Ethnic Differences in Posttraumatic Distress: Hispanics' Symptoms Differ in Kind and Degree

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Abstract

Objective—This longitudinal study of physical injury survivors examined the degree to which Hispanic and non-Hispanic Caucasians reported similar PTSD symptoms.

Methods—Adult physical trauma survivors (N = 677) provided information regarding posttraumatic distress by completing an interview-administered version of the PTSD Symptom Checklist (Civilian version) at three timepoints: within days of trauma exposure, and again at 6- and 12-months posttrauma. Structural equation modeling with propensity weights was used to analyze data.

Results—Results replicated prior research indicating that Hispanics report greater overall PTSD symptom severity. However, the size of this effect varied significantly across the 17 individual PTSD symptoms, and several symptoms were not reported more highly by Hispanics. Relative to non-Hispanic Caucasians, Hispanics tended to report higher levels of symptoms that could be regarded as exaggerated or intensified cognitive and sensory perceptions (e.g., hypervigilance, flashbacks). In contrast, few differences were observed for symptoms characteristic of impaired psychological functioning (e.g., difficulty concentrating, sleep disturbance).

Conclusions—Findings suggest that the pattern of PTSD symptoms experienced most prominently by Hispanics differs in kind and not merely in degree. Results have implications for theory aimed at explaining this ethnic disparity in posttraumatic psychological distress as well as for clinical intervention with trauma-exposed Hispanics at risk for PTSD.

Keywords

posttraumatic stress disorder; Hispanics; ethnic disparities; structural equation modeling

Are Hispanics more likely than their non-Hispanic Caucasian counterparts to experience severe symptoms of posttraumatic stress disorder (PTSD) following exposure to trauma? A wide range of research studies suggests that they do, including investigations of individuals exposed to military combat experience (Kulka et al., 1990; Schell & Marshall, 2008), civilian occupational trauma (Pole et al., 2001), natural disaster (Norris, Perilla, & Murphy, 2001; Perilla, Norris, & Lavizzo, 2002) and terrorist attack (Galea et al., 2004). For a review of these findings, see Pole, Gone, and Kulkarni (2008).

In their study of nearly 1200 Vietnam combat veterans, for example, Kulka et al. (1990) reported that Hispanics were twice as likely as their non-Hispanic Caucasian counterparts to

meet criteria for current PTSD. Ethnic differences remained large and statistically significant even after controlling for extent of exposure to combat. Similarly, in research on 655 police officers, Pole et al. (2001) found that individuals of Hispanic descent reported more severe symptoms of PTSD than did non-Hispanic Caucasians even after adjusting for several covariates.

This line of research suggests the existence of an ethnic health disparity. Yet, core issues about the nature of this disparity remain unaddressed. Specifically, whereas it appears that Hispanics exhibit greater PTSD symptoms in the aggregate, virtually no research exists as to the nature or scope of the differences at the level of individual PTSD symptoms. Although one study has examined differences at the level of the symptom cluster (Ortega & Rosenheck, 2000)[1], confining analyses to overall PTSD symptom severity or even to specific symptom clusters hampers recognition of how these aggregate differences are manifested at the level of the individual PTSD symptom.

Understanding how Hispanics differ from their non-Hispanic Caucasian counterparts in the manifestation of individual symptoms of PTSD has important theoretical and practical implications. Numerous explanations of the observed ethnic disparity in the manifestation of posttraumatic distress have been offered. These accounts include a culturally-based propensity to exaggerate or overreport mental health symptoms (Ruef, Litz, & Schlenger, 2000; Ortega & Rosenheck, 2000), a disposition toward acquiescent responding (Ortega & Rosenheck, 2000), and the tendency of Hispanics to manifest distress in physical rather than psychological form (Hough, Canino, Abueg, & Gusman, 1996). Other explanations include differential exposure to traumatic life events (Frueh, Brady, & de Arellano, 1998), ethnic discrimination (Marsella, Friedman, & Spain, 1996), differences in coping resources following trauma exposure (Pole et al., 2005), or sociodemographic disadvantage (Pole et al., 2008).

Whether any of these hypotheses is ultimately borne out, it appears evident that some of these accounts would lead to different expectations about how symptoms of PTSD would be expressed in Hispanics. For example, if elevated symptom levels were attributable to a cultural propensity to more freely report psychological distress (Ortega & Rosenheck, 2000), then one might expect to find higher levels of endorsement of all PTSD symptoms. In this scenario, the Hispanic difference might be one of degree rather than kind, with Hispanics and non-Hispanic Caucasians showing a similar pattern of PTSD symptoms.

By contrast, if differences in overall PTSD symptoms were due to the tendency for Hispanics to endorse somatic, rather than psychological, symptoms (e.g., Hough, Canino, Abueg, & Gusman, 1996), then it might be anticipated that this predisposition would be evidenced in endorsement of certain symptoms rather than others. Specifically, Hispanics might be predicted to endorse greater physical reactivity to trauma reminders and more exaggerated startle responses relative to their non-Hispanic Caucasian counterparts. However, differential endorsement of physical symptoms would not appear to predict differences in symptoms that reflect more psychological reactions to trauma. Increased knowledge regarding ethnic differences in the symptomatology of PTSD is critical to the efforts to build theory explaining the origins of ethnic differences in risk for PTSD. In essence, the phenomenon that these theories attempt to explain has not yet been well described.

Elucidating the phenomenology of these ethnic differences would also have significant clinical ramifications. If PTSD symptoms in Hispanics were observed to differ in kind rather than

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[1]Ortega and Rosenheck (2000) conducted a cluster-level analysis, finding that Hispanics reported slightly elevated levels of reexperiencing and hyperarousal clusters. Yet, these analyses provide no information about which of the 10 symptoms show ethnic differences. Moreover, the absence of cluster-level differences in avoidance or numbing symptoms does not preclude the possibility that significant differences exist with respect to individual symptoms of avoidance or numbing.

merely in degree, then modifications in treatment may be required to ensure the delivery of culturally competent care. Questions might reasonably be raised about whether the currently available interventions are appropriate for Hispanics, how existing therapies might be tailored to the specific needs of this group, and even whether novel interventions must be developed and evaluated. Similarly, outreach and psychoeducational programs might need to be crafted to emphasize the unique features of posttraumatic distress as it is experienced by Hispanic trauma survivors. In addition, providers may need to be educated to increase cultural competence so that treatment effectiveness can be maximized.

The current investigation examined ethnicity-related symptom differences in the context of a prospective study of the natural course of posttraumatic distress following sudden physical injury requiring hospitalization. Research documents that traumatic physical injury requiring hospitalization is associated with posttraumatic distress and PTSD in the months following acute inpatient care (e.g., G. Marshall & Schell, 2002; O'Donnell, Creamer, Pattison, & Atkin, 2004; Zatzick et al., 2008), thus attesting to the relevance of this population to the research questions of interest.

This investigation examined the main effect of ethnicity across all symptoms as well as the interaction between ethnicity and specific symptoms. The study was guided by prior empirical research indicating that Hispanics report more severe symptoms of PTSD overall as well as theory which implies that Hispanics manifest significant differences across all individual PTSD symptoms (e.g., Ortega & Rosenheck, 2000; Ruef et al., 2000). The research focused on two questions: (1) do Hispanics report higher overall levels of PTSD symptoms than do their non-Hispanic Caucasian counterparts?; and, if so (2) do Hispanics manifest uniform differences across all 17 PTSD symptoms relative to non-Hispanic Caucasians? Finally, few, if any, investigations have compared the PTSD symptoms of Hispanics to African Americans with research almost always treating non-Hispanic Caucasians as the conceptual reference category. In part due to the lack of descriptive research, there are no theoretical predictions for the comparisons between Hispanics and African Americans. However, we conducted exploratory analyses to foster research on this generally neglected topic.

Method

Participants

The sample consisted of Hispanic (N = 330), non-Hispanic Caucasian (N = 135), and African American (N = 171) survivors of sudden physical injury who required hospitalization for acute medical care. Participants who indicated another ethnic group as their sole or main group were assigned to an "other ethnicity" group (N = 41) and excluded from further analyses. The sample of interest averaged 33.3 years of age (SD = 11.72), and was largely male (77.6%). Close to half of participants were either married (29%) or living as married (20.0%). The highest grade completed by most participants was high school or its equivalent (49%). Participants averaged $12,961 in income in the six months preceding the injury (SD = $15,637). The overall analytic sample from which the sample of interest was drawn did not differ significantly from the LA County census of trauma center patients with respect to age, gender, ethnicity and mechanism of injury (i.e., assault versus motor vehicle collision/other accident). The overall sample had, however, sustained slightly more severe injuries than had the trauma center population (Ramchand et al., 2009).

Procedures

The sample was recruited between February 2004 and August 2006 from four trauma centers in Los Angeles County: Los Angeles County + University of Southern California Medical Center (LAC+USC), UCLA Medical Center, King-Drew Medical Center, and California

Hospital Medical Center. Patients were eligible for the study if they met the requirements for the Los Angeles County Trauma registry. The purpose of the registry is to facilitate monitoring of individuals who suffered potentially life-threatening injuries that required hospitalization and surgical intervention. Trauma patients were ineligible to participate, if they (1) could not communicate in English or Spanish; (2) had been incarcerated at hospital admission; (3) had been homeless prior to hospitalization and expected to be homeless upon discharge; (4) had cognitive impairment that precluded informed consent; or (5) had experienced injuries from family violence or attempted suicide.

Recruitment followed a two stage procedure that was intended to yield a sample representative of hospitalized trauma survivors in Los Angeles county: stage one identified admitted patients thought to be eligible for the study and stage two used a face-to-face screening interview to verify eligibility. Different methods for identifying potentially eligible individuals were used at the four hospitals due to different types of medical records and IRB-approved protocols. At LAC+USC, research staff had direct access to computerized admission records. At the remaining three trauma centers, medical personnel notified research staff of the presence of individuals who appeared to meet study eligibility requirements whereupon research staff conducted screening interviews.

Interviewers attempted to screen and consent all patients identified as potentially eligible, with one exception: the research staff approached every other Hispanic patient at LAC+USC, determined by the order of admission. This random sampling strategy adjusted for the known overrepresentation of Hispanics at this trauma center relative to the overall population served by the Los Angeles county trauma service. Patients identified as likely to meet eligibility requirements ($N = 1133$) were then screened in a face-to-face interview to assess eligibility criteria more completely. Of those screened, 10.3% were excluded because they were unable to converse in either English or Spanish, 10.0% because they were homeless, 2.4% because the injuries were due to an attempted suicide, 1.4% because injuries were caused by family violence, and 0.9% because they had a cognitive impairment that prevented informed consent or understanding the interview. Eighty-nine percent of attempted screenings were completed.

Participating individuals were asked to complete three face-to-face structured interviews. The first was conducted within days of hospitalization; the second was conducted at 6-month follow-up; and a third was completed at 12-month follow-up. Of the 850 patients identified as eligible for the larger study based on screening, 677 (80%) completed the baseline interview at a median of 9 days following the trauma. Of the 677 who completed an initial interview, 476 (70%) completed 6-month follow-up interviews and 462 (68%) completed 12-month follow-up interviews.

Interviews were completed in English or Spanish, as needed. All participants provided informed consent and all study procedures were approved and monitored by the Institutional Review Boards of the RAND Corporation, LAC+USC Medical Center, UCLA Medical Center, King-Drew Medical Center, and California Hospital Medical Center.

Measures

PTSD symptoms—The civilian version of the Posttraumatic Stress Disorder Checklist (PCL Weathers et al., 1993) was used to assess for PTSD symptom severity at all three assessments. The PCL is a 17 item measure of PTSD symptoms in which participants rate the degree to which they were bothered by each symptom. Responses were provided on a scale ranging from 1 (*not at all*) to 5 (*extremely*), yielding a theoretical range of 17 to 85. The 17 PCL items map directly onto the DSM-IV symptom criteria for PTSD (American Psychiatric Association, 2000), assessing *reexperiencing* (Criterion B; five symptoms), *avoidance* and *numbing* (Criterion C; seven symptoms, and *hyperarousal* (Criterion D; five symptoms). Symptoms

were assessed with regard to the injury (e.g., "how much have you been bothered by repeated, disturbing dreams of *the injury*"). At the initial interview, participants answered items with respect to the time since the injury; at follow-up assessments, answers were provided with respect to the past two weeks.

The PCL has been used in diverse samples and possesses solid psychometric properties (Andrykowski, Cordova, Studts & Miller, 1998; Blanchard, Jones-Alexander, Buckley & Forneris, 1996; Denson, Marshall, Schell, & Jaycox, 2007). Previous research has demonstrated that Spanish and English versions of the PCL are psychometrically equivalent (Miles, Marshall, & Schell, 2008). Specifically, no evidence was found of differential item function or an overall scoring bias across the two versions. The PCL summed scores for the three assessment indicated a relatively high level of traumatic distress (M's = 39, 38 and 36; SD's = 16, 19 and 18; for the baseline, 6-month and 12-month assessments, respectively, in the weighted analytic sample).

For descriptive purposes, we used PCL screener responses to estimate the percentage of the sample who met symptom criteria for PTSD using the procedure recommended by Weathers et al. (1993). Specifically, individual symptoms receiving a score of 3 (*moderately*) or greater were treated as indicating symptom presence and mapped against the *DSM–IV* criteria B–D. Following this scoring strategy, 29.5% of participants met screening criteria for PTSD at the initial assessment (excluding duration), whereas 33.0% and 27.8% met criteria at the 6- and 12-month follow-up assessments, respectively.

Ethnicity—Respondents were asked about race and ethnicity using standard census questions, i.e., they were asked separately about being of Hispanic or Latino ethnicity (yes/no) and their race (multiple categories). Respondents were allowed to indicate multiple racial categories. For those who indicated multiple race/ethnicity categories (e.g., Hispanic and White/ Caucasian), we asked "Which of the racial or ethnic groups you selected do you consider your main group?" For the purposes of the current analyses, the latter question was used to place respondents into a single category when multiple categories were selected. An individual who self-identified, for example, as both Hispanic and another group but whose primary self-identification was Hispanic was assigned to the Hispanic group. Using this classification framework, participants were categorized as either Hispanic (N = 330), non-Hispanic Caucasian (N = 135), African-American (N = 171) or another group (N = 41). The latter group included Asians and Pacific Islanders, Native American, and Asian Indian participants. Of the 330 Hispanic participants, 70% (N = 230) were of Mexican geographic origin; 18% (N = 60) and 12% (N = 40) were of Central American and South American ancestry, respectively. These proportions of Hispanic subgroups are generally reflective of the composition of Hispanics residing in Los Angeles, and are similar to the broader US Hispanic population. However, the current sample under represents Hispanics of Caribbean origin, which constitute approximately 13% of Hispanics in the US (US Census Bureau, 2008).

Injury Mechanism—Injury mechanism was assessed by self-report, and classified as either: motor vehicle collisions (61%); injuries stemming from assaults (e.g., having been shot with a gun, stabbed with a knife) (43%); or other types of injuries (9%).[2] For analytic purposes, we distinguished assault from other causes of injury.

Loss of Consciousness—After relating the incidents that resulted in their injuries, respondents were asked, "Did you lose consciousness during the event?"

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[2]Because a small number of individuals suffered injuries from multiple causes, the total percentage exceeds 100.

Injury Type and Severity—Several objective indicators of injury type and severity were taken from hospital records, including: *length of hospital* stay (assessed in days from admission to discharge), *Injury Severity Score* (ISS, American Association for the Advancement of Automotive Medicine, 1990; Baker, O'Neill, Haddon, & Long, 1974), and *Glasgow Coma Score* (Teasdale & Jennett, 1974).

The ISS serves as an overall injury severity score. Injuries to each of six body regions (i.e., head, face, chest, abdomen, extremities, external) are assigned a score ranging from 1 "Minor" and 5 "Critical". Scores for the three most severely injured body regions are squared and summed to produce the ISS score (Baker et al., 1974). Thus, the ISS incorporates information concerning both the site and extent of injuries into a single score ranging from 1 to 75, with higher scores reflecting lower probability of survival. For this study, ratings were made by medical center trauma nurses and were abstracted from medical records. The mean ISS score for this sample was 9.66 (*SD* = 7.42). A score of 9 is considered to be of mild severity from the standpoint of mortality prediction (American Association for the Advancement of Automotive Medicine, 1990).

The Glasgow Coma Score (GCS) is widely used to quantify level of consciousness following traumatic brain injury. The scale measures the ability to respond to verbal commands in three areas: eye, verbal and motor. Spontaneous responses received the highest possible score (e.g., opens eyes spontaneously) while failure to respond results in the lowest possible score. Scores for each of the three areas are summed to yield an overall score, ranging from 3 to 15 (Teasdale & Jennett, 1974). A score of 3 corresponds to a person in a coma whereas a 15 corresponds to a fully awake and responsive person (Teasdale & Jennett, 1974). In this sample, 84% of participants received scores of 15; 9.5% received scores of 14; and 6.7% received scores below 14. Level of consciousness was assessed by medical center personnel immediately upon arrival to the trauma center. Scores were obtained from medical records.

Data Analysis

Propensity Weighting—To make meaningful comparisons across ethnic groups, it is important to ensure that observed ethnic differences are not attributable to incidental differences between groups. As an initial step prior to estimating the ethnic effect of interest, we desired to create analytic groups that were matched with respect to trauma type, trauma severity, and demographic characteristics other than those that might be products of ethnicity (Rosenbaum & Rubin, 1983).[3]

One way to achieve equivalent groups is by propensity weighting (e.g., Hirano, Imbens, & Ridder, 2003; Farley et al., 2002). Using this approach, individuals in one group who are most similar to the individuals in the comparison group are given increased weight in the analyses. Although individual respondents are not directly matched to one another across groups, this procedure can result in groups that, when weighted, have equivalent distributions on the background variables. We used the estimation method outlined in McCaffrey, Ridgeway and Morral (2004) to derive propensity weights. This method is based on Generalized Boosted Models which is a powerful regression technique that does not require assumptions about linearity or additivity in the relationship between the covariates and ethnicity. We derived weights to match the three ethnic groups on the following covariates as well as on all two-way interactions among these covariates: ISS, length of hospitalization, GCS, loss of consciousness, mechanism of injury, age, and gender.

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[3]We did not control for SES or related constructs because these factors might plausibly be regarded as consequences of ethnicity. To the extent that ethnicity might influence SES, controlling for SES might introduce bias in the estimation of the effect of ethnicity (Campbell & Kenny, 1999).

As shown in Table 1, the weights were effective at creating groups that were balanced on these factors, i.e., the differences that remained were similar in magnitude to those expected if participants had been randomly assigned to ethnicity. Prior to weighting, these potentially confounding variables were highly associated with ethnicity: a multinomial logistic regression gave a statistically significant effect of $\chi^2 (7) = 44$, $p < 0.001$, with pseudo-$R^2 = 0.06$ and $c = 0.62$. With weights applied, $\chi^2 (7) = 5.8$, $p = 0.604$; pseudo-$R^2 = 0.01$, $c = 0.50$, which is comparable to the balance expected when group membership is randomly assigned.

Modeling Approach—We implemented structural equation modeling as our general analytic strategy to assess the relationship between PTSD symptoms and ethnicity. A longitudinal model was fit to all three assessments. In this model, the variance of each symptom at each time can be partitioned into two meaningful components: variability across particular PTSD symptoms that is stable across the three assessments, and variability across time that is stable across the 17 symptoms. Thus, each symptom at each assessment is modeled as a linear combination of two factors: a symptom-specific factor and a time-specific factor. The resulting model has a separate factor for each of the 17 symptoms that measures participants' overall level of each symptom across time. In addition, three time factors measured the participants' overall level of symptoms at each assessment across the various symptoms.

We refer to this representation as a *multi-symptom, multi-time model*. This type of longitudinal model is discussed in Scherpenzeel and Saris (2007) as having identification conditions similar to latent variable multitrait-multimethod (MTMM) models, although with substantively different factor interpretations. This multi-symptom, multi-time model can also be seen as a particular type of hierarchical or multi-level model (Curran, 2003; Raudenbush, 2001). Specifically, a model in which each level-one observation is cross-categorized on two level-two random variables nested within individuals, one representing the 17 symptom clusters and the other representing the 3 time clusters.

The model was identified by constraining the 17 symptom factors to be orthogonal to the three time-specific factors, and the three time-specific factors to be orthogonal to one another. No constraints were placed on the means or intercepts of the latent variables or on the correlations among the 17 symptom factors. In addition, several a priori constraints were imposed to improve interpretability of the model: the loadings of each measured symptom on its time-specific factor were constrained to be equal to 1; the loadings of symptoms from time 2 and 3 on the appropriate symptom factors were constrained to be equal to 1; and the loadings of Time 1 symptoms on the appropriate symptom factors were constrained to be equal across all symptoms.

Thus, when two groups have different means on a latent variable, it can generally be interpreted as their average difference on that factor's observed indicators. For example, if Hispanics had a mean that was 1 point higher than non-Hispanic Caucasians on the latent factor for Time 1, then it implies that Hispanics averaged 1 point higher than non-Hispanic Caucasians on each of the 17 symptoms measured at Time 1. Finally, the model was estimated as a means model in which the intercepts of the 51 measured symptoms (i.e., 17 symptoms at three occasions) are constrained to be zero while the means of the latent variables are freely estimated. Thus, the structural equation model explains the means, variances and covariances of all observed symptoms at all time points.

As noted earlier, we were primarily interested in assessing two primary research questions: (1): Do Hispanics report higher overall levels of symptoms of posttraumatic distress than do their non-Hispanic Caucasian counterparts?; and, if so (2) do Hispanics manifest uniform differences across all 17 PTSD symptoms relative to non-Hispanic Caucasians? Secondarily, we also investigated whether differences between Hispanics and African-Americans existed

with respect to both overall level of distress and the pattern of differences across individual PTSD symptoms. The relationship between ethnicity and the three time factors, which document possible ethnic differences in symptom trajectories, is not discussed in the existing literature. Rather than assume that ethnic differences in symptom profiles did not differ over time, we explicitly tested for the existence in ethnic differences on the three time factors. These tests revealed no significant differences in aggregate symptom change over time across ethnic groups ($p = .89$) while controlling for the effects of ethnicity on the symptom factors. Thus, these paths were excluded from the model used in the study.

Covariates—Although the propensity weighting methods created ethnic groups that were well balanced across the various covariates, they do not necessarily result in a perfect match, i.e., covariates may not be fully independent of the predictor of interest. This issue can be addressed by using doubly-robust estimation (Robins, Rotnitsky & Zhao, 1994; Robins & Rotnitsky, 2001; Emsley, Lunt, Pickles & Dunn, 2008) in which potential confounding variables are entered into the regressions as covariates after propensity weighting.

To achieve doubly-robust estimation, latent variables were regressed onto the 7 covariates listed in Table 1. To maximize the efficiency of the parameter estimation as well as the parsimony of the model, unnecessary model parameters were removed. The effect of a covariate on a given symptom or time factor was set to zero when doing so was more parsimonious than allowing free estimation. The model was pruned by removing paths iteratively in a reverse-stepwise manner. All remaining effects of covariates have non-significant p-values (i.e., $p < .25$). The use of this p-value criterion results in less pruning than would occur using either the AIC or BIC as the pruning criteria.

Model Estimation and Testing—To account for sample attrition, we conducted all analyses using expectation maximization (EM) parameter estimates (McLachlan & Krishnan, 1996). EM avoids sample biases that can occur when cases are omitted from the analysis for having missed at least one follow-up interview (Little & Rubin, 1987). When data are either missing at random or missing completely at random, EM is an unbiased method for increasing inferential power.

All parameters were estimated using Robust Maximum Likelihood (MLR) methods in Mplus 5.2 (Muthen & Muthen, 2006). These parameter estimates and their standard errors are robust to deviations from normality, with the standard errors derived with sandwich estimation (Huber, 1964). When testing the relative model fit across nested models, we used the Yuan-Bentler T2* test statistic (Yuan, Marshall, & Bentler, 2002; implemented in Mplus as MLR estimation), the difference in χ^2 statistics was adjusted using the method proposed by Satorra (2000; see also Satorra & Bentler, 1999).

When assessing overall model fit, we used the TLI, the CFI (Bentler, 1990), the RMSEA (Steiger & Lind, 1980), and the SRMR (Bentler, 1995) fit indices. We considered CFI or TLI values of .90 and greater to reflect adequate fit, and values of .95 and greater to indicate excellent fit. For RMSEA or SRMR, values of .08 or less were regarded as reflecting adequate fit, and values of .05 or less represented excellent fit. To assess the significance of differences between nested models, we used $\Delta\chi^2$ tests with a p value of 0.05. Although there is not universal agreement on what constitutes good model fit (e.g. Marsh, Hau, & Wen, 2004), these criteria are broadly consistent with Hu and Bentler (1999).

Results

The basic multi-symptom, multi-time model with covariates and propensity weights fit the data satisfactorily ($\chi^2 = 2250$, $df = 1470$, TLI = .92; CFI = .93, RMSEA = .028, SRMR = .047). In

this model, each symptom factor was allowed to be independently related to Hispanic ethnicity, and these ethnicity effects were assumed not to vary over time.

A test of the "main effect" of ethnicity on the 17 symptoms was performed by comparing the relative fit of two models: a model in which the effects of ethnicity were constrained to be equal across all 17 symptom factors relative to a model in which all were constrained to be equal to zero. The main effect for Hispanic versus non-Hispanic Caucasian ethnicity across symptoms was significant ($\Delta\chi^2 = 4.86$, $df = 1$, $p = .028$). On average, Hispanics had higher scores than non-Hispanic Caucasians (.17 of a scale point) across all symptoms. This difference was approximately equivalent to a 3 point increase in the PCL summed score. The main effect for Hispanics versus African-Americans across symptoms was not significant ($\Delta\chi^2 = 0.23$, $df = 1$, $p = .63$), and was descriptively small; on average, Hispanics had higher scores than Blacks (.03 of a scale point) across all symptoms. This difference is equivalent to a half-point increase in the PCL summed score.

Each of the main effects was qualified by a substantial interaction for Hispanics relative to non-Hispanic Caucasians. That is, the effects of ethnicity varied across the 17 symptoms. A test of the interaction effect of ethnicity by symptom was performed by comparing the relative fit of the base model, in which each symptom factor is allowed to be independently related to the ethnicity variables, to a model in which the effects of ethnicity were constrained to be equal across groups for all 17 symptom factors. This comparison indicated that the differences between Hispanics and non-Hispanic Caucasians varied significantly in magnitude across symptoms ($\Delta\chi^2 = 49$, $df = 16$, $p < .001$). The differences between Hispanics and African-Americans also varied in magnitude across symptoms ($\Delta\chi^2 = 33$, $df = 16$, $p = .008$), although the latter were smaller in magnitude than those observed between Hispanics and non-Hispanic Caucasians. As shown in Table 2, Hispanics scored significantly higher relative to non-Hispanic Caucasians on 11 of 17 symptoms; no significant differences were observed with respect to 6 symptoms.

The distribution of Hispanic versus non-Hispanic Caucasians effects for individual symptoms ranged from hypervigilance, which was .61 scale points higher for Hispanics relative to non-Hispanic Caucasians, to sleep disturbance, which was .13 scale points higher for non-Hispanic Caucasians as compared to Hispanics. The distribution of effects does not correspond neatly to the DSM-IV PTSD symptom clusters (American Psychiatric Association, 2000). Both the largest and the smallest effects were from DSM-IV cluster D (i.e., Hyperarousal). In addition, the four items with the largest Hispanic difference relative to non-Hispanic Caucasians included two reexperiencing symptoms, one avoidance/numbing symptom, and one hyperarousal symptom. A comparison of African-American and Hispanic responses revealed only a single statistically significant difference, i.e., Hispanics scored significantly higher on item C1 (avoiding thoughts of trauma).

To investigate the role of covariates in these results, we re-ran the models without the propensity weights and without covariates in the models. This analysis generally resulted in slightly larger ethnicity effects. On average, Hispanics had higher scores than non-Hispanic Caucasians, i.e., .22 of a scale point, on all symptoms ($\Delta\chi^2 = 9.2$, $df = 1$, $p = .002$. Differences involving African-Americans and Hispanics were slight .02 points), $\chi^2 (1) = 0.12$, $p = 0.73$). As with the analyses on covariate-equated groups, these overall differences were qualified by the finding that the effect of ethnicity varied significantly across symptoms. Specifically, the differences between Hispanics and non-Hispanic Caucasians varied in magnitude across symptoms ($\Delta\chi^2 = 106.5$, $df = 16$, $p < .001$).[4]

As an additional test of the potential clinical relevance of these findings involving differences between Hispanics and non-Hispanic Caucasians, we reran the model including only those

participants who met PTSD screening criteria (excluding duration) at one or more assessment. Results of the sensitivity analysis revealed an essentially identical pattern. Specifically, the coefficients relating ethnicity variables to the 17 symptoms were correlated .93 across the highly symptomatic subsample and the overall sample.

Discussion

This study used structural equation modeling of longitudinal data to determine whether Hispanics experience different symptoms of PTSD than do non-Hispanic Caucasians. Unlike prior investigations, which have tended to focus either on a single index reflecting overall PTSD symptoms or on symptom clusters, this research examined whether ethnic differences could be identified for individual symptoms. Two important results emerged.

First, these results replicated previous findings showing that Hispanics report higher levels of overall posttraumatic distress (e.g., Galea et al., 2004; Kulka et al., 1990; Lewis-Fernandez et al., 2008; Norris et al., 2001; Pole et al., 2001; Schell & Marshall, 2008). This difference remained, although somewhat attenuated, when using doubly-robust estimates in which the ethnic groups were first matched on the type and severity of trauma and these variables were also included as model covariates in predicting symptom severity.

The most notable finding, however, was that Hispanics showed a distinctive pattern of symptoms relative to similarly traumatized non-Hispanic Caucasians. That is, instead of merely reporting more symptoms of posttraumatic distress, Hispanics appear to experience somewhat different symptoms. For this reason, the prevailing emphasis on differences in overall symptoms following trauma exposure appears an oversimplification. Although Hispanics experience more symptoms of posttraumatic distress, this simple "main effect" characterization of the ethnic disparity is misleading because it is qualified by a substantial interaction. In other words, levels of some symptoms, but not all, are higher in Hispanics.

How are the apparent divergences in ethnic symptom presentation best described? The differences are not confined to a single DSM-IV symptom cluster. Hispanics reported experiencing substantially greater levels of at least one symptom reflecting all three clusters: intrusive thoughts (Criterion B), loss of interest (Criterion C), and hypervigilance (Criterion D). Thus, the pattern is not easily explained with respect to existing PTSD symptom clusters. Nonetheless, a clear pattern does emerge. Specifically, Hispanics tend to report higher levels than non-Hispanic Caucasians of what might be viewed as positive symptoms of PTSD. That is, invoking the distinction between positive and negative symptoms of PTSD (e.g., Barrowclough, Gregg, & Tarrier, 2008; Glynn et al., 1999), Hispanics tend to report elevated levels of symptoms that reflect positive symptoms, i.e., an excess of normal functions (e.g., hypervigilance, intrusive thoughts, flashbacks, emotional reactivity). Specifically, 9 of the 11 symptoms on which Hispanics scored higher than non-Hispanic Caucasians were positive symptoms of PTSD. In contrast, few differences were observed with respect to so-called negative symptoms that reflect a deficit in important functional abilities (e.g., emotional detachment, restricted affect, difficulty sleeping, and impaired concentration). Of the 6 symptoms for which no significant group differences were observed, all but one (i.e., heightened physiological reactivity) were negative symptoms. Although the distinction between positive and negative PTSD symptoms offers a provisional descriptive account of the Hispanic difference in symptom expression, to the extent that other characterizations are possible, additional research is required.

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[4]A separate series of sensitivity analyses, which focused solely on participants of Mexican ancestry ($N = 230$), resulted in an essentially identical pattern of results. The study contained too few Hispanic participants of non-Mexican ancestry to formally compare the Mexican-American subsample to other Hispanics.

The observed pattern is also notable in that it may help to explain why prior studies have found ethnic disparities in PTSD symptoms whether symptom manifestation was expressed as more severe symptoms (e.g., Pole et al., 2001) or as higher rates of PTSD (e.g., Galea et al., 2004). Given that the particular symptoms that Hispanics are more likely to experience are spread across DSM-IV criteria B, C and D, Hispanics are more likely than non-Hispanic Caucasians to meet diagnostic criteria even though they are not more likely to experience all PTSD symptoms. By contrast, if Hispanics were at increased risk for only one of the three required clusters of symptoms (e.g., hyperarousal), then elevated levels of the given symptom cluster would be likely to have only a minimal impact on overall rates of PTSD. In other words, whereas Hispanic differences confined to a single symptom cluster are unlikely to result in substantially higher rates of PTSD, elevation across symptom clusters could readily affect both PTSD diagnostic status and symptom severity.

These findings have important ramifications for current theory regarding the etiology of Hispanic differences in the manifestation of trauma-related distress. Although this study does not support a particular theoretical explanation of the underlying origins of Hispanic differences, the research is valuable because it provides evidence that is inconsistent with several of the theories that have been proposed. Specifically, most existing theories would predict that Hispanics should experience higher levels of all PTSD symptoms. For example, Ortega and Rosenheck (2000) suggest that differences might be explained by tendency for Hispanics to acquiesce. Yet, if response acquiescence underlies the observed differences, then this account must be modified to explain the observed pattern in which some symptoms but not others were endorsed.

Similarly, Marsella et al. (1996) and others (e.g., Pole et al., 2005) have suggested that greater exposure to racial discrimination might serve as a chronic stressor that puts members of racial minority groups at risk for PTSD. Insofar as this conjecture would suggest that Hispanics might experience elevated levels of all symptoms, this explanation does not appear sufficient to explain the observed data pattern. In a slight variation, Loo et al. (2001) proposed that racial discrimination might precipitate a constant state of hypervigilance which could contribute to PTSD, thus highlighting the possibility that ethnic minority samples might differ on hypervigilance. It is noteworthy that the single symptom that most differentiates Hispanics from non-Hispanic Caucasians in this study is, in fact, hypervigilance. Without additional conceptual elaboration, however, this account does not currently explain why Hispanics would show higher levels of a single hyperarousal symptom (i.e., hypervigilance) without also manifesting increases in other hyperarousal symptoms (e.g., sleep problems; concentration difficulties). The proposition that ethnic differences are due to a tendency for Hispanics to express distress in the form of somatic symptoms (Hough et al., 1996) also appears at odds with the data. That is, physical complaints (e.g., physiological reactivity) do not emerge as more prominent in Hispanics relative to their non-Hispanic Caucasian counterparts.

For at least 25 years, research has been directed at documenting and describing the clinical phenomenology of posttraumatic distress experienced by Hispanic trauma survivors (Escobar et al., 1983). Ample evidence exists that Hispanics suffer more posttraumatic distress. Progression of the field now hinges on understanding why Hispanics manifest a particular constellation of symptoms. Empirical tests of the various etiological hypotheses are required. The most valuable theories will be those that can explain symptom level differences in posttraumatic distress. Those that merely predict differences in overall PTSD symptoms are unlikely to prove adequate, given that the size of these ethnic differences across symptoms varies dramatically. Moreover, conceptualizations that do posit a particular constellation of symptom differences (e.g., the tendency to experience distress in somatic terms) must be evaluated by examining whether the theory can account for patterns that are observable at the level of the individual symptom. Also, although it is tempting to think in terms of a single

mechanism that might explain the observed pattern, researchers should remain open to the possibility that multiple mechanisms may be operating. One mechanism may, for example, explain why Hispanics are more susceptible to recurrent nightmares, whereas a second may protect them against a broader pattern of sleep problems.

These findings also raise important practical issues. First, although delivery of culturally competent care poses many challenges, in general (Sue, 1998), and with respect to PTSD, in particular (Ford, 2008), at a minimum, competence requires knowledge. To provide clinical assessment and psychotherapy to Hispanic trauma survivors, providers must be aware of the potential for symptoms of posttraumatic distress to be manifested differently in this high-risk subgroup. To the extent that posttraumatic distress takes on different contours for Hispanics, treatments may require modification to address appropriately those symptoms that cause the most functional impairment or distress for this group.

Moreover, although Hispanics comprise a large and rapidly growing segment of the US population (US Census Bureau, 2006), they are underrepresented in clinical research (US Department of Health and Human Services, 2001). Insofar as minimal information is available concerning whether the PTSD treatments that are effective and efficacious for non-Hispanics also work for Hispanics, the possibility that Hispanics may actually experience a different manifestation of the disorder vis-a-vis non-Hispanic Caucasians is particularly problematic. It appears critical to verify that PTSD interventions that have been demonstrated to be the most appropriate in other populations are also the most appropriate treatment for Hispanics.

In considering these findings, certain caveats should be kept in mind. Similar to other research in this area, this study treated broad ethnic classifications as homogeneous analytic categories. Thus, the current investigation cannot determine whether the differences observed with respect to Hispanics might be isolated to a specific subgroup within this heterogeneous group. Conversely, there may be important heterogeneity among non-Hispanic Caucasian subgroups, some of which may show symptom profiles similar to Hispanics. Further research designed to sample specific ethnic subpopulations is required to address concerns regarding heterogeneity. [5] Moreover, to the extent that symptom severity and profiles vary by trauma type, additional research is needed to investigate the generalizability of these findings. For example, individuals exposed to military combat, natural disaster or other traumatic experiences which do not result in physical injury might be examined. Finally, this investigation focused on the nature and scope, rather than the causal origins, of Hispanic differences in posttraumatic distress. Future research will be required to disentangle the many constructs bound up in Hispanic ethnicity that may contribute to the observed differences in reactions to trauma (e.g., education, income, culture, religiosity, family composition, employment type, self-concept, discrimination).

Despite these limitations, this investigation has a number of features that enhance the generalizability and interpretability of the findings. The sample consists of Hispanic and non-Hispanic Caucasians who experienced broadly similar traumatic injuries. The findings are drawn from a longitudinal structural equation model that explains the means, variances and covariances for all 17 PTSD symptoms measured at three assessments over the course of a year following the trauma. Finally, analyses incorporated propensity score weighting and doubly-robust estimation methods to better ensure that the ethnic differences reported are not attributable to differential trauma exposure.

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[5]Future attempts to examine ethnic differences in posttraumatic distress in more detail are likely to require new data obtained from studies designed expressly to address these issues. To our knowledge, no existing studies of the epidemiology of mental health have recruited sufficiently large and ethnically diverse representative samples of symptomatic trauma survivors. Such research must also collect information to enable subgroups to be analytically equated with respect to the type, timing, and severity of the trauma exposure.

Conclusion

In conclusion, this research indicates that Hispanic trauma survivors may experience symptoms of posttraumatic distress that differ in kind and not merely in degree from those experienced by non-Hispanic Caucasians. Documenting the character of these ethnic differences in the expression of posttraumatic distress has the potential to spur advances in understanding and treating PTSD among Hispanics trauma survivors.

Acknowledgments

References

American Association for the Advancement of Automotive Medicine. The Abbreviated Injury Scale, 1990 Revision. Des Plaines, Illinois: American Association for the Advancement of Automotive Medicine; 1990.

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4. Washington, D.C: Author; 2000.

Andrykowski MA, Cordova MJ, Studts JL, Miller TW. Posttraumatic stress disorder after treatment for breast cancer: prevalence of diagnosis and use of the PTSD Checklist-Civilian Version (PCL-C) as a screening instrument. Journal of Consulting and Clinical Psychology 1998;66:586–590. [PubMed: 9642900]

Baker SP, O'Neill B, Haddon W, Long WB. The injury severity score: A method for describing patients with multiple injuries and evaluating emergency care. Journal of Trauma 1974;14:187–96. [PubMed: 4814394]

Barrowclough C, Gregg L, Tarrier N. Expressed emotion and causal attributions in relatives of post-traumatic stress disorder patients. Behavioural Research and Therapy 2008;46:207–218.

Bentler PM. Comparative fit indexes in structural models. Psychological Bulletin 1990;107:238–246. [PubMed: 2320703]

Bentler, PM. EQS Structural Equations Program Manual. Encino, CA: Multivariate Software; 1995.

Blanchard EB, Jones-Alexander J, Buckley TC, Forneris CA. Psychometric properties of the PTSD checklist (PCL). Behaviour Research and Therapy 1996;34:669–673. [PubMed: 8870294]

Campbell, DT.; Kenny, DA. A primer on regression artifacts. NY: Guilford Press; 1999.

Curran PJ. Have multilevel models been structural equation models all along? Multivariate Behavioural Research 2003;38:529–569.

Denson TF, Marshall GN, Schell TL, Jaycox LH. Predictors of posttraumatic distress one year after exposure to community violence: The importance of acute symptom severity. Journal of Consulting and Clinical Psychology 2007;75:683–692. [PubMed: 17907850]

Emsley R, Lunt M, Pickles A, Dunn G. Implementing double-robust estimators of causal effects. The Stata Journal 2008;8:334–535.

Escobar JI, Randolph ET, Puente G, Spiwak F, Asamen JK, Hill M, Hough RL. Posttraumatic stress disorder in Hispanic veterans: Clinical phenomenology and sociocultural characteristics. Journal of Nervous and Mental Disease 1983;171:585–596. [PubMed: 6619821]

Farley DO, Short PF, Elliott MN, Kanouse DE, Brown JA, Hays RD. Effects of CAHPS health plan performance information on plan choices by New Jersey Medicaid beneficiaries. Health Services Research 2002;37:985–1007. [PubMed: 12236394]

Ford JD. Trauma, posttraumatic stress disorder, and ethnoracial minorities: Toward diversity and cultural competence in principles and practices. Clinical Psychology: Science & Practice 2008;15:62–67.

Frueh BC, Brady KL, de Arellano MA. Racial differences in combat-related PTSD: Empirical findings and conceptual issues. Clinical Psychology Review 1998;18:287–305. [PubMed: 9564582]

Galea S, Vlahov D, Tracy T, Hoover DR, Resnick H, Kilpatrick D. Hispanic ethnicity and post-traumatic stress disorder after a disaster: Evidence from a general population survey after September 11, 2001. Annals of Epidemiology 2004;14:520–531. [PubMed: 15350950]

Glynn SM, Eth S, Randolph ET, Foy DW, Urbaitis M, Boxer L, Paz GG, Leong GB, Firman G, Salk JD, Katzman JW, Crothers J. A test of behavioral family therapy to augment exposure for combat-related posttraumatic stress disorder. Journal of Consulting and Clinical Psychology 1999;67:243–251. [PubMed: 10224735]

Hirano K, Imbens G, Ridder G. Efficient estimation of average treatment effects using the estimated propensity score. Econometrica 2003;71:1161–1189.

Hough, RL.; Canino, GJ.; Abueg, FR.; Gusman, FD. PTSD and related stress disorders among Hispanics. In: Marsella, AJ.; Friedman, MJ.; Gerrity, ET.; Scurfield, RM., editors. Ethnocultural aspects of posttraumatic stress disorder: Issues, research and clinical applications. Washington DC: American Psychological Association; 1996. p. 301-340.

Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional versus new alternatives. Structural Equation Modeling 1999;6:1–55.

Huber PJ. Robust estimation of a location parameter. Annals of Mathematical Statistics 1964;35:73–101.

Kulka, RA.; Schlenger, WE.; Fairbank, JA.; Hough, RL.; Jordan, BK.; Marmar, CR.; Weiss, DS. Trauma and the Vietnam war generation: Report of findings from the National Vietnam Veterans Readjustment Study. NY: Brunner/Mazel; 1990.

Lewis-Fernandez R, Turner JB, Marshall R, Turse N, Neria Y, Dohrenwend BP. Elevated rates of current PTSD among Hispanic veterans in the NVVRS: True prevalence or methodological artifact? Journal of Traumatic Stress 2008;21:123–132. [PubMed: 18404629]

Little, RJA.; Rubin, DB. Statistical analysis with missing data. New York: Wiley; 1987.

Loo CM, Fairbank JA, Scurfield RM, Ruch LO, King DW, Adams LJ, Chemtob CM. Measuring exposure to racism: Development and validation of a Race-Related Stressor Scale (RRSS) for Asian American Vietnam veterans. Psychological Assessment 2001;13:503–520. [PubMed: 11793894]

Marsella, AJ.; Friedman, MJ.; Spain, E. Ethnocultural aspects of PTSD: An overview of issues and research directions. In: Marsella, AJ.; Friedman, MJ.; Gerrity, ET.; Scurfield, RM., editors. Ethnocultural aspects of posttraumatic stress disorder: Issues, research and clinical applications. Washington, DC: American Psychological Association; 1996. p. 105-129.

Marsh HW, Hau K-T, Wen Z. In search of golden rules: Comment on hypothesis-testing approaches to setting cutoff values for fit indexes and dangers of overgeneralizing Hu and Bentler's (1999) findings. Structural Equation Modeling 2004;11:320–341.

Marshall GN, Schell TL. Reappraising the link between peritraumatic dissociation and PTSD symptom severity: Evidence from a longitudinal study of community violence survivors. Journal of Abnormal Psychology 2002;111:626–636. [PubMed: 12428776]

McCaffrey D, Ridgeway G, Morral A. Propensity score estimation with boosted regression for evaluating adolescent substance abuse treatment. Psychological Methods 2004;9:403–425. [PubMed: 15598095]

McLachlan, GJ.; Krishnan, T. The EM algorithm and extensions. New York: Wiley-Interscience; 1996.

Miles JNV, Marshall GN, Schell TL. Spanish and English Versions of the PTSD Checklist–Civilian Version (PCL-C): Testing for Differential Item Functioning. Journal of Traumatic Stress 2008;21:369–376. [PubMed: 18720394]

Muthen, LK.; Muthen, BO. Mplus: The comprehensive modeling program for applied researchers [Computer Program]. Los Angeles: Muthen & Muthen; 2006.

Norris FH, Perilla JL, Murphy AD. Postdisaster stress in the United States and Mexico: A cross-cultural test of the multicriterion conceptual model of posttraumatic stress disorder. Journal of Abnormal Psychology 2001;110:553–563. [PubMed: 11727945]

O'Donnell ML, Creamer M, Pattison P, Atkin C. Psychiatric morbidity following injury. American Journal of Psychiatry 2004;161:507–514. [PubMed: 14992977]

Ortega AN, Rosenheck R. Posttraumatic stress disorder among Hispanic American veterans. American Journal of Psychiatry 2000;157:615–619. [PubMed: 10739422]

Perilla JL, Norris FH, Lavizzo EA. Ethnicity, culture, and disaster response: Identifying and explaining ethnic differences in PTSD six months after Hurricane Andrew. Journal of Social and Clinical Psychology 2002;21:20–45.

Pole N, Gone GP, Kulkarni M. Posttraumatic stress disorder among ethnoracial minorities in the United States. Clinical Psychology: Science & Practice 2008;15:35–61.

Pole N, Best SR, Metzler T, Marmar CR. Why are Hispanics at greater risk for PTSD? Cultural Diversity and Ethnic Minority Psychology 2005;11:144–161. [PubMed: 15884985]

Pole N, Best SR, Weiss DS, Metzler T, Ligerman AM, Fagan J, Marmar CR. Effects of gender and ethnicity on duty-related posttraumatic stress symptoms among urban police officers. Journal of Nervous and Mental Diseases 2001;189:442–448.

Raudenbush, SW. Toward a coherent framework for comparing trajectories of individual change. In: Collins, LM.; Sayer, AG., editors. New methods for the analysis of change. Washington, DC: APA; 2001.

Ramchand R, Marshall GN, Schell TL, Jaycox LH, Hambarsoomians K, Shetty V, Hinika GS, Cryer HG, Meade P, Belzberg H. Alcohol abuse and illegal drug use among Los Angeles County trauma patients: Prevalence and evaluation of single item screener. Journal of Trauma 2009;66:1461–1467. [PubMed: 19430255]

Robins JM, Rotnitsky A. Comment on "Inference for semiparametric models: Some questions and an answer" by P. Bickel and J. Kwon. Statistica Sinica 2001;11:920–936.

Robins JM, Rotnitsky A, Zhao LP. Estimation of regression coefficients when some regressors are not always observed. Journal of the American Statistical Association 1994;89:846–866.

Rosenbaum PR, Rubin DB. The central role of the propensity score in observational studies for causal effects. Biometrika 1983;70:41–55.

Ruef AM, Litz BT, Schlenger WE. Hispanic ethnicity and risk for combat-related posttraumatic stress disorder. Cultural Diversity and Ethnic Minority Psychology 2000;6:235–251. [PubMed: 10938633]

Satorra, A. Scaled and adjusted restricted tests in multi-sample analysis of moment structures. In: Heijmans, RDH.; Pollock, DSG.; Satorra, A., editors. Innovations in multivariate statistical analysis. A Festschrift for Heinz Neudecker. London: Kluwer Academic Publishers; 2000. p. 233-247.

Satorra, A.; Bentler, P. A scaled difference chi-square test statistic for moment structure analysis. UCLA Statistics Electronic Publication #260; 1999.

Schell, TL.; Marshall, GN. Survey of individuals previously deployed for OEF/OIF. In: Taniellian, T.; Jaycox, LH., editors. Invisible wounds of war: Psychological and cognitive injuries, their consequences, and services to assist recovery. Santa Monica, CA: The RAND Corporation; 2008. p. 87-115.

Scherpenzeel, A.; Saris, W. Multitrait-multimethod models for longitudinal research. In: van Montfort, K.; Oud, J.; Satorra, A., editors. Longitudinal models in the behavioral and related sciences. Mahwah, NJ: Lawrence Erlbaum Associates; 2007. p. 381-402.

Steiger, JH.; Lind, JM. Statistically based tests for the number of common factors. Paper presented at the annual meeting of the Psychometric Society; Iowa City, IA. 1980 May.

Sue S. In search of cultural competence in psychotherapy and counseling. American Psychologist 1998;53:440–448. [PubMed: 9572007]

Teasdale C, Jennett B. Assessment of coma and impaired consciousness: A practical scale. Lancet 1974;2:81–84. [PubMed: 4136544]

U.S. Census Bureau. U.S. Census Bureau News. Washington, DC: Department of Commerce; 2006 [Accessed May 29, 2009]. Nation's population one-third minority. http://www.census.gov/Press-Release/www/releases/archives/population/006808.html

U.S. Census Bureau. Hispanic population of the United States. 2006 American Community Survey. 2008 [Accessed July 14, 2009]. http://www.census.gov/population/www/socdemo/hispanic/files/Internet_Hispanic_in_US_2006.pdf

U.S. Department of Health and Human Services. Mental health: Culture, race, and ethnicity. Rockville, MD: U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General; 2001.

Weathers, FW.; Litz, BT.; Herman, DS.; Huska, J.; Keane, TM. The PTSD Checklist (PCL): Reliability, validity, and diagnostic utility. Ninth annual meeting of the International Society for Traumatic Stress Studies; San Antonio. 1993 [Accessed July 11, 2009].
http://www.pdhealth.mil/library/downloads/PCL_sychometrics.doc

Yuan KH, Marshall LL, Bentler PM. A unified approach to exploratory factor analysis with missing data, nonnormal data, and in the presence of outliers. Psychometrika 2002;67:95–122.

Zatzick D, Jurkovich GJ, Rivara FP, Wang J, Fan MY, Joesch J, Mackenzie E. A national US study of posttraumatic stress disorder, depression, and work and functional outcomes after hospitalization for traumatic injury. Annals of Surgery 2008;248:429–437. [PubMed: 18791363]

Table 1

Odds ratios (OR) Predicting Hispanic Ethnicity from Injury Characteristics, with and without Propensity Weights

Variable	Hispanic vs. Non-Hispanic Caucasian		Hispanic vs. African American	
	Unweighted OR	Weighted OR	Unweighted OR	Weighted OR
Injury severity score	0.99	0.99	1.02	0.73
Assault	2.14[**]	1.30	0.26[**]	1.00
Time in Hospital	1.02	1.01	0.98	1.02
Age (10s of years)	1.58[**]	1.05	1.64[**]	1.26
Female	1.15	1.39	0.94	1.11
Lost Consciousness	0.81	0.94	1.06	1.16
Glascow Coma Score	1.41[*]	1.09	1.30[*]	0.73

[*]
$p < .05$.

[**]
$p < .01$.

Table 2

The Effect of Hispanic Ethnicity by PTSD Symptom

PTSD symptoms	Non-Hispanic Caucasian minus Hispanic			African American minus Hispanic		
	Estimate	Lower 95% C.I.	Upper 95% C.I.	Estimate	Lower 95% C.I.	Upper 95% C.I.
D4: Hypervigilance	-0.61^*	−0.88	−0.33	0.17	−0.08	0.41
B1: Intrusive thoughts	-0.46^*	−0.69	−0.24	0.16	−0.10	0.42
B3: Flashbacks	-0.44^*	−0.66	−0.23	−0.14	−0.32	0.05
C4: Loss of interest	-0.42^*	−0.64	−0.20	−0.04	−0.28	0.20
B4: Emotional reactivity	-0.36^*	−0.62	−0.09	0.00	−0.24	0.24
D5: Startle response	-0.36^*	−0.57	−0.14	−0.08	−0.30	0.13
C1: Avoiding thoughts	-0.35^*	−0.56	−0.15	-0.19^*	−0.38	0.00
C7: Foreshortened future	-0.30^*	−0.54	−0.05	−0.04	−0.26	0.19
B2: Recurrent dreams	-0.25^*	−0.45	−0.05	0.02	−0.19	0.23
C2: Avoiding reminders	-0.24^*	−0.45	−0.04	−0.04	−0.26	0.18
D2: Irritability	-0.24^*	−0.43	−0.04	0.11	−0.12	0.34
B5: Physiological reactivity	−0.18	−0.39	0.02	0.12	−0.10	0.35
C5: Detachment	−0.08	−0.33	0.18	0.05	−0.16	0.26
D3: Difficulty concentrating	−0.06	−0.25	0.14	0.13	−0.08	0.33
C3: Inability to recall	0.02	−0.20	0.23	−0.11	−0.32	0.10
C6: Restricted affect	0.11	−0.08	0.31	0.17	−0.02	0.36
D1: Sleep disturbance	0.13	−0.13	0.38	0.14	−0.11	0.40

Note. Estimated coefficients are in PTSD Checklist scale units. Each symptom is presented with its DSM-IV cluster, item number, and abbreviated content. The estimates are sorted in descending order such that symptoms that are more common among Hispanics are listed earlier. CI = Confidence Interval.

* $p < .05$.