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Increased risk of alcohol dependency in a cohort of National Guard troops with PTSD: A longitudinal study

Anna Kline [a,b,*], Marc D. Weiner [d], Donald S. Ciccone [c], Alejandro Interian [a,b], Lauren St. Hill [a,d], Miklos Losonczy [e]

[a] Department of Veterans Affairs-New Jersey Health Care System, Lyons, NJ, United States
[b] Department of Psychiatry, Robert Wood Johnson Medical School, Rutgers University, New Brunswick, NJ, United States
[c] University of Medicine and Dentistry of New Jersey, New Jersey Medical School, Newark, NJ, United States
[d] Bloustein Center for Survey Research, Rutgers University, New Brunswick, NJ, United States
[e] Lincoln Medical and Mental Health Center, New York, NY, United States

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ABSTRACT

Studies show high rates of co-morbid post-traumatic stress disorder (PTSD) and alcohol use disorder (AUD) but there is no consensus on the causal direction of the relationship. Some theories suggest AUD develops as a coping mechanism to manage PTSD symptoms and others that AUD is a vulnerability factor for PTSD. A third hypothesis posits independent developmental pathways stemming from a shared etiology, such as the trauma exposure itself. We examined these hypotheses using longitudinal data on 922 National Guard soldiers, representing a subsample (56%) of a larger pre- and post-deployment cross-sectional study of New Jersey National Guard soldiers deployed to Iraq. Measures included the PTSD Checklist (PCL), DSM-IV-based measures of alcohol use/misuse from the National Household Survey of Drug Use and Health and other concurrent mental health, military and demographic measures. Results showed no effect of pre-deployment alcohol status on subsequent positive screens for new onset PTSD. However, in multivariate models, baseline PTSD symptoms significantly increased the risk of screening positive for new onset alcohol dependence (AD), which rose 5% with each unit increase in PCL score (AOR = 1.05; 95% CI = 1.02–1.07). Results also supported the shared etiology hypothesis, with the risk of a positive screen for AD increasing by 9% for every unit increase in combat exposure after controlling for baseline PTSD status (AOR = 1.09; 95% CI = 1.03–1.15) and, in a subsample with PCL scores <34, by 17% for each unit increase in exposure (AOR = 1.17; 95% CI = 1.05–1.31). These findings have implications for prevention, treatment and compensation policies governing co-morbidity in military veterans.

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1. Background and study objectives

The conflicts in Iraq (Operations Iraqi Freedom: OIF) and Afghanistan (Operation Enduring Freedom: OEF) have resulted in increasing numbers of soldiers returning from combat with post-traumatic stress disorder (PTSD) and other combat-related stress conditions. Recent health surveillance data show 9.3% of active component and 16.3% of Reserve component troops screen positive for PTSD symptoms in post-deployment health assessments (Armed Forces Health Surveillance Center, 2011). Less well recognized, however, is the extent to which PTSD in returning veterans is complicated by co-morbid substance use problems, especially alcohol misuse. A recent study found that nearly half of active component and National Guard soldiers screening positive for PTSD also met criteria for alcohol misuse (Thomas et al., 2010) and in 2008, the Veterans Administration (VA) reported that almost 70% of veterans hospitalized for PTSD had a diagnosis of co-morbid substance use disorder (SUD) (Management of Post-Traumatic Stress Working Group, 2010). Veterans suffering from both disorders, moreover, have poorer long-term prognoses than those diagnosed with one disorder only (Schäfer and Najavits, 2007).

Despite the wide prevalence of co-morbid PTSD and SUD, the functional relationship underlying the co-occurrence of these disorders is not well understood. At least three hypotheses have been proposed to account for the association. One, the susceptibility hypothesis, states that prolonged substance abuse may increase

* Corresponding author. Dual Diagnosis Development, VA NJ Health Care System (116A), Lyons, NJ 07090, United States. Tel.: +1 908 647 0180x4560; fax: +1 908 604 5313.
E-mail address: Anna.kline@va.gov (A. Kline).

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one's vulnerability to the PTSD-inducing effects of trauma exposure through such mechanisms as substance-induced changes in brain neurochemistry or deficits in stress-related coping skills (Chilcoat and Breslau, 1998). A second explanation, the self-medication hypothesis, holds that individuals may turn to psychoactive substances to relieve the disturbing symptoms of traumatic stress (Brown and Wolfe, 1994; Ferrier-Auerbach et al., 2009; Stewart, 1996). A third hypothesis posits independent developmental pathways stemming from a shared etiology, such as common genetic factors (Comings et al., 1996; Kehle et al., 2012; McLeod et al., 2001; Miller, 2003) or shared environmental risk factors, including the trauma exposure itself (Jordan et al., 1991). These three hypotheses are not mutually exclusive, since a number of pathways may be involved in both the development and maintenance of co-morbidity, once it becomes established.

Disentangling the causal direction of the association between PTSD and SUD has important implications for prevention and treatment as well as direct relevance to policies governing disability compensation. Currently the VA does not consider SUD to be a primary service-connected disorder (38 U.S.C. § 1110). For this reason, compensation for SUDs is limited to cases where the disorder is deemed to be caused or aggravated by another primary service-connected condition. If, however, SUDs may develop as a direct consequence of combat exposure, VA policies regarding disability compensation may require further evaluation.

To date, the preponderance of research points to PTSD as a causal factor in the development of SUDs, rather than the reverse (Schäfer and Najavits, 2007), while the independent pathways hypothesis has received only mixed support (Streimer et al., 1985; Wilk et al., 2010b). Most previous prospective studies examining the PTSD-SUD relationship, however, have focused on civilian trauma (Breslau et al., 2003; Stewart, 1996); therefore, the results may not generalize to combat trauma since research suggests that psychological responses to trauma vary according to the type of traumatic event (Stewart, 1996). While a substantial literature has examined co-morbid SUD and PTSD in military veterans, the majority of this research was conducted with Vietnam veterans, employed cross-sectional, retrospective designs and/or focused on veteran populations in treatment. More recent studies of OEF/OIF veterans have been similarly limited. Two recent studies exploring co-morbid PTSD and Alcohol Use Disorders (AUD) in National Guard soldiers, for example, measured PTSD and alcohol use concurrently (Kehle et al., 2012; Marshall et al., 2012), thus limiting examination of the temporal order, or causal pathways, in the development of each disorder. Jacobson et al.'s (2008) research on Millennium Cohort Study participants represents one of the few prospective studies to measure PTSD and AUD at pre-deployment. This study found that both a history of PTSD symptoms/diagnosis and combat exposure independently predicted new onset AUD in Reserve/National Guard personnel, indicating the possibility of separate pathways to alcohol and co-morbid disorders. However, the study did not explore the alternative hypothesis that pre-deployment alcohol problems are a risk factor for PTSD.

The present study addresses some of the gaps in our understanding of the functional relationship between co-morbid PTSD and alcohol dependence (AD) in military veterans using longitudinal data from a sample of New Jersey National Guard (NJNG) soldiers deployed to Iraq. By investigating the temporal order of the development of the two conditions, the paper examines evidence for the three causal hypotheses described above, namely: 1. Pre-existing alcohol problems increase susceptibility to the development of co-morbid PTSD; 2. PTSD leads to the development of co-morbid AD; and 3. Combat exposure independently contributes to AD, irrespective of PTSD status.

2. Methods

2.1. Sample

Data for this study are based on pre- and post-deployment surveys assessing the mental and physical health of 922 NJNG soldiers deployed to Iraq in 2008–2009. Self-administered survey data were collected at pre-deployment on 2665 of 2995 Guard members (89%) undergoing mandatory pre-deployment medical screening, and at post-deployment on 1665 of 1723 soldiers (97%) who attended mandatory reintegration events approximately 3 months after their return. Because survey administration was conducted anonymously, we used a matching algorithm of non-personal identifiers (birth month and letters from respondent's birth city and mother's name) to match respondents from pre- to post-deployment, with demographic characteristics asked on both surveys to confirm the accuracy of our matches. This method resulted in the successful match of 56% of veterans attending the reintegration events, or 925 respondents. After eliminating three surveys for poor data quality as judged by two independent raters, our final sample consisted of 922 respondents. A more complete description of the sample and data collection methods is provided elsewhere (Kline et al., 2013; Ciccone and Kline, 2012; Kline et al., 2010). All study participants received written and verbal explanations about the study purpose and procedures but to preserve anonymity, written informed consent was not obtained. The study received Rutgers and VA institutional review board approval.

Since many respondents in our pre- and post-deployment cross-sectional surveys could not be matched for our longitudinal sample, there is the possibility that selection bias may have influenced our results. Moreover, only about 60% of deployed soldiers attended the mandatory re-integration events, raising the possibility that non-attendees had health concerns or experienced other systematic differences associated with their non-attendance. We examined this issue extensively in two earlier, unrelated studies by comparing pre-deployment ($N = 2543$), post-deployment ($N = 1665$), and matched samples ($N = 922$) on a wide range of demographic and illness-related characteristics (Ciccone and Kline, 2012; Kline et al., 2013). While earlier analyses showed few remarkable demographic or clinical differences across samples overall, pre-deployment clinical differences between matched and unmatched respondents suggested that soldiers with pre-deployment alcohol and/or PTSD problems may have been somewhat underrepresented at the post-deployment events. In an earlier unrelated paper on the determinants of PTSD by the present authors (Kline et al., 2013) we demonstrated that clinical differences in the samples did not affect the integrity of the relationships observed between key predictor variables and PTSD outcomes. We further address the possibility of selection bias affecting our current results in the Data Analysis and Results sections below.

2.1.1. Measures

Alcohol use and misuse We used several alcohol use and misuse variables as outcome or predictor variables or to define subsamples for specific analyses. All measures were obtained using questions and scoring algorithms from the DSM-IV-based National Survey on Drug Use and Health (NSDUH) (United States Department of Health and Human Services, 2007). Since NSDUH scoring algorithms do not provide continuous measures of alcohol misuse, our primary alcohol outcome measure, *positive screen for new onset AD*, was dichotomous, requiring the post-deployment endorsement of 3 or more DSM-IV-identified problem behaviors (e.g spending significant time using, developing tolerance, using more than intended, being unable to limit use, etc.). Our predictor