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Biological Correlates of Intimate Partner Violence Perpetration

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Abstract

An extensive literature documents biological correlates of general aggression, but there has been less focus on biological correlates of intimate partner violence (IPV). The purpose of this review is to summarize the research literature to date that has reported on biological factors in IPV perpetration. We review the existing literature on four domains of biological processes that have been examined with respect to IPV perpetration, including: head injury and neuropsychology; psychophysiology; neurochemistry, metabolism and endocrinology; and genetics. We critique the literature, discuss the clinical relevance of research findings, and provide some recommendations for future biologically-oriented IPV research.

Keywords

Intimate partner violence; biological factors; neuropsychology; psychophysiology; neurochemistry; genetics

1. Introduction

Efforts to understand the etiology of intimate partner violence (IPV) perpetration are critical given the scope of the IPV problem and its numerous negative impacts. According to a national survey, approximately 1.5 million women and 834,732 men are physically assaulted or raped by their intimate partner every year in the United States, with 41.5% of women injured during their most recent assault, compared to 19.9% of men (Tjaden & Thoennes, 2000). In 2005, data collected by the FBI revealed that 1181 females and 329 males were murdered by an intimate partner (Fox & Zawitz, 2007). Physical injuries, ranging from bruises to gastrointestinal disorders, are often accompanied by psychological problems including depression, post-traumatic stress disorder, low self-esteem, and harmful health behaviors such as substance abuse and risky sexual activity (Campbell et al., 2002; Coker et al., 2002; Plichta, 2004). Moreover, the annual cost of medical care, mental health services, and lost employment productivity due to IPV has been estimated at more than $8.3 billion

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(Max, Rice, Finkelstein, Bardwell, & Leadbetter, 2004; National Center for Injury Prevention and Control, 2003). IPV-related criminal justice service use is also significant, including police and investigative costs, prosecutors, courts, legal fees (public defenders), and incarcerations (Miller, Cohen, & Wiersema, 1996), though nationally representative cost estimates are not available (National Center for Injury Prevention and Control, 2003).

IPV perpetration has been studied from different perspectives. Bell and Naugle (2008) recently reviewed and critiqued the most established theoretical explanations of IPV perpetration. Feminist theories argue that socially-defined gender roles within patriarchal societies in combination with men's systematic efforts to control women through the use of power and domination are the main causes of IPV (Dobash & Dobash, 1979; Walker, 1984). Power theorists posit that violence stems from within the family; violence used to resolve family conflicts is learned in childhood by witnessing or experiencing abuse. Furthermore, psychosocial stressors (e.g. power imbalances between spouses) increase the risk of IPV (Straus, 1976a, 1977). Social learning theories hold that violence develops when individuals model parental behavior and fail to learn constructive conflict resolution methods (O'Leary, Van Hasselt, Morrison, Bellack, & Hersen, 1988). Personality theories emphasize the roles of insecure attachment styles, early childhood family experiences, impulsivity, and borderline, antisocial, and narcissistic traits in IPV perpetration (Dutton, 1995; Holtzworth-Munroe & Stuart, 1994). Notably absent are the potential contributions of physiological factors, despite the fact that they constitute one leg of the bio-psych-social trinity, so often invoked to explain human behavior.

The contribution of a biological perspective to IPV perpetration has received limited attention despite a well-characterized body of literature on the relationship between biological factors and general aggression (for reviews, see Miczek et al., 2007; Patrick, 2008; Raine, 2002; Siever, 2008). A recent meta-analytic review of IPV risk factors did not include any biological variables (Stith, Smith, Penn, Ward, & Tritt, 2004). The extant research in this area underscores the need to include biological factors in theorizing and model building regarding the causes of IPV.

Furthermore, interventions for abusers based on a psychological and social understanding of IPV perpetration have not generally been successful. A meta-analytic review on the efficacy of abusers' treatment on recidivism of IPV found a small effect size (Babcock, Green, & Robie, 2004). This quantitative summary of controlled studies utilizing victim reports or police records of physical violence post-treatment indicated that therapeutic interventions had a minimal impact on reducing recidivism beyond legal ones. Developing a biological understanding of perpetration may ultimately serve to bolster program effectiveness.

To our knowledge, the current paper is the first literature review focusing on the association between biological factors and the perpetration of IPV. We searched MEDLINE, PsycINFO, and PubMed, from inception of the databases through January 2008. For each literature search, we combined every intimate partner abuse term, such as *intimate partner abuse, intimate partner violence, relationship abuse, and relationship aggression*, with every subtopic search term. For head injury and neuropsychology, search terms included *neuropsychology, neuropsychological, cognition, head injury, and traumatic brain injury*. For psychophysiology, search terms included *reactivity, physiological reactivity, psychophysiology, and cardiovascular reactivity*. For neurochemistry, metabolism, endocrinology, we used the terms *serotonin, testosterone, and hypothalamic pituitary axis*. For genetics, we used the terms *genetics, gene, DNA, allele, heredity, inheritance, behavioral genetics, nature nurture, twin studies, lineage studies, genetic markers, polymorphisms, chromosomes, polygenic, multifactorial, DRD4 gene locus, serotonin transporter gene, markers, and linkage analysis*. We also searched the bibliographies of

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located articles for further references. Having identified ten empirical investigations relevant to head injury and neuropsychology, six empirical investigations that addressed psychophysiology, five empirical studies concerning neurochemistry, metabolism, and endocrinology, and one empirical study on genetics, evidence summarizing the biological processes that underlie IPV perpetration were divided into each of these four domains. A discussion of suggestions for future biologically-oriented research is provided. Given the lack of research on the biological underpinnings of female-perpetrated IPV, our emphasis is on male-perpetrated IPV.

2. Head Injury and Neuropsychological Factors for IPV

Rosenbaum and Hoge (1989) conducted the first empirical evaluation of head injury in men who engage in IPV. Study participants, all of whom had physically abused their female partner, were self-referred or court-mandated to an outpatient, hospital-based psychoeducational program for with problems of aggression in marital relationships. A concussion, as diagnosed by a physician, or a loss of consciousness (LOC), as reported by a participant, defined a significant head injury. It was found that 19 out of the 31 participants (61%) had positive histories of head injury. This rate was considerably higher than the authors' estimated 6% prevalence rate of head injury in the general population. Head injured participants had a mean age at injury of 16.1 ± 5.2 yrs, with LOC ranging from several minutes to several months. Predictors of head injury examined in this study included childhood abuse, general aggression, and alcohol and drug use. Only alcohol abuse was significantly associated with experiencing head injury. Since alcohol use is also significantly associated with IPV (perpetration and victimization) (Foran & O'Leary, 2008), it is possible that alcohol use functions as a third variable, increasing the probability of both head injury and IPV. However, it is also possible that alcohol abuse leads to IPV in part because alcohol abuse increases the likelihood of experiencing head injury. Future longitudinal research is needed to clarify the relationship between alcohol abuse, head injury, and IPV.

In a second study, Rosenbaum et al. (1994) compared the head injury rates of 53 partner-abusive men to those of two non-violent groups, 32 maritally discordant and 45 maritally satisfied men. Group membership was based on cut off scores on the Conflict Tactic Scales (CTS; Straus, 1979). The latter two groups were added to this study to serve as control groups, which had been absent in the first study. The researchers found that 53% of abuse perpetrators had a history of closed head injury compared to 25% and 16% of discordant and satisfied men, respectively, suggesting that marital discord could not account for the association between head injury and abuse. An overwhelming majority (93%) of head-injured abusers had endured their head injury prior to the first occurrence of marital abuse, with 74% of these men receiving the head injury before the age of 16. The three groups of participants did not differ significantly on severity of head injury, perhaps due to the fact that most of the head injured men (67%) reported mild injury, and the groups also did not differ on alcohol use or childhood abuse. When comparing abusers with non-abusers (discordant and satisfied combined; discordant alone), having had prior head injury, regardless of severity, appeared to increase the likelihood of being an abuser six-fold.

Marsh and Martinovich (2006) attempted to replicate and extend findings from Rosenbaum and colleagues by investigating the prevalence of head injury among abusers in addition to studying abusers' executive dysfunctions and general intellectual functioning. Of the 38 abusers, 22 (58%) reported at least one head injury, consistent with the rates reported by Rosenbaum and Hoge (1989) and Rosenbaum et al. (1994). To determine the rate of executive impairments associated with head injury, these researchers administered three measures of executive functioning: the Behavioral Assessment of the Dysexecutive Syndrome (BADS; Wilson, Alderman, & Burgess, 1996) and the Hayling and Brixton Tests

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(Burgess & Shallice, 1997). General intellectual functioning (i.e. current IQ) was assessed by the Vocabulary and Block Design subtests of the WAIS-R (Wechsler, 1981). Compared to the non-head-injured group (n = 16) matched on age, pre-morbid IQ and presence and severity of alcohol problems, the head-injured group scored significantly worse on the Hayling and Brixton Tests (Burgess & Shallice, 1997), and on current IQ (Vocabulary and Block Design subtests of the WAIS-R; Wechsler, 1981). Within the head-injured group, 18% (n = 4), 27% (n = 6), and 27% (n = 6) were impaired on the BADS, Hayling test, and Brixton test, respectively. Participants performed at different levels across these measures such that no individual was impaired on all three, suggesting a diversity of executive functioning.

Warnken, Rosenbaum, Fletcher, Hoge, & Adelman (1994) compared 33 head-injured men to 42 orthopedically-injured men on relationship aggression to determine whether or not head injury itself, relative to other forms of injury, predisposed individuals to aggression. Patients seeking medical treatment for head, or orthopedic, injury between 1985 and 1990 were eligible to participate, as long as they had no history of IPV nor any head injury prior to 1985, seizure disorder, hyperactivity, attention deficit disorder, secondary loss of consciousness or hypoxia. According to responses on the CTS (Straus, 1979) post-injury, no significant group differences on IPV were found, as reported by men and their female partners. However, head-injured men reported significantly more loss of temper and control, increased difficulty communicating verbally, and increased frequency of getting into trouble, arguing with others (including spouse), and yelling, compared to orthopedically injured men on a measure of pre- and post-injury changes in the relationship and in the men's behavior. Female partners of the head-injured men confirmed these results, and added that after the injury, these men had increased their frequency of fighting and smashing things, and verbal abuse as reported on the measure of pre- and post-injury changes in the relationship and in the men's behavior. Head-injured men and their partners also reported significantly more negative changes in their relationship since the injury. While not corroborated by partners, head-injured men reported more frequent post-injury alcohol use, compared to controls. Although the results of the study did not support previous work suggesting that head injury is associated with an increased likelihood of IPV, head-injured men appeared to experience more behavioral changes that could negatively impact their intimate relationships. To help explain these disparate results, the authors speculated that in addition to a self-selection bias, the study may have suffered from a short time interval between injury and assessment. Had there been more than a three to five year follow-up since injury, more aggression may have occurred given the aforementioned negative relational and behavioral changes.

Cohen, Rosenbaum, Kane, Warnken, & Benjamin (1999) assessed neuropsychological functioning in 39 partner-abusive men and 63 non-violent men (of whom 26 were maritally discordant and 37 were maritally satisfied). Abusers were recruited from the psychoeducational outpatient treatment program used in the studies of Rosenbaum and colleagues (1989, 1994). Of the total 102 participants, 31 were classified as head-injured (HI; mild, moderate, and severe) and 71 as non-head-injured (NHI; subclinical, no head injury), based on LOC duration as defined by Rosenbaum and colleagues (1989, 1994). When comparing abusers to non-abusers, these researchers found that abusers were significantly less educated and more emotionally distressed, reported a higher incidence of prior head injury (46% vs. 19%), and performed worse on a measure of verbal intellectual ability (Shipley Institute of Living Scale; Shipley, 1946). Current alcohol or drug use did not statistically differentiate abusers from non-abusers; however, a majority of abusers, compared to controls, reported that they had become aggressive while consuming alcohol. Abusers also scored lower on tests assessing the domains of executive functioning (Wisconsin Card Sorting Test (WCST); Heaton, 1981), attention (Digit Symbol subset of the

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WAIS-R; Wechsler, 1981), and learning and memory (Nonverbal Selective Reminding Test for Long-Term Storage and Retrieval (NVSRT), Kane & Perrine, 1988; Warrington Recognition Memory Test for Faces and Words (RMT), Warrington, 1984). Particularly large performance differences on RMT-Words, WCST-Total Errors, and Digit Symbol suggest that frontal-temporal brain dysfunction may play a role in IPV. When comparing head-injured with non-head-injured participants, regardless of abuser status, group differences only reached significance for long-term recall on the NVSRT (Kane & Perrine, 1988). Thus, while prior head injury was correlated with IPV, head injury alone did not fully explain abusers' neuropsychological deficits.

Having found that abusers perform poorly on measures of executive functioning, attention, learning and memory, and verbal ability, Cohen and colleagues (2003) attempted to further characterize specific types of executive impairment, including impulsivity. Abusers were recruited from the outpatient treatment program of Cohen et al. (1999) and were split into abuser (n = 41) and control (n = 20) groups based on the CTS (Straus, 1979). The groups were matched on age, education, and socioeconomic status, and did not differ significantly on current alcohol or drug use even though a majority of abusers reported committing violent acts while intoxicated. However, childhood behavioral problems were significantly associated with being an abuser. Findings indicated that forty percent of the abusers reported prior head injury compared to 10% of the controls. Abusers performed significantly worse than controls on the Vocabulary and Comprehension subtests of the WAIS-R (Wechsler, 1981), supporting previously found lower scores on the Shipley Institute of Living Scale (Cohen et al., 1999). With respect to measures of attention and executive functioning, two domains whose deficits have been hypothesized to underlie impulsivity, significant group differences were found on the Paced Auditory Serial Addition Test (PASAT; Gronwall, 1977), the TMT Part B (Reitan, 1958), and the Stroop (Golden, 1978). With respect to measures particularly sensitive to impulsivity, abusers made significantly more false positive errors on the Adaptive Rate Continuous Performance Test (ARCPT; Cohen, 1993a), reflecting difficulty inhibiting responses to non-target stimuli. Abusers also had significantly underestimated time on the Time Estimation test of the Walter Reed Performance Assessment Battery (Thorne, Genser, Sing, & Hegge, 1985), and showed greater variability of inter-response interval on the Motor Continuation test of the same battery. Such problems with inhibiting and delaying responses suggest that abusers have difficulty with impulsivity which, in combination with mild executive impairment, implies mild frontal lobe dysfunction. Furthermore, the verbal deficits and impulsive tendencies of these abusers suggest that poor verbal skills may limit effective problem solving, leaving abusers more likely to impulsively respond to conflict with aggressive behavior.

A number of studies looked at the relationship between neuropsychological functioning and IPV irrespective of any history of head injury. Schafer and Fals-Stewart (1997) studied the relationship between neuropsychological functioning and IPV in recovering substance abusers. Thirty-one married men recovering from substance abuse for at least three weeks, with no history of neurological disease or head injury, were recruited by advertisement. IPV was assessed by responses to the CTS (Straus, 1979). The majority of couples (81%) reported a minimum of one violent occurrence during the year preceding the study, i.e. the time in which study participants abused substances. This rate was approximately five times the prevalence rate of IPV in the general population (Straus & Gelles, 1990). Partial correlations revealed that poor performances on measures of executive functioning (Booklet Category Test, De Filippis, McCampbell, & Rogers, 1979; Trail Making Test Part B, Reitan, 1958; Stroop Color Word Test, Stroop, 1935) and verbal ability (Vocabulary subtest of the WAIS-R; Wechsler, 1981) were associated with moderate and severe violence.

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Donovan Westby and Ferraro (1999) hypothesized that abusers free of neurological illness, including head injury, would perform relatively poorly on neuropsychological measures related to functional frontal lobe deficits. Abusers were recruited from IPV treatment programs, while controls were recruited from advertisements and abuse data gathered via arrest records and telephone interviews. None of the participants, 38 abusers and 38 men matched on age and education, reported a history of neurologic dysfunction. Abusers scored significantly lower on the vocabulary subset of WAIS-R (Wechsler, 1981) and significantly higher on a measure assessing the presence and severity of alcohol problems (Short Michigan Alcoholism Screening Test (SMAST); Selzer, Vinokur, & van Rooijen, 1975). Controlling for lower vocabulary scores and higher SMAST scores, abusers also took significantly longer to complete Trail Making B of the Trail Making Test (TMT; Reitan, 1958). Because Trails B measures ability to regulate competing response sets, abusers' longer completion times suggest they have difficulties inhibiting competing responses, thus taking longer to finish the task. No significant group differences were found on other neuropsychological measures, including the WCST and the Stroop Color Word Test (Stroop, 1935).

Teichner and colleagues (2001) compared the neuropsychological functioning of 50 abusers from a court-ordered outpatient treatment program with that of 23 non-patient controls. Participants were administered a 30-minute neuropsychological battery that measured general neuropsychological functioning (Screening Test for the Luria-Nebraska Neuropsychological Battery II; Golden, Purisch, & Hammeke, 1985), inhibition of verbal responses (Stroop Color Word Test; Golden, 1978), immediate and delayed non-verbal memory (Luria-Nebraska Neuropsychological Battery II; Golden et al., 1985), attention, motor speed and coordination, visual scanning (Trials A and B; Reitan, 1958), and cognitive flexibility (Trails B). Based on cutoff scores in the impaired range on two or more measures of the battery, participants were categorized as cognitively impaired and cognitively non-impaired. Of the abusers, 24 scored in the cognitively impaired range and 26 scored in the cognitively non-impaired range. The 48% rate of cognitive impairment among the abusers was significantly higher than that of the controls (4%).

Stanford, Conklin, Helfritz, & Kockler (2007) examined event-related potentials (ERPs), specifically the P3 component, in 18 abusers and 18 non-violent controls. Attendees to a court mandated anger management clinic who had a history of arrest and conviction for IPV comprised the abuser group. One-third of the abusers met diagnostic criteria for alcohol abuse. High P3 amplitude has been shown to reflect the efficient use of the brain's attentional capacity, as required for goal-directed behavior, or more generally, for behavioral control. Based on previous research showing low P3 amplitude in impulsively aggressive individuals, it was hypothesized that the abusers would show significantly lower P3 amplitude compared to controls, in addition to showing executive functioning problems. P3 amplitude and latency to auditory stimuli were measured at midline scalp electrode sites. As predicted,the abuser group showed significantly lower P3 amplitude compared to controls. On measures of executive functioning, the abuser group showed significantly more errors on Trail Making Test Part B (Reitan, Wolfson, Wedding, Horton, & Webster, 1986) and more failures to maintain set on WCST (Heaton, Chelune, Talley, Kay, & Curtiss, 1993), indicative of problems with attention associated with disinhibition. Overall, findings suggest that abusers may have an underlying attentional deficit that results in behavioral control problems.

2.1. Summary

The last two decades of research on the relationship between head injury and/or neuropsychological functioning and the perpetration of IPV have provided a limited, yet fairly consistent set of results. Rates of head injuries among abusers, ranging from 40% to

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61%, have been significantly higher than those found in the general population. Such high rates have been maintained regardless of how the head injury was determined, that is, by either a physician diagnosing a concussion or a study participant reporting a loss of consciousness. However, the alternative scenario, whether or not head-injured men are more likely to abuse their partners, has yielded inconclusive results, due mostly to methodological problems (e.g., self-selection bias, short time interval between head injury and assessment, variations in average age of sample). Additionally, the head injured population exhibits substantial diversity of injuries both in terms of severity and site of the injury, and we would expect a diversity of cognitive and behavioral sequelae. Neuropsychological studies have consistently found that abusers demonstrate poor performance on measures of verbal intellectual ability, executive functioning, and attention. Furthermore, some abusers with executive impairment have been shown to have difficulties with impulsivity, including response inhibition and time (under)estimation. Some studies have also reported abusers' problems with learning and memory. Finally, the role of alcohol use in the association between head injury and/or neuropsychological functioning and IPV is unclear. Findings that head injured abusers do not significantly differ from head injured non-abusers on alcohol use, and that abusers exhibit executive functioning and general intelligence deficits beyond the effects of alcohol use, suggest that alcohol problems alone do not account for abuse perpetration in these populations. However, very little is known about how alcohol use may interact with head injury and/or neuropsychological dysfunctioning in leading to abuse perpetration.

3. Psychophysiological Factors for IPV

Physiological reactivity refers to any changes in the body brought about by a stimulus. Examples include changes in heart rate, blood pressure, or cholesterol in response to a stressor. Given evidence that hostile and antisocial individuals are more physiologically reactive than non-hostile individuals and non-antisocial individuals, early studies on physiological reactivity among IPV perpetrators sought to determine whether one or more of these patterns also held for this population. In one of the first investigations to explore the relationship between physiological reactivity and IPV perpetration, Jacobson et al. (1994) conducted a laboratory based study examining the interpersonal behavior and affect of heterosexual couples (in which the male partner had been physically abusive) during conflictual discussions. Among their hypotheses was the expectation that partner abusive husbands would display greater physiological reactivity in response to conflict than non-abusive husbands would. They recruited couples with an abusive male partner, based on partner reports on the CTS (Straus, 1979). Among the couples experiencing IPV, only abusers who engaged in severe violence, ranging from pushing and grabbing six or more times in the past year to using a gun or knife at least once in the past year, were included in the study. Experimenters asked participating couples to engage in a discussion of two topics about which they typically disagreed. During the discussion experimenters measured cardiac interbeat interval (i.e., heart rate), pulse transmission time to the finger, and finger pulse amplitude. Husbands of violent couples and husbands of non-violent but distressed couples did not differ in physiological reactivity as Jacobson et al. (1994) predicted. However, wives from abusive couples exhibited greater finger pulse amplitude and faster finger pulse transit time (i.e., cardiovascular reactivity) than wives from non-violent couples. Jacobson et al. (1994) concluded that greater cardiovascular reactivity in wives may reflect feelings of helplessness, citing their finding that wives of abusive husbands exhibited more fear, anger, and sadness during the discussion.

In a separate study by these researchers, Gottman, Jacobson, Rushe, & Shortt (1995) hypothesized that there were two types of IPV perpetrators: those who respond to relationship conflict with physiological hyporeactivity, and those who respond with

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hyperreactivity. They tested their hypothesis by examining cardiovascular activity, skin conductance, and general somatic activity in 61 married, heterosexual male abuse perpetrators during a laboratory conflict discussion task with their spouses. Investigators divided abuse perpetrators into two groups based on whether their heart rate decreased (Type I), or increased (Type II), between baseline and marital conflict task periods. When compared to baseline, Type I men demonstrated more emotional aggression and became decreasingly aggressive over time during the conflict discussion. In other words, despite behaving aggressively toward their partners during the conflict discussion, Type I men appeared calm. Type II men displayed a pattern of escalating aggression across time. In their daily lives, there were no differences between Type I and Type II in terms of physical violence or psychologically abusive behavior toward their wives. However, Type I men did report more aggression toward others in their daily lives, scored more highly on measures of antisocial personality traits, drug dependence, and aggressive-sadistic personality traits, and obtained lower dependency trait scores. The authors concluded that there were two types of male abuse perpetrators: a cold, calculating, manipulative type who abuses to exert control or dominance (Type I), and a more reactive, emotional type who abuses out of fear of abandonment or jealousy (Type II).

Meehan, Holtzworth-Munroe, and Herron (2001) attempted to replicate the Gottman et al. (1995) findings using a similar methodology. In their sample, 33% of men (vs. Gottman et al.'s 20%) fell into the Type I category, and 67% of men (vs. Gottman et al.'s 80%) qualified as Type II. Similar to Gottman et al. (1995), Type I wives reported more marital distress. There was no difference in the frequency or severity of physical or psychological abuse between Type I and II men. In contrast to Gottman et al. (1995), there were no differences between groups during the conflict discussion in affect, or conflict discussion behavior (including aggression). Additionally, the groups did not differ on personality traits (including antisociality) or in levels of general violence. While Meehan et al. (2001) were able to replicate the Type I and II responder groups, they did not replicate the findings of group differences regarding affect, behavior or personality traits (e.g., antisocial traits).

To determine whether disrupted vagal regulation of the heart is a contributing factor in domestic abuse, Umhau et al. (2002) recruited thirteen male and three female individuals with a history of physically abusive behavior toward their spouses. Normally, rapid changes in heart rate are systematically regulated by vagal mechanisms and increases in heart rate correlate with decreases in cardiac vagal activity, as indexed by respiratory sinus arrhythmia (RSA). Vagal regulation is contrasted with slower acting sympathetic regulation, which increases cardiac arousal. Experimenters used information obtained during interviews with the abused partners to determine abuse type and severity. A group of healthy non-abusive men ($n = 13$) and women ($n = 2$) and a group of ten male and three female nonviolent alcohol abusers provided comparison data. The alcohol abuser group was included to rule out the possible dampening influence of chronic alcohol consumption on vagal reactivity. RSA, serum norepinephrine, heart period, and blood pressure provided measures of autonomic activity. The physiological reactivity task was a postural challenge that consisted of a supine 30-minute baseline period followed by five minutes of standing. All three groups, upon standing, showed significant increases in heart rate, norepinephrine and diastolic blood pressure, and significant decreases in cardiac vagal activity (RSA). However, while standing, evaluations of heart rate and RSA conducted every minute revealed that changes in RSA were significantly correlated to changes in heart rate in controls only. Unlike controls, the perpetrators' change in RSA or vagal activity did not predict changes in heart rate, suggesting that the complex neural regulation of heart rate is different in perpetrators than in controls. The authors concluded that IPV perpetrators' inconsistent vagal control of heart rate may be related to difficulties in autonomic regulation during relationship conflict, thus affecting emotion and aggression modulation. This is similar to

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Gottman et al.'s (1995) finding that all violent husbands' increased vagal activity during relationship conflict was not systematically correlated with decreased heart rate.

Babcock, Green, Webb, and Graham (2004) attempted a second replication of the Gottman et al. (1995) study. They compared physiological response differences during a conflict task in 30 non-violent men and 22 male IPV perpetrators in steady heterosexual relationships. The conflict task consisted of discussing an area of disagreement in the couples' relationship for fifteen minutes while experimenters obtained physiological measurements. They split groups according the Gottman et al. typology and explored group differences in affect and personality. Type I and II men did not differ on any personality measure or in their degree of aggression during the conflict discussion. In contrast to Meehan et al. (2001) and Gottman et al. (1995), low resting HR related to greater male-to-female physical and psychological aggression, more antisocial personality traits, and greater general violent behavior. They concluded that their results did not replicate Gottman et al.'s finding of personality, behavior, and affect differences between Type I and II men. They concluded that low resting heart rate is a better predictor of psychopathy and interpersonal violence than HR reactivity to interpersonal conflict.

In a separate study, Babcock, Green, Webb, and Yerington (2005) examined whether there were differences in autonomic reactivity between low-level violent (LLV) and severely violent (SV) male IPV perpetrators. LLV men were previously defined (Babcock, Green, Webb et al., 2004) as committing at least one act of violence in the past year that did not meet the Gottman et al. (1995) criteria of six or more minor acts (push, grab), two or more moderate acts (kick, bite, hit with fist), or one severe act (use a weapon). SV men were defined as committing one or more acts of violence on the CTS2 Severe Violence subscale (punched, slammed against a wall, kicked, used a knife or a gun, choked, beaten up, and burned or scalded; Straus, Hamby, Boney-McCoy, & Sugarman, 1996) in the past year. The authors expected antisociality and IPV to relate to physiological hyperreactivity in LLV men and hyporeactivity in SV men. One hundred two heterosexual couples participated. Heart rate (HR), skin conductance (SC), and affect in male IPV perpetrators was assessed during a marital conflict discussion and a relationship-themed anger induction task. SV men exhibited more anger during the two laboratory tasks, lower resting HR, and lower HR during the anger-induction task. Antisocial traits related to low resting HR among SV men. Across tasks, antisocial traits were negatively related to changes in HR and SC from baseline to conflict task (i.e., hyporeactivity) among SV men, while antisocial traits among LLV men related to increased HR and SC from baseline to conflict task (i.e., hyperreactivity). By exploring the variables specified by Gottman et al. (1995) in a broader sample of IPV perpetrators, i.e. men who are LLV or SV rather than SV or non-violent, the psychophysiological pattern predicted by Gottman et al. (1995) was replicated, but only for SV men. The authors concluded that physiological reactivity appears to be a better predictor of antisocial behavior than of IPV perpetration.

3.1. Summary

There have been multiple attempts to distinguish IPV perpetrators from nonviolent individuals and to create subgroups of IPV perpetrators based on physiological differences. However, to date, no consistent pattern of baseline physiological differences or physiological reactivity to laboratory tasks appears to distinguish these groups. These mixed findings may be the result of one or more of the following methodological limitations. First, physiological hyporeactivity in the summarized studies may have resulted from regression to the mean phenomenon and may not have reflected a true response characteristic. To account for regression to the mean, future studies would benefit from ensuring stable baseline arousal measurements, as some have argued (Ornduff, Kelsey, & O'Leary, 1995). Second, an average of physiological reactivity responses over time and across laboratory tasks

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provides the most reliable measure of trait autonomic reactivity (Hurwitz, Nelesen, Saab, & Nagel, 1993; Manuck et al., 1993; Rutledge, Linden, & Paul, 2001). All of the studies reviewed here administered reactivity tasks at one time point and most used only one type of reactivity task. Future studies would benefit from averaging reactivity data across two or more tasks and times. Finally, the research of Babcock et al. (2005) suggests that low level and severely violent IPV perpetrators may exhibit different patterns of physiological reactivity. Future research should attempt to replicate this finding and explore other ways in which these groups may also differ.

Beyond methodological limitations, however, that these studies suggest neural regulation of heart rate is different in some perpetrators than in controls may help explain why there are inconsistent results among these studies. Several studies have noted that changes in vagal activity do not correlate with the expected changes in heart rate. This was noted in a subset of perpetrators (Type I vs Type II) by Gottman et al. (1995) and in all perpetrators (perpetrators vs. normals) by Umhau et al. (2002). Under normal conditions when, for example, a person moves from a supine position to a standing one, a decrease in arterial blood pressure is sensed by pressure or stretch receptors (e.g. baroreceptors). The medullary vasomotor center then directs efferent fibers along myelinated fibers of the vagus nerve to decrease firing on the SA node of the heart, decreasing parasympathic outflow to the heart and thereby increasing heart rate to restore arterial blood pressure. However, in some perpetrators, a decrease in efferent vagal activity, or conversely, an increase in efferent vagal activity, is not followed by a corresponding increase or decrease in heart rate, respectively. Such dysreguated vagal control of heart rate may be reflective of a more generalized abnormality of autonomic regulation which in some perpetrators may be causing difficulties in regulating emotion and contributing to their violent behavior.

4. Neurochemical, Metabolic, and Endocrine Factors for IPV

Having found a high rate of head injury among abusers, Rosenbaum et al. (1997) investigated whether or not low serotonin activity, previously found to be associated with both head injury and increased aggression, played a role in IPV. Thirty-six abusers and 38 non-abusers were compared on their levels of serotonergic activity, as measured by their prolactin response, over six hours, to 60mg of fenfluramine (fenfluramine challenge) or placebo. Abusers were recruited from a hospital-based psychoeducational program for outpatients with problems of aggression in marital relationships. Head injury was assessed by a physician blind to participants' aggression history. At five hours post fenfluramine administration, abusers without head injury ($n = 23$) had significantly reduced prolactin levels compared to non-abusers without head injury ($n = 21$), suggesting that in the absence of head injury, abusers have decreased serotonin levels compared to controls. Although not a significant difference, head-injured non-abusers ($n = 17$) tended to have lower peak prolactin levels than non-head injured non-abusers ($n = 21$) over hours 4 to 6 post fenfluramine administration. This trend among non-abusers, that head injury decreases serotonin and possibly increases aggression, is consistent with previous research showing that head injury negatively impacts serotonergic functioning (van Woerkom, Teelken, & Minderhous, 1977).

Testosterone, a male steroid hormone of the androgen group, is found in both males and females, although females generally show much lower levels. It is responsible for, among other things, male secondary sex characteristics. There is substantial evidence that high testosterone levels are associated with a high probability of aggression (Kaiser & Powers, 2006; McKenry, Julian, & Gavazzi, 1995). The relationship, however, is complicated by the fact that high testosterone levels are associated with a host of potential moderating and mediating factors. For example, as McKenry et al. (1995) note, testosterone is consistently and moderately associated with dominance, alcohol and drug use, and sensation seeking.

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With regard to intimate relationships, Booth and Dabbs (1993) found testosterone levels to be positively related to both marital quality and marital aggression in an ex-military sample; and Julian and McKenry (1989) reported an inverse relationship between testosterone levels and marital satisfaction. Elevated testosterone levels have also been found to be associated with both verbal and physical aggression toward a female intimate partner (Soler, Vinayak, & Quadagno, 2000). Interestingly, there is some evidence that interpartner aggression by the male is related not only to his own elevated levels of testosterone but also to the testosterone level of his female partner (Cohan, Booth, & Granger, 2003).

Kaiser and Powers (2006) examined the relationship between testosterone levels and conflict tactics in a sample of late adolescent, heterosexual couples and found that the relationship between testosterone levels and male perpetrated dating aggression was moderated by the level of testosterone of the female partner: "…when paired with a high T[estosterone] partner, the higher the male's T, the more often he used physical assault, whereas when his partner was low in T, the reverse was true" (p.240). Specifically, the pairing of a male with a high testosterone level and a female with a low testosterone level predicted a reduced frequency in male perpetrated physical assault. Further, both physical and psychological aggression by the male were most destructive when the partners had similar levels of testosterone (either both high or both low). Complementary levels of testosterone between partners, such that one partner is high in testosterone and the other partner is low testosterone, were associated with less hostility and aggression. This relationship held for both male and female perpetrators, but was stronger for males.

There is an interesting connection between these findings and more popular social-psychological explanatory models. While testosterone levels are clearly associated with aggressive behavior in males, the evidence suggests that it is not a direct relationship and that the relationship is triggered primarily in the context of social dominance. In other words, males high in testosterone use aggression instrumentally to assert their dominance when they are challenged (Tremblay et al., 1998). According to Kaiser and Powers (2006), couples with complementary testosterone levels would have a more clearly defined power differential and less need for the male to assert his dominance through aggression. This is consistent with power and control models of IPV and suggests one possible biological substrate for that model.

George et al. (2001) compared cerebral spinal fluid (CSF) concentrations of serotonin (5-HT metabolite 5-HIAA) and testosterone from alcoholic abusers ($n = 13$), non-alcoholic abusers ($n = 10$) and healthy controls ($n = 20$). As a large percentage of IPV perpetrators abuse alcohol, alcoholic abusers were included to determine the effect of alcohol abuse on serotonin and testosterone. All abusers scored significantly higher on measures related to aggression, compared to controls. Non-alcoholic abusers had significantly lower serotonin and higher physical aggression, according to responses on the CTS (Straus, 1979), than alcoholic abusers and controls. Alcoholic abusers had significantly higher testosterone than non-alcoholic abusers and controls, and significantly higher physical aggression (CTS; Straus, 1979) than controls. Testosterone in chronic alcoholics typically increases to normal levels during abstinence, as required for 3 weeks prior to the study. However, because alcoholic abusers' testosterone level was higher than that of controls following the abstinence period, the authors concluded that alcohol abstinence was not a likely explanation for group differences in testosterone. Serotonin in chronic alcoholics typically decreases during abstinence; however, because alcoholic abusers' serotonin level approximately equaled controls' serotonin level following the abstinence period, the authors again concluded that alcohol abstinence could not explain such a similarity in serotonin.

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The cortical-hippocampal-amygdala pathway has been implicated in anxiety, depression and violence. This system comprises the biological substrate for fear-induced rage. Noting the similarities between the physiologic symptoms experienced by perpetrators preceeding an episode of IPV, as reported by Bitler, Linnoila, and George (1994), and the symptoms of panic anxiety (e.g., palpitations, increased respiratory rate, fear, feelings of being out of control), George et al. (2000) hypothesized that in some cases, episodes of IPV may be triggered by fear, and suggested that IPV perpetrators and individuals with panic disorder may both show an exaggerated fear response. Infusions of lactate have been used to experimentally induce panic attacks in a laboratory setting, but when these researchers gave such an infusion to IPV perpetrators, they induced attacks of fear and rage as well as panic. Non-violent comparison participants did not report such reactions. IPV perpetrators also reported greater feelings of unreality, fear, and loss of control at the time of the lactate infusions. Hypoglycemia has been linked to both panic disorder and violent behavior (Lindman, von der Pahlen, Ost, & Eriksson, 1992). Lindman et al. (1992) examined ethanol levels, serum testosterone concentrations, cortisol levels, and glucose in 16 men arrested for IPV offenses and compared them to a group of randomly selected males. Results indicated that offenders were significantly more intoxicated and that they did not differ from similarly intoxicated controls with respect to testosterone, glucose, and cortisol levels. However, sober state cortisol and glucose levels were significantly elevated as compared to a comparison group of non-alcoholic males. The authors suggested that these differences might be attributed to the higher stress levels of the IPV offenders.

George and colleagues (2004) used ^{18}F-2-fluoro-2-deoxyglucose (FDG) positron emission tomography (PET) to compare glucose uptake (activity) in cortical and subcortical brain regions hypothesized to modulate fear-induced aggressive behaviors in some abusers. IPV was assessed by responses to the CTS (Straus, 1979). Regional cerebral glucose metabolism was measured in abusers with alcoholism ($n = 8$), non-abusers with alcoholism ($n = 11$), and controls ($n = 10$). Only abusers who had normal brain MRI and no history of head trauma, bipolar disorder, and schizophrenia were included in the study. Alcoholic abusers had significantly lower glucose uptake in the right hypothalamus compared to alcoholic non-abusers and controls, suggesting an abnormality in hypothalamic activity of alcoholic abusers. Reduced correlations in glucose activity between various cortical structures and the amygdala were found in alcoholic abusers compared to the other groups. Several of these brain structures form circuits important for control and mediation of fear-induced aggression. Since alcoholic non-abusers did not did show decreased hypothalamic activity, the authors concluded that alcohol was not a likely explanation for group differences in activity.

4.1. Summary

Taken together, these diverse studies attempt to more precisely define the brain dysfunction that is associated with IPV, and each of these areas of investigation focuses on a different level of dysfunction. At the most basic level, neurochemical alterations in perpetrators, specifically excessive testosterone or reduced serotonin activity, reflect an alteration of neuronal function that can be simplistically thought of as promoting rapid responding to external stimuli. Hypoglycemia may have a similar effect on the brain by impairing cortical evaluation of stimuli and the nuanced responses that promote non-violent responding. At the morphological level, PET scanning shows that the connections between critical parts of the brain are disrupted in perpetrators. Such impaired neuronal connections may be related to alterations in neuro chemistry, or they may be the result of head injury. Head injury, even if it is relatively minor, can result in disruption of the long myelinated axons that normally connect one part of the brain to another. At the functional level, a grossly abnormal response to the panicogenic stimulant, sodium lactate, illustrates how brain dysfunction in

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perpetrators can manifest. While alcoholism is often associated with IPV, and may have important confounding effects on the brain, many of these studies controlled for this effect thereby demonstrating that alcohol consumption, by itself, cannot explain the abnormal brain function seen in perpetrators.

5. Genetic Factors for IPV

Previous research has shown a pattern of intergenerational transmission of IPV (Kalmuss, 1984; Kwong, Bartholomew, Henderson, & Trinke, 2003; Stith et al., 2000). Hines and Saudino (2004) conducted the first empirical evaluation of genetic and environmental contributions to the use (and receipt) of psychological and physical IPV. Among 175 pairs of same-sex adult twins, all of whom had experienced an intimate adult relationship, 134 were monozygotic (18 male, 116 female), sharing 100% of their genes, and 41 were dizygotic (5 male, 36 female), sharing 50% of their genes. Recruitment took place at twins' conventions. Correlational analyses within each class of twins indicated that monozygotes, unlike dizygotes, were significantly similar to one another in their frequency of physical and psychological IPV, as measured by the CTS2 (Straus et al., 1996). Because similarity between twins varies with the degree of genetic relatedness, these correlations suggest that genetic influences may be important to the use of aggression. Univariate model-fitting analyses revealed the models that best fit the data attributed familial resemblance of physical and psychological IPV to shared genes. The best fitting models included both genetic and unique environmental effects (but not shared environmental effects). Approximately 16% and 22% of the variance in physical and psychological aggression, respectively, were due to twins' shared genes. The remaining variance (78% – 84%) in both forms of aggression were due to twins' unique, non-shared environmental influences, such as interacting with different peer groups, receiving different parental treatment, and experiencing different illnesses, accidents, or traumatic events. These results provide preliminary evidence suggesting that variability in IPV is, in part, genetically influenced. However, the molecular genetic basis for IPV remains to be studied.

The candidate-gene approach is one method that can be used to study the genetic influences on complex traits such as IPV by (a) generating hypotheses about the role of specific genes in the etiology of the behavior, (b) identifying variants in or near those genes that might either cause a change in the protein or its expression, or be associated with functional changes, (c) genotyping the variants in the population of interest, and (d) using statistical methods to determine if there is a correlation between those variants and the phenotypes of interest. Candidate genes are selected where there is a priori evidence to hypothesize an association with the outcome of interest. The best candidates for study of behavioral phenotypes are functional polymorphisms or genetic variants that have already been shown to impact neurobiological pathways implicated in the outcome. While IPV is undoubtedly influenced by the small effects of many genes, a growing body of literature suggests links between MAOA gene polymorphisms and behaviors associated with IPV including aggression, impulsivity, and antisociality and this research provides a useful example for how researchers might integrate molecular genetics into future research and conceptualizations of IPV.

Monoamine oxidase A (MAOA) is a mitochondrial enzyme involved in the metabolism of serotonin, norepinephrine, and dopamine. Several human MAOA polymorphisms have been described including a functional VNTR polymorphism that modulates transcriptional activity of MAOA and ultimately enzyme activity (Sabol, Hu, & Hamer, 1998). Six variants containing 2, 3, 3.5, 4, 5, and 6 repeats of this sequence have been identified, with the 3- and 4-repeat alleles being the most common (Huang et al., 2004). These alleles have previously

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been dichotomized with the three-repeat variant as low-activity and longer alleles (3.5 repeats and longer) as high-activity.

Evidence from animal and human studies suggests that MAOA may play a role in the etiology of traits related to IPV, including impulsivity, addictive behavior, and aggression. MAOA gene knock-out mice have elevated brain levels of serotonin and enhanced aggression (Cases et al., 1995). Recent research on the social behavior of primates implicates the MAOA gene in primate aggression. Macaques are particularly well-suited to the study of the genetic basis of conflict behavior because they exhibit broad interspecies variation in patterns of aggression, dominance, and temperament (Thierry, Iwaniuk, & Pellis, 2000). Investigators have developed a four-grade classification scheme for describing conflict behavior across 16 species of Macaque on the basis of coding social behavior variables that resemble human behavior such as male-female dominance and aggressive and conciliatory acts. Grade 1 species are characterized by highly hierarchical societies characterized by intense episodes of conflict between animals and low levels of conciliatory behavior, while grade 4 species are comparatively tolerant, displaying relaxed dominance, open relationships, and high levels of conciliatory behavior. Using this taxonomy, Wendland and colleagues (2006) recently examined functional genetic variants underlying these inter-species differences in behavior and found that Macaque species exhibiting low confict (grade 4) displayed little or no intraspecies variation in the MAOA-LPR genotype. In contrast, grade 1 and 2 species were polymorphic at one or both of these loci suggesting a role for the MAOA-LPR gene in the etiology of conflict behavior in primates.

In humans, low MAOA activity has been associated with impulsive behavior and conduct disorder (Gabel, Stadler, Bjorn, Shindledecker, & Bowden, 1995; Lawson et al., 2003). Mild retardation and impulsive aggression (including arson and attempted rape) have been found among males of a large Dutch family with a chain termination mutation in the eighth exon of the MAOA gene (Brunner, Nelen, Breakefield, Ropers, & van Oost, 1993). The low-activity variant of the MAOA-VNTR has also been linked to antisocial behavior in alcohol-dependent males (Samochowiec et al., 1999), pathological gambling (Ibanez, Perez de Castro, Fernandez-Piqueras, Blanco, & Saiz-Ruiz, 2000), and impulsivity, hostility, and a lifetime history of aggression in a community sample of men (Manuck, Flory, Ferrell, Mann, & Muldoon, 2000).

Other research suggests that the influence of the MAOA-VNTR on antisocial behavior may be moderated by exposure to traumatic stressors. For example, Caspi et al. (2002) reported that although the MAOA genotype did not affect antisocial behavior in their sample as a whole, in subjects who had been maltreated in childhood, high MAOA activity had a protective effect against the development of antisocial behavior, while low MAOA activity was a significant risk factor for antisocial behavior. In Caspi et al.'s (2002) study, participants with the low activity genotype who experienced severe abuse were 50% more likely to have been convicted for a violent offense in adulthood than those with the high activity genotype. Similar evidence for a gene × environment interaction involving the MAOA genotype was reported by Huang et al. (2004) who found that the low-activity variant was associated with increased impulsivity only in a subgroup of male participants who reported childhood physical or sexual abuse. Likewise, Foley et al. (2004) showed that low MAOA activity increased risk for conduct disorder only in the presence of adverse childhood environment. Collectively, these findings suggest (a) a link between the MAOA gene and social conflict behavior, and (b) an association between this gene and antisocial behavior with the latter being most likely to be observed in individuals with histories of trauma and abuse.

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5.1. Summary

Results of Hines and Saudino's (2004) important twin study showed that while the majority of variance in IPV is explained by non-shared environmental influences (i.e., unique experiences that operate on one, but not both, twins), a significant proportion of variance in IPV is accounted for by heredity. These findings should raise concern about conceptualizing IPV exclusively as the outcome of social learning processes and they underscore the importance of addressing genetic factors in future research in this area. Though it is unlikely any genes would confer risk for IPV specifically, it is conceivable that genetic polymorphisms indirectly influence the development of maladaptive patterns of behavior, including IPV, through their influence on mediating factors such as personality and psychiatric symptomatology. Research on MAOA illustrates possible mechanisms by which the genes that regulate neurotransmitters and neuropeptides might give rise to problems in the domain of IPV.

6. Conclusions and Recommendations

Biological correlates of IPV perpetration can be categorized into the following domains: head injury and neuropsychology; psychophysiology; neurochemistry, metabolism, and endocrinology; and genetics. Head injury rates among abusers have been consistently higher than those of the general population. Abusers have been distinguished from non-abusers on the basis of their performance on several neuropsychological measures, particularly those of verbal intellectual ability, executive functioning, and attention. Abusers' poor performance on these measures reflects frontal lobe dysfunction, which head injury alone cannot fully explain. There is little to no evidence that physiological reactivity data reliably predicts IPV perpetration or severity. That antisociality, not IPV specifically, relates to hyperreactivity among low-level violent abuse perpetrators and hyporeactivity among more severely violent abuse perpetrators suggests that the relationships are not simplistic main effects. The decreased serotonin and increased testosterone levels found in some research with partner abusive males, in combination with their reduced hypothalamic activity and reduced correlations of activity between cortical and subcortical structures involved in control and mediation of fear-induced aggression, may predispose abusers to overreact, with violence, to actual and perceived threats (i.e. facial and verbal expressions of partners). Variance in physical and psychological IPV has been shown to be accounted for by both shared genes between twin siblings and non-shared environments, suggesting that there may also be a genetic component to IPV.

While research on the biological factors associated with IPV perpetration has produced findings to help explain the complex etiology of IPV, this literature suffers from a number of methodological limitations. Many of the studies relied on small samples and included inadequate comparison groups, limiting statistical power and the ability to draw definitive conclusions. Although most studies have used versions of the CTS to assess IPV, there is a lack of agreement on cutoff scores to determine IPV status. For example, some studies define abuse as one act of physical violence committed in the last year, while others look for more than 11 violent acts. Research would also benefit from a consistent method for classifying violence as mild, moderate, or severe. In addition, some studies sampled only frequently and/or severely violent abusers who may be qualitatively different from those who engage in mild violence. Further, consideration of both partners' reports of IPV may yield more accurate classifications, especially given the widely held belief that abusers' tend to underestimate or even deny the seriousness of their abusive behaviors. Furthermore, only two studies utilized both the Physical Assault and Psychological Aggression subscales of the CTS2 (Straus et al., 1996). It would be informative to determine whether or not certain biological factors play different roles in these two forms of IPV, particularly in light of research showing that psychological IPV can be at least as harmful to victims' physical and

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psychological health as physical IPV (Taft, Murphy, King, Dedeyn, & Musser, 2005). Finally, as researchers have relied on clinical samples, it will be necessary to determine if findings reported in this literature review generalize to non-clinical samples.

To date, research has been unable to reliably differentiate between nonviolent individuals and IPV perpetrators, or subgroups of IPV perpetrators, on measures of baseline physiological arousal and physiological reactivity. It is therefore not currently possible to use physiological data to distinguish IPV perpetrators and nonviolent individuals. Inconsistency between studies in finding differences in stress reactivity between IPV perpetrators and non-violent individuals may be due to the unreliable nature of stress reactivity data. It is not uncommon, for example, for a constellation of physiological reactivity scores to vary for the same individual on the same stress task at different points in time (e.g., Hurwitz et al., 1993; Manuck et al., 1993; Rutledge et al., 2001).

In addition, studies of physiological reactivity and IPV have primarily examined cardiovascular variables, such as changes in heart rate. However, other measures of autonomic activity, such as galvanic skin response (GSR), either do not map onto heart rate or do not occur immediately during a stressful situation (e.g., cortisol secretion) and may be better for distinguishing between IPV perpetrators and non-violent individuals. Further, rather than an absolute difference in response magnitude (i.e., differences in reactivity), it is possible that the relevant dimension for distinguishing abusers from non-violent individuals involves a cumulative effect of escalating stress or differences in returning to baseline physiological arousal. The literature would therefore benefit from research that compares the reactivity of IPV perpetrators and non-violent individuals at multiple points in time, by using multiple measures of reactivity, and by measuring not only reactivity to relationship discord, but also recovery from physiological reactivity.

Regarding neurochemistry, metabolism and endocrinology, George et al. (2001) showed that serotonin and testosterone concentrations differ between alcoholic and non-alcoholic partner abusers. The next step would be to explore the mechanisms underlying such differences. For example, it would be helpful to look at the interaction between alcohol use (frequency and quantity) and serotonin and IPV or the interaction between alcohol use and testosterone and IPV. Using similar methodology, Savarese, Suvak, King, & King (2001) found a complex interaction between the hyperarousal symptom of posttraumatic stress disorder (PTSD) and the drinking frequency and drinking quantity of Vietnam veterans in predicting IPV. Given that serotonin and testosterone may influence, among abusers, fear-associated behaviors and aggression, similar interactions may emerge. Furthermore, to rule out subtle structural brain abnormalities modulating aggression in participants with alcoholism, diffusion tensor imaging (DTI), a relatively new MRI technique that delineates axonal fiber tract direction and organization (Kubicki et al., 2007; Le Bihan et al., 2001; Mori & Zhang, 2006) could provide microscopic in vivo evidence of abnormalities in connectivity between different brain regions (Mori & Zhang, 2006), such as cortical structures and amygdala in alcoholic abusers. DTI could also be used in combination with functional MRI (fMRI) to investigate the connectivity of functional effects arising under threat arousal conditions.

It is also important to consider that serotonin itself is a very complex neurochemical and that in addition to absolute levels of serotonin, serotonergic functioning also depends on the number and nature of serotonin receptors. Further, the technology utilized in the studies cited did not directly measure serotonin levels, but rather levels of either metabolites of serotonin or blood levels of chemicals (e.g., prolactin) demonstrated to be correlated with levels of serotonin. While metabolite levels are generally accepted as a proxy for brain serotonin levels, and George et al. (2001) measured CSF concentrations of serotonin (5-HT

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metabolite 5-HIAA), it is important to remember that serotonin occurs in other body areas, such as the gut, and metabolite levels reflect both brain and non-brain serotonin activity.

To further determine, and perhaps more definitively distinguish, the genetic and environmental contributions to the familial resemblance of IPV, future studies would benefit from using larger samples of twins in which the number of pairs of monozygotic twins approximately equaled the number of pairs of dizygotic twins. A more heterogeneous sample including more non-White males would make findings more generalizable and allow for the comparison of potential gender differences underlying genetic risk for IPV. Furthermore, since non-shared environments among twins accounted for much of the variance in aggression in the Hines and Saudino (2004) study, a design that attempts to elucidate the characteristics of the unique environments contributing to aggression within twin members of the same family would be helpful. For example, determining whether one twin experiences a poorer maternal relationship than his/her twin sibling may explain more of the variance in aggression than does the twins' exposure to different peer groups. In this way, manipulating or modifying the unique environment, i.e. rebuilding the maternal relationship, could be helpful in decreasing aggression in someone that may already be genetically predisposed to violent behavior. It would also be useful to examine the mechanisms of genetic transmission, i.e., what is being transmitted. For example, it is possible that it is some heritable aspect of cognitive functioning, autonomic reactivity, or neurochemical imbalance that accounts, in some part, for IPV.

Despite a plethora of pharmacologic agents used in the treatment of aggressive behavior in adults with major mental disorders (for a review, see Volavka, Citrome, & Huertas, 2006), no medications have emerged with universal robust efficacy in treating violence (Goedhard et al., 2006). To date, there are only two published studies that have determined the efficacy of treating aggressive behavior in IPV perpetrators with a drug. Lee, Kavoussi, & Coccaroa (2008) administered 20–60 mg (daily by mouth) of the selective serotonin reuptake inhibitor (SSRI) fluoxetine or a placebo to abusers for 12 weeks. Abusers in the fluoxetine group (n = 13) and in the placebo group (n = 13) only differed from each other in that the fluoxetine group had a greater number of lifetime Axis I diagnoses. No significant differences in efficacy for fluoxetine vs. placebo were found in reducing overt aggression or subjective irritability, according to respective responses on the Aggression subscale and the Irritability subscale of the Overt Aggression Scale – Modified (OAS-M; Coccaro et al., 1989).

George et al. (2010) studied 66 alcoholics with at least two instances of IPV in the previous year in a 12-week, double-blind, randomized, placebo-controlled intervention study testing the efficacy of fluoxetine to decrease anger and irritability. All subjects received inpatient alcohol treatment and supportive cognitive-behavioral therapy (CBT). For perpetrators who completed the 12-week study (n = 24), fluoxetine significantly reduced irritability, as measured by the Irritability subscale of the OAS-M (Coccaro et al., 1989). In addition, all participants, regardless of condition, exhibited significant reductions in physical and non-physical abuse, as measured by the Partner Abuse Scale (Hudson, 1990). These results suggest that fluoxetine, in conjunction with CBT and alcohol treatment, may be useful in reducing anger and physical aggression in alcoholic perpetrators of IPV.

Less invasive than the use of drug therapies for aggressive individuals, the use of nutritional supplements to treat aggression offers promising results. A recent study of 221 Dutch prisoners treated with daily supplements of vitamins, minerals, and essential-fatty acids for up to three months found a significant reduction in total number of incidents of aggression and rule-breaking behavior, as reported by prison staff, compared to prisoners treated with placebos (Zaalberg, Nijman, Bulten, Stroosma, & van der Staak, 2010). While this study replicated results from an earlier one (Gesch, Hammond, Hampson, Eves, & Crowder, 2002)

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that found a 26% reduction of incident reports in supplemented prisoners compared to controls, more research is required in different offender groups, such as IPV abusers.

Each of the topic areas summarized in the current review shows promise for enlightening our understanding of intimate partner aggression. Inconsistencies and methodological weaknesses in the research literature also suggest ideas for future research. For example, DTI or PET imaging studies would improve the reliability and validity of self-reported head injuries, often subject to memory loss. Longitudinal studies in which assessments of IPV are made twelve months, two years, four years and eight years post - head injury may reveal differences in IPV among head-injured abusers and controls that are not immediately apparent. Furthermore, longitudinal studies will allow us to evaluate the process by which IPV develops in head injured individuals, perhaps as a result of the deterioration in the marital relationship. Considering the nature of the head injury and age of the patient, longitudinal differences in IPV would guide the appropriate timing of violence prevention efforts during head injury rehabilitation.

Consistent findings of executive functioning deficits are consistent with social information processing models of IPV (Holtzworth-Munroe, 1992), and suggest the potential salience of cognitive rehabilitation techniques shown to be effective in reducing the secondary symptoms of head injury (Rees, Marshall, Hartridge, Mackie, & Weiser, 2007) for abusers with head injury and executive functioning deficits. While neurological impairments have been tested largely by executive functioning measures, deficits in the domains of learning and memory should be further explored. Cohen et al. (1999, 2003) suggest that these deficits diminish abusers' ability to modify their behavior to novel situations or to develop new, more effective ways to resolve conflicts. They also recommend that studies examine validated reports of abusers' developmental histories including educational, emotional, and behavioral problems in order to discern causative versus correlated factors. An alternative method to standard neuropsychological testing, beyond that of Stanford et al. (2007), would have abusers undergo functional magnetic resonance imaging (fMRI) while performing different tasks. Functional imaging would help researchers relate dysfunctional cognitive processes to specific brain regions. Such imaging could help identify risk factors for IPV. For example, using fMRI, Raine et al. (2001) found that antisocial violent offenders (e.g. IPV abusers, rapists, attempted murders/murders) who had suffered severe child abuse had reduced right hemisphere functioning, particularly in the temporal cortex, during a working memory task.

It is also important to study the possible relationships between these various biological domains. Although head injury often leads to neuropsychological deficits, it is not the only cause of such deficits. There is evidence that head injury leads to serotonergic deficits, thus suggesting a possible connection between those two variables. It is also clear that head injury often has a detrimental effect on the marital relationship. Genetic factors may exercise their effect on IPV via neurochemical functioning, brain morphology, or even temperament. Several of these factors have also been implicated in alcohol use and abuse, which itself has been implicated as a major contributor to all forms of aggression, especially IPV (Foran & O'Leary, 2008). A theoretical model linking abnormalities in the neuropathways that mediate conditioned fear and fear avoidance to IPV perpetrators' behaviors and diagnoses has recently been proposed (George, Phillips, Doty, Umhau, & Rawlings, 2006). Specifically, the authors suggest that the changes in perpetrators' serotonin and testosterone metabolism contribute to their increased sensitivity to what they perceive as irritating or threatening stimuli. The ensuing response to this perceived threat is then unregulated due to the lack of cortical input to the amydala. Behaviorally, instantaneous conditioned fear responses (fight, flight, or freeze) and learned fear avoidance responses (alcohol and drug

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use) may result from these events. Future research is needed to develop and empirically evaluate more complex models of relationships among these biological factors of IPV.

This literature review suggests that biological variables in the domains of head injury, neuropsychology, psychophysiology, neurochemistry, metabolism and endocrinology, and genetics play an important role in the etiology of IPV. However, biological factors are only one piece of the complex interaction among psychological, social, and environmental influences that contribute to the perpetration of IPV. Thus, biological factors alone do not determine, nor justify, the occurrence of violence. However, as irresponsible as it would be to suggest that biological factors are primarily responsible for IPV, it would be equally imprudent to minimize their contribution to this ubiquitous form of aggression. Indeed, studies of IPV intervention strategies that use pharmacotherapy and/or nutritional supplementation to target these biological factors will be a fascinating area of future investigation. It is hoped that a consideration of empirically-established biological correlates of IPV may help inform etiological models of IPV and lead to effective prevention and intervention programs for IPV perpetrators.

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