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# The relationship between post-traumatic stress disorder, depression and cardiovascular disease in an American Indian tribe

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## ABSTRACT

**Background.** Empirical findings suggest that psychiatric illness is associated with cardiovascular disease (CVD). The purpose of this study was to compare the strength of the association of lifetime post-traumatic stress disorder (PTSD) and lifetime major depression on CVD among Northern Plains American Indians.

**Method.** A total of 1414 participants aged 18–57 years completed a structured interview that assessed psychiatric diagnoses, alcohol abuse/dependence, self-reported CVD, and traditional CVD risk factors including age, sex, education, diabetes, high blood pressure, and smoking. Logistic regression analyses compared the odds ratios of CVD in participants with and without diagnosed PTSD or major depression.

**Results.** The rates of lifetime PTSD and major depression were 15% and 8% respectively. CVD was more commonly reported by participants with PTSD than by those without PTSD (12% *v.* 5%, $p \leqslant 0{\cdot}01$). Likewise, more participants with major depression reported CVD than did their non-depressed counterparts (14% *v.* 6%, $p \leqslant 0{\cdot}05$). PTSD was significantly associated with CVD even after controlling for traditional CVD risk factors and major depression (odds ratio 2·0, confidence interval 1·1–3·8). In contrast, the association of major depression with CVD was not significant after accounting for both traditional risk factors and PTSD.

**Conclusions.** Rates of PTSD are high in American Indian communities. Rising CVD rates in this population may be better understood if PTSD is considered along with other traditional risk factors. Future research should examine the association and mechanisms of PTSD and CVD prospectively. Such data could lead to more effective CVD prevention efforts for American Indians.

## INTRODUCTION

Heart disease, stroke, and other manifestations of cardiovascular disease (CVD) have emerged as major health problems among racial/ethnic minority communities in the USA in the last decade. The Centers for Disease Control and Prevention-sponsored Racial and Ethnic Approaches to Community Health (REACH 2010) initiative recognized that diverse, underserved populations show considerable variability in CVD risk factors, disease prevalence, and ability to access preventative health care services. In contrast to the declining CVD mortality rates in the general USA population, the incidence

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and mortality estimates of CVD are steadily rising in American Indian (AI) communities (Lee *et al.* 1990, 1998; Oopik *et al.* 1996; North *et al.* 2003). This increase in the prevalence of CVD cannot be completely explained by traditional risk factors such as diabetes, hypertension, smoking, and familial history of CVD (Howard *et al.* 1992, 1995; Welty *et al.* 1995), but many newly recognized CVD risk factors such as inflammation (Ross, 1999), hostility (Kahn *et al.* 1987; Yan *et al.* 2003), and depression (Rudisch & Nemeroff, 2003) have not yet been systematically examined among AIs. In the majority population, the cross-sectional association between major depression and CVD has been confirmed by prospective studies demonstrating that depression predicts both the development of CVD and poor outcomes among persons who already have CVD (Rudisch & Nemeroff, 2003). However, most of these studies have not rigorously examined the influence of other psychiatric illnesses on CVD, and none have focused on AIs.

A growing body of literature suggests that anxiety-related symptoms (Kawachi *et al.* 1994; Kubzansky *et al.* 1997, 1998; Kubzansky & Kawachi, 2000), especially post-traumatic stress disorder (PTSD) (McFarlane *et al.* 1994; Wolfe *et al.* 1994; Boscarino, 1997; Boscarino & Chang, 1999; Schnurr *et al.* 2000), are associated with CVD. Yet, these investigations have been compromised by high lifetime rates of major depression observed among individuals with PTSD, making it difficult to untangle the effects of major depression and PTSD. In addition, some studies have relied on self-report dimensional measures rather than diagnostic interviews to establish the presence of PTSD (Wolfe *et al.* 1994; Schnurr *et al.* 2000). Others involved only subjects engaged in high stress activities (e.g. military veterans, firefighters); even in research that has included nationally representative samples (Wolfe *et al.* 1994; Boscarino & Chang, 1999; Schnurr *et al.* 2000), the participants were Vietnam-era veterans, rather than individuals drawn from a community-based sample. Only one study has included women (Wolfe *et al.* 1994).

This study addresses some of these limitations by examining a large sample of AIs between the ages of 18 and 57 years who participated in the American Indian Service Utilization Psychiatric Epidemiology Risk and Protective Factors Project (AI-SUPERPFP). In this population-based study, tribal members completed in-depth interviews that yielded information on psychiatric illness, trauma exposure, development of PTSD following trauma exposure, and health status. The high prevalence of lifetime PTSD in this sample, particularly in women, and the low rate of major depression in comparison to national figures (Beals *et al.* 2005) provide a unique opportunity to examine the independent effects of PTSD and major depression on CVD. We, therefore, address the following questions: (1) Are lifetime PTSD and major depression associated with self-reported CVD? (2) Do traditional risk factors confound the association of PTSD and major depression with CVD? and, (3) Is PTSD more strongly associated with CVD above that observed for major depression after accounting for traditional risk factors?

## METHOD

### Study design, sample, and procedures

The primary objective of the AI-SUPERPFP was to estimate the prevalence of psychiatric disorders and health service utilization in two AI reservation populations. The AI-SUPERPFP involved a Southwest and a Northern Plains sample and included those listed on tribal rolls (the legal record of tribal membership) who were between the ages of 15 and 57 years in June 1997. Interviews were restricted to those who lived on or within 20 miles of their reservation at the time of the study. For the purposes of our study involving CVD risk, only those participants aged 18 years and older at the time of the interview with complete data for all study variables in the Northern Plains tribe ($n=1414$) were included. Using stratified random sampling procedures (Cochran, 1977), this population was stratified by age (four strata) and sex (two strata). Sample weights were used to account for differential probabilities of selection and non-response within strata (Kish, 1965). The AI-SUPERPFP study design and sampling methods are described in greater detail elsewhere (Beals *et al.* 2003; AIANP, 2005).

Considerable effort was made by the AI-SUPERPFP team during the project development phase to involve the AI communities in constructing content-valid, culturally relevant

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interview questions. A structured, comprehensive interview was administered by lay members of the tribal communities, intensively trained in research methods and assisted by laptop computers. The domains assessed included sociodemographic background, psychiatric disorders, substance use, physical health, functional status, and use of cultural and medical health-care services. All interview procedures were administered in a standardized, reliable manner (Beals *et al*. 2003). Data were collected from July 1997 to August 1999. The AI-SUPERPFP obtained the necessary tribal and university approvals, and written informed consent was obtained from each participant.

## Measures

### *CVD and risk factors*

Sociodemographic information included sex, age, and education as a measure of socioeconomic status. Education was dichotomized as attending $<12$ years of school *versus* $\geqslant 12$ years. Two interview questions were used to establish the presence of provider-diagnosed CVD. To create the CVD-positive group, we selected all participants endorsing the following question: 'Did a doctor, medicine man, or other health-care professional ever tell you that you had heart disease or stroke?' Similar questions were used to assess the presence of diagnosed diabetes and high blood pressure. Smoking status also was ascertained, and the sample was partitioned into never, former, and current smokers.

### *Psychiatric disorders*

PTSD was diagnosed using a modified version of the World Health Organization's Composite International Diagnostic Interview (CIDI; WHO, 1990) and based on DSM-IV criteria. The World Health Organization PTSD module was used because the University of Michigan CIDI can only assess a single, traumatic event; however, cumulative traumatic experiences are particularly salient among AIs (Beals *et al*. 2002). Data were collected from participants on three or less traumatic events, including type, frequency, direct or vicarious, and experienced symptoms of trauma exposure. Specifically, respondents were asked about 16 categories of traumatic events including experiencing a natural disaster, physical abuse, serious accident, witnessing traumatic events, or experiences with serious events that happened to someone close to the respondent. Lifetime PTSD was diagnosed based on established algorithms (Beals *et al*. 2002).

The University of Michigan CIDI was used to establish lifetime major depressive and alcohol abuse and dependence disorder based on DSM-III-R criteria (Kessler *et al*. 1994; Wittchen, 1994). Additional questions were added to conform to DSM-IV diagnostic standards (Beals *et al*. 2005). Those meeting diagnostic criteria for lifetime alcohol abuse or dependence were combined into a single abuse/dependence category.

## Statistical analyses

Descriptive statistics and 95% confidence intervals (CIs) for continuous variables were calculated as mean values and frequencies were computed for categorical variables. Logistic regression analyses were used to examine the association between PTSD and CVD and major depression and CVD. The prevalence odds ratio (OR) was used to compare the odds of CVD in participants with and without either lifetime PTSD or lifetime major depression. Initially, separate models adjusting for age (continuous), sex, and education (categorical) were fitted to estimate the PTSD-CVD and major depression-CVD associations. Next, we estimated the PTSD-CVD association after adjusting for major depression, age, sex, and education. Likewise, the major depression-CVD association was estimated after adjusting for PTSD, age, sex, and education. We constructed a final model to estimate the associations of interest, adjusting for the confounding effects of other known CVD risk factors and the covariates considered in the previous model. In addition to age, sex, and education, the CVD risk factors we adjusted for were diabetes, high blood pressure, smoking history, and a lifetime alcohol abuse/dependence diagnosis. We also examined whether a diagnosis of major depression modified the association between PTSD and CVD. Adjusted prevalences of CVD were computed after jointly stratifying by PTSD and major depression status (CVD was not reported by any depressed participant who never smoked; therefore, the CVD prevalences were adjusted

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Table 1. *Participant characteristics by lifetime PTSD and major depression*

| Characteristic | Lifetime PTSD | | | | Lifetime depression | | | |
|---|---|---|---|---|---|---|---|---|
| | Yes | (95% CI) | No | (95% CI) | Yes | (95% CI) | No | (95% CI) |
| Demographic | | | | | | | | |
| Age, mean years (S.D.) | 37 | (36–38) | 34 | (34–34) | 37 | (35–38) | 34 | (34–35) |
| Female, % | 69 | (63–75) | 48 | (47–49) | 60 | (51–69) | 50 | (49–51) |
| Attended ⩾12 years of school, % | 78 | (71–83) | 78 | (75–80) | 79 | (70–86) | 78 | (75–80) |
| Clinical | | | | | | | | |
| Diabetes, % | 14 | (10–19) | 9 | (7–10) | 14 | (9–22) | 9 | (8–11) |
| High blood pressure, % | 23 | (18–29) | 16 | (14–18) | 29 | (21–37) | 16 | (14–18) |
| Smoking status, % | | | | | | | | |
| Never | 21 | (16–27) | 31 | (29–34) | 19 | (12–28) | 31 | (28–34) |
| Former | 18 | (13–24) | 21 | (18–23) | 23 | (16–32) | 20 | (18–23) |
| Current | 61 | (54–67) | 48 | (45–51) | 58 | (49–67) | 49 | (46–52) |
| Lifetime psychiatric diagnoses | | | | | | | | |
| Major depression, % | 23 | (18–30) | 6 | (4–7) | — | — | — | — |
| PTSD, % | — | — | — | — | 41 | (32–51) | 12 | (11–14) |
| Alcohol abuse/ dependence, % | 61 | (54–67) | 31 | (29–34) | 69 | (60–77) | 33 | (30–35) |

PTSD, Post-traumatic stress disorder; CI, confidence interval.

for all risk factors listed above except smoking history). Formal testing for effect modification of major depression on the PTSD-CVD association was accomplished via logistic regression and adjusted for all CVD risk factors. All statistical tests were two-sided adjusted Wald tests. Analyses were conducted in STATA 8.1 for Windows (Stata Corporation, College Station, TX, USA) using 'SVY' commands to accommodate the weights for complex sampling and survey non-response.

## RESULTS

### Participant characteristics

The average age of the 1414 participants was 35 years, slightly more than half were female (51%), and the majority had at least 12 years of education (78%). Overall, 6% of the total sample reported a history of CVD. Self-reported rates of diabetes and high blood pressure were 9% and 17% respectively. Half of the participants were current smokers and an additional 20% were former smokers. More than a third of the participants had a diagnosis of lifetime alcohol abuse or dependence (36%). The respective rates of lifetime PTSD and major depression were 15% and 8%, while 3% of participants had both diagnoses. In addition, we examined the association of self-reported CVD with traditional risk factors (data not shown) and found a similar pattern of findings to epidemiological studies utilizing clinically verified CVD status, including male gender, older age, and higher incidences of diabetes, high blood pressure, and current smoking history (American Heart Association, 2005).

### PTSD, major depression, and CVD

Table 1 summarizes demographics, health conditions, and psychiatric diagnoses for participants with a lifetime diagnosis of PTSD and major depression. On average, participants with PTSD were older than those without PTSD, and were more likely to be female. Other established risk factors for CVD, including diabetes, high blood pressure, and current smoking status were all more frequent among those with PTSD. Similarly, the PTSD group was more likely to be diagnosed with lifetime major depressive disorder and alcohol abuse/dependence. The AI-SUPERPFP assessed 16 trauma event categories (Manson *et al.* 2005). The most commonly reported trauma-related experiences among the PTSD group included observed violence between other members of their family (70%), abused physically or hurt by a spouse or partner (54%), witnessed a serious accident or disaster where someone else was hurt very badly or killed (53%), and experienced a life-threatening accident (40%). Participants reporting lifetime depression were older and a higher proportion was female compared to those without depression. Diabetes, high blood

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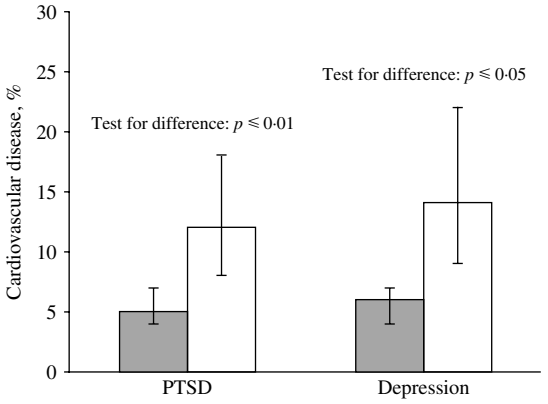


FIG. 1. Unadjusted prevalence of cardiovascular disease and 95% confidence intervals by lifetime PTSD and lifetime major depression. ■, No; □, yes.

pressure, current smoking status, PTSD, and alcohol abuse/dependence all occurred more frequently among participants with a diagnosis of major depression than those without depression.

Fig. 1 illustrates differences in the prevalence of CVD according to PTSD and major depression. Approximately 12% of the PTSD group reported CVD in contrast to only 5% among their non-PTSD counterparts. Similarly, there was more than a 2-fold increase in the prevalence of CVD among participants reporting a lifetime diagnosis of major depression compared to participants without a depression diagnosis.

Table 2 presents the findings from a series of logistic regression models examining PTSD and major depression with CVD. After adjusting for age, sex, and education in a baseline model, PTSD and CVD were strongly related, with an adjusted OR of 2·4 (95% CI 1·4–4·1). Similarly, after adjusting for age, sex, and education, major depression was strongly related to CVD, with an adjusted OR of 2·6 (95% CI 1·4–4·9). We then fitted a regression model to CVD that included age, sex, education, and both PTSD and depression. In this model, the strength of the association between PTSD and CVD was slightly reduced (OR 2·1) although it remained statistically significant. Similarly, in this model, the OR between depression and CVD was reduced to 2·2 but also remained significant. The last stage of model building adjusted for established CVD risk factors such as age, male gender, socio-economic status, diabetes, high blood pressure, smoking status, and lifetime alcohol abuse/dependence. The association between PTSD and CVD remained stable and unchanged in this fully adjusted model (95% CI 1·1–3·8). However, in the fully adjusted model the OR between depression and CVD was reduced (OR 1·8) and was no longer significant (95% CI 0·9–3·7).

Fig. 2 presents the adjusted prevalence of CVD among those with and without PTSD after stratification by major depression. A higher prevalence of CVD was noted in those with PTSD and major depression compared to those with PTSD without major depression. However, this difference in prevalence was not statistically significant when tested by including a product term between PTSD and depression in the fully adjusted logistic regression model ($p = 0{\cdot}85$).

## DISCUSSION

In this study, we found that 15% of our community-based sample met criteria for lifetime PTSD†, a prevalence similar to the 22% previously reported in another AI community (Robin *et al.* 1997). These rates of PTSD are substantially higher than those observed in other USA minority groups and nearly double the rate found among White Americans (Davidson & Fairbank, 1993; Kessler *et al.* 1995, 2005; Yehuda, 2002). The high prevalence of PTSD is likely attributable to the frequent exposure of AIs to traumatic events, rather than an increased vulnerability to PTSD *per se* (US Department of Health and Human Services, 2001).

We also found that the prevalence of CVD among participants with PTSD was twice that of their non-PTSD counterparts. This association remained significant after controlling for demographic features, traditional CVD risk factors, major depression, and alcohol abuse/dependence. Similarly, participants meeting criteria for major depression experienced a 2-fold increase in the rate of CVD compared to their non-depressed counterparts. In contrast to our

† The actual rate of lifetime PTSD in the present study was 14·7%. A lifetime PTSD prevalence of 14·2% for this same Northern Plains tribal sample has been reported elsewhere (Beals *et al.* 2005). This difference is due to the present study using only those tribal participants between the ages of 18 and 57 years, whereas the Beals *et al.* (2005) study reported rates for those aged 15–57 years.

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Table 2. *Adjusted odds ratios for cardiovascular disease with lifetime PTSD and major depression*

| | Baseline model[a] | | Model adjusted for other diagnosis[b] | | Fully adjusted model[c] | |
|---|---|---|---|---|---|---|
| Lifetime diagnosis | OR | (95% CI) | OR | (95% CI) | OR | (95% CI) |
| PTSD | 2·4 | (1·4–4·1) | 2·1 | (1·2–3·7) | 2·0 | (1·1–3·8) |
| Major depression | 2·6 | (1·4–4·9) | 2·2 | (1·1–4·1) | 1·8 | (0·9–3·7) |

OR, odds ratio; CI, confidence interval; PTSD, post-traumatic stress disorder.
[a] Adjusted for sex, age, and education; [b] adjusted for age, sex, education, lifetime PTSD or lifetime major depression; [c] adjusted for age, sex, education, lifetime PTSD or lifetime major depression, diabetes, high blood pressure, smoking, and lifetime alcohol abuse/dependence.

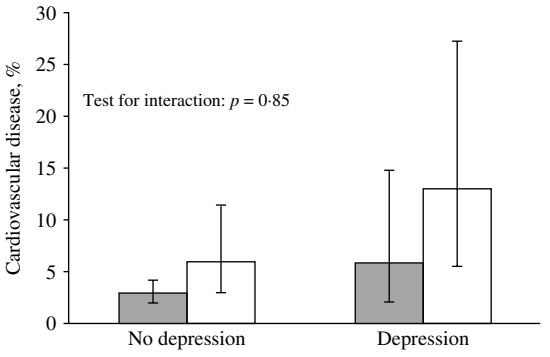


FIG. 2. Joint effects of lifetime PTSD and lifetime major depression on the prevalence of cardiovascular disease and 95% confidence intervals adjusted for age, sex, education, lifetime alcohol abuse/dependence, diabetes, and high blood pressure. ▩, No PTSD; □, PTSD.

PTSD findings, the major depression-CVD association was not significant after adjusting for demographic, traditional CVD risk factors, PTSD, and substance abuse/dependence diagnosis. Of note, our population of inference has less variability than other racial/ethnic groups in socio-economic status and education level, variables that profoundly influence the risk of CVD (Steenland *et al*. 2004), PTSD (North & Smith, 1992), and major depression (Yen & Kaplan, 1999). In addition, subsyndromal depressive symptoms also confer risk for coronary artery disease (Rudisch & Nemeroff, 2003) and the severity of depression has been related to the likelihood of cardiac events in a dose-dependent fashion (Wassertheil-Smoller *et al*. 1996; Pennix *et al*. 2001). Because our structured interview assessed the presence or absence of lifetime PTSD and lifetime major depression in a categorical fashion, the association of subsyndromal PTSD or depression symptoms and CVD is unclear. To better understand the PTSD-CVD relationship, and whether it changes in a dose-dependent manner, future studies should include dimensional measures of PTSD symptom severity as well as the frequency of traumatic events. Nonetheless, in this AI sample, when compared to major depression, lifetime PTSD emerged as the psychiatric illness more strongly related to CVD.

The prevalence of lifetime major depression in our AI sample is lower in comparison to other population estimates (16·2%; Kessler *et al*. 2003). Considerable efforts were taken during the early stages of the AI-SUPERPFP to develop a culturally sensitive, DSM-IV consistent diagnostic measure of major depression. Beals and colleagues (2005, in press *a*) offer three possible explanations for the lower observed estimates of lifetime major depression. First, respondents may have endorsed fewer symptoms of depression to meet CIDI-defined lifetime diagnostic criteria due to differences in the cultural interpretation of those symptoms. Second, the higher rates of PTSD may be reflective of the tendency to attribute symptoms of distress to trauma as opposed to depression. Similarly, it is possible that the higher prevalence of alcohol abuse/dependence may have partially masked otherwise symptomatic levels of major depression. Third, the AI-SUPERPFP interview relied on tribal peers to administer the CIDI in a structured, standardized format. While this method is a noted strength in conducting epidemiological studies with the AI community, it does preclude the lay interviewer from offering clarifications and probing for additional information. Previous epidemiological studies yielding higher prevalence rates of depressive disorders in AI samples have used trained mental health professionals who were encouraged to clarify diagnostic interview questions (e.g. Kinzie *et al*. 1992). Qualitative research efforts may help clarify culture-specific

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meanings of depression among AI respondents, and how these symptoms may overlap with or be distinct from symptoms associated with PTSD and substance-use related disorders.

The depression-CVD relationship was no longer nominally significant in the final adjusted model. While the point estimate of the association is reduced in the adjusted analysis, it still is elevated and a suggestive, clinically relevant finding. In addition, AIs with both PTSD and depression had a prevalence of CVD in excess of what would be expected under a multiplicative model. This suggests a possible interaction between PTSD and depression, but the interaction effect was not statistically significant in the regression model. The lack of significance may be because only a small percentage of participants were diagnosed with both lifetime PTSD and depression. Future studies employing a larger sample may also allow for a more definitive examination of whether major depression and co-morbid psychiatric conditions are associated with CVD likelihood in a dose-dependent manner.

A substantial body of research has documented diverse physiological effects associated with PTSD (Bremner, 1999, 2002). Although biological mechanisms underlying the PTSD and CVD relationship are speculative, neurohormonal alterations, autonomic cardiac rhythm disturbances, hemostatic dysregulation, and inflammation are candidates for further inquiry. For example, neurohormonal alterations may accelerate vascular damage (Remme, 1998) and hyperactivity of the hypothalamic–pituitary–adrenal axis with changes in plasma catecholamines has been found among Vietnam-era veterans with PTSD (Bremner, 1999). Ventricular arrhythmias and reduced heart rate variability are risk factors for mortality among patients with CVD (Curtis & O'Keefe, 2002). PTSD patients appear to have lower heart rate variability at rest compared to their non-PTSD counterparts (Cohen *et al.* 1997), but the evidence for autonomic rhythm disturbances in major depression is stronger (Joynt *et al.* 2003). Hypercoagulation, an indicator of hemostatic dysregulation, is important in the pathophysiology of CVD. Although platelet activity may be abnormal in depression (Mendelson, 2000), these processes have not been examined in PTSD. Lastly, pro-inflammatory cytokines have been implicated in CVD pathogenesis (Koenig, 2001). PTSD has been associated with higher concentrations of inflammatory markers, including C-reactive protein, interleukin-6, and interleukin-1 (Maes *et al.* 1999), as well as elevated leukocyte and total T-cell counts (Boscarino & Chang, 1999). Taken together, these mechanisms may explain, in part, the positive relationship noted in previous investigations of PTSD and the cardiovascular system (McFarlane *et al.* 1994; Wolfe *et al.* 1994; Boscarino, 1997; Boscarino & Chang, 1999; Schnurr *et al.* 2000). A possibility exists, however, that continued refinements in our ability to study the biological effects of PTSD and depression on the vascular system may reveal more shared than distinctive neurohormonal, autonomic nervous system processes. The constellation of anxiety, depression, and anger as a dispositional indicator of negative affectivity is emerging as an important construct in understanding cardiovascular health (Suls & Bunde, 2005). Furthermore, the impact of overall psychiatric burden, especially those conditions more associated with chronic (e.g. PTSD and major depression) as opposed to acute (e.g. panic and phobias) stress, may be helpful toward understanding CVD risk above specific, individual psychiatric disorders.

This study has several notable strengths as well as limitations. We used a structured, diagnostic interview for PTSD rather than symptom scores, recruited a population-based sample that included women, focused on a minority group, considered a broad range of traumatic events, and controlled for the effects of lifetime major depression and alcohol abuse/dependence. However, a number of limitations suggest a cautious interpretation of our findings. First, the cross-sectional design does not allow us to determine the direction of the PTSD-CVD association. Prospective studies are crucial to determine if PTSD is a risk factor for incident CVD. Similar studies have demonstrated that major depression predicts both the onset and prognosis of CVD (Rudisch & Nemeroff, 2003). Second, like most previous investigations, we relied solely on a self-reported diagnosis of CVD. Because persons with PTSD may be more likely to report poorer health than those without PTSD (McFarlane *et al.* 1994; Wolfe *et al.* 1994; Boscarino, 1997; Schnurr *et al.* 2000), response

bias cannot be completely ruled out. Some studies suggest self-reported and medically diagnosed health conditions are poorly correlated (McFarlane *et al.* 1994). However, the agreement between self-reported and objectively documented myocardial infarction and stroke is among the most accurate given the fairly dramatic onset, associated pain, and involvement in more intensive in-patient and out-patient follow-up care for cardiovascular events (Okura *et al.* 2004). Nonetheless, objective clinical assessment of CVD is needed in future investigations. Third, it is possible that the higher number of CVD risk factors endorsed by those reporting a CVD history is reflective of a general tendency to endorse a range of physical symptoms and somatic complaints during the interview. The need for objective assessment of CVD risk factors (e.g. body mass index, high blood pressure, diabetes) is underscored. Fourth, the role of protective factors in moderating the PTSD-CVD and major depression-CVD relationships was not investigated. Protective factors that reduce disease likelihood or manifestations, such as spirituality and social support, may be important variables to assess in AI communities (Beals *et al.* in press *b*). Lastly, our results cannot be generalized to all AIs. Although the tribes that participated in this study are among the largest in the USA, future efforts to reproduce our findings should include geographically and culturally diverse tribal communities.

In conclusion, little information exists about the relationship between PTSD and CVD in general, and even less is known about this association in women, minorities, and community samples. Previous studies have not examined the PTSD-CVD or major depression-CVD relationship among AIs. Yet, traumatic experiences are common among AI people (Beals *et al.* 2005), and the 'rising tide of CVD' (Howard *et al.* 1999) is of great concern to tribal communities. In this regard, data from a large epidemiological study of cardiovascular risk factors document that CVD is now the leading cause of death among AI men and women in two out of three geographically diverse tribes (Lee *et al.* 1990). Both from a clinical and research perspective, increased attention to the physical health consequences of PTSD and major depression is needed. The development of better primary and secondary disease management programs also rests with identifying protective factors linked to positive PTSD, major depression, and CVD health outcomes.

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## DECLARATION OF INTEREST

None.

## APPENDIX. The AI-SUPERPFP Team

In addition to the authors of this paper, the AI-SUPERPFP team includes Cecelia K. Big Crow, Buck Chambers, Michelle L. Christensen, Denise A. Dillard, Karen DuBray, Paula A. Espinoza, Candace M. Fleming, Ann Wilson Frederick, Joseph Gone, Diana Gurley, Lori L. Jervis, Shirlene M. Jim, Carol E. Kaufman, Ellen M. Keane, Suzell A. Klein, Denise Lee, Monica C. McNulty, Denise L. Middlebrook, Laurie A. Moore, Tilda D. Nez, Ilena M. Norton, Douglas K. Novins, Theresa O'Nell, Heather D. Orton, Carlette J. Randall, Angela Sam, James H. Shore, Sylvia G. Simpson, Paul Spicer, and Lorette L. Yazzie.

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