Early physical health consequences of disaster exposure and acute disaster-related PTSD

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Abstract:

A sample of adults (N= 666) was interviewed 6 months after the devastating 1999 floods and mudslides in Mexico. Comparisons between sample data and population norms pointed to significant postdisaster elevations in physical health symptoms across a variety of domains. With age, gender, and predisaster mental health and living conditions controlled, severity of exposure was related to higher physical symptoms. The effects of severity of exposure dropped out of the equations when postdisaster posttraumatic stress disorder (PTSD) symptoms were taken into account. The effects of acute PTSD on health symptoms were largely, but not completely, accounted for by concurrent depressed affect, with criterion symptoms reflecting intrusion and arousal most likely to show a specific effect. Although previous research examined stressors from the distant past, here the role of PTSD as a mediator of the trauma-health relation was demonstrated with recent disaster exposure and acute PTSD, in a very different population.

Keywords: Disaster | physical health | PTSD | Mexico | developing countries

Article:

Introduction

Survivors of major disasters are quite likely to experience psychological problems, such as posttraumatic stress disorder (PTSD), anxiety, and depression (Galea, Nandi, & Vlahov, 2005; Norris et al., 2002) and many also report declines in their physical health. In past studies, disaster victims have scored higher than norms or controls on objective measures of morbidity (Holen, 1991; Palinkas et al., 1993) as well as on self-reported somatic complaints or checklists of medical conditions (Clayer, Bookless-Pratz, & Harris, 1985; Murphy 1984; Phifer, Kaniasty, & Norris 1988). Often, physiological indicators of stress are elevated, immune functioning is compromised, and sleep quality is poor (Inoue- Sakurai, Maruyama, & Morimoto, 2000; Ironson et al., 1997; Krakow et al., 2004; Mellman, David, Kulick-Bell, Hebding, & Nolan, 1995).

Disaster exposure may also increase the likelihood of relapse (clinical worsening of symptoms) and perceived illness burden in previously disabled populations (Lutgendorf et al., 1995).

Despite substantial progress in documenting the health consequences of natural disasters, the research is limited in two critical ways that the present study aimed to address. Our understanding of disaster effects and physical health consequences worldwide has remained quite limited because relatively little of the research has been conducted outside of the United States and other developed countries (Norris et al., 2002). This shortcoming is especially important because disasters are more prevalent, and typically more severe, in developing areas of the world (De Girolamo & McFarlane, 1996; International Federation of Red Cross and Red Crescent Societies, 2004), and these populations may be particularly susceptible to adverse health effects given their poverty and lack of healthcare resources. For several reasons, we anticipated that survivors of disasters in the developing country of Mexico would be especially likely to experience declines in their subjective physical health. For one thing, somatic expressions of distress have been found quite commonly in Central and South America (Escobar et al., 1987; Jenkins, 1996) as well as among Mexican American, Puerto Rican, and other Latino populations in the United States (Canino et al., 1999; Eisenman et al., 2003). More specifically relevant, exposure to a disaster in Puerto Rico increased the prevalence of gastrointestinal (e.g., abdominal pain, vomiting, nausea) and ''pseudoneurological'' symptoms (e.g., paralysis, fainting, double vision) (Escobar et al., 1992). When Norris et al., queried Mexicans about their responses to disasters in unstructured interviews, two of the five clusters that emerged in the data were composed of various somatic complaints. The first group generally could be characterized as acute suffering (e.g., pains, aches, gastrointestinal distress), the second as ''wasting away'' (weakness, weight loss). Together, these various findings suggested that more in-depth research on postdisaster physical health in Mexico was warranted.

The second deficiency in the disaster research to date is that it has been largely descriptive with little attention to the mechanisms that account for health declines. Research on other traumatic stressors suggests that the effects of trauma exposure on health might be mediated by emotional reactions, especially PTSD (Schnurr & Green, 2004). Although the effects of health and emotional reactions may be reciprocal because of these previous results, the current investigation examined acute PTSD as the mechanism linking disaster exposure to physical health complaints. Characterized by a combination of intrusion, avoidance, and arousal symptoms, PTSD emerges in about 20% of female and 10% of male trauma victims and very often persists (Kessler et al., 1995; Norris et al.,
2003). Even higher estimates of PTSD have been found following trauma and disasters in developing countries (Basoglu et al., 2003; de Jong et al., 2001; Mollica, Poole, & Tor, 1998). Combat veterans with PTSD have been shown to have more chronic health conditions and poorer functional status than combat veterans without PTSD (Boscarino, 1997; Ouimette et al., 2004; Schnurr & Spiro, 1999; Taft et al., 1999; Wolfe et al., 1994). In many of these studies, the effects on health of exposure per se dropped out when PTSD was controlled. Similar effects have been observed in samples of sexual assault victims (Kimerling & Calhoun, 1994; Zoellner, Goodwin, & Foa, 2000) and refugees (Van Ommeren et al., 2002), suggesting that the effects are not confined to combat veterans, although clearly the latter have been most thoroughly researched. With a few exceptions (e.g., Wagner et al., 2002), these studies have been conducted long after the initial trauma and therefore have been especially informative about the cumulative impact of

chronic PTSD. Little is known about the impact on health of recent traumatic stressors and acute PTSD.

Researchers are not yet certain why PTSD affects health, but several explanations have been offered (Schnurr & Green, 2004). In part, the association may stem from the negative affect associated with PTSD, especially anger and comorbid depression (Zoellner, Goodwin, & Foa, 2000; Friedman & Schnurr, 1995; Miranda et al., 2002). A substantial body of research indicates that depression has an impact on health, especially coronary artery disease (see Ford, 2004 for a review). The issue is more complex with regard to interpretation of self-reported or subjective health symptoms because they may be confounded with (rather than caused by) negative affect, i.e., negative affect may influence how bodily sensations are perceived. The comorbidity, or confounding, of PTSD with depressed affect is especially important to take into account because, like PTSD, depressed affect is prevalent in the aftermath of disasters (Norris et al., 2002).

There are reasons to suspect, however, that the effects on physical health of PTSD are not solely explained by depressed affect. Physiological reactivity, hypervigilance, and exaggerated startle are core features of the disorder that have been linked in clinical and laboratory studies to sympathetic nervous system dysregulation and significant cardiovascular changes, such as hypertension, higher tonic heart-rates, and arrhythmias (Friedman & McEwen, 2004; Litz et al., 1992). The notion that the effects of PTSD on health symptoms are specific, rather than merely reflective of negative or depressed affect, is supported by data showing that PTSD affects health with concurrent negative affect controlled (Zoellner, Goodwin, & Foa, 2000; Miranda, Meyerson, Marx, & Tucker, 2002). It is also quite possible that different clusters of PTSD symptoms vary in the specificity of their effects. The relative impact of different symptom clusters is not yet understood because data are sparse and inconsistent (Zoellner, Goodwin, & Foa, 2000; Kimerling, Clum, & Wolfe, 2000; McFarlane et al., 1994; Mohr et al., 2003) and occasionally confounded by health measures that include behavioral symptoms of arousal, such as disturbed sleep and inability to concentrate.

The present study examined disaster victims' health and mental health six months after the 1999 Mexican flood. In October 1999, a stationary tropical depression in the Gulf of Campeche generated torrential rains, widespread flooding, and devastating mudslides in nine Mexican states. More than 400 people died, and at least 200,000 people lost their homes. Officials in Mexico characterized this event as the worst flooding disaster of the decade, if not of the century. The severity of this event made it an important and relevant context for studying the health consequences of disaster exposure and disaster-related PTSD in Latin America. Our primary hypotheses were these: (1) that this sample of disaster victims, as a whole, will exhibit levels of physical health symptoms that are higher than population norms for Mexico; (2) that severity of exposure to the disaster will predict health symptoms after effects of demographic variables and predisaster PTSD, MDD, and living conditions have been controlled; (3) that postdisaster PTSD will predict past-month health symptoms and be the mechanism responsible for the exposure-health relation; and (4) that the effects of postdisaster PTSD will remain significant when past-week depressed affect is controlled, thereby supporting the position that PTSD has a specific influence on health not accounted for by the confounding of self-reported physical and psychological symptoms. After testing these hypotheses in hierarchical regression analyses, we conducted some explicitly exploratory analyses of the relations between specific criterion

symptoms and health for which we offer no a priori hypotheses but which may inform understanding of the mechanisms by which PTSD exerts its effects on health.

Method

Sampling and interviewing procedures

To capture the variability in the way this event was experienced, we studied two different communities: Villahermosa, the capital of the coastal state of Tobasco, population 500,000, and Teziutla´n, a mountain city in the state of Puebla, population 180,000. These communities anchored the geographic range of the disaster. Visits to the two selected communities revealed that identical sampling procedures would not be possible. In Villahermosa, the flood damage was extensive, and victims were dispersed across a large sector of the city. The context necessitated a probability sampling design to draw a random sample of adults representative of the afflicted population in purposively selected sectors, which had experienced flood damage. In Teziutla´n, the stricken hillside neighborhoods were condemned, and all families were relocated to a new community outside of the original city. The size of the community did not necessitate sampling, and all households were included in the sampling frame. Despite the difference in approach, both strategies provided samples that were highly representative of the populations and settings.

The interviews were conducted six months postdisaster, in April 2000. From affected census tracts in Villahermosa, 653 households were sampled randomly in proportion to the tracts' population sizes. Of the 601 eligible households (non-eligible units were vacant lots or businesses), 530 were successfully contacted and the adult who answered the door was asked to provide a sociodemographic interview about the household. Of these households, 470 agreed to complete this initial interview. One adult resident was then randomly selected from each participating household and asked to participate in an in-depth psychological interview. Of these, 461 completed the psychological interview, for a final Wave 1 response rate of 77% of those assessed as eligible and 87% of those actually contacted. In Teziutla´n, all 235 households provided with plots in the new community were selected and, of these, 209 were successfully contacted. Only 1 household refused the demographic interview. Of the 208 households that completed the demographic interview, 205 participants completed the psychological interview, for a final response rate of 87% of those eligible and 98% of those actually contacted.

The proportion of women in the sample (67%) was higher than it should have been (55%) according to Mexican census data. Analyses of the sociodemographic data indicated that the bias occurred at the point of selection for the psychological interview, although the reason for this was not clear. This selection was made randomly at the end of the demographic interview, well after the informant had provided the birthdays, birth years, and present residence status of each household member. Fieldwork supervisors reviewed audiotapes of each interview and verified that the interviewer selected the appropriate adult (the one with the most recent birthday) for the psychological interview regardless of who gave the sociodemographic interview or who was home at the time of that initial interview. Analyses of the household demographic data indicated that female participants were quite representative of the larger population of women, but male participants underrepresented younger, lower-income, less-educated men. With effect sizes (d) in

the range of .09 to .12, the magnitude of the bias appeared to be relatively small. The data were weighted to produce a 55:45 ratio of women to men.

All interviews were completed by trained, local lay interviewers in respondents' homes in private. The demographic interviews lasted about 1 hour, and psychological interviews lasted an average of 2 hours. Demographic and psychological interviews were typically completed on separate days. Study procedures were approved by institutional review boards in the United States and Mexico and were reviewed for adherence to federal (U.S.) guidelines for conducting research in international settings.

Measures

Physical health symptoms. The scale of physical health symptoms was adapted and translated from the Physical Symptoms Checklist (Leventhal, E, Hansell, Diefenbach, Leventhal, H, Glass, 1996). Each of the scale's 34 items described a specific physical symptom experienced in the last month from *not at all* (1) to *extremely* (5).

Because the scale had not been used in Mexico previously, we conducted an exploratory factor analysis by using the data from a normative sample of 2,509 adults randomly selected from four cities in Mexico (Hermosillo in the north, Guadalajara in the central region, Oaxaca in the south, and Me´rida in the Yucatan). Response rates were, respectively, 76%, 82%, 79%, and 70%. The four-city epidemiologic study was conducted between 1999 and 2001 to establish population norms for measures of various constructs, such as PTSD, trauma exposure, health, and social support, that we planned to use in future disaster research in Mexico. As in the disaster study, all persons were interviewed in their homes by trained indigenous interviewers. These data were also weighted to yield a 55 female: 45 male ratio. More detail about the sampling and assessment procedures used in the normative study may be found in Norris et al. (2003).

Before conducting the factor analysis on this normative sample, two items (problems remembering things, sleeping problems) were removed because of their close overlap with items on the measure of PTSD. Several other items either did not load on any factor or formed weak 2-item factors (e.g., teeth or gum problems, vision problems, hearing problems, skin problems, bruising/sores). The end result was a 24-item measure with 5 subscales (see Table I): (1) Cardio_Pulmonary Symptoms, e.g., chest discomfort or pain, irregularities in heartbeat, high blood pressure, difficulty breathing, other lung problems; (2) Muscular_Skeletal Symptoms, e.g., pain or stiffness in shoulders, arms or hands, back problems, hip, leg, knee, or feet problems, swelling in legs or ankles, loss of strength; (3) Nose-Throat Symptoms, e.g., nose or sinus problems, throat problems, neck problems or swollen glands, fever or chills; (4) Gastrointestinal/Urinary Symptoms, e.g., stomach or digestive problems, intestinal or bowel problems, urination problems, changes in appetite or thirst; and (5) Hormonal Symptoms, e.g., low blood pressure, headaches, dizziness/balance problems, menstrual [women] or genital [men] problems. This last factor was not very internally consistent, so was not used in the regression/correlational analyses.

Scales were initially scored as the mean of component items, for example a total health symptoms score of 1.3 meant that across all 24 items, the person averaged a score of 1.3 (range

1_5). Then, for ease of comparison to country-wide norms, raw health scores in the disaster sample were standardized by subtracting the normative sample's mean, dividing that result by the normative sample's standard deviation, multiplying that result by 10, and adding 50. In other words, the norm for all measures is 50 with a standard deviation of 10.

Table I. Health symptom scale scores for the total sample ($n = 666$) 6 months postdisaster.

			Raw score (Item *M*)		Standardized score	
Scale or subscale	# items	α	*M*	*(SD)*	*M*	*(SD)*
Total Health Symptoms	24	.90	1.71	(0.60)	54.5[a]	(12.6)
Cardio-Pulmonary Symptoms	5	.78	1.52	(0.73)	54.5[a]	(14.1)
Muscular-Skeletal Symptoms	7	.77	1.90	(0.77)	52.9[a]	(11.1)
Nose-Throat Symptoms	4	.71	1.73	(0.83)	53.8[a]	(12.7)
Gastrointestinal /Urinary Symptoms	4	.62	1.68	(0.74)	52.1[a]	(11.3)
Hormonal Symptoms	4	.56	1.64	(0.66)	53.7[a]	(12.0)

[a]Above Mexican population norm of 50 ($SD = 10$), $p < .001$.

Background variables and covariates. Sex (45% *male*_ 0, 55% *female*_ 1) was dummy-coded for use in these analyses, whereas age (range 18_94, M_ 36.8, SD_ 13.4) and education (range 0_19, M_ 7.9, SD_ 4.8) were scored in years. Assessed retrospectively, predisaster living conditions were scored as the mean of 5 items (α_ .76), each measured on a fourpoint scale (1_ not at all, 4_ a lot). The items captured the extent to which respondents had experienced shortages of food and water, crowding, lack of electricity, and problems with sanitation. Fifty-eight percent of the sample had experienced at least one of these problems to some degree (M_ 1.5, SD_ 0.6). The measure was included to control for impoverished conditions that could have adversely influenced health.

In addition, we included two predisaster measures of mental health, also retrospectively assessed by using Version 2.1 for DSM-IV (American Psychiatric Association, 1994) of the Composite International Diagnostic Interview (CIDI), developed and translated into Spanish by the World Health Organization (1997). The CIDI is a structured interview designed for use by trained lay interviewers. It has been used widely in prior epidemiologic studies (Kessler et al., 1995; Andrade et al., 2003), including one in Mexico (Norris et al., 2003). Lifetime PTSD was measured by using Module K, which assessed all DSM-IV Criteria for PTSD as they emerged after the worst event experienced in the respondent's lifetime, excluding the flood. Module E of the CIDI was used to measure lifetime major depressive disorder (MDD). We identified predisaster disorders by using items on the CIDI that indicated the onset of symptoms. Prevalences were 16% for predisaster PTSD and 13% for predisaster MDD.

Disaster exposure, postdisaster PTSD symptoms, and past-week depressed affect. Severity of exposure to the disaster was captured in an ordinal measure created by counting (1) whether respondents had experienced the death of a friend or family member (28%); (2) whether they or other household members were injured or had experienced an illness as a direct and immediate consequence of the flood (61%); (3) whether they felt that they were in danger of losing their

lives during the event (68%); (4) whether their dwellings were damaged to an extent perceived as *much* or *enormous* (47%); and (5) whether they had beendisplaced (31%). Injury, although physical in nature, does not appear to explain the trauma_health relation (Friedman & Schnurr, 1995). The correlation between self/ household injury/illness with total physical health in this sample was r (666)_ .22, p< .001. Severity of exposure was normally distributed, with 8% of participants scoring 0, 15% 1, 30% 2, 29% 3, 14% 4, and 4% 5.

Current (6-month) disaster-specific PTSD was measured by using a modified version of CIDI Module K for PTSD. These questions (located early in the interview) specifically referred to symptoms attributed to the flood, whereas the original module (located late in the interview) explicitly excluded the flood. All participants were asked all PTSD symptom questions. A count of affirmative responses to CIDI symptom questions (range 0-17) provided the continuous measure of postdisaster PTSD symptoms, $\alpha = .89$, that was used in this analysis. The sample averaged 7.8 criterion symptoms (SD= 4.9). We elected to use this variable rather than the diagnosis for several reasons. First, it is preferred for a mediating variable to be continuous in form because it must serve as a dependent variable in one aspect of the analysis. Continuous variables have greater variability than dichotomous ones, thereby increasing the power to detect an effect. Also, there was an exceptionally high degree of partial PTSD related to the disaster, which clouds the meaning of a dichotomous score. Altogether 24% of study participants met all 6 DSM-IV criteria for current disaster-specific PTSD; an additional 41% met 4-5 criteria. Of persons who did not meet all PTSD criteria, 82% met Criterion B (1+ intrusion symptoms), 28% met Criterion C (3+ avoidance symptoms), and 56% met Criterion D (2+ arousal symptoms). For more details about PTSD in this sample, (see Norris et al., 2004).

Data analysis and hypothesis tests

Separate hierarchical regression analyses were conducted for each dependent measure of physical health symptoms, i.e., for total health symptoms and each subscale. In the first step, we entered the demographic, predisaster, and severity of exposure measures. Age, gender, education, predisaster living conditions, predisaster PTSD, and predisaster MDD were all expected to predict health, such that adults who were older, female, or less educated or who had poorer living conditions, PTSD, or MDD before the disaster would exhibit more physical symptoms. We hypothesized also that health symptoms would increase as severity of exposure increased. In the second step, we entered postdisaster (disaster-specific) PTSD. We hypothesized that this variable would explain additional variance in all health domains and that the effects of exposure would decrease when postdisaster PTSD was controlled. In the third step, we entered past-week depressed affect. We hypothesized that depressed affect would also be related to physical symptoms. Although PTSD and depressed affect do share variance, we expected postdisaster PTSD to have independent or specific effects; if so, the effects of PTSD might decline but would remain significant when depressed affect was entered into the equation. A reasonable alternative sequence would have been to enter depressed affect first but we believed the chosen sequence provided more thorough information. Final betas are not influenced by the particular sequence.

Because we had directional hypotheses but five equations, we used a Bonferroni correction for five tests and set alpha to .01, one-tailed, for testing the significance of specific coefficients. For correlational analyses for which there were no hypotheses, we set alpha to .01, two-tailed.

Results

Hypothesis 1: Sample-level effects of disaster exposure

Table I shows sample means for the total health symptom scale and the subscales. In absolute terms, Muscular-Skeletal Symptoms were most prevalent, and Cardio-Pulmonary Symptoms were least prevalent. All standardized measures were significantly ($p < .001$) above Mexican population norms in one-sample t -tests. Effect sizes ranged from small but not trivial (.2) for Gastrointestinal/Urinary Symptoms to medium (.5) for Cardio-Pulmonary Symptoms (Cohen, 1992). In other words, Cardio-Pulmonary Symptoms were less prevalent than other health symptoms within this sample but showed the greatest effect because they were the most elevated relative to population norms.

This physical health effect for the sample overall could be explained merely by elevated depressed affect. Therefore we compared the physical health of the normative and disaster cities directly in a MANCOVA with depressed affect as a covariate. Although the covariate had an extremely large effect, *Multivariate* $F (5, 3164)\_ 200.91$, $p < .001$, the effect of city type (disaster vs. normative) was significant with depressed affect controlled, *Multivariate* $F (5, 3164) = 15.04$, $p < .001$. The city-level effect was strongest for Cardio-Pulmonary Symptoms, $F (1, 3168) = 43.71$, $p < .001$

Hypothesis 2: Effects of severity of exposure on health

Table II shows the intercorrelations of the predictor variables. The strongest correlations were between age and education (- .45), severity of exposure and postdisaster PTSD symptoms (.58), and postdisaster PTSD and past-week depressed affect (.42). Table III shows the results of the hierarchical regression analyses, including zero-order correlations between the predictors and physical health measures. Because of the multiple measures (5) and multiple steps (3), we present only the standardized results (βs) in the table. The dependent variables were standardized, and thus the unstandardized coefficients were less informative than were the relative strengths and significance levels of the standardized coefficients. However, the unstandardized regression coefficients (Bs) and their standard errors were used for testing mediation.

The set of demographic, predisaster, and exposure measures (Step 1) explained approximately 26% of the variance in total health symptoms. As Table III shows, age was positively related to total health symptoms, Cardio-Pulmonary Symptoms, and Muscular- Skeletal Symptoms. Female gender was positively associated with all health domains except Nose-Throat Symptoms. Education did not show independent effects. Predisaster living conditions, MDD, and PTSD were each positively and independently related to all health symptom domains.

With these demographic and predisaster variables simultaneously controlled, severity of exposure to the disaster was positively related to health symptoms for all domains except Nose-Throat Symptoms; the more severe the exposure, the higher were self-reported physical symptoms. When entered hierarchically, exposure uniquely accounted for approximately 1-2% of the variance across health domains.

Table II. Correlations between predictor variables ($N = 657$).

	1	2	3	4	5	6	7	8	9
1. Age	1.0								
2. Female gender	.03	1.0							
3. Education	−.45**	.01	1.0						
4. Predisaster living conditions	.10	−.04	−.27**	1.0					
5. Predisaster PTSD	−.03	.08	−.11	.07	1.0				
6. Predisaster MDD	.12	.13**	−.01	.03	.09	1.0			
7. Severity of exposure	.10	.04	−.30**	.22**	.09	.05	1.0		
8. Postdisaster PTSD symptoms	.19**	.16**	−.33**	.25**	.27**	.16**	.58**	1.0	
9. Past week depressed affect	.04	.16**	−.22**	.23**	.20**	.14**	.24**	.42**	1.0

PTSD = posttraumatic stress disorder. MDD = major depressive disorder. **$p < .001$.

Table III. Effects of severity of exposure and psychological symptoms on health symptom domains: standardized betas by step.

	Total Health Symptoms				Cardio-Pulmonary Symptoms				Muscular-Skeletal Symptoms			
Variables	*r*	Step1	Step2	Step3	*r*	Step1	Step2	Step3	*r*	Step1	Step2	Step3
Age	.22**	.15**	.14**	.17**	.25**	.17**	.15**	.18**	.27**	.21**	.20**	.22**
Female sex	.22**	.18**	.15**	.12**	.14**	.11*	.08*	.06	.19**	.16**	.14**	.12**
Education	−.21**	−.02	.01	.04	−.24**	−.07	−.05	−.02	−.21**	−.02	.00	.02
Predisaster living conditions	.28**	.21**	.19**	.15**	.23**	.16**	.14**	.10*	.22**	.16**	.14**	.11**
Predisaster PTSD	.22**	.17**	.12**	.10*	.18**	.13**	.09*	.07	.17**	.13**	.10*	.08
Predisaster MDD	.25**	.18**	.16**	.13**	.22**	.17**	.15**	.12**	.24**	.17**	.15**	.13**
Severity of exposure	.25**	.15**	.04	.05	.21**	.12**	.01	.02	.22**	.13**	.06	.07
Postdisaster PTSD symptoms	.41**		.23**	.12*	.37**		.22**	.11*	.33**		.15**	.07
Past-week depressed affect	.49**			.34**	.44**			.31**	.38**			.25**
R^2 Change		.256**	.030**	.091**		.196**	.027**	.075**		.215**	.013**	.047**
Adjusted R^2 (9,647)				.369**				.288**				.265**
Final Multiple R				.614**				.546**				.525**

	Nose-Throat Symptoms				Gastrointestinal/Urinary Symptoms			
Variables	*r*	Step1	Step2	Step3	*r*	Step1	Step2	Step3
Age	.08*	.05	.03	.05	.09*	.09	.07	.09
Female sex	.09*	.07	.05	.03	.19**	.16**	.13**	.11*
Education	−.11*	.00	.02	.04	−.06	.08	.11*	.14**
Predisaster living conditions	.21**	.17**	.16**	.13**	.19**	.16**	.14**	.10*
Predisaster PTSD	.18**	.15**	.11*	.10*	.19**	.15**	.10*	.08
Predisaster MDD	.15**	.12**	.10*	.08	.18**	.13**	.10**	.08
Severity of exposure	.14**	.08	−.01	.00	.19**	.14**	.02	.03
Postdisaster PTSD symptoms	.26**		.17**	.10	.34**		.25**	.16**
Past-week depressed affect	.33**			.23**	.49**			.27**
R^2 Change		.101**	.017**	.040**		.140**	.035**	.057**
Adjusted R^2 (9,647)				.145**				.222**
Final Multiple R				.396**				.482**

MDD = major depressive disorder. PTSD = posttraumatic stress disorder. *$p < .01$, one-tailed. **$p < .001$, one-tailed.

Hypothesis 3: PTSD as a mediator of the exposure-health relation

The continuous measure of postdisaster (disaster-specific) PTSD symptoms was enteredinto the equations in the second step, explaining the 3% additional variance in total healthsymptoms (see Table III). Postdisaster PTSD made unique contributions to all symptom domains: the greater the number of PTSD symptoms, the poorer the health. Notably, the effects of severity of exposure dropped out of all equations when postdisaster PTSD was taken into account.

This apparent evidence of mediation was further verified in a series of analyses (following Baron & Kenny, 1986) that explicitly tested the indirect effects of exposure on health through PTSD. This test is based on the notion that the total effect on a dependent variable is equal to the direct effect from the independent variable plus any indirect effects through another variable. These tests were conducted for all health scales except Nose-Throat Symptoms, the one health domain for which no effect of severity of exposure was observed before PTSD was entered. To test for the necessary relationship between the independent variable and the proposed mediator, postdisaster PTSD was regressed on the same exogenous variables included in the first step of the analysis for physical health. In this equation, which explained 43% of the variance, $p < .001$, severity of exposure to the disaster was strongly and positively related to PTSD, $B\_$ 1.904, *SE* B_ 0.123, $\beta\_$ .48, $p < .001$. Thus a significant relation between exposure (the independent variable) and postdisaster PTSD symptoms (the hypothesized mediating variable) was established. This information was combined with the nonstandardized betas and standard errors corresponding to the tests of the relations between PTSD and health (the dependent variables) to get the indirect effect for each scale. Sobel's formula (cited by Baron and Kenney, 1986) was used to derive the standard errors of the indirect effects. The indirect effects of exposure were significant in all tests, as shown in Table IV. Together these data provided strong evidence of mediation.

Hypothesis 4: Specificity of the effects of PTSD on health

Past-week depressed affect was entered in the third and final step of the hierarchical regressions (Table III), explaining approximately 9% of additional variance in total health symptoms and 4% to 8% of the variance in specific domains. The effects of predisaster MDD decreased in all equations when postdisaster depressed affect was entered, suggesting that the effects of past MDD on current health were largely mediated by its relation with current depressive symptoms. The effects of postdisaster PTSD were greatly diminished when the effects of depressed affect were simultaneously controlled, but remained significant for total health symptoms, Cardio-Pulmonary Symptoms, and Gastrointestinal/ Urinary Symptoms.

Table IV. Indirect effects of severity of exposure on physical health symptoms through PTSD.

Dependent variable (Health domain)	Indirect effect	*SE*	*Z*
Total Health Symptoms	1.12	0.23	4.95**
Cardio-Pulmonary Symptoms	1.19	0.26	4.51**
Muscular-Skeletal Symptoms	0.65	0.20	3.25**
Gastrointestinal/Urinary Symptoms	1.10	0.22	4.97**

**$p < .001$.

For these three variables, tests of mediation were repeated, this time using the Step 3, rather than Step 2, coefficients and standard errors, to control for depressed affect. PTSD symptoms again mediated the effects of exposure on the three measures of health: indirect effect of exposure on total physical symptoms= 0.56, *SE*= .21, *z*= 2.65, *p*< .01; indirect effect on Cardio-Pulmonary Symptoms= 0.61, *SE*= .25, *z*= 2.42, *p*< .01; indirect effect on Gastrointestinal/Urinary Symptoms= 0.69, *SE*= .21, *z*= 3.24, *p*< .001.

To examine the relative impact on health of the various symptoms of PTSD, we conducted additional exploratory analyses. For the total health symptom scale and each of the 17 criterion PTSD symptoms, Table V shows the zero-order correlation and the partial correlation controlling for age, sex, predisaster living conditions, predisaster PTSD and MDD, and past-week depressed affect. Results for the total health scale are shown; the subscales showed the same pattern of results. The analysis further clarified the influence of depressed affect on the PTSD-health relation. Of the zero-order correlations, 16 of 17 were significant at the p< .001 level. Of the partial correlations, only 4 were significant at *p*< .001, 8 at *p*< .01. The PTSD symptoms showing specific effects were predominantly from the arousal cluster (4 of 5) and intrusion cluster (3 of 5), with only 1 of 7 avoidance symptoms showing a specific effect on health with depressed affect controlled. The lack of correlation between hyper-vigilance (D4) and health may be attributable to its exceptionally high frequency (81%) in this sample.

These results suggested that the specific health impact of intrusion and avoidance symptoms may be masked by the inclusion of avoidance/numbing symptoms in summary measures of PTSD. Thus in a final exploratory analysis, the regressions were repeated. The equations were identical to those shown in Table III except that the count of all PTSD symptoms (range 0 to 17) was replaced by a count of the PTSD symptoms that showed specific effects in Table V (range 0 to 8). With this revision, PTSD symptoms were related to all clusters of health symptoms including Muscular_Skeletal Symptoms and Nose-Throat Symptoms (see Table VI). With depressed affect controlled, the betas for PTSD symptoms decreased in magnitude but remained statistically significant.

Table V. Correlations and partial correlations of specific PTSD criterion symptoms with total health symptoms.

PTSD Cluster or symptom	%	*r*	*pr*
Intrusion (B, count 0–5)		.36**	.14**
B1. Remembered when didn't want to	83.7	.16**	.08
B2. Nightmares	46.0	.28**	.14**
B3. Felt as though happening again	58.1	.29**	.10*
B4. Upset when reminded	29.7	.19**	−.01
B5. Physiological reactivity	46.3	.34**	.13**
Avoidance/Numbing (C, Count 0–7)		.34**	.07
C1. Avoided thinking about event	51.9	.23**	.08
C2. Avoided reminders	38.2	.23**	.05
C3. Memory blank for all or part	30.9	−.02	−.03
C4. Lost interest in things	35.5	.35**	.14**
C5. Felt distant from others	39.7	.27**	.03
C6. Emotionally numb	34.3	.20**	−.01
C7. No point in thinking about future	26.5	.23**	−.01
Arousal (D, Count 0–5)		.39**	.16**
D1. Trouble sleeping	42.4	.31**	.13**
D2. Anger or irritability	42.5	.31**	.12*
D3. Difficulty concentrating	49.9	.33**	.12*
D4. More concerned about danger	80.8	.17**	.06
D5. Jumpy or easily startled	50.1	.33**	.11*

% is the prevalence of the symptom in the sample. Partial correlations (*pr*) were computed controlling for age, sex, predisaster living conditions, predisaster PTSD and MDD, and past-week depressed affect. With listwise deletion, $n = 656$. *$p < .01$. **$p < .001$.

Table VI. Effects of PTSD symptom subset on health symptoms before and after depressed affect is controlled.

Health symptom scale (DV)	Before depressed affect β	After depressed affect β
Total Health Symptoms	.28**	.17**
Cardio-Pulmonary Symptoms	.26**	.16**
Muscular-Skeletal Symptoms	.20**	.13*
Nose-Throat Symptoms	.21**	.15*
Gastrointestinal/Urinary Symptoms	.26**	.18**

DV = Dependent variable. Independent variables in the analysis were the same as shown in Table III except that an 8-item subset of the PTSD symptom measure replaced the original 17-item version. *$p < .01$, one-tailed. **$p < .001$, one-tailed.

Discussion

Consistent with our first hypothesis, this sample of disaster victims, as a whole, exhibited levels of physical health symptoms that were significantly higher than population norms for Mexico. Across domains, the mean level of health symptoms in this sample was nearly a half standard deviation higher than the norm. An effect of this magnitude is ''likely to be visible to the naked eye of a careful observer'' (Cohen, 1992; p. 156), a notion that is truly striking when extrapolated to an entire population. Although the regression analyses controlling for a variety of predisaster variables provided methodologically stronger evidence that the health symptoms

were, in fact, attributable to the disaster and its sequellae, these descriptive, population-level findings may best reveal the public health implications of large-scale community disasters. Moreover, differences between the disaster sample and the normative sample in health remained significant even when differences between them in depressed affect were controlled.

The study also shed some light on the domains of public health that might be most susceptible to disaster effects. In absolute terms, Cardio-Pulmonary symptoms were least prevalent, but they were nonetheless the most elevated relative to normative levels in the general population. Notably, this finding from an epidemiologic investigation converges with findings from previous laboratory investigations and suggests that, like other traumaexposed individuals, disaster victims may experience excessive sympathetic nervous system reactivity that increases vulnerability for cardiovascular illness (Friedman & McEwen, 2004). Given that health consequences are usually the result of chronic wear and tear, it is also of note that these effects were observed here only six months after the focal event.

Whereas the first hypothesis concerned sample, or population, level effects, our remaining questions concerned individual differences within the sample. As expected on the basis of past research, health symptoms were more prevalent among older adults, women, those with impoverished living conditions before the disaster, and those with predisaster PTSD and MDD. Consistent with our second hypothesis, severity of exposure to the disaster was modestly related to health symptoms with these demographic and predisaster conditions controlled. This finding further supports our overriding conclusion that disaster victims are at risk for physical health problems, at least for subjective physical health problems, and additionally suggests that these risks grow as exposure grows increasingly severe. This is consistent with previous research showing somatic expressions of distress to be common in Mexico and Central and South America (Escobar et al., 1987; Jenkins, 1996; Norris et al., 2001) and among Mexican American and other Latino populations in the United States (Canino et al., 1999; Eisenman et al., 2003). In no case, however, did the effects on health of recent trauma surpass those associated with the longerterm consequences of predisaster impoverished living conditions, including shortages of food or water, crowding, and inadequate sanitation. It is important to retain a sense of perspective about the relative contributions of trauma and poverty when conducting research on health in the developing world.

Our third hypothesis was two-fold. Consistent with our expectations, as well as with past research on other types of trauma, postdisaster PTSD symptoms were related to past-month physical health symptoms. For each health domain, the effects of recent, postdisaster PTSD symptoms overshadowed those of past, predisaster PTSD. Moreover, consistent with research on sexual assault (Kimerling & Calhoun, 1994; Zoellner, Goodwin, & Foa, 2000) and traumatic exposure of refugees (Van Ommeren et al., 2002), the effects of exposure dropped out of each equation when PTSD symptoms were entered. This finding provides strong support for the mediational model proposed by Schnurr and colleagues (Schnurr & Green, 2004; Wolfe et al., 1994; Friedman & Schnurr, 1995). Whereas previous findings show health effects long after trauma, this effect provides evidence of these physical health effects following recent traumatic stressors. Whether or not these physical symptoms are medically verifiable, there exists an adverse relationship between subjective health and acute PTSD. These findings have important implications for public health interventions in the aftermath of disaster because they

suggest, first, that addressing the emotional consequences of the event is critical for restoring physical health and, second, that informing medical practitioners about these effects may be an effective outreach strategy.

Our final hypothesis was that PTSD would show a specific effect on health not accounted for by the strong and pervasive influence of depressed affect on self-reported PTSD and physical symptom scores. This hypothesis was supported, but only modestly. Consistent with previous findings (see review of Ford, 2004), recent depressed affect showed a strong relationship to physical health. The effects of postdisaster PTSD remained significant in 3 of the 5 primary analyses (total health, cardiopulmonary, and gastrointestinal symptoms), but the un-confounded effects were weak in magnitude. However, the exploratory analysis suggested that the specific effects are confined to particular criterion symptoms and are therefore masked by summary measures. After controlling for depressed affect, specific effects were predominantly from the arousal cluster (e.g., sleep disruption, startle) and intrusion cluster (e.g., physiological reactivity, nightmares), although there were symptoms in each cluster that did and did not show specific effects.

In summary, within the limits of our study's correlational methodology, we have concluded: (1) that this disaster in Mexico affected the physical health of survivors; (2) that these effects were most pronounced in persons who were severely exposed to this disaster; (3) that the health consequences of exposure were mediated by subsequent symptoms of PTSD; and (4) that these effects were not solely attributable to the depressed affect that was high and pervasive in the stricken communities and substantially correlated with both PTSD and health.

In closing, the weaknesses and some strengths of our study should be reviewed. For the topic at hand, the study's greatest limitation was its reliance on self-report measures of health and the lack of a physical health measure for this sample prior to the disaster. Given the nature of disaster research, the size of the sample and location of the study, it was neither economically nor practically feasible for us to collect prior or objective measures of physical health. Most conservatively, our findings describe subjective health rather than verified morbidity, but this is also true of most other studies conducted outside of clinics or laboratories. Further research is needed to confirm the mediating role of PTSD symptoms on the relationship of disaster exposure and physical health using objective measures of physical health. In addition, research is needed that relates somatic complaints to functional impairments. A related shortcoming is that the health measure had not been used in Mexico previously (excluding our own parallel normative study); thus, although its factor structure provided reasonable evidence of construct validity, we cannot assert with certainty that the measure is cross-culturally valid.

Another issue, of course, is the difficulty in establishing causation in a non-experimental design. It is plausible that the disaster's effects were confounded with the impoverished conditions of many of the study's participants. We attempted to address this problem by controlling for pre-existing living conditions, education level, and mental health status. An additional shortcoming of the present analysis was its cross-sectional design. The sample was assessed longitudinally but, given the multiple domains of health to be examined and the large set of potential predictors, it seemed advisable to first explore the initial wave of findings in considerable depth. Our work

continues, proceeding to test the presumably complex, reciprocal, and possibly lagged relations of PTSD and health over time.

Another shortcoming is that we did not attempt to examine interactive effects between the various predictors. It is possible that pre-existing mental health problems serve as moderators of the effects of current depressed affect and acute PTSD on health. Given the multiple domains of health to be examined and the complexity of testing moderation in combination with mediation, it seemed advisable to address this question after the main effects of exposure and PTSD had been thoroughly explored.

Our study also had several strengths. Our parallel four-city normative study has helped us immensely in interpreting various self-report measures where there was little prior research in Mexico to guide us. A particular strength was our sample that was composed of representative community-dwelling men and women, ranging in age from 18 to 94, whose selection was independent of whether or not they sought physical or mental health treatment. Although our procedures detected some selection bias for men, these effects were very small in magnitude and unlikely to have influenced these results. Our participants all experienced an objectively definable event at the same point in time. This feature does not eliminate ‘‘third variable’’ problems (i.e., that an unmeasured variable leads both to the trauma exposure and to the problems with health) but should minimize them. Because we controlled for various predisaster conditions, as well as for past-week depressed affect, our tests of the relation between PTSD and health were more conservative than most that have been reported in the literature. In addition, we attempted to remove confounds of overlap in the measures of health, depressed affect, and PTSD. Even in the United States, the relations between trauma and physical health have seldom been studied so closely in time to the focal event; thus our finding that the health effects of PTSD begin while the symptoms are still fairly acute extends previous research in an important way. More broadly, our research is important for its ability to show that theories linking psychological and physical disorders are cross-culturally viable. On several key dimensions of culture (e.g., individualism, power distance, uncertainty avoidance; Hofstede, 1980), Mexico and the United states are very different, yet we have observed more similarities than differences in our trauma research there to date.

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