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Co-Twin Control Study of Relationships Among Combat Exposure, Combat-Related PTSD, and Other Mental Disorders

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The well-documented association between combat-related PTSD (C-PTSD) and other mental disorders may be an artifact of shared familial vulnerability. This study uses a co-twin control design to examine whether the association between C-PTSD and other mental disorders persists after adjusting for shared familial vulnerability. Data were from male monozygotic twin pairs in the Vietnam Era Twin Registry. Logistic regression analyses demonstrated that combat exposure, adjusted for C-PTSD, was significantly associated with increased risk for alcohol and cannabis dependence and that C-PTSD mediated the association between combat exposure and both major depression and tobacco dependence. We conclude C-PTSD comorbidity persists after controlling for shared vulnerability. Combat exposure is directly and indirectly, through C-PTSD, associated with increased risk for other mental disorders.

KEY WORDS: PTSD; combat exposure; twins; comorbidity.

The high prevalence of comorbidity between combat-related PTSD (C-PTSD) and other mental disorders in Vietnam veterans has been well documented (Keane & Wolfe, 1990; Kulka, et al., 1990). However, the inability to control for shared vulnerability that may contribute to the association between C-PTSD and other mental disorders is a major methodological issue that complicates interpretation of reported associations. For example, an observed association between combat exposure and drug dependence may occur not because combat exposure increases risk for drug dependence but because there is a shared vulnerability (e.g., sensation seeking) that increases risk for both. In this study, we use monozygotic (MZ) twins

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in a co-twin control analysis to adjust for the influence of shared vulnerability and examine whether combat exposure and C-PTSD have significant effects on risk for the following five mental disorders: major depression, alcohol dependence, drug dependence, cannabis dependence, and tobacco dependence.

Twin studies offer a natural experiment that can test whether a risk factor, such as combat exposure or C-PTSD, has an effect on risk for other mental disorders over and above any association due to shared vulnerability. Because MZ twins share 100% of their genes and many family background risk factors, within MZ pair differences in diagnosis must originate from the nonshared environment or the environmental influences that differ between twins. Thus, by selecting discordant MZ twins, we eliminate the possibility that an association found between combat exposure or C-PTSD and another mental disorder is due to genetic or shared environmental factors.

Having eliminated association due to shared vulnerability, we can ask questions about the uniqueness of the association between combat exposure, C-PTSD, and other mental disorders. One possibility is that combat exposure, which is necessary for a diagnosis of C-PTSD, may alone increase risk for other mental disorders. Alternatively, the association between combat exposure and other mental disorders may be mediated by C-PTSD. We test whether combat exposure and C-PTSD uniquely increases risk for other mental disorders is by using hierarchical regression analysis and examining whether combat exposure is associated with increased risk for another mental disorder after controlling for C-PTSD and, conversely, whether C-PTSD is associated with increased risk of another mental disorder after controlling for exposure.

Method

Participants

Participants were members of the Vietnam Era Twin (VET) Registry. The VET Registry, assembled from Department of Defense computerized military records, is a nationally distributed cohort, comprised of male–male twin pairs born between 1939 and 1957 in which both members served in the military during the Vietnam War era (Eisen, True, Goldberg, Henderson, & Robinette, 1987; Henderson et al., 1990). Zygosity was determined using a questionnaire and blood group typing methodology that achieved 95% accuracy (Eisen, Neuman, Goldberg, Rice, & True, 1989). Of 10,300 eligible individuals (5,150 pairs), 8,169 (79.6%) were successfully interviewed by telephone in 1993 as part of the Harvard Twin Study on Substance Abuse. The mean age of respondents was 44.6 years (SD = 2.8, range = 36–55 years); 90.4% were non-Hispanic white, 4.9% African American, 2.7% Hispanic, 1.3% Native American/Alaskan Native, and 0.7% "other"; 33.3% were high school graduates and 38.6% college graduates; 92.6% were employed full-time and 1.8% part-time. The sample used for this study consisted of all 1874 monozygotic twin pairs from the VET Registry for whom complete *DSM-III-R* diagnostic information was available.

Measures

Combat Exposure Index

Combat exposure was measured by asking each veteran whether he engaged in 18 specific combat activities, such as, flying in an attack helicopter, being wounded, and receiving incoming fire. A global index of combat exposure was constructed by summing over all positive responses to form approximate quartiles: 0 (*did not serve in Southeast Asia*), 1 (*served in Southeast Asia but did not experience combat*), score of 2–3 (*low combat*), score of 4–6 (*medium combat*), and score of 7 or more (*high combat*). The four level combat index demonstrated good internal consistency (coefficient alpha = .86) and test–retest reliability (k = .84). The validity of the index is supported by a strong association between the index and being awarded a military combat metal (Janes, Goldberg, Eisen, True, & Henderson, 1991).

Diagnostic Interview Schedule Version III—Revised (DIS-III—R)

A structured psychiatric interview was administered to VET Registry twins by telephone (Robins, Helzer, Cottler, & Goldring, 1988). The questions from the DIS-III-R assess mental disorders according to the *Diagnostic and Statistical Manual for Mental Disorders-III-R* (*DSM-III-R*, American Psychiatric Association, 1987). In the National Vietnam Veterans Readjustment Study (NVVRS), the DIS was found to have high specificity (97.9%) but low sensitivity (21.5%) in identifying cases of PTSD (Kulka et al., 1990).

The DIS-III-R inquires about the occurrence of a number of traumatic events. If a respondent endorses one of these events or another qualifying experience, the interviewer gathers information about age at exposure, age at onset of symptoms, symptom type, and duration. This information is collected on up to three traumatic events. Participants received a C-PTSD diagnosis if they

(1) reported combat as a traumatic event and (2) met *DSM-III-R* criteria for PTSD in relation to this event. Details regarding the types and prevalences of events and interview procedures are published elsewhere (Koenen et al., 2002). This study focused on five lifetime diagnoses: alcohol dependence, drug dependence, major depression, cannabis dependence, and tobacco dependence.

Control Variables

While the co-twin control method controls for the contribution of shared familial vulnerability (e.g., genes, parental education) to the association between combat exposure/C-PTSD and other mental disorders, this method does not account for nonshared environmental factors that might confound these relationships. Findings from the NVVRS (Kulka et al., 1990) and this sample (Koenen et al., 2002) suggest that the following variables are potential nonshared environmental confounders of the relationship between combat exposure/C-PTSD and other mental disorders: premilitary trauma history, age of entry into the military, and level of education at entry into the military. Age of entry into the military and level of education at entry into the military were abstracted from military records. The variable premilitary trauma history was created from the DIS-III-R PTSD section. The number of traumatic events before the age of entry into the military were counted and summed.

Statistical Analyses

The analyses focused on twin pairs who were discordant for each of the five specific psychiatric disorders (one twin had the disorder while the other twin did not). Initial analysis obtained estimates of the prevalence of C-PTSD in those with and without each of the diagnoses. The conditional logistic regression model (Breslow & Day, 1980) was used to account for the paired structure of the data and calculate the matched pairs odds ratios (OR) and 95% confidence intervals (CI) for the association between combat exposure and/or C-PTSD and the psychiatric diagnoses. Power analysis indicated there was 80% power for detecting a medium size effect for any disorder where there were at least 90 diagnosis discordant MZ pairs. Analyses were, therefore, limited to those disorders for which at least 90 or more twin pairs were diagnosis discordant (Cohen, 1977). Pre-military trauma history, age of entry into the military, and education level at entry into the military were entered as control variables in all models. Three models were examined for each psychiatric diagnosis:

Model 1 examines the association of the ordinal index of combat exposure with each diagnosis. This model tests whether combat exposure is a risk factor for the diagnosis. Model 2 examines the association of C-PTSD with each diagnosis. This model tests whether C-PTSD is a risk factor for the diagnosis. Model 3 includes both combat exposure and C-PTSD to examine the unique and simultaneous effects of both factors on risk for the psychiatric diagnoses. If combat is associated with the diagnosis after adjusting for C-PTSD, then this suggests there is a unique association between combat exposure and the disorder over and above what is produced by C-PTSD. If C-PTSD is associated with the other diagnosis after entering combat exposure into the model, then this suggests there is a unique association between C-PTSD and the disorder over and above what is produced by combat exposure. If, when C-PTSD is entered into the model, the OR for the association between combat exposure and the diagnosis is substantially reduced, C-PTSD is a mediator of the relationship between combat exposure and that disorder. "Substantially reduced" is defined as a reduction of the OR for the association between combat exposure and the mental disorder of 10% or more (Rothman, 2002; p. 194).

Missing Data

Analyses were undertaken to determine if missing data on combat exposure, C-PTSD, or control variables were associated with outcomes. Missingness was not related to the presence or absence of the mental disorders examined in this study.

Results

For all MZ and dizygotic pairs in the VET Registry, the prevalence of subjects who met lifetime *DSM-III-R* criteria for the disorders examined in this study was as follows: PTSD (9.6%), major depression (9.2%), alcohol dependence (35.2%), drug dependence (9.5%), tobacco dependence (47.8%), and cannabis dependence (6.6%).

Table 1 presents the OR and 95% CI for the conditional logistic regression analyses as well as the descriptive statistics for combat exposure and C-PTSD prevalence for MZ twins discordant for each mental disorder. For each specific diagnosis, the twin with the diagnosis had a higher mean level of combat exposure and a higher prevalence of C-PTSD than the twin without the diagnosis.

Results for Model 1 indicate that combat exposure unadjusted for C-PTSD is significantly associated with

Table 1. Descriptive Statistics and Conditional Logistic Regression Analyses for the Relationship of Combat Exposure and PTSD to Comorbid Mental Disorders

	Combat *M* (*SD*)	PTSD (%)	Model 1 Combat unadjusted for PTSD OR (95%CI)	Model 2 PTSD unadjusted for Combat OR (95%CI)	Model 3 Combat adjusted for PTSD OR (95%CI)	Model 3 PTSD adjusted for Combat OR (95%CI)
Major depression (n = 237)			1.22 (1.03,1.44)*	3.04 (1.48,6.24)*	1.10 (0.92,1.33)	2.55 (1.16,5.61)*
Absent	1.31 (2.73)	6.33				
Present	1.81 (1.01)	14.77				
Alcohol dependence (n = 524)			1.16 (1.02,1.31)*	1.39 (0.76,2.56)	1.15 (1.01,1.30)*	1.21 (0.63,2.31)
Absent	1.26 (2.64)	3.70				
Present	1.39 (2.54)	5.34				
Drug dependence (n = 224)			1.24 (1.03,1.48)*	2.26 (1.05,4.88)*	1.19 (0.99,1.44)**	1.73 (0.76,3.92)
Absent	1.09 (2.51)	6.70				
Present	1.56 (2.96)	11.61				
Tobacco dependence (n = 548)			1.18 (1.04,1.34)*	3.09 (1.55,6.16)*	1.14 (1.00,1.29)	2.76 (1.37,5.57)*
Absent	1.02 (2.26)	2.37				
Present	1.34 (2.62)	6.39				
Cannabis dependence (n = 157)			1.36 (1.09,1.71)*	2.24 (0.95,5.31)	1.31 (1.04,1.66)*	1.67 (0.66,4.20)
Absent	1.08 (2.54)	6.37				
Present	1.77 (3.06)	12.10				

Note. *n*s provide the number of pairs discordant for diagnosis. All analyses adjusted for premilitary trauma history, age of entry into the military, and level of education at entry into the military.
*$p < .05$. **$p < .10$.

major depression, alcohol dependence, drug dependence, tobacco dependence, and cannabis dependence. The OR for combat exposure indicates the odds of having a diagnosis for a one-step increase in the combat exposure scale. For example, if Twin A has a combat exposure score that is one-point higher than his co-twin, Twin A's odds of having a major depression diagnosis are 1.22 times higher than those of his co-twin. Alternatively, Twin A is 22% more likely to have a major depression diagnosis than his co-twin.

Results for Model 2 indicate that C-PTSD unadjusted for combat exposure is significantly associated with major depression, drug dependence, and tobacco dependence. The odds ratios for the association between C-PTSD and these disorders are substantial, ranging from 2.26 to 3.53. This means that, for example, if Twin A has C-PTSD, his odds of having a diagnosis of major depression are 3.04 times higher than that of his co-twin without C-PTSD. Alternatively, Twin A is 204% more likely to have a major depression diagnosis than his co-twin.

Results for Model 3 indicate that after adjusting for C-PTSD, combat exposure is significantly associated only with alcohol dependence and cannabis dependence. There was a trend for a unique association between combat exposure and drug dependence. The OR for the association between combat exposure and major depression is substantially reduced, suggesting C-PTSD is a mediator of the relationship between combat exposure and major depression. C-PTSD also qualifies as a mediator of the association between combat exposure and tobacco dependence. C-PTSD (adjusted for combat exposure) is significantly associated with major depression and tobacco dependence.

Discussion

C-PTSD comorbidity persists after controlling for the potential confounding effects of genetic and shared environmental vulnerability by using a co-twin control design. Because differences in diagnostic status between MZ twins must be due to nonshared environmental factors, this study provides strong evidence that combat exposure and C-PTSD are nonshared environmental factors that contribute to differences in risk for other mental disorders among Vietnam veterans.

Our results further suggest that combat exposure and C-PTSD differentially affect risks for specific mental disorders. When combat exposure and C-PTSD were entered separately into models predicting comorbid disorders (Models 1 & 2), MZ twins who had lifetime major depression, drug dependence, or tobacco dependence were more likely to have been exposed to combat and C-PTSD than their co-twins who did not have the disorder. Moreover, the effects of combat exposure on risk for alcohol dependence and cannabis dependence persisted after controlling for C-PTSD, indicating that combat exposure has a unique effect on risk for these disorders. In contrast, C-PTSD was not uniquely associated with increased risk of alcohol, drug, or cannabis dependence. An association between combat exposure and increased risk of substance dependence is consistent with previous co-twin control

studies showing an association between stressful life events such as childhood sexual abuse and negative psychosocial outcomes (e.g., Kendler et al., 2000).

In contrast, C-PTSD remained a significant predictor of major depression and tobacco dependence after adjusting for combat exposure. Moreover, the association between combat exposure and both major depression and tobacco dependence was not significant once C-PTSD was entered into the model, suggesting that C-PTSD mediates the association between combat exposure and these disorders; that is, it is the increased likelihood of C-PTSD after combat exposure and not the exposure itself increases risk. The finding that C-PTSD increases risk for major depression is consistent with other epidemiological studies using community samples (Breslau, Davis, Peterson, & Schultz, 2000) and with previous research on Vietnam veterans (e.g., CDC, 1988; Mellman, Randolph, Brawman-Mintzer, Flores, & Milanes, 1992). As far as we are aware, the association between tobacco dependence and C-PTSD has not been examined in epidemiological samples. Further research into the relationship between C-PTSD symptomology and tobacco use is warranted (Beckham, 1999).

Our study does not support C-PTSD as a unique risk factor for alcohol dependence, drug dependence, or cannabis dependence, while general population studies have tended to suggest that PTSD increases risk for drug dependence, even after controlling for exposure to trauma (Chilcoat & Breslau, 1998; Kessler et al., 1995). The relationship between PTSD and drug dependence depends, however, on the class of substances examined. Chilcoat and Breslau (1998) identified PTSD as a unique risk factor for psychoactive drug dependence, but, like us, found no association between PTSD and cannabis dependence. Previous studies have not controlled for the contribution of shared genetic vulnerability to the association between PTSD and substance dependence. In fact, the association among these disorders appears to be due to genetic and nonshared environmental influences that these disorders have in common (Xian et al., 2000). This study suggests combat exposure is one component of the nonshared environmental influences underlying the PTSD, alcohol, and drug dependence association.

Limitations

These results should be interpreted in the context of four methodological limitations. First, the sample consisted entirely of male military personnel. The relationships among combat exposure, C-PTSD, and other mental disorders cannot be generalized to trauma exposure and PTSD among civilians or females. Second, our assessment of combat exposure and other potentially traumatic events was undertaken retrospectively. If individuals who had experienced a mental disorder were more likely to report combat exposure than their co-twins who did not have a mental disorder, this would inflate our associations. However, our combat exposure measure has been shown to be highly stable. Third, our assessment of noncombat trauma exposure was not comprehensive and might have resulted in the underreporting of traumatic events, particularly those that occurred in childhood. If noncombat traumatic events were underreported, not shared between twins, and increased risk of both combat exposure/C-PTSD and the mental disorder studied, our findings for the association between combat exposure/C-PTSD and other mental disorders would be inflated. Fourth, our assessment of PTSD has shown high specificity but low sensitivity. Thus, we may have classified some individuals as not having PTSD when they actually had the disorder. The effect of this possibility on our results is unclear. If random, misclassification would bias our results toward the null. If misclassification differed by the outcomes examined, our results might be inflated or attenuated depending on the direction of misclassification in relation to the outcomes.

Most importantly, we cannot infer a causal link between combat exposure/C-PTSD and the mental disorder studied. Our study design merely eliminates genetic and shared environmental risk factors from confounding the relationships examined. While we attempted to control for other nonshared environmental factors that might have confounded the association between combat exposure/C-PTSD and other mental disorders, it remains possible that such factors might increase risk for combat exposure/C-PTSD and other mental disorders. Moreover, we did not attempt to address the temporal relationship between combat exposure/C-PTSD and the onset of the other mental disorders. Whether such an analysis would be useful or valid with retrospective data is uncertain. A review of retrospectively reported onsets for the mental disorders examined determined that most were reported to begin after entry into the military (e.g., from 99.7% for major depression to 83% for tobacco dependence). This makes it unlikely that the associations between combat exposure/C-PTSD and other mental disorders are due entirely to pre-military psychopathology increasing risk for combat exposure or C-PTSD.

Conclusions

This study extends the results of previous research by using a MZ co-twin control design to examine the unique contribution of combat exposure and C-PTSD to risk for major depression, alcohol dependence, drug dependence,

tobacco dependence, and cannabis dependence while controlling for shared familial/genetic vulnerability. Our findings indicate that C-PTSD comorbidity is not merely the product of previously uncontrolled for shared vulnerability, but that combat exposure and C-PTSD have nonshared environmentally mediated effects of risk for other mental disorder. Moreover combat exposure and C-PTSD differentially affect risk for these disorders. Combat exposure uniquely increases risk for substance dependence, while C-PTSD uniquely increases risk for major depression and tobacco dependence. Interventions aimed at preventing combat exposure and C-PTSD should, therefore, decrease veterans' risk of developing other mental disorders. Future studies should combine genetically informative samples with longitudinal designs in order to specify the temporal relationship among disorders and fully understand the mechanisms underlying PTSD comorbidity.

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