

## Serotonin, testosterone and alcohol in the etiology of domestic violence

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### Abstract

In a previous study we administered the panicogenic agent sodium lactate to a select group of perpetrators of domestic violence and comparison groups. Results of that study showed that perpetrators exhibited exaggerated lactate-induced fear, panic and rage. In this current study, we compared the cerebral spinal fluid (CSF) concentrations of 5-hydroxyindoleacetic acid (5-HIAA) and testosterone obtained from perpetrators of domestic violence and a group of healthy comparison subjects. All subjects were assessed for DSM-III-R diagnoses. Perpetrators with alcohol dependence (DV-ALC) ( $n = 13$ ), perpetrators without alcohol dependence (DV-NALC) ( $n = 10$ ) and healthy comparison subjects (HCS) ( $n = 20$ ) were clinically assessed using the Spielberger Trait Anxiety, Brown–Goodwin Aggression Scale, Buss Durkee Hostility Inventory and Straus Conflict Tactics. Following an overnight fast and bed rest, subjects received a lumbar puncture to obtain CSF concentrations of 5-HIAA and testosterone. Perpetrators scored significantly higher on measures of aggression than HCS. DV-NALC had significantly lower concentrations of CSF 5-HIAA and higher Straus Conflict Tactics (CT) physical violence scores than DV-ALC and HCS. DV-ALC had significantly higher concentrations of CSF testosterone than DV-NALC. DV-ALC also had significantly higher Straus CT physical violence scores than HCS. DV-NALC and DV-ALC differed on 5-HIAA concentrations, testosterone concentrations, Straus CT physical violence scores and alcohol dependence. These results suggest that DV-NALC and DV-ALC groups could have different biological mechanisms mediating domestic violence. © 2001 Elsevier Science Ireland Ltd. All rights reserved.

**Keywords:** Aggression; Alcoholism; Rage; Cerebral spinal fluid (CSF); Fear conditioning

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## 1. Introduction

Two nationally conducted surveys in 1975 and 1985, involving a representative sample of approximately 6663 American families, found that 16% of the men and women had been physically assaulted by their spouse or significant other in the year prior to the survey (Straus and Gelles, 1986). Fifty percent of the perpetrators were either binge drinkers or consumed 3–5 drinks of alcohol per day. Perpetrators with the highest chronic alcohol consumption were the most likely to be violent (Kantor and Straus, 1990).

In a previous study investigating the link between fear and aggression in perpetrators of domestic violence, we administered the panicogenic agent sodium lactate to a select group of perpetrators and comparison groups (George et al., 2000). Results of the study showed that perpetrators exhibited exaggerated lactate-induced fear, panic and rage; some perpetrators reported that they experienced similar symptoms at the time of domestic violence. Perpetrators also showed a preponderance of DSM-III-R diagnoses of anxiety/phobic disorders. These results led us to examine the biological mechanisms, which could contribute to these exaggerated fear-related responses and ultimately to domestic violence.

One possibility is that perpetrators could have a disturbance in serotonin (5-HT) metabolism. Both human (Virkkunen et al., 1987, 1989a,b, 1994a; Roy et al., 1988; Limson et al., 1991) and animal studies (Higley et al., 1992; Mehlman et al., 1994; Doudet et al., 1995) have shown an inverse relationship between the cerebrospinal fluid (CSF) metabolite of 5-HT, 5-hydroxyindolacetic acid (5-HIAA), and aggression. Administration of pharmacological agents that reduce central 5-HT concentrations, either by depleting tryptophan (Chamberlain et al., 1987; Cleare and Bond, 1995; Moeller et al., 1996) or by blocking 5-HT synthesis (Katz and Thomas, 1976; Valzelli et al., 1981), typically cause an increase in aggression. Conversely, administration of agents that increase 5-HT, either by facilitating its release (Cherek and Lane, 1999) or by blocking its re-uptake (Fava et al., 1991; Salzman et al., 1995; Coccaro and Kavoussi, 1997), generally cause a decrease in aggression.

Studies in humans and animals show that 5-HT modulates (Morrison and Foote, 1986) the structures and neuropathways that mediate fear-related behaviors (Shaikh et al., 1987; LeDoux et al., 1990; Shaikh and Siegel, 1994; Morgan and LeDoux, 1995; Armony and LeDoux, 1997; Davis, 1997, 1998; LaBar et al., 1998; LeDoux, 1998; Hashimoto et al., 1999; Siegel et al., 1999). For example, 5-HT modulates the startle reflex (Davis et al., 1980) as well as sensory input to the thalamus (Marks et al., 1987) and striatum (Reisine et al., 1982) which serves to warn animals of environmental dangers. Serotonin in the medial prefrontal cortex modulates freezing behavior associated with a conditioned fear stimulus (Inoue et al., 1996; Hashimoto et al., 1999). Serotonergic input to amygdala, the medial hypothalamus and the periaqueductal grey (PAG) is involved with the control as well as the expression of fear-associated 'fight' or 'flight' behaviors (Spooon, 1992; Shaikh et al., 1997; Viana et al., 1997; Stutzman et al., 1998).

Another possible biological mechanism is that perpetrators could have a disturbance in testosterone metabolism. Evidence for testosterone's possible role in modulating fear-related responses and aggression is derived from both animal and human studies. For example, animal studies show that testosterone-dependent aggression occurs in non-human mammals (Albert et al., 1993; Higley et al., 1996). Evidence for testosterone's role in human physical aggression comes from studies showing that some subgroups of violent subjects have higher plasma (Ehrenkranz et al., 1974; Mattsson et al., 1980), saliva (Soler et al., 2000) and CSF testosterone (Virkkunen et al., 1994b) concentrations than non-violent controls. Also, it has been shown that adolescent males with high plasma testosterone concentrations are more irritable and more likely to respond aggressively to provocation and threats than subjects with lower testosterone concentrations (Olweus et al., 1980, 1988). Finally, subjects receiving testosterone are more likely to have an aggressive response to perceived threats than subjects receiving placebo (Pope and Katz, 1990; Su et al., 1993; Pope et al., 2000).

How testosterone could facilitate these fear-related responses and aggression is not known.

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