., a set of trials) and three experimental blocks. Each practice and experimental block consist of 32 trials each. As shown in *Figure 1,* each trial begins with a fixation circle in which the participant should fixate upon that is then followed with an arrow (go signal) being presented within the circle at 250 milliseconds (ms) into the trial. This behavioral task asks participants to respond whether an arrow is pointing left or right on a computer monitor with corresponding keyboard keys to indicate the perceived arrow direction on "go trials", but to inhibit this response on "stop trials". On stop trials, a go signal (e.g., an arrow) is shown at 250 ms and then a stop signal (i.e., an audible beep) is played after a delay (default to start at 250 ms after the go signal). The delay in the stop signal after the go signal is shown (i.e., stop signal delay [SSD]) decreases if the participant is unable to inhibit their response (i.e., responds by pressing a key) by 50 ms or

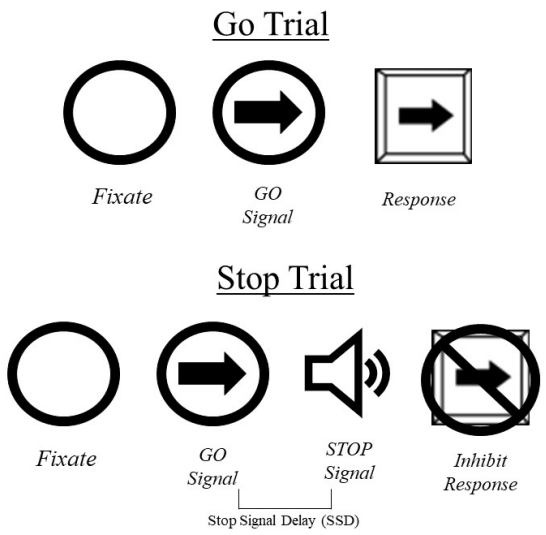

Figure 1. Stop Signal Trial Sequence

increases by 50 ms if a correct response is recorded. Each SSD is set to default at 250 ms after the go signal at the beginning of the experimental block.

As the primary outcome index on this task, the time difference between the average go trial response time and SSD capture the minimum time required to inhibit an initiated response (i.e., stop signal reaction time [SSRT]; Verbruggen et al., 2008). SSRT is a measure of the latency required to stop an ongoing action after it has been initiated by a cue (e.g., the go signal). Two cognitive processes theoretically occur during the stop signal task. A go signal initiates a process to accomplish the goal of correctly reporting an arrow's direction. However, the stop signal (e.g., a beep) initiates a process to inhibit this response. If the response inhibition process is completed before the goal attainment process, then the response is inhibited. Thus, SSRT is a measure of the average time needed to complete the response inhibition process and smaller SSRT(s) will signify more efficient (i.e., faster) behavioral inhibition. Thus, our main predictor SSRT was used to measure our participants response inhibition ability (Verbruggen, 2008).

Data Analysis

Specific Aim 1: To examine the relationship between response inhibition and IPV perpetration while taking into account related covariates.

We hypothesize that deficits in response inhibition will significantly explain the perpetuation of IPV after controlling for the influence of other related covariates. To test this hypothesis, we used hierarchical linear regression with IPV as the dependent variable. In Step 1 we entered each covariate of IPV: impulsivity, alcohol use, emotional dysregulation, PTSD symptoms, and minority stress. In Step 2, we entered indices of response inhibition deficits (i.e., SSRT) which allowed us to examine the proportion of additional variance explained by response inhibition deficits in the last step (= R^2

increase in Step 2) after controlling for other related covariates in Step 1. This enabled us to test the incremental validity of response inhibition deficits as a useful correlate of IPV perpetration. Moreover, we repeated this hierarchical regression analysis for each subtype of IPV (i.e., psychological, physical, and sexual) to observe whether the association between response inhibition deficits and IPV varies as a function of the type of perpetration.

Specific Aim 2: To examine how RI deficits at Time 1 predict subsequent IPV perpetration at Time 2 (= 3 months later)

We hypothesize that response inhibition deficits will predict significantly IPV perpetuation at Time 2 while controlling for relevant covariates as well as the level of IPV at Time 1. To test this hypothesis, we used hierarchical linear regression with IPV that occurred with the participants current partner at Time 2 as the dependent variable. In Step 1, we entered IPV perpetration at Time 1, to control for its baseline severity in predicting its subsequent level at Time 2. In Step 2, we included the previously described covariates to examine how much additional variance in IPV at Time 2 is explained by these variables. In Step 3, we entered response inhibition last to observe whether response inhibition will explain a significant proportion of variance of IPV after controlling for other related covariates. In these hierarchical regression analyses, we examined the beta coefficient and the statistical significance of each of the predictors in the model to evaluate their relative contribution to the model (beta, t-test). We also evaluated the model by inspecting the R^2 (the proportion of variance explained by the predictors) for each step and for the whole model. Finally, we repeated this process for each IPV subtype.

To address the potential issue of multicollinearity, we examined the relevant diagnostic indices to ensure that predictors within our regression models are not highly correlated with each other. In the literature, a VIF value less than 10 (Alin, 2010) indicates the absence of significant multicollinearity issues.

We also examined each variable that was entered into the regression. If the variable was not normally distributed based on the 1-sample Kolmogorov-Smirnov Test for Normality, we analyzed the variable after conducting a natural log transformation[1].

Power Analysis

There is no pilot data or relevant literature to guide us to assume a particular level of effect size in examining the association between response inhibition deficits and IPV perpetration. Thus, we conducted power analyses based on conventional effect sizes. We utilized GPower*3 to compute power using the R^2 increase test for hierarchical regression analyses.

To detect a medium effect size ($f = 0.15$) setting $\alpha = 0.05$, with 1 main predictor with 5 covariates in the hierarchical regression analysis, the required sample size is 55 to attain a power of 0.80. To detect a small effect size ($f = 0.02$) setting $\alpha = 0.05$, with 1 main predictor with 5 covariates in the hierarchical regression analysis, the required sample size is 395 to attain a power of 0.80. Therefore, the current sample size (N= 217) was sufficient to detect a small-to-medium sized effect .

Results

Descriptive Data on Demographic and Clinical Variables

Means and standard deviations of study variables, including self-report measures and SST task indices, are presented in Table 2. Overall, at T1, the current study sample reported low frequencies of sexual and physical IPV (M = .51), but higher frequencies of psychological IPV (M = 15.98). At T2, physical IPV (M = 3.41) perpetration increased, but both sexual (M = .61) and psychological IPV (M = 2.49) decreased numerically. Paired samples t-test showed that physical IPV perpetration significantly increased from T1 to T2 (t=4.4, $p < .001$) and psychological IPV perpetration significantly decreased from T1 to T2 (t= -8.8, $p < .001$) IPV. However, rates of sexual IPV perpetration did not significantly change from T1 to T2. SSRT, the index of response inhibition deficits, had an average of 208 milliseconds (ms) and the overall accuracy of the task performance was at 97%.

Table 2
Means and standard deviations of study variables

Variables	Mean	SD
IPV Frequencies at T1		
Physical IPV Perpetration	.51	2.85
Sexual IPV Perpetration	.93	3.49
Psychological IPV Perpetration	15.98	24.16
IPV Frequencies at T2		
Physical IPV Perpetration T2	3.41	8.05
Sexual IPV Perpetration T2	.61	2.59
Psychological IPV Perpetration T2	2.49	5.94
Clinical Measures at T1		
AUDIT Total Score	7.17	7.83
PCL-5 Total Score	29.19	18.13
DERS Total Score	50.29	12.44
UPPS-P Total Score	42.17	8.38
IHS Total Score	19.45	9.03
Stop Signal Task at T1		
SSRT (ms)	208.74	74.90
SSD (ms)	389.30	196.62
Hit (%)	97.46	2.89
Miss (%)	1.65	2.85

Participant Attrition and Data Loss

Participants' data were removed from the analysis either due to non-completion of response inhibition tasks or to ensure the validity of the SSRT data from an original pool of 311 participants. A total of 51 participants were removed due to non-completion of the behavioral task (n=260). A total of 51 participants were removed due to not meeting the criteria required for valid data: (1) *p* values <.05 (outside of acceptable range for ratio of successful and failed stop trials; n=51), (2) SSRTs with negative values (excessively large SSD indicating waiting for stop signal; n=2). Within the stop signal task, the *p* value is a measure of statistically significant variance from a 50/50 balance of successful and unsuccessful stop trials. It is important that this ratio remains as close to 50/50 as possible to ensure the validity of the SSRT calculation based on the adjustable tracking algorithm. Due to ongoing data collection within the original study dataset, 78 participants have not reached a 5 month follow up assessment (T2). The final sample included in the analysis was n=207 for T1, and n=129 for T2.

Correlations among Study Variables

We examined the zero-order correlations among all study variables (See Table 3). Overall, IPV variables were not significantly associated with other clinical variables, or showed only small-sized correlation coefficients. Psychological IPV at T1 showed a significant correlation with PTSD severity scores (i.e., PCL-5), emotional dysregulation (i.e., DERS), and internalized homophobia (IHS), but the magnitude of correlations indicated a small effect size (all *r*s < .26). Physical IPV at T2 showed a significant correlation with PTSD severity with a small effect size ($r = .15$) and sexual IPV at T2 showed a significant correlation with PTSD severity ($r = .16$) and internalized homophobia ($r = .21$) with a small effect size. Psychological IPV at T2 showed a significant correlation with PTSD severity ($r = .24$) and emotional

dysregulation (r = .14) with a small effect size. Finally, psychological IPV at T1 showed a significant correlation with psychological IPV at T2 (r = .68) with a large effect size. However, T1 and T2 physical IPV frequency, as well as T1 and T2 sexual IPV frequency, did not show significant correlations between the two time points.

The SSRT index was not significantly correlated with any of the IPV frequency indices at T1 or T2. Notably, trait impulsivity (i.e., UPPS-P) did not show a significant correlation with any of the IPV subtypes. See Table 3 for the results of the full bivariate correlation test.

Hierarchical Regression on the T1 IPV Frequency Indices (Study Aim 1).

A series of hierarchical regression analyses were conducted to examine the hypothesized relationship between SSRT (=IV) and physical, sexual, and psychological IPV frequencies (=DVs), while controlling for relevant covariates. [2,3]

Table 4
Prediction of Physical IPV at T1

Step	Predictors	Predicting Physical IPV			
		ΔR^2	F	β	t
1	AUDIT			0.12	1.72
	PCL-5			-0.06	-0.72
	DERS			0.01	0.09
	UPPS-P			0.15	2.17*
	IHS			-0.01	-0.07
	Step 1 Model	0.02	1.17		
2	SSRT			0.05	0.8
	Step 2 Model	0.00	0.64		

*$p < 0.05$

Table 3

Correlations between the main study variables and IPV subtypes during T1 and T2

	Physical IPV T1	Sexual IPV T1	Psychological IPV T1	Physical IPV T2	Sexual IPV T2	Psychological IPV T2	AUDIT	PCL-5	DERS	UPPS-P	IHS
Sexual IPV T1	.19**										
Psychol IPV T1	.07	.28**									
Physical IPV T2	.07	.28**	.07								
Sexual IPV T2	.08	0.06	.30**	.23**							
Psychol. IPV T2	.00	.25**	.68**	.65**	.22**						
AUDIT	.04	-.04	.03	-.01	.11	.01					
PCL-5	-.01	.06	.25**	.15*	.16*	.24**	.24**				
DERS	.02	.11	.18**	.07	.11	.14*	.05	.66**			
UPPS-P	.09	.05	.10	.02	.07	.07	-.27**	.19**	.26**		
IHS	.02	.06	.22**	.04	.21**	.11	.13*	.10	.07	.03	
SSRT	.05	.03	.00	-.01	-.04	-.01	.04	-.06	-.06	-.06	.03

Note. **. Correlation is significant at the .01 level (2-tailed); *. Correlation is significant at the .05 level (2-tailed)

Within Table 3 are the correlations between the study intimate partner violence subtypes at both time points and our main covariates and predictor, The measures of IPV are taken from baseline (T1) and the follow-up period (T2). This includes physical (physic), sexual, and psychological (psychol) IPV. This table also includes each covariate which will be entered into our hierarchical regression model as controls to examine whether our main measure of response inhibition, stop signal reaction time (SSRT), will uniquely contribute to the variance of IPV frequency. AUDIT will account for alcohol use, PCL-5 will account for PTSD severity, DERS will account for emotional dysregulation, UPPS-P will account for trait impulsivity, and IHS will account for internalized homophobia.

Predicting Physical IPV Frequencies at T1. The five covariates were entered in Step 1 of the model followed by SSRT in Step 2. In Step 1, the AUDIT, PCL-5, DERS, UPPS-P, and HIS accounted for 2.3% of variance in Physical IPV perpetration (R^2 = .02, F= 1.17, p = .32). In Step 2, SSRT explained an additional 0.2% of the variance in physical IPV perpetration (R^2 = .002, F= 0.64, p = .42). Although the UPPS-P was the only significant predictor of physical IPV perpetration (β= .15, t = 2.17, p < .05) in Step 1, the overall hierarchical regression model did not significantly explain the variance in physical IPV.

Table 5
Prediction of Sexual IPV at T1

Step	Predictors	Predicting Sexual IPV			
		ΔR^2	F	β	t
1	AUDIT			-0.05	-0.75
	PCL-5			-0.01	-0.14
	DERS			0.14	1.59
	UPPS-P			0.01	0.11
	IHS			0.06	0.89
	Step 1 Model	0.02	1.16		
2	SSRT			0.04	0.59
	Step 2 Model	0.00	0.35		

Predicting Sexual IPV Frequencies at T1. In predicting sexual IPV frequencies, the five covariates were entered in Step 1 with SSRT in Step 2. In Step 1, the AUDIT, PCL-5, DERS, UPPS-P, and HIS accounted for 2.2% of variance in sexual IPV perpetration (R^2 = .02, F= 1.16, p = .33). In Step 2, SSRT explained an additional 0.2% of the variance in sexual IPV perpetration (R^2 = .002, F= 0.64, p = .56). None of the predictors emerged as a significant predictor of sexual IPV perpetration.

Table 6
Prediction of Psychological IPV at T1

Step	Predictors	Predicting Psychological IPV			
		ΔR^2	*F*	*β*	*t*
1	AUDIT			-0.01	-0.13
	PCL-5			0.26	3.14**
	DERS			0.04	0.49
	UPPS-P			0.06	0.91
	IHS			0.17	2.80**
	Step 1 Model	0.13	7.35***		
2	SSRT			0.01	0.23
	Step 2 Model	0.00	0.05		

*$p < 0.05$
**$p < 0.01$

Predicting psychological IPV frequencies at T1. In predicting psychological IPV frequencies, the AUDIT, PCL-5, DERS, UPPS-P, and HIS accounted for 12.6% of variance in psychological IPV perpetration ($R^2 = .13$, $F = 7.35$, $p < .001$) in Step 1. In Step 2, SSRT explained a negligible amount of the variance in psychological IPV perpetration ($R^2 = .0002$, $F = 0.05$, $p = .82$). Among the predictors, PCL-5 ($\beta = .26$, $t = 3.14$, $p < .01$) and IHS ($\beta = .17$, $t = 2.80$, $p < .01$) emerged as the only significant predictors of physical IPV perpetration.

In sum, SSRT was not shown to predict baseline IPV frequencies at T1. However, covariates PTSD symptom severity and internalized homophobia predicted psychological IPV at T1.

Predicting Physical IPV Frequency at T2. Physical IPV perpetration at T1 was entered in Step 1 of the model followed by the five covariates in Step 2, and SSRT in Step 3. In Step 1, physical IPV perpetration at T1 accounted for a negligible amount of the variance in physical IPV perpetration at T2 ($R^2 = .00$, $F = .05$, $p = .82$). In Step 2, the AUDIT, PCL-5, DERS, UPPS-P, and HIS at T1 accounted for 6.3% of variance in physical IPV perpetration at T2 ($R^2 = .06$, $F = 2.20$, $p = .06$). In Step 3, SSRT explained a negligible amount of the variance in physical IPV

perpetration at T2 (R^2 = .00, F= 0.00, p = .98). Among the predictors, the PCL-5 (β= .31, t = 2.99, p < .01) emerged as the only significant predictors, but the overall regression model explained only 6% of the variance in physic al IPV at T2.

Table 8
Prediction of Sexual IPV at T2

Step	Predictors	Predicting Sexual IPV			
		ΔR^2	F	β	t
1	Sexual IPV T1			0.1	0.7
	Step 1 Model	0.00	0.43		
2	AUDIT			0.18	2.02*
	PCL-5			0.12	1.17
	DERS			-0.02	-0.22
	UPPS-P			0.16	1.83
	IHS			0.20	2.65**
	Step 2 Model	0.10	3.72**		
3	SSRT			-0.04	-0.57
	Step 3 Model	0.00	0.33		

*p < 0.05
**p < 0.01

Predicting Sexual IPV Frequency at T2. In Step 1, sexual IPV perpetration at T1 accounted for a negligible amount of the variance in physical IPV perpetration (R^2 = .00, F= .43, p = .82). In Step 2, the AUDIT, PCL-5, DERS, UPPS-P, and HIS accounted for 10.1% of variance in physical IPV perpetration (R^2 = .10, F= 3.72, p < .01). In Step 3, SSRT explained a negligible amount of the variance in sexual IPV perpetration (R^2 = .00, F= 0.33, p = .57). Among the predictors, the AUDIT (β= .18, t = 2.02, p < .05) and IHS (β= .20, t = 2.65, p < .01) emerged as the only significant predictors of sexual IPV perpetration at T2.

Table 9
Prediction of Psychological IPV at T2

Step	Predictors	Predicting Psychological IPV			
		ΔR^2	F	β	t
1	Psychological IPV T1			0.71	13.15***
	Step 1 Model	0.50	172.82***		
2	AUDIT			0.02	0.36
	PCL-5			0.07	0.88
	DERS			-0.11	-1.49
	UPPS-P			0.01	0.17
	IHS			-0.03	-0.60
	Step 2 Model	0.01	0.54		
3	SSRT			0.00	0.05
	Step 3 Model	0.00	0.00		

*$p < 0.05$
**$p < 0.01$
***$p < 0.001$

Predicting Psychological IPV Frequency at T2. In Step 1, psychological IPV perpetration at T1 accounted for 50% of the variance in psychological IPV perpetration at T2 (R^2 = .50, F= 172.82, p <.001). In Step 2, the AUDIT, PCL-5, DERS, UPPS-P, and HIS accounted for 1% of variance in psychological IPV perpetration (R^2 = .01, F= .54, p = .75). In Step 3, SSRT explained a negligible amount of the variance in psychological IPV perpetration (R^2 = .00, F= 0.00, p = .96). Among the predictors, the psychological IPV perpetration at T1 (β= .72, t = 13.15, p < .001) emerged as the only significant predictors of psychological IPV perpetration at T2.

In sum, SSRT at T1 was not shown to be predictive of IPV perpetration at T2. However, PTSD symptom severity predicted physical IPV perpetration and alcohol use predicted sexual IPV perpetration. Additionally, previous IPV perpetration at T1 was only predictive for future IPV at T2 for psychological IPV. Physical and sexual IPV at T1 were not predictive for subsequent IPV of the same type at T2.

Discussion

This study sought to explore the relationship between response inhibition and IPV perpetration frequencies while controlling for covariates of IPV. Our goal was to examine if response inhibition deficits predict IPV perpetration at baseline and at a 5-month follow up assessment. Response inhibition relates to the suppression of actions that are inappropriate within a certain context which interfere with goal-driven behavior. Deficits in response inhibition have been shown to be predictive of the severity of other psychiatric, emotional, and behavioral problems within several disorders including ADHD, OCRDs, substance use disorder, and eating disorders (Wodka et al., 2007; Berlin & Lee, 2018; Nigg et al., 2006; Lock et al., 2011). Within these disorders, response inhibition ability plays a crucial role in resisting maladaptive impulsive behavior which causes functional impairment. In the context of IPV, deficits in response inhibition may lead to impulsive perpetration of IPV as maladaptive conflict resolution strategy (e.g., Schafer & Fals-Stewart, 1997; Teichner et al., 2001, Horne et al., 2020). The overarching goal of the current study was to test the hypothesis that response inhibition deficits would be significantly associated with the perpetration of impulsive aggressive behavior inherent in IPV. However, contrary to our prediction, response inhibition deficits did not predict IPV perpetration for any IPV subtype either cross-sectionally or longitudinally.

Within our study we quantified the magnitude IPV perpetration using the well-established approach of measuring IPV perpetration frequency (e.g., Shorey et al., 2012; Portnoy et al., 2022). Hierarchical regression in combination with measures of IPV frequency is also a commonly used analytic approach within studies of IPV perpetration to understand the relationship between IPV and associated risk factors (e.g., Doumass et al., 2008; Mechanic et al., 2008). Although this method is commonly used, these null findings may be due to the way IPV

was assessed and quantified using retrospectively recalled frequency as the primary index of IPV perpetration in the current study. Within the data collected, the overall frequency of physical and sexual IPV perpetration was markedly low (i.e., on average less than 1 instance of IPV perpetrated) with only psychological IPV perpetration occurring at a considerable rate at T1. A previous study with primarily female LGB+ populations showed higher rates of physical (frequency = .83 instances/6 months) and sexual (frequency = .64 instances/6 months) IPV perpetration compared to those observed in the current study sample (Whitton et al., 2019). In contrast, Whitton et al., (2019) reported lower amounts of psychological IPV (frequency = 1.57 instances /6 months) perpetration compared to our study sample (Whitton et al., 2019). However, psychological IPV perpetration dropped from an average of 15 instances of IPV to 3 instances of IPV from T1 to T2 during a similar period considering the average length of participant relationships was 5 months. Furthermore, although O'Leary et al. (1999) demonstrated that an individual's past aggressive behaviors predict future aggressive behavior, the frequencies of physical and sexual IPV perpetration were not significantly correlated between T1 and T2. Only the frequency of psychological IPV perpetration was correlated between T1 and T2. Taken together, an issue that could have hindered the accuracy of IPV reporting is the retrospective nature of the data included in the current study.

Each IPV subtype at T1 asked participants to remember the amount of IPV they perpetrated within the last year. Retrospective reporting relies on memory of these events which could have diminished the reliability of reporting an accurate frequency of IPV perpetrations (e.g., Csikszentmihalyi & Larson, 1987; Sullivan et al., 2011). However, the current hypotheses may have been better tested using a daily diary approach which relies less on the recollection of past instances of IPV. Compared with a daily dairy approach which asks participants to report

certain events/behaviors on an ongoing basis over time, retrospective reporting may be more prone to recall errors (Robinson, 2014). Furthermore, especially for socially unacceptable behaviors, retrospective reporters tend to underreport their maladaptive behaviors in comparison to reporting using the daily diary approach (Leigh et al., 1998). Future research would benefit from using either a more sophisticated version of retrospective reporting (e.g., a modified timeline follow back) or creating a baseline of IPV perpetration using a daily diary approach. Using a daily diary approach for baseline assessment of IPV perpetration may also reduce baseline and follow up recall differences due to observational effects (i.e., Hawthorne effect) (Sedgwick and Greenwood, 2015).

Although this study did not demonstrate a relationship between IPV frequency and response inhibition deficits, we cannot exclude the possibility that response inhibition affects the *process* of IPV. Without the presence of other proxy variables such as frustration, anger, or a general lack of emotional regulation, the manifestation of a violent behavior may not be as affected by response inhibition deficits. In a study comparing violent and non-violent schizophrenia patients with inhibition deficits, Krakowski et al. (2016) showed through an emotionally valanced inhibition task (i.e., Go/No Go) that violent offenders committed more hastily responded commission errors compared to non-violent patients. However, for neutral stimuli, violent and non-violent patients had similar performance. This suggests that emotional components may offer a more sensitive and ecological valid context to detect response inhibition deficits which may underlie maladaptive behaviors. Future research into IPV perpetration should utilize an emotionally valanced stop signal task that could tap into difficulties with inhibiting responses in the presence of emotional stimuli relevant for IPV perpetration.

Similarly, it should also be noted that, trait impulsivity, a previously shown covariate of IPV perpetration (Shorey et al., 2011), did not predict the perpetration of IPV either. This suggests that general trait measures of impulsivity or context-independent/neutral response inhibition measure may not be a consistent predictor of IPV perpetration. Although general trait impulsivity did not predict frequency of IPV, more affectively dysregulated forms of trait impulsivity may be better predictors of IPV perpetration. Indeed, previous research has suggested that individuals who act rashly when experiencing extreme negative emotions have been shown to perpetrate more acts of aggression (Derefinko et al., 2008). These findings based on the Stop Signal task and self-reported impulsivity suggest that disinhibition/impulsivity may need to be assessed using a relevant emotional context when they are evaluated for their association with IPV.

Measures of IPV tend to survey a variety of behaviors and actions related to partner violence, but often do not measure the *nature* of IPV episodes. There are understudied components related to the process of IPV that may be affected by response inhibition deficits, including the length of IPV perpetration lasts, the intensity of the perpetration of violence, the perceived controllability of the IPV, and the way IPV is utilized as a maladaptive strategy. A recent study conducted by Nedegaard et al. (2019) showed that individuals who had lower levels of impulsivity rated the utility of mild IPV as a conflict resolution strategy higher than individuals with higher levels of impulsivity. This suggests that some individuals may perpetrate IPV as a purposeful conflict strategy to end disagreements, while more impulsive individuals may fail to control maladaptive behavior regardless of their intention to use violence as a means of resolving a conflict. Indeed, prior research has shown that violent acts can be less controlled affective/reactionary responses to conflict or as a preplanned predatory act to assert dominance

and power (Penagos-Corzo et al., 2019). It is possible that response inhibition deficits may cause more of an effect on the manner and intensity of IPV among reactionary individuals compared to those who use IPV as preplanned conflict strategy. Future research should further explore other aspects of IPV processes, including the dominant mode of IPV perpetration (i.e., reactionary vs. strategic/preplanned), the pattern of behavioral and emotional regulation within IPV episodes, and IPV severity as well as its frequency. Although current measures like the CADRI and the conflict tactics scale (Strauss, 1996) measure different types of IPV which vary in their severity, a new measurement scale could be devised which surveys the manner in which IPV is committed. This would allow a deeper and more nuanced understanding of the processes of IPV, and a more comprehensive evaluation of the role of response inhibition deficits in the perpetration of IPV. Current findings suggest that response inhibition deficits may not put an individual at risk for perpetrating IPV more frequently. Nevertheless, it is possible that the pattern and intensity of IPV episodes may be negatively affected by these deficits once they occur. Future research needs to examine how response inhibition affects the severity of intimate partner violence and the individual's ability to withhold maladaptive, shortsighted responses to interpersonal conflicts.

There is also a possibility that the minority population (i.e., sexual minority) the sample was derived from affected the current findings. Although we controlled for variables of minority stress by adding internalized sexual minority stress as a covariate, other factors of minority stress could have unique effects on the perpetration of IPV within this population. Previous research has shown that young LGB+ adults are at a higher risk for the perpetration and victimization of IPV. However, within this sample the rate of physical and sexual IPV were lower than expected with physical IPV perpetration occurring on average less than three times and sexual perpetration

occurring less than once during course of the participants relationship at either T1 and T2. Kaschak (2001) suggests that reporting IPV may be more troublesome for LGB+ individuals due to fears of re-stigmatizing a group which already experiences severe amounts of discrimination. Furthermore, this effect could also discourage LGB+ individuals from participating in studies regarding IPV which would inherently reduce rates of IPV within study samples. Additionally, McClennen (2005) also suggests that instances of violence may need to reach a higher severity before being considered IPV within same-sex relationships. Same-sex partners may view their partner as being equally capable of harm which may minimize perceptions of IPV severity. Although intuitively this may seem to only pertain to physical perpetrations of violence, this effect has been shown as well for psychological forms of violence (Finneran & Stephenson, 2014). Therefore, rates of IPV within this sample may have been artificially low due to psychological barriers to reporting and participation, and to higher perceived thresholds for IPV perpetration.

There are several limitations to this study. Overall, the level of IPV reported from our sample was low which made it difficult to examine the relationship between response inhibition, other IPV related covariates, and IPV perpetration. Additionally, the sample was derived from a minority population that could affect the generalizability of the results to non-minority populations and as previously mentioned could possibly have reduced the reported frequency of IPV due to population differences in reporting IPV. Future research should use language that addresses concerns around re-stigmatization during participant recruitment, and during the data collection process, and increase participant buy-in by communicating the importance of IPV research to the health and wellbeing of minority communities. Another limitation within this study was the use of a non-affective response inhibition task to measure the relationship between

response inhibition deficits and IPV perpetration. Future research should use affective (e.g., angry faces, invalidating/threatening words) stimuli within the stop signal task to further explore how negative emotions affect response inhibition to maladaptive behavior. However, careful considerations should be made to ensure IPV related stimuli are reasonably aversive to not re-traumatize victims of IPV. Additionally, although IPV related stimuli may mimic IPV related stop signals, the ecologically similar emotional state which could possibly prompt IPV may also affect an individual's ability to inhibit violence perpetration. Indeed, it may be prudent to examine SST performance while inducing an emotional state akin to the anger and frustration a partner may experience during a relationship conflict. Therefore, future experiments may also consider using an emotional context manipulation before a stop signal task which mimics the experience of relationship conflicts. Finally, the retrospective nature of the measures used to gauge IPV perpetration could affect the validity of reported IPV. As previously stated, each participant may have poorly estimated the amount of IPV performed within the last year due to lapse of memory or personal biases of their own maladaptive behavior. Therefore, the baseline data requiring individuals to report various time limited instances of IPV within the previous year could have affected the accurate recall of IPV. Further research is needed using a daily diary approach to examine IPV perpetration data prospectively collected over time and then compare its association with response inhibition tasks.

Although the hypothesized relationship between response inhibition and the perpetration of IPV was not demonstrated, our data provides a useful insight into how to further this line of investigation to better understand the pattern of IPV and the association between IPV and response inhibition deficits. The question remains of how response inhibition affects the manner in which IPV is committed at a within-episode level. Future research should consider how

response inhibition affects the way IPV is committed and the effect of proximal negatively valanced emotions in the context of response inhibition.

Footnote

[1] Tests to see if the data met the assumption of collinearity indicated that multicollinearity was not a concern. For each predictor within each regression the VIF < 2.4.

[2] We performed a dummy coded regression to test if sexual orientation was a significant predictor of IPV subtypes and therefore needed to be controlled. For aim 1 *p* values ranged from .17 to .51 with β values ranging from -6.25 to 4.62. For aim 2 *p* values ranged from .30 to .78 with β values ranging from -1.40 to .08.

[3] We added relationship length as a covariate within each regression analysis to test whether it was a significant contributor to the variance of IPV perpetration. For both aims, *p* values ranged from .4 to .9 and β values ranged from -.02 to .04. Thus, relationship length was not a significant predictor of physical, sexual, or psychological IPV perpetration at T1 or T2. The overall pattern of findings in the hierarchical regression models that predict IPV frequencies remain largely identical with or without the inclusion of relationship length as a covariate.

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