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

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This longitudinal study of physical injury survivors examined the degree to which Hispanic and non-Hispanic Caucasians reported similar posttraumatic stress disorder (PTSD) symptoms. 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 3 time points: within days of trauma exposure and again at 6 and 12 months posttrauma. Structural equation modeling with propensity weights was used in analyzing data. 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). 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.

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 suggest that they are. These studies include 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 a study of nearly 1,200 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 adjustment 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. 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 (Ortega & Rosenheck, 2000; Ruef, Litz, & Schlenger, 2000), a disposition toward acquiescent responding (Ortega & Rosenheck, 2000), and the tendency of...

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1 Ortega and Rosenheck (2000) conducted a cluster-level analysis and found 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.
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, Best, Metzler, & Marmar, 2005), and sociodemographic disadvantage (Pole et al., 2008).

Whether any of these hypotheses are 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), 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 et al., 1996), it might be anticipated that this predisposition would be evidenced in endorsement of certain symptoms rather than others. In particular, 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 merely in degree, modifications in treatment might 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 require crafting 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 has documented that traumatic physical injury requiring hospitalization is associated with post-traumatic distress and PTSD in the months following acute inpatient care (e.g., Marshall & Schell, 2002; O’Donnell, Creamer, Pattison, & Atkin, 2004; Zatzick et al., 2008) and thus attests 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 by theory implying 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: Do Hispanics report higher overall levels of PTSD symptoms than do their non-Hispanic Caucasian counterparts? and, if so, do Hispanics manifest uniform differences relative to non-Hispanic Caucasians across all 17 PTSD symptoms? Finally, few, if any, investigations have compared the PTSD symptoms of Hispanics to those of African Americans, as research has almost always treated non-Hispanic Caucasians as the conceptual reference category. In part due to the lack of descriptive research, there were 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 6 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 Los Angeles County census of trauma center patients with respect to age, gender, ethnicity, and mechanism of injury (i.e., assault vs. 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 and 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 (a) could not communicate in English or Spanish; (b) had been incarcerated at hospital admission; (c) had been homeless prior to hospitalization and expected to be homeless upon discharge; (d) had cognitive impairment that precluded informed consent; or (e) 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: Admitted patients thought to be eligible for the study were identified in Stage 1, and a face-to-face screening interview was used in Stage 2 to verify eligibility. Different methods for identifying potentially eligible individuals were used at the four hospitals due to different types of medical records and institutional review board 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 obtain consent from all patients identified as potentially eligible, with one exception: The research staff approached every other Hispanic patient at LAC + USC, as 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 = 1,133) 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 the 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 the third was completed at 12-month follow-up. Of the 850 patients identified as eligible for the larger study on the basis of 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 conducted 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, UCLA Medical Center, King–Drew Medical Center, and California Hospital Medical Center.

Measures

PTSD symptoms. PTSD symptom severity was assessed with the civilian version of the Posttraumatic Stress Disorder Checklist (PCL; Weathers, Litz, Herman, Huska, & Keane, 1993) at all three interviews. 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 Diagnostic and Statistical Manual of Mental Disorders (DSM–IV–TR) 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 2 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). No evidence was found of differential item function or an overall scoring bias across the two versions. The PCL summed scores for the three assessments indicated a relatively high level of traumatic distress (in the weighted analytic sample, Ms = 39, 38, and 36 and SDs = 16, 19, and 18, for the baseline, 6-month, and 12-month assessments, respectively).

For descriptive purposes, we used PCL screener responses to estimate the percentage of the sample who met symptom criteria for PTSD according to the procedure recommended by Weathers et al. (1993). Individual symptoms receiving a score of 3 (moderately) or greater were treated as indicating symptom presence and mapped against the DSM–IV–TR 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 with standard census questions; that is, 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, we categorized participants as Hispanic (n = 330), non-Hispanic Caucasian (n = 135), African American (n = 171), or another group (n = 41).

The latter group of participants included Asians and Pacific Islanders, Native Americans, and Asian Indians. Of the 330 Hispanic participants, 70% (n = 230) were of Mexican geographic origin and 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 that of the broader U.S. Hispanic population. However, the current sample underrepresents Hispanics of Caribbean origin, who constitute approximately 13% of Hispanics in the United States (U.S. Census Bureau, 2007).

Injury mechanism. Injury mechanism was assessed by self-report and was classified as motor vehicle collisions (61%; injuries stemming from assaults (e.g., having been shot with a gun, 1171 ETHNIC DIFFERENCES IN POSTTRAUMATIC DISTRESS
stabbled 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 respondents related the incidents that resulted in their injuries, they were asked, “Did you lose consciousness during the event?”

**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) to 5 (Critical). Scores for the 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 the 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 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 receive the highest possible score (e.g., opens eyes spontaneously), and 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 comatose person, whereas a score of 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 at the trauma center. Scores were obtained from medical records.

**Data Analysis**

**Propensity weighting.** To make meaningful comparisons across ethnic groups, one must ensure that observed ethnic differences are not attributable to incidental differences between groups. We desired, as an initial step prior to estimating the ethnic effect of interest, 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., Farley et al., 2002; Hirano, Imbens, & Ridder, 2003). With 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. This powerful regression technique 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, Glasgow Coma Score, loss of consciousness, mechanism of injury, age, and gender.

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 \(\chi^2(7) = 44, p < .001\), with pseudo \(R^2 = .06\) and \(c = 0.62\). With weights applied, \(\chi^2(7) = 5.8, p = .604,\) pseudo \(R^2 = .01, c = 0.50\), 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 multisymptom, multitime model. This type of longitudinal model is discussed in Scherpenzeel and Saris (2007) as having identification conditions similar to those of latent variable multitrait–multimethod models, although with substantively different factor interpretations. This multisymptom, multitime model can also be seen as a particular type of hierarchical, or multilevel, model (Curran, 2003; Raudenbush, 2001), in particular, one in which each Level 1 observation is cross-categorized on two Level 2 random variables nested within individuals, one representing the 17 symptom clusters and the other representing the three 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

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\(^2\) Because a small number of individuals suffered injuries from multiple causes, the total percentage exceeds 100.

\(^3\) We did not control for socioeconomic status (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).
ETHNIC DIFFERENCES IN POSTTRAUMATIC DISTRESS

Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hispanic vs. non-Hispanic Caucasian</th>
<th>Hispanic vs. African American</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISS</td>
<td>0.99</td>
<td>1.02</td>
</tr>
<tr>
<td>Assault</td>
<td>2.14**</td>
<td>0.26**</td>
</tr>
<tr>
<td>Time in hospital</td>
<td>1.02</td>
<td>0.98</td>
</tr>
<tr>
<td>Age (10s of years)</td>
<td>1.58***</td>
<td>1.64**</td>
</tr>
<tr>
<td>Female</td>
<td>1.15</td>
<td>0.94</td>
</tr>
<tr>
<td>Lost consciousness</td>
<td>0.81</td>
<td>1.06</td>
</tr>
<tr>
<td>Glasgow Coma Score</td>
<td>1.41*</td>
<td>1.30*</td>
</tr>
</tbody>
</table>

Note. ISS = Injury Severity Score.
*p < .05. **p < .01.

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 Times 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, this generally can 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 that of non-Hispanic Caucasians on the latent factor for Time 1, this would imply 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 but 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: Do Hispanics report higher overall levels of symptoms of posttraumatic distress than do their non-Hispanic Caucasian counterparts? and, if so, do Hispanics manifest uniform differences across all 17 PTSD symptoms relative to non-Hispanic Caucasians? 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) when 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 (Emsley, Lunt, Pickles, & Dunn, 2008; Robins & Rotnitsky, 2001; Robins, Rotnitsky, & Zhao, 1994) in which potential confounding variables are entered into the regressions as covariates after propensity weighting.

To achieve doubly robust estimation, we regressed latent variables onto the seven covariates listed in Table 1. To maximize the efficiency of the parameter estimation as well as the parsimony of the model, we removed unnecessary model parameters. 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 nonsignificant p values (i.e., p < .25). The use of this p-value criterion results in less pruning than would occur if either the Akaiake information criterion or the Bayesian information criterion were used as the pruning criterion.

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 individuals 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 according to robust maximum likelihood (MLR) methods in Mplus Version 5.2 (Muthén & Muthén, 2006). These parameter estimates and their standard errors are robust to deviations from normality, as the standard errors are 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) and adjusted the difference in chi-square statistics using the method proposed by Satorra (2000; see also Satorra & Bentler, 1999).

When assessing overall model fit, we used the Tucker–Lewis index (TLI), the comparative fit index (CFI; Bentler, 1990), the root mean square error of approximation (RMSEA; Steiger & Lind, 1980), and the standardized root mean square residual (SRMR; Bentler, 1995). We considered CFI or TLI values of .90
Results

The basic multisymptom, multitime model with covariates and propensity weights fit the data satisfactorily, \( \chi^2(1470) = 2,250, TLI = .92, CFI = .93, RMSEA = .028, SRMR = .047 \). 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 and 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, \( \Delta \chi^2(1) = 4.86, p = .028 \). On average, Hispanics had higher scores than did 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, \( \Delta \chi^2(16) = 49, p < .001 \). The differences between Hispanics and African Americans also varied in magnitude across symptoms, \( \Delta \chi^2(16) = 33, p = .008 \), although these differences were smaller in magnitude than those observed between Hispanics and non-Hispanic Caucasians. As shown in Table 2, Hispanics scored significantly higher than did non-Hispanic Caucasians on 11 of 17 symptoms; no significant differences were observed with respect to six symptoms.

The distribution of Hispanic versus non-Hispanic Caucasians effects for individual symptoms ranged from hypervigilance, which was .61 scale points higher for Hispanics than for non-Hispanic Caucasians, to sleep disturbance, which was .13 scale points higher for non-Hispanic Caucasians than for Hispanics. The distribution of effects does not correspond neatly to the DSM–IV–TR PTSD symptom clusters (American Psychiatric Association, 2000). Both the largest and the smallest effects were from DSM–IV–TR Cluster D (i.e., Hyperarousal). In addition, the four items with the largest Hispanic relative to non-Hispanic Caucasians difference included two reexperiencing symptoms, one avoidance/numbing symptom, and one hyperarousal symptom. A comparison of African American and Hispanic responses revealed

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

Note. Estimated coefficients are in Posttraumatic Stress Disorder (PTSD) Checklist scale units. Each symptom is presented with its DSM–IV cluster, item number, and abbreviated name. The estimates are sorted in descending order, such that symptoms that are more common among Hispanics are listed earlier. CI = confidence interval.

\(^* p < .05\).
only a single statistically significant difference: Hispanics scored significantly higher on Item C1 (avoiding thoughts of trauma).

To investigate the role of covariates in these results, we reran 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 did non-Hispanic Caucasians (i.e., .22 of a scale point) on all symptoms, $\Delta \chi^2(1) = 9.2, p = .002$. Differences involving African Americans and Hispanics were slight (.02 points), $\chi^2(1) = 0.12, p = .73$. As with those for the analyses on covariate-equated groups, these overall differences were qualified by the finding that the effect of ethnicity varied significantly across symptoms. That is, the differences between Hispanics and non-Hispanic Caucasians varied in magnitude across symptoms, $\Delta \chi^2(16) = 106.5, p < .001$.

As an additional test of the potential clinical relevance of these findings involving differences between Hispanics and non-Hispanic Caucasians, the model was rerun 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. In particular, the coefficients relating ethnicity variables to the 17 symptoms were correlated .93 across the highly symptomatic subsample and the overall sample.

Discussion

In this study, we 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; Kulfka 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 we used doubly robust estimates in which the ethnic groups were first matched on the type and severity of trauma and these variables were then included as model covariates in predicting symptom severity.

The most notable finding, however, was that Hispanics showed distinctive patterns of symptoms relative to similarly traumatized non-Hispanic Caucasians. That is, instead of merely reporting more symptoms of posttraumatic distress, Hispanics appeared 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–TR symptom cluster. Hispanics reported experiencing substantially greater levels of at least one symptom reflecting all three clusters: intrusive thoughts (Criterion B), avoidance (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. Hispanics tend to report higher levels than do 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, such as hypervigilance, intrusive thoughts, flashbacks, emotional reactivity). Nine 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, impaired concentration). Of the six 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.

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–TR 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), 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. For example, most existing theories would predict that Hispanics should experience higher levels of all PTSD symptoms. For example, Ortega and Rosenheck (2000) suggested that differences might be explained by a tendency for Hispanics to acquiesce. Yet, if response acquiescence underlies the observed

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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 for us to formally compare the Mexican American subsample to other Hispanics.
differences, 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 that 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), competence requires knowledge, at a minimum. 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, modification of treatments may be required to address appropriately those symptoms that cause the most functional impairment or distress for this group.

Moreover, although Hispanics constitute a large and rapidly growing segment of the U.S. population (U.S. Census Bureau, 2006), they are underrepresented in clinical research (U.S. 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-à-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 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 that of Hispanics. Further research designed to sample specific ethnic subpopulations should address concerns regarding heterogeneity. 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 that 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, our 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.

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 analytical equation of subgroups 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 Hispanic trauma survivors.

References


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