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Family Psychiatric History, Peritraumatic Reactivity, and Posttraumatic Stress Symptoms: A Prospective Study of Police

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Abstract

Background—Family history of psychiatric and substance use disorders has been associated with posttraumatic stress disorder (PTSD) in cross-sectional studies.

Method—Using a prospective design, we examined the relationships of family history of psychiatric and substance use disorders to posttraumatic stress symptoms in 278 healthy police recruits. During academy training, recruits were interviewed on family and personal psychopathology, prior cumulative civilian trauma exposure, and completed self-report questionnaires on nonspecific symptoms of distress and alcohol use. Twelve months after commencement of active duty, participants completed questionnaires on critical incident exposure over the previous year, peritraumatic distress to the worst critical incident during this time, and posttraumatic stress symptoms.

Results—A path model indicated: 1) family loading for mood and anxiety disorders had an indirect effect on posttraumatic stress symptoms at 12 months that was mediated through peritraumatic distress to the officer's self-identified worst critical incident; 2) family loading for substance use disorders also predicted posttraumatic stress symptoms at 12 months and this relationship was mediated through peritraumatic distress.

Conclusion—These findings support a model in which family histories of psychopathology and substance abuse are pre-existing vulnerability factors for experiencing greater peritraumatic distress to critical incident exposure which, in turn, increases the risk for development of symptoms of posttraumatic stress disorder. Replication in other first responders, military and civilians will be important to determine generalizability of these findings.

Keywords

Family History; Police Stress; Peritraumatic Distress; Posttraumatic Stress Disorder; Substance Use

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1. Introduction

Even after the most severe traumatic events, only a minority of those exposed develop PTSD while most develop only transient symptoms and recover quickly (e.g., Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Since individual differences in post-incident responses are not fully explained by the severity of trauma exposure (Kessler et al., 1995), characteristics that predate the trauma may be influential in determining reactivity to trauma and the eventual development of symptoms. Family history of psychopathology is a pre-existing variable that has been found to be associated with the presence of PTSD in cross-sectional studies. Family history predictors have included general psychiatric symptoms (Davidson, Hughes, Blazer, & George, 1991; Speed, Engdahl, Schwartz, & Eberly, 1989), PTSD (e.g., Yehuda, Halligan, & Bierer, 2001) and other anxiety disorders (e.g., Dierker & Merikangas, 2001; Reich, Lyons, & Cai, 1996), mood disorders (Davidson, Tupler, Wilson, & Connor, 1998), and addictions (Davidson et al., 1991; Dierker et al., 2001). Twin studies have demonstrated heritable risk for both traumatic exposure and for PTSD (Koenen et al., 2002; Stein, Jang, Taylor, Vernon, & Livesley, 2002; True et al., 1993) as well as shared heritability for PTSD and comorbid disorders, including generalized anxiety and panic (Chantarujikapong et al., 2001), major depression and dysthymia (Koenen et al., 2003), and alcohol and drug dependence (McLeod et al., 2001; Xian et al., 2000).

While a range of mechanisms of transmission have been considered (e.g., genetics, abuse, neglect, attachment disturbances, poverty), one that has been underemphasized is difficulty regulating emotional distress during traumatic exposure. Peritraumatic emotional distress is an intense response (e.g., fear, helplessness, horror, and anger) associated with physiological reactivity (e.g., racing heart, sweating, and shaking) at the time and in the immediate aftermath of traumatic exposure (Brunet et al., 2001) and shown to be an important risk factor for posttraumatic stress responses (Marmar et al., 2006; Ozer, Best, Lipsey, & Weiss, 2003). Perceived threat at the time of trauma exposure has been found to predict greater peritraumatic reactivity (Marmar et al., 1996; McCaslin et al., 2006), a process believed to involve excessive activation of the amygdala and other paralimbic structures and insufficient activation of emotion modulating centers in the prefrontal cortex, which collectively results in greater and more prolonged states of fear driven activation (e.g., Bremner, 2002; Pitman, Shin, & Rauch, 2001). This excessive activation is believed to enhance fear conditioning and memory consolidation, subsequently resulting in chronic hyperarousal symptoms of PTSD (Pitman, 2000).

Koenen (2006) postulated that impairments in emotional “self-regulation”, a related concept involving emotional processing and executive functioning, results from family history of psychological disorders and emotional dysregulation as well as early life adversity and is a key mechanism responsible for the development of posttraumatic stress symptoms in adulthood. She hypothesized that those with a family history of psychopathology may inherit deficits in emotional processing and executive functioning resulting in poorer emotional self-regulation. Deficits in the ability to regulate emotion could result in greater peritraumatic emotional responses during and after trauma exposure and could also increase risk behaviors, increasing the likelihood of additional trauma exposure.

Others have noted the importance of sociocultural mechanisms such as modeling and social support that may affect emotion regulation. A parent who is unable to model appropriate emotional regulation skills because of their own limited ability to cope with stress may inadvertently teach their child dysfunctional coping responses (Salmon & Bryant, 2002). In addition to learning to appraise situations from face-to-face contact with their caregivers, children also rely on verbal communication with adults to learn about and understand their own experiences (Cole, Michel, & Teti, 1994). In addition, social support is a known stress

buffer (Cohen & Wills, 1985). Individuals with family members with psychiatric symptoms may not receive social support from these traditional sources of support during times of stress. However, whether individuals with family histories of mental disorders and substance abuse are at increased risk for greater reactivity during trauma exposure, and, as a result, at greater risk for the development of posttraumatic stress symptoms has not been evaluated.

With the exception of a small number of prospective studies (e.g., by Koenen and colleagues (2007), most family and all twin studies to date have relied on cross-sectional data in which family disorders and posttraumatic stress symptoms in probands were assessed simultaneously, relying on retrospective reports of pre-trauma variables and symptoms. The present study employs a prospective design and examines family history of mood, anxiety, and substance-related disorders in police recruits prior to critical incident stress exposure, peritraumatic responses and posttraumatic stress symptoms, while considering prior cumulative civilian trauma exposure and critical incident stress exposure. As part of their duties, police officers are repeatedly exposed to life-threatening experiences which place them at heightened risk for developing posttraumatic stress symptoms. From a methodological perspective, this is an ideal population in which to study the influence of reported family history on posttraumatic distress; because recruits are psychologically healthy at the time of our baseline assessment (i.e., heavily screened by the police academy for psychopathology, alcohol and substance abuse), the probability that prior psychopathology will bias retrospective reporting of their own or familial psychiatric symptoms is minimized. Furthermore, examining a homogenous group of trauma survivors allows for a systematic assessment of more proximal risk factors such as level of critical incident exposure, and peritraumatic reactions to similar types of events.

In this study, we hypothesized that family loading for mood or anxiety disorders or alcohol or drug use disorders in first-degree relatives, assessed during academy training and prior to critical incident exposure, would predict peritraumatic distress to officers' worst critical incident experienced during the year, and lead to the development of posttraumatic stress symptoms following the first year of service. We focus on peritraumatic distress as the primary mechanism because of its proposed central role in affecting vulnerability for PTSD through adrenergic hyperarousal and peritraumatic dissociation. In addition, we expected that young healthy recruits self-selected for police service are relatively resilient to developing psychopathology, and therefore, we focused on a continuous measure of posttraumatic stress symptoms rather than on the categorical criteria for PTSD. We also examined other putative covariates or mechanisms, including prior cumulative civilian trauma exposure and nonspecific psychiatric distress measured prior to active duty police service, alcohol use, and critical incident stress exposure over the course of the first year of police service.

2. Methods

2.1 Participants

Data were collected as part of an ongoing prospective study of police officer stress and health in which clinical, self-report, and biological data were collected from 400 police recruits during academy training. Participating police officers were recruited from four urban police departments, the New York Police Department and three departments in the San Francisco Bay Area (Oakland, San Francisco, and San Jose) during police academy training. Of the 400 recruits initially enrolled, 389 (97%) completed baseline clinical interviews and self-report measures, and, of these, 278 (71%) completed self-report questionnaires at the 1 year follow-up.

Participants who dropped out of the study did not differ from those who remained in the study on age, income level, education, and family history of mood or anxiety disorder or family history of drug or alcohol use disorders (all p 's > .05). However, minority recruits were more

likely to complete the follow-up assessment, $X^2(1, N = 374) = 6.65, p < .05$. In addition, a nonsignificant trend suggested that women were more likely to drop out of the study than men, $X^2(1, N = 400) = 3.38, p = .07$.

2.2 Procedures

2.2.1 Sampling and Recruitment—Academy trainees were introduced to the study through a presentation made by study personnel during academy training classes, letters from the commissioner or police chief of the affiliated department and from the study team, and informational flyers posted at the academies. The study was open to all trainees except for those who were combat veterans or who had served in the military, law enforcement or emergency services occupations at the time of academy assessment. All procedures were approved by the University of California, San Francisco Human Subjects Committee and Institutional Review Board and a Federal Certificate of Confidentiality was obtained.

Participants were evaluated at baseline, which occurred while they were in training at the police academy, and twelve months after commencement of training. Prior to the initial assessment, study procedures were described in detail and written informed consent was obtained.

2.3 Measures

2.3.1 Initial Assessment During Academy Training—The initial assessment involved a structured clinical interview conducted by Ph.D. level clinicians and self-report questionnaires.

2.3.1.1 Demographics: Participants were asked to report on age, gender, ethnicity, and education.

2.3.1.2 Axis I Disorders: Current or past mood disorders, anxiety disorders, and substance use disorders was determined with the *Structured Clinical Interview for DSM-IV, version 2.0* (SCID I-NP) (First, Spitzer, Gibbon, & Williams, 1996).

2.3.1.3 General Distress: Participants also completed the self-report *Symptom Checklist 90-Revised Global Symptom Index (SCL-90-R)* (Derogatis, 1994) with the General Symptom Index (GSI) used to measure levels of general psychiatric symptoms.

2.3.1.4 Family History: Academy recruits were interviewed for lifetime psychiatric history of first-degree relatives using the *Family History Screen (FHS)* (Weissman et al., 2000), a structured interview screening tool used in previous research (Benazzi, 2004; Rende et al., 2007; Weissman et al., 2000) found to be comparable with other longer measures of family history (Milne et al., 2008). We determined first-degree relatives to include full biological parents and full biological siblings. The genetic material shared between a parent and offspring is 50% and between siblings or fraternal twins is approximately 50% on average. By combining these groups we increased the base rates of the disorders of interest giving us a sufficient distribution to examine these variables. We did not include children of probands in the analyses because most officers did not yet have children or had very young children with no known psychiatric symptoms. The FHS has a median sensitivity of 67.6% and specificity of 87.6% (Weissman et al., 2000). Participants were asked screening questions that correspond to core symptoms of Axis I disorders including alcohol and drug dependence, major depressive disorder, panic disorder, generalized anxiety, agoraphobia, simple phobia, social phobia, obsessive-compulsive disorder, posttraumatic stress disorder, and psychosis. The interviewer indicates whether participants endorsed the presence of these symptoms using a 3-point scale (0=not present, 1=present, and 9=don't know) and which relative was affected. We considered family members to be positive for a disorder if the interviewer coded a "1" for "definite"

endorsement of an item consistent with other research (Rende et al., 2007). The calculation for family loading for five family history variables (PTSD, anxiety, depression, alcohol use disorders, and drug use disorders) was adapted from previous research (Johnson & Pickens, 2001; Milne et al., 2008), and determined by summing the total number of first degree full biological relatives who, based on the reports of the recruits, screened positive (“definite”) for symptoms, separately for each family history variable, divided by the total number of first degree relatives, providing an estimate of family loading for that variable.

2.3.1.5 Civilian Trauma: Exposure to civilian trauma prior to starting police work was assessed by interview using the *Life Stressor Checklist-Revised* (Wolfe, Kimerling, Brown, Chresman, & Levin, 1996). LSC-R interviews were conducted by Ph.D.-level clinical psychologists, who calibrated their assessments biweekly. For each event, the respondent indicated 1) whether the event occurred; 2) whether at the time of exposure the event triggered intense emotions consistent with DSM-IV criterion A2 for PTSD; 3) whether the event occurred once or multiple times; and 4) the ages at which the event first and last occurred. We focused on respondents’ breadth of exposure to stressful events (e.g., the *Lifetime Exposure to Stressful Events* variable described in McHugo et al. (2005)), in which it is assumed that a wider exposure to trauma is indicative of increased severity of lifetime trauma exposure. Due to previous findings that personal life threat was most predictive of posttraumatic symptoms (McCaslin et al., 2006), we created an index of personally life threatening trauma by selecting 9 out of 30 stressful events, specifically those involving direct personal threat to the subjects (e.g., sexual and/or physical assault, serious accident or disaster, serious physical and mental illness, physical neglect, and abortion or miscarriage).

2.3.1.6 Alcohol Use: Participants reported on alcohol use at baseline using the *Michigan Alcoholism Screening Test (MAST)* (Selzer, 1971), a 25-item questionnaire assessing alcohol use disorders over the previous 30 days. We used a conservative cut score for “alcoholism” as 5 and higher (Selzer, 1971). In addition, data from the SCID provided a diagnostic evaluation of alcohol abuse and dependence. Because recruits were rigorously screened for illicit drug use by the police academy as a condition of enrollment and throughout police service, we did not assess for police recruits’ drug use.

2.3.2 Twelve-Month Follow-Up—Participants were reassessed 12 months after the start of active police duty, using the SCID and MAST. In addition, the following measures were administered at the 12-month follow-up:

2.3.2.1 Critical Incident Exposure: The *Critical Incident History Questionnaire (CIHQ)* is a 34-item self-report measure assessing cumulative exposure to critical incidents (Brunet et al., 1998; Weiss et al., in submission). Participants tabulate the frequency of exposure to each critical incident in the line of duty, resulting in a total cumulative exposure score across all items. The CIHQ demonstrated good agreement on incident severity ratings (.94), and adequate convergent and divergent validity (Weiss et al., in submission). For this study, we tabulated 14 items of experiences that posed a direct threat to life or physical integrity to be consistent with DSM-IV-TR criterion A for PTSD and since direct personal threat was found to be a strong predictor of distress and symptoms in police officers in previous research (e.g., (McCaslin et al., 2006)). Statistical analyses were performed using the log-transformed values of the sum to reduce the influence of several high frequency outliers.

2.3.2.2 Posttraumatic Stress Symptoms: The *PTSD Checklist-specific (PCL-S)* is a 17-item self-report assessment of DSM-IV PTSD-related symptoms of intrusion, avoidance, and hyperarousal (Blanchard, Jones-Alexander, Buckley, & Forneris, 1996; Ruggiero, Del Ben, Scotti, & Rabalais, 2003). Participants endorse the level of distress experienced with each of

the items on a 5-point scale ranging from 1 (not at all) to 5 (extremely) in the past week. The PCL-S was found to have adequate internal consistency (Cronbach's $\alpha = .94$), test-retest reliability (.88 for 1 week), and adequate convergent and discriminant validity in civilians (Ruggiero et al., 2003). For this study, police were asked how much they have experienced each item related to their most stressful police-related critical incident over the course of the first year of service.

2.3.2.3 Peritraumatic Reactivity: Distress reactions related to the officer's self-identified most distressing critical incident that occurred during the first 12 months of active police duty was assessed using the *Peritraumatic Distress Inventory (PDI)*, a self-report questionnaire on dysphoric emotional states such as fear and physiological responses including rapid heart rate, sweating and shaking during and immediately after trauma exposure (Brunet et al., 2001). This measure has been found to be internally consistent, with good test-retest reliability, and good convergent and divergent validity in police officers (Brunet et al., 2001). While this measure was completed at the 12 month follow-up visit, responses reflect peritraumatic reactions that may have occurred at any point during the previous year during police service.

2.4 Data Analysis

All data were checked for expected ranges, presence of outliers and abnormal values, and to determine that the distribution of variables met assumptions of statistical tests. Subsequent analyses were conducted to identify potential confounds for family history variables or for posttraumatic stress symptom levels. Differences in characteristics of those positive or negative for family history of mood/anxiety disorders or substance use disorders were examined with independent sample t-tests for continuous variables, Chi square tests for categorical variables, and Kruskal-Wallis tests for rank-ordered data. Correlational analyses or independent sample t-tests were used to determine associations of these variables with posttraumatic stress symptom levels.

We conducted a principal factors analysis of five family history variables (PTSD, anxiety, depression, alcohol use disorders, and drug use disorders) to determine whether they reflect separate underlying factors of mood and anxiety disorder on the one hand, and alcohol and drug use disorders on the other. Exploratory factor analysis with both orthogonal (varimax) and oblique (promax) rotations suggested a two factor solution, with family history of alcohol and drug use disorders loading highly on one factor, and PTSD, anxiety, and depression loading highly on the second factor. However, using the oblique rotation, the correlation between the two factors was .70. We therefore conducted a confirmatory factor analysis to directly compare the one and two factor solutions. Using robust maximum likelihood estimation for non-normal variables in Mplus 5.1 (Muthén & Muthén, 1998–2007), we found that the two factor solution was significantly better than the single factor solution (Satorra-Bentler Chi-square = 5.22, $df = 1$, $p = .022$). We therefore proceeded to treat family history of anxiety or mood disorder separately from family history of alcohol or drug use disorders and calculated a family loading score for both categories to be used in our primary analyses.

We used path analysis to test the fit of our proposed models using M-Plus 5.1 (Muthén et al., 1998–2007). Because some of the variables were not normally distributed, models were fit using robust maximum likelihood estimation. Our final outcome variable was posttraumatic stress symptoms on the PCL-S at 12 months of police service. The predictors for the path model were screened for multicollinearity. Since our study used a prospective longitudinal design, the directionality of several of the paths was determined by the timing of assessment.

We tested two path models depicted in figures 1 and 2, one examining the direct and indirect effects of family loading of mood and anxiety disorders on posttraumatic stress symptoms at one year of police service and the other examining the direct and indirect effects of family

history of drug and alcohol disorders on posttraumatic stress symptoms at one year. To examine the role of family mood and anxiety on posttraumatic stress symptoms, we entered the following variables in the models: 1) *pre-academy variables* that were assessed at baseline during academy training: loading of family history of mood and anxiety, prior cumulative civilian trauma exposure, 2) *academy variables* assessed at baseline: general psychiatric symptoms during academy training, 3) *peritraumatic variables* that were assessed at 12 months: critical incident stress exposure based on number of life threatening duty-related events experienced and peritraumatic distress to officer's worst self-identified event that occurred at any point during the first year of police service and 4) *posttraumatic variables*: posttraumatic stress symptoms at 12 months.

For the path model examining family substance use disorders on the development of posttraumatic stress symptoms, we entered the following variables: 1) *pre-academy variables*: loading of family history of drug or alcohol abuse and dependence and prior cumulative civilian trauma, 2) *academy variables*: alcohol use during academy training using the MAST, 3) *peritraumatic variables*: critical incident stress exposure and peritraumatic distress during the first year of police service, and 4) *posttraumatic variables*: posttraumatic stress symptoms at 12 months. On the family history screen, several recruits reported that they were uncertain of the presence of family alcohol or drug use disorders ($n = 3$) and for family mood or anxiety disorders ($n = 7$) in some of their relatives. The models were tested without the inclusion of these data points. There were also scattered missing data (less than 5% per variable) on the other predictors. An advantage of the maximum likelihood estimation procedure used here is that it can accommodate these missing values without excluding subjects and without biased results, under the assumption that data are missing at random.

3. Results

3.1 Sample Characteristics

Sample characteristics, including demographics, prior cumulative civilian trauma exposure, symptom measures and family disorders are listed in Table 1. Those reporting family drug and alcohol use disorders were older than those without such family histories, $t(64.41) = -3.43, p < .05$, however, since there was no association between age and posttraumatic stress symptoms at one year ($r = .09, p = .16$), we did not include age as a covariate in our path model. There were no other differences in demographics (gender, income¹, education, or minority status) found between family history groupings (all p 's $> .05$) and no significant associations between these demographic variables and posttraumatic stress symptoms (all p 's $> .05$). There were no systematic differences found between participants who were only children ($n = 38$) and those who had biological siblings on age, gender, education, minority status, income, and posttraumatic stress symptoms (all p 's $> .05$).

3.2 Psychiatric symptoms and nonspecific distress of recruits during academy training

As expected in a carefully screened group of academy recruits, general distress measured during academy training using the SCL-90-R GSI was relatively low ($M = .18, SD = .21$) compared with published norms for a general nonpsychiatric population ($M = .31, SD = .31$; (Derogatis, 1994)). SCID interviews at baseline confirmed very low rates of current Axis I disorders present in recruits, including single cases of Bipolar II (hypomania), depression NOS, specific phobia, and anxiety NOS. A minority of participants met criteria for prior lifetime diagnoses, including a single case of lifetime mood disorder due to a general medical condition, one case of anxiety disorder due to a general medical condition, and several cases of prior major depression (6.5%, $n = 18$), depression NOS (2.9%, $n = 8$), PTSD (1.1%, $n = 3$), alcohol

¹Kruskal-Wallis test used for rank-ordered data: e.g., income level, education

abuse (11.5%, $n=32$), and alcohol dependence (2.9%, $n=8$), all in remission during academy training. At baseline, daily alcohol use over the past 7 days assessed with the MAST was low ($M=1.73$; $SD=2.57$), well below the established cut-off of 5 for alcoholism (Selzer, 1971).

The rates of disorders among family members are presented in Table 2. Mood or anxiety disorders were found in the first-degree full biological relatives of 41% ($n=114$) of recruits. Drug or alcohol use disorders in first-degree full biological relatives was reported by 18.9% ($n=52$) of recruits. There was substantial overlap among those with family mood or anxiety disorders and those with family substance use disorders, $X^2(1, N=268) = 17.36, p < .001$. While 29.1% ($n=78$) had family mood or anxiety symptoms only and 6.0% ($n=16$) had family substance-related difficulties only, 12.7% ($n=34$) had both types of disorders in first-degree relatives and 52.2% ($n=140$) had relatives with neither type of disorder. Having a personal lifetime history of mood/anxiety disorders was associated with family loading of mood or anxiety disorders, $X^2(1, N=271) = 6.26, p < .05$, greater psychiatric distress during academy training, $t(273) = -2.19, p < .05$, but not with the presence of posttraumatic stress symptoms at 12 months, $t(34.13) = -1.59, p < .12^2$. Lifetime alcohol abuse or dependence in recruits was not associated with family loading for drug or alcohol use disorders, $X^2(1, N=275) = 1.21, p = .27$, global symptoms of distress, $t(273) = -.30, p = .77$, or with posttraumatic stress symptoms at 12 months of police service, $t(276) = -.87, p = .38$.

Approximately 2.6% ($n=7$) of our sample indicated the presence of possible psychotic symptoms in family members. Six of these cases were in families with a loading for mood or anxiety disorders and one case was in a family with a loading for substance-related difficulties. We did not evaluate family loading of psychotic disorders as a separate category due to the low prevalence in this sample and the overlap with other disorders.

Despite frequent critical incident exposure during the first year of police service (see table 1), levels of posttraumatic stress symptoms on the PTSD Symptom Checklist at 12 months of police service were generally low, $M=19.88, SD=5.64$, and well below established cutoff scores for PTSD in civilians (e.g., 44–50) (Ruggiero et al., 2003). One officer (0.4%) met criteria for PTSD and 7 met criteria for partial PTSD (2.5%) at 12 months using the PCL-S cutoff.

3.3 Path Analysis Examining Family Psychiatric History and PTSD

Intercorrelations among variables included in the path model are presented in Table 2. Figure 1 illustrates the associations among variables with partial correlation coefficients (beta weights) given for each path. The results of the path analysis indicate that family loading of mood and anxiety disorders had a significant direct effect on peritraumatic distress and a significant indirect effect on posttraumatic stress symptoms that was fully mediated through peritraumatic distress to the worst critical incident in the first year of service. Interestingly, prior cumulative civilian trauma also had a significant indirect effect on posttraumatic stress symptoms that was partially mediated through academy distress. The full model accounted for 30% of the variance in posttraumatic stress symptoms. Fit indices for the overall model were good: $\chi^2(2, n=278) = 108, p = .95$; Comparative Fit Index (CFI) = 1.000; and the Root Mean Square Error of Approximation (RMSEA) = 0.000, LO 90 = .000 to HI 90 = .013.

3.4 Path Analysis Examining Family Substance Use Disorder History and PTSD

Figure 2 shows the path model for family loading of drug and alcohol use disorders. A significant direct effect was present for family loading of drug and alcohol use disorders on peritraumatic distress and family loading of drug and alcohol use disorders indirectly predicted posttraumatic stress symptoms via peritraumatic distress. The full model accounted for 25%

²T test for unequal variances.

of the variance in posttraumatic stress symptoms. The overall model yielded strong fit indices: $\chi^2(3, n = 278) = .255, p = .880$; CFI = 1.000; and RMSEA = 0.000, LO 90 = .000 to HI 90 = .058.

Since there was a significant correlation between family loading of drug and alcohol use disorders and global symptoms of distress, we conducted posthoc analysis to investigate whether global distress might be an additional mediator in the model described above. The addition of GSI to the model above indicated that the indirect path between family loading of drug and alcohol use disorders and posttraumatic stress symptoms through GSI was not significant, $\beta = .02, p = .28$.

4. Discussion

Consistent with prior research (e.g., Dierker et al., 2001), we found that greater family loading of mood and anxiety disorders and substance-related disorders were associated with the subsequent development of greater posttraumatic stress symptoms, extending previous findings with our use of a prospective design. A novel contribution of this study is that peritraumatic distress to the officers' worst critical incident experienced during the first year of police service was an important mediator of these relationships. This is consistent with the model that vulnerability for PTSD is expressed via increased peritraumatic arousal responses, leading to greater fear conditioning and overconsolidation of traumatic memories (Koenen et al., 2002; Pitman, Shalev, & Orr, 2000). We also found that general symptoms of emotional distress during training were another significant mediator between family history of mood and anxiety disorders and posttraumatic stress symptoms. This finding is not surprising given that problems in prior adjustment have been found to predict PTSD in other research (Ozer et al., 2003). Interestingly, peritraumatic responses accounted for significant variability even after considering prior symptomatology in the model.

Our findings emphasize the important role of emotional regulation at the time of a traumatic stressor, in the prevention of subsequent distress. Emotional dysregulation may be transmitted among members of a family in a number of ways. While our use of the family history method did not include genotyping, several genetic markers for PTSD (e.g., polymorphisms in FKBP5 (Binder et al., 2008; Koenen et al., 2005)) and for associated brain abnormalities (e.g., serotonin transporter gene (Hariri et al., 2002; Pezawas et al., 2005)) have been proposed. Further, family dynamics and relationships may be impacted by the turning of attention, either positive or negative, to the affected individual. (Bremner, 2002; Pitman et al., 2001).

In addition to biological mechanisms, sociocontextual variables also influence the development of emotional regulation skills. Parents play a particularly important role as they are responsible for the direct care of the child and typically serve as a protector and a role model. Recruits raised by parents who had difficulty regulating emotional responses during stress may have had fewer opportunities to learn effective emotion regulation skills (Cole et al., 1994). Officers with affected family members may have received inadequate social support, an important stress buffer, from family members during childhood and they may not have learned how to seek out or effectively use other potential sources of support later in their lives. Further, having a sibling with a psychological disorder can impact family dynamics by drawing greater attention, either positive or negative, from parents to the affected sibling, influencing relationships between officers, their parents, and their siblings.

In addition, children of individuals with psychiatric disorders or addictions may be exposed to more traumatic events because of risky or abusive parental behaviors (e.g., Koenen et al., 2007; Yehuda, Halligan, & Grossman, 2001). Our results highlight the importance of examining the role of prior cumulative trauma exposure in such models, specifically in association with familial mood, anxiety, and substance-related disorders as well as general

distress and PTSD symptoms. Although distress and symptoms were relatively lower than typically seen in other populations, prior cumulative civilian trauma exposure before academy training was associated with more general emotional distress during the academy, perhaps in response to the stress of training, and recruits with greater prior cumulative civilian trauma exposure were more vulnerable when exposed to subsequent critical incidents during the first year of police service. Officers with more prior cumulative civilian trauma exposure who were initially resilient may have had some degree of sensitization by repeated exposure to critical incident stress related to police work. A sensitization hypothesis was supported by a study of emergency service personnel involved in the clean up and recovery of an major airline crash (Dougall, Herberman, Delahanty, Inslicht, & Baum, 2000). In this study, more prior exposure to trauma that was dissimilar from rescue work was associated with greater distress symptoms and intrusive thoughts at 2, 6, 9 and 12 months after the crash, and greater urinary catecholamine levels at 12 months after the crash. Biological studies conducted in more symptomatic populations have suggested that early life trauma exposure leads to prefrontal cortex damage via stress-related hormonal alterations (De Bellis et al., 1999; Nelson & Carver, 1998), resulting in dysregulated stress responsivity to future traumatic events (De Bellis et al., 1999; Perry, 1995). Our group has previously found that police recruits with childhood trauma had greater emotion modulated startle reactivity to startling sounds (Pole et al., 2007) and greater catecholamine responses to a laboratory stressor (Otte et al., 2005) during academy training. While less symptomatic, it is possible that police recruits who experience repeated trauma and ongoing chronic occupational and familial stress may have similar stress related biological alterations that may influence risk for the development of posttraumatic stress symptoms to later trauma exposure. Future research should consider a finer analysis of prior trauma characteristics that may explain a potential differential impact on peritraumatic reactivity as well as biological underpinnings of increased vulnerability in these police officers.

Another possibility is that prior cumulative civilian trauma exposure may have increased risk for future trauma exposure. We found a trend suggesting a relationship between prior cumulative civilian trauma exposure and greater distress during academy training, greater exposure to life-threatening critical incidents, greater peritraumatic distress, and posttraumatic stress symptoms. Koenen suggested that individuals with an overly reactive amygdala and impairments in emotional processing may also have difficulties with stimulus registration and the assessment of threat (Fox, 1994; Koenen, 2006), potentially increasing the likelihood that they are exposed to dangerous situations. Previous research also suggests that impulsivity or sensation-seeking, a potentially inherited trait, could increase risk of exposure to trauma and PTSD (Koenen, 2006; McLeod et al., 2001).

Prior research has also suggested that substance abusers (and potentially those at high risk for substance abuse) have heightened stress reactivity and turn to substances to dampen their reactivity to stress (Jacobsen, Southwick, & Kosten, 2001). Our findings partially support this possibility since family history of addictions was associated with greater posttraumatic stress symptoms and this was mediated through peritraumatic distress to officers' worst critical incident over the first year of police work. However, a lack of relationships between alcohol use and family history of drug and alcohol use disorders or posttraumatic stress symptoms, contradicts prior research conducted in other trauma-exposed populations (e.g., McFarlane et al., 2009; Prescott & Kendler, 1999; Stewart, 1996). We may have been unable to detect these relationships due to minimal alcohol use during academy training and at 12 months, which suggest that alcohol use does not play an important role in this sample at this time. Low rates of alcohol use may be a result of self-selection into law enforcement, underreporting, and rigorous police academy screening and continual monitoring of alcohol and substance use.

Our study was limited in that the family history method did not allow us to determine whether family effects were due to genetics versus shared environmental influences. It also relies on

recruit reports of their relatives functioning. While a more ideal method would be to personally interview family members about their symptoms, we interviewed largely asymptomatic recruits prior to critical incident exposure, which would have likely mitigated potential recall biases associated with current distress (Milne et al., 2008).

It is notable that even though most officers experienced life-threatening exposure to critical incident stressors, PTSD symptoms scores were positively skewed. Despite a restricted range in symptomatology, we had adequate variability in posttraumatic symptomatology to detect significant relationships after only one year of police service. There are several reasons that symptom reports were relatively low and only 3% of the sample had full or subsyndromal PTSD at the 12-month assessment, based on the PCL-S. Because police officers are a self-selected group, carefully screened by the police academies, and trained to manage life threat exposure, it is a particularly resilient population, and as a result the generalizability of our findings to other trauma-exposed populations may be limited. In addition, emergency services personnel have been noted to underreport symptoms (Wagner et al., 1998), perhaps due to bravado, concerns about confidentiality and potential impact on job security and advancement. Further, it is possible that individuals with higher levels of symptomatology may have dropped out of the study or police service. While our sample is seemingly resilient, they are early in their careers. Research in officers at later stages in their careers suggest that police are at heightened risk for posttraumatic distress symptoms, chronic stress, burnout and stress-related health problems due to the nature of their repeated exposure to stress and violence extended over their careers (Pole, 2008). Police officers assessed at mid-career had rates approximating 5% for full PTSD and an additional 2% for subthreshold PTSD; full criteria for PTSD were found in 15% of retired police officers (Pole, 2008). In addition, we acknowledge that our measure of PTSD (i.e., PCL-S) was designed to capture symptoms related to a single focal event and is not a cumulative measure; thus, it may not capture symptoms related to other critical incidents or traumatic experiences that may have occurred in civilian life.

Despite these potential limitations, to our knowledge, this is one of the first studies to examine the impact of family history on posttraumatic stress symptoms using a prospective design, while considering historical variables systematically measured prior to exposure to a focal trauma, trauma characteristics, and peritraumatic emotional reactivity. These data provide initial support for a model in which peritraumatic reactivity mediates the relationship between pre-exposure family history risk factors and the development of critical incident related posttraumatic stress symptoms. Given that those with family disorders are at heightened risk for peritraumatic distress which in turn increases risk for developing posttraumatic stress symptoms, interventions to reduce peritraumatic distress including stress inoculation during training and as soon as possible after critical incident exposure will likely benefit at risk police officers. It will be important to evaluate the possibility that familial factors may increase risk for peritraumatic reactivity and posttraumatic stress symptomatology in other trauma-exposed populations. Given the critical function that police serve in our society, it is of great importance to better understand the early processes that lead to the development of symptoms that may incubate over time and could result in psychiatric distress and dysfunction at later stages in their careers.

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Figure 1.

Path Model of Familial Loading of Mood and Anxiety Disorders Predicting Posttraumatic Stress Symptoms (n = 278)

Note. $^+p < .10$, $^*p < .05$, $^{**}p < .01$, $^{***}p < .001$. Pre-academy and academy variables were measured at Baseline during academy training, police duty and 1 year follow-up variables were measured at the end of Year 1.

Family Loading of Drug and Alcohol Use Disorders Predicting PTSD Symptoms (n = 278)

Figure 2.

Path Model of Familial Loading of Alcohol and Substance Use Disorders Predicting Posttraumatic Stress Symptoms (n = 278)

Note. $^+p < .10$, $^*p < .05$, $^{**}p < .01$, $^{***}p < .001$. Pre-academy and academy variables were measured at Baseline during academy training, police duty and 1 year follow-up variables were measured at the end of Year

Table 1

Sample Characteristics

	M or n	SD or %	n	%
<u>Baseline Measures</u>				
Age	27.0	4.60		
Gender (Male)			243	87.49%
Income				
Up to 15,000			2	0.7%
\$15,000 to \$30,000			11	4.0%
\$30,001 to 50,000			122	44.7%
\$50,001 to 70,000			79	28.9%
70,001 to 90,000			31	11.4%
\$90,001 and above			28	10.3%
Ethnicity				
Caucasian			114	41.6%
Hispanic/Latino			63	23.0%
African American			38	13.9%
Other or mixed ethnicity			24	8.8%
Education				
Up to 12 th grade			3	1.1%
Completed High School or equivalent			27	9.9%
Completed 2 year college/vocational training			91	33.2%
College graduate			139	50.7%
Post-graduate degree			14	5.1%
Number of Officers with prior civilian trauma			163	58.6%
Average number of prior civilian traumas	.91	.97		
<u>Disorders in First Degree Relatives</u>				
Major Depression			82	30.5%
Mania			3	1.1%
Generalized Anxiety			9	3.2%
Panic			18	6.5%
Social Phobia			8	2.9%
Specific Phobia			27	9.7%
Agoraphobia			4	1.4%
Obsessive Compulsive Disorder			7	2.5%
PTSD			27	10.6%
Drug Use Disorder			25	9.1%
Alcohol Use Disorder			42	15.3%
Psychotic Symptoms			7	2.6%
<u>12 Month Measures</u>				
Officers exposed to personally life threatening critical incidents over 1 st year			185	67.0%

	M or n	SD or %	n	%
Average number of personally life threatening critical incidents over 1 st year	7.47	13.40		
Mean Peritraumatic Distress Index	.57	.49		
PTSD Symptom Checklist	19.88	5.64		

Table 2
Correlation Matrix of Predictors of Posttraumatic Stress Symptoms (N=257–278)

	1.	2.	3.	4.	5.	6.	7.	8.
1. PTSD Symptoms	---	.16**	.24***	.21***	.39***	.03	.24***	.44***
2. Family Mood and Anxiety Symptoms		---	.34***	.14*	.22***	.03	.05	.25***
3. Family Drug and Alcohol Difficulties			---	.22***	.13*	.02	-.01	.18**
4. Prior Cumulative Civilian Trauma				---	.23***	.05	.02	.14*
5. GSI during academy					---	.16**	.13*	.20**
6. Alcohol Use during academy						---	.06	.07
7. Life-threatening Critical Incident Exposure (log)							---	.29***
8. Peritraumatic Distress								---

Table 3

Direct and Indirect Effects for the Path Model of Family Loading of Mood and Anxiety Disorders and PTSD Symptoms (N=278)

	β	SE	p-value
<u>Family History of Mood/Anxiety→PCL</u>			
Total Effects	.132	1.75	.079
Total Direct Effects	.005	.084	.933
Total Indirect Effects	.127	3.154	.002
<u>Specific Indirect Effects</u>			
Family History of Mood/Anxiety →GSI→PCL	.051	1.496	.135
Family History of Mood/Anxiety →PDI→PCL	.064	2.674	.008
Family History of Mood/Anxiety →GSI→Lifethreat→PCL	.003	1.147	.251
Family History of Mood/Anxiety →GSI→PDI→PCL	.007	1.088	.277
Family History of Mood/Anxiety →GSI→Lifethreat→PDI→PCL	.002	1.631	.103
<u>Cumulative Prior Trauma→PCL</u>			
Total Effects	.192	2.900	.004
Total Direct Effects	.094	1.526	.127
Total Indirect Effects	.099	2.902	.004
<u>Specific Indirect Effects</u>			
Prior Trauma→GSI→PCL	.057	2.399	.016
Prior Trauma→PDI→PCL	.029	1.282	.200
Prior Trauma→GSI→Life Threat→PCL	.003	1.227	.220
Prior Trauma→GSI→PDI→PCL	.007	1.340	.180
Prior Trauma→GSI→Life Threat→PDI→PCL	.002	1.817	.069
<u>Cumulative Life Threat→PCL</u>			
Total Effects	.194	2.809	.005
Total Direct Effects	.107	1.569	.117
Total Indirect Effects	.087	3.713	.000
<u>Specific Indirect Effects</u>			
Life Threat→PDI→PCL	.087	3.713	0.000
<u>GSI→PCL</u>			
Total Effects	.340	3.286	.001
Total Direct Effects	.279	2.982	.003
Total Indirect Effects	.061	2.446	.014
<u>Specific Indirect Effects</u>			
GSI→Life Threat→PCL	.014	1.304	.192
GSI→PDI→PCL	.036	1.492	.136
GSI→Life Threat→PDI→PCL	.011	2.059	.039

Note. β = Standardized Beta; SE = Standard Error for Standardized Effects

Table 4

Direct and Indirect Effects for the Path Model of Family Loading of Drug and Alcohol Use Disorders and PTSD Symptoms (N=278)

	β	SE	p-value
<u>Family History of Drug/Alcohol→PCL</u>			
Total Effects	.201	2.059	.040
Total Direct Effects	.143	1.473	.141
Total Indirect Effects	.058	2.450	.014
<u>Specific Indirect Effects</u>			
Family History of Drug/Alcohol →PDI→PCL	.058	2.447	.014
Family History of Drug/Alcohol →MAST→PCL	.000	-.173	.862
Family History of Drug/Alcohol →MAST→Lifethreat→PCL	.000	.188	.851
Family History of Drug/Alcohol →MAST→PDI→PCL	.000	.187	.851
Family History of Drug/Alcohol →MAST→Lifethreat→PDI→PCL	.000	.189	.850
<u>Cumulative Prior Trauma→PCL</u>			
Total Effects	.162	2.927	.003
Total Direct Effects	.126	2.418	.016
Total Indirect Effects	.037	1.603	.109
<u>Specific Indirect Effects</u>			
Prior Trauma→PDI→PCL	.036	1.564	.118
Prior Trauma→MAST→PCL	-.001	-.32	.745
Prior Trauma→MAST→Life Threat→PCL	.000	.680	.496
Prior Trauma→MAST→PDI→PCL	.001	.673	.501
Prior Trauma→MAST→Life Threat→PDI→PCL	.000	.724	.469
<u>Cumulative Life Threat→PCL</u>			
Total Effects	.239	3.298	.001
Total Direct Effects	.138	2.043	.041
Total Indirect Effects	.100	4.216	.000
<u>Specific Indirect Effects</u>			
Life Threat→PDI→PCL	.100	4.216	.000
<u>MAST→PCL</u>			
Total Effects	.015	.291	.771
Total Direct Effects	-.018	-.383	.701
Total Indirect Effects	.033	1.14	.254
<u>Specific Indirect Effects</u>			
MAST→Life Threat→PCL	.008	.987	.323
MAST→PDI→PCL	.019	.861	.389
MAST→Life Threat→PDI→PCL	.006	1.107	.268

Note. β = Standardized Beta; SE = Standard Error for Standardized Effects