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The Stress-Buffering Effects of Received Social Support on Posttraumatic Stress Disorder Among Hurricane Ike Survivors

Chenyi Ma[1], Tony E. Smith[2], and Dennis P. Culhane[1]
[1] University of Pennsylvania School of Social Policy & Practice
[2] University of Pennsylvania School of Engineering and Applied Science

Hurricane Ike made landfall in the Galveston Bay Area in 2008, destroying parts of the built and natural environment, and leaving survivors with a high prevalence of posttraumatic stress disorder (PTSD); however, equally striking was the rapid decline of PTSD among survivors during the 18 months following this disaster. Existing literature suggests that social support tends to either directly or indirectly mitigate such mental health outcomes by moderating disaster-related stress. The present article builds on this to analyze the possible factors contributing to PTSD recovery, including both Ike-related stress suffered by victims and the social support they received over time. We used the Galveston Bay Recovery Study public use file, which includes three waves of interviews for selected victims ($n = 658$) and employed a series of nested random-effect logistic regression models to test two hypotheses: (a) that social support received by victims buffered the positive relationship between Ike-related stress and PTSD risk and (b) that this buffering effect (BE) diminished over time. Our results provide strong evidence for such BEs ($\beta = -0.03$, $p = .009$), and, through the use of three-way interaction analysis corrected for multiple testing, we show that the observed BE did indeed diminish over time ($p = .045$). PTSD resulting from disaster-related stress can be substantially reduced by social support interventions. Policy formulation should focus on early social support interventions, given they are most effective in PTSD recovery.

Keywords: buffering effect, disaster-related stress, Hurricane Ike, posttraumatic stress disorder, received social support

Posttraumatic stress disorder (PTSD) is a psychiatric disorder that is usually observed among survivors who experienced or witnessed traumatic events like those that may occur during a disaster. Examples include sustaining physical injury, witnessing the death of a family member or a close friend, seeing dead bodies, and being robbed or assaulted, among others (American Psychiatric Association, 2024; Tracy et al., 2011). Additionally, since PTSD comes with a series of symptoms that may include flashbacks, nightmares, severe anxiety, and uncontrollable thoughts about traumatic events, those who experience it are also at risk of developing other severe mental health disorders. For example, the U.S. National Comorbidity Survey reports individuals with PTSD were 6 times as likely as those without PTSD to attempt suicide (Kessler, 2000). In addition to adversely impacting an individual's health and behavioral outcomes, the costs to society are also substantially high. For example, prior research demonstrates that in 2018 alone PTSD caused a total economic burden of $232.2 billion (Davis et al., 2022), more than 1% of the U.S. Gross Domestic Product for that year (Bureau of Economic Analysis, 2019). While it is necessary to determine a cost-effective intervention to address PTSD among disaster survivors, it is imperative that the psychopathological factors of PTSD are fully explored first, especially for those who experienced stressful events due to disasters.

The existing literature well documented that many survivors' post-disaster lives were disrupted by stressful events caused by disasters, such as property/material losses (McKinley et al., 2019; Norris et al., 2010), job losses (Ferreira et al., 2018; Hobfoll, 1988; Ma et al., 2021) and displacements from homes (Ferreira & Figley, 2015; Galea et al., 2007; Ma, 2018; Ma & Culhane, 2024). Consequently, the survivors who were exposed to these disaster-related stressors were particularly susceptible to PTSD and in need of social support (McLaughlin et al., 2011). For example, many Hurricane Karina survivors with PTSD who lost their real properties and were displaced from their homes were in great need of financial support for permanent housing (Glass et al., 2009). Melrose et al.' study (2015) suggests people in need of social support usually received it. Nevertheless, survivors may receive social support at different time points since their exposure to disaster-related stressors (Norris & Kaniasty, 1996). Yet, it has remained unclear to what temporal extent received social support could minimize the impact brought by disaster-related stressors on PTSD. Observing Hurricane Ike survivors in Galveston Bay Area (GBA), this secondary data analysis study aims to fill this research gap.

Hurricane Ike made landfall in the GBA in September 2008, and was the ninth costliest hurricane in U.S. history, killing a total of 112 persons in Texas and leaving survivors with severe psychological impacts (Insurance Information Institute, 2024; Liddell et al., 2020, Lin et al., 2014). Among these impacts, PTSD was found to be most prevalent among survivors in GBA immediately following Ike. However, it has also been documented that the prevalence of PTSD

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Chenyi Ma https://orcid.org/0000-0003-1051-9538
Dennis P. Culhane https://orcid.org/0000-0002-2332-5567
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Correspondence concerning this article should be addressed to Chenyi Ma, University of Pennsylvania School of Social Policy & Practice, 3718 Locust Walk, McNeil Building, Philadelphia, PA 19104, United States. Email: machenyi@upenn.edu

declined dramatically within the first 18 months following the disaster (Pietrzak et al., 2012). In the present article, we focus on the possible factors contributing to this decline. Of central interest are the Ike-related stress suffered by victims and the social support received by these victims over time. The relations between support and PTSD have been previously studied for Ike survivors by Platt et al. (2016), who identified both temporal and bidirectional relations between post-traumatic stress (PTS) symptoms and three distinct types of received social support (emotional, informational, and tangible). In a separate study, Lowe et al. (2013) identified temporal relations between the PTS/depressive symptoms of Ike survivors and both the immediate (Ike-related) stressors and longer term stressors suffered by these victims. In the present study, our primary goal is to combine these two lines of investigation by focusing on the possible interactive effects of both Ike-related stress and received social support on PTSD over time.

Our specific approach is largely motivated by the buffering model of social support which postulates both that exposure to stressful disaster events is a major risk factor for PTSD, and that social support serves primarily to buffer the effects of this risk factor (Cohen & Wills, 1985; Fleming et al., 1982; Kaniasty & Norris, 2009; Kawachi & Berkman, 2001). In these terms, the study of Lowe et al. (2013) focuses on the temporal aspects of stressful disaster events as a risk factor for PTSD. In contrast, the study of Platt et al. (2016) focuses on the temporal relations between support and PTS symptoms, and can be viewed as a temporal extension of the main model of social support, which postulates a direct relation between support and mental health outcomes (Cohen & Wills, 1985; Gibbons et al., 2010; Kaniasty & Norris, 2009; Kawachi & Berkman, 2001; Uchino, 2004). The main objective of the present study is thus to extend these temporal considerations to the role of support in buffering the effects of stressful disaster events on PTSD. Like Kaniasty and Norris (2009), we believe that the identification of such temporal effects may help to improve the delivery process of support resources with respect to mental health recovery.

For purposes of analysis, we postulate the following set of explicit hypotheses which effectively add temporal dimensions to both main-model and buffering-model perspectives of the interrelations between support, stress, and PTSD:

> *Hypothesis 1 (H1):* (Hypothesis 1a [H1a]) There is a positive association between survivors' levels of Ike-related stress and their likelihood of PTSD after Ike, and (Hypothesis 1b [H1b]) This positive association decreases over time.

> *Hypothesis 2 (H2):* (Hypothesis 2a [H2a]) There is a negative association between survivors' levels of received social support and their likelihood of PTSD after Ike, and (Hypothesis 2b [H2b]) This negative association decreases over time.

> *Hypothesis 3 (H3):* (Hypothesis 3a [H3a]) Higher levels of received social support tend to buffer the positive relationship between Ike-related stressors and the likelihood of PTSD after Ike. and (Hypothesis 3b [H3b]) This buffering effect (BE) decreases over time.

Method

As in Lowe et al. (2013) and Platt et al. (2016), the data used for this study were taken from the Galveston Bay Recovery Study (GBRS) public use file (PUF), accessed online at the Inter-University Consortium for Political and Social Research (National Center for Disaster Mental Health Research et al., 2016). GBRS is an epidemiological and mental health study of ($N = 658$) disaster survivors drawn from the adult population residing in Galveston or Chamber counties at least 1 month before Hurricane Ike made landfall.

Sampling

GBRS employed a stratified sampling method to ensure the inclusion of those GBA residents who were most likely to have experienced hurricane-related traumatic events (as detailed in GBRS PUF). Telephone and face-to-face interviews were conducted in three waves, respectively, 2–5, 6–9, and 14–18 months after the hurricane. Given that our present study focuses on both the short- and long-term impacts of Hurricane Ike on survivors, we have used all three waves of interview data, including both time-invariant and time-varying attributes of survivors.

Measurement

Time-invariant variables were measured in Wave 1 exclusively. As detailed in Table 1, these variables include demographic and socioeconomic variables, PTSD status prior to Ike, Ike-related stress, and Ike-related trauma. In particular, Ike-related stress is operationally defined as the number of "yes" answers to seven questions, such as being "displaced from home for more than 1 week" by Ike. Ike-related trauma was originally defined as the number of "yes" answers to four questions related to survivors' traumatic experiences from Ike, such as "a family member or close friend killed as a result of Ike." For purposes of analysis, this was simplified to a categorical variable with ordinal values "zero," "one," and "two or more." The time-varying variables were measured in all three waves, as detailed in the following paragraphs and in Table 2.

According to the GBRS code book (National Center for Disaster Mental Health Research et al., 2016), Ike-related PTSD, is operationally defined by a set of six criteria based on survivors' responses to a series of questions assessing standard symptoms of PTSD from the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2000). This measurement exhibits high internal consistency across waves, Cronbach's $\alpha = 0.95$, 0.95, and 0.96 for wave 1, wave 2, and wave 3, respectively (2).

The continuous variable, post-Ike stress, was measured as the number of "yes" answers to 12 questions, such as "being displaced from home" or "experiencing financial loss" as a result of the hurricane. The categorical variable, post-Ike trauma was constructed from a list of 10 types of traumatic events that may have been experienced by respondents, such as "the unexpected death of someone close" or "being robbed or mugged." These two variables are controlled for, given their potential effects on PTSD (Hobfoll, 1989).

We adopt a holistic view which is more appropriate for gauging the overall stress-BEs of received social support. It should also be noted that the high degree of internal consistency between these three support types ("emotional," "informational," and "tangible") found by Platt et al. (2016), $\alpha = .85$, .84, and .86 for Waves 1, 2, and 3, respectively, adds further support to this holistic viewpoint. In each wave, survivors were asked to respond to whether they received each of 11 possible support types since Ike (or since last interview), with four possible answers, 1 = *never*, 2 = *once or twice*, 3 = *a few*

Table 1
Summary Statistics for the Imputed Values of Time-Invariant Respondent Characteristics

Time-invariant variable	*N*	%
Race/ethnicity		
White	393	59.73
Black	105	15.96
Hispanic	124	18.84
Other	36	5.47
Total	658	100
Marital status		
Married	310	47.11
Living with a partner	33	5.02
Separated	35	5.32
Divorced	84	12.77
Widowed	75	11.4
Never been married	121	18.39
Total	658	100
Gender		
Female	394	59.88
Male	264	40.12
Total	658	100
Income		
<\$40,000[a]	291	44.22
≥\$40,000	367	55.78
Total	658	100
Educational attainment		
<High school	89	13.53
High school	157	23.86
Some college	190	28.88
≥College degree	222	33.74
Total	658	100
Employed before Ike		
No	280	42.55
Yes	378	57.45
Total	658	100
PTSD prior to Ike		
No	574	87.23
Yes	84	12.77
Total	658	100
Ike-related trauma		
None	581	88.3
One	65	9.88
Two or more	12	1.82
Total	658	100
Ike-related stress[b]	658	$M = 3.09$ $SD = 1.749$

Note. PTSD = posttraumatic stress disorder.
[a] The median household income category is \$40,000–59,999. Household income less than \$40,000 is below the median income category. [b] Continuous variable.

times, or 4 = *many times*. Our continuous received total support variable is then defined to be the total of these numerical responses (with values from 11 to 44).

Existing studies suggest that these mental health services may play a role in reducing PTSD symptoms (2013), and thus constitute a relevant control variable for the present study. The dichotomous mental health services variable is defined as "yes" if survivors received any mental health services since Ike or since last interview, and "no" otherwise.

Finally, we include one time-varying dichotomous economic variable, namely whether household income has declined since Ike (Wave 1) or since the last interview (Waves 2 and 3). Such loss of income may contribute to PTSD symptoms, and should thus be controlled for.

Missing Data and Data Imputations

GBRS-PUF includes 658 Ike survivors ($n = 658$) who completed the Wave 1 interview, of which 529 (80%) completed Wave 2 interview, and 487 (74%) completed Wave 3 interview. Only 448 completed all three waves, yielding an attrition rate of about 30% (210 out of 658). Following both Lowe et al. (4) and Platt et al. (3), we employed the five sets of imputed data provided by GBRS-PUF (which is based on the sequential regression imputation method implemented in IVEWARE software, Raghunathan et al., 2007, using Rubin's [15] correction of standard error). Additional testing by Platt et al. (2016) suggests that there are no statistically significant differences between the group completing all three waves (448) and the group that did not (210). This adds further support for the missing-at-random (MAR) assumption required for statistical consistency of such imputed values.

Statistical Analysis

Our analysis begins in Tables 1 and 2 with statistical summaries of our time-invariant and time-varying variables, respectively. To test for possible time dependencies in each categorical time-varying variable, Pearson χ^2 goodness-of-fit tests were employed (with an implicit null hypothesis of independence). Similarly, paired t tests were employed to test for possible time dependencies in each continuous time-varying variable, where separate tests were conducted for Wave 2 relative to Wave 1 and for Wave 3 relative to Wave 1.

Our main modeling efforts focus on the three hypotheses specified in the introductory part. Here we employ subject-specific random-intercept logistic regression models to analyze the influence of both time-invariant and time-varying attributes of respondents on PTSD outcomes over time. Such models can be represented generically as follows. If p_{ij} denotes the probability of PTSD for individual i in time period (Wave) j, then each model takes the form,

$$\log\left(\frac{p_{ij}}{1-p_{ij}} \mid v_i\right) = (\beta_0 + v_i) + \beta_1 t_j + \sum\nolimits_k \beta_k x_{ik} + \sum\nolimits_h \beta_h x_{ijh}, \tag{1}$$

where the dependent log-odds variable is here referred to as PTSD risk. The conditioning statement reflects the fact that v_i is a random variable summarizing relevant unobserved time-invariant attributes of individual, i. The time variable, t_j, is here made explicit (with values $t = 0, 1, 2$ for waves $j = 1, 2, 3$, respectively). The last two terms denote, respectively, time-invariant variables (such as Ike-related stress) and time-varying variables (such as received social support). Note also the possible interactions between time, t_j, and time-invariant variables are included in this last term.

There are several reasons for this choice of models. First, standard fixed effects models do not allow us to identify relations between PTSD risk and time-invariant predictors (Allison, 2009), including the key variables Ike-related stress and trauma. Second, our focus is on subject-specific differences in PTSD remission, which cannot be analyzed by population-average models, such as standard logistic regression or generalized estimation equation models (Rabe-Hesketh & Skrondal, 2008). Finally, with respect to missing data considerations, it should be noted that the multiple imputation procedure used for GBRS data requires that such data be MAR. While the random intercept model is consistent with MAR, this is

Table 2
Summary Statistics for the Imputed Values of Time-Varying Respondent Characteristics

Time-varying variable	Wave 1, *N* (%)	Wave 2, *N* (%)	Wave 3, *N* (%)	Test statistics[a]
PTSD[b]				14.20***[c]
No	598 (90.88)	505 (95.46)	465 (95.48)	
Yes	60 (9.12)	24 (4.54)	22 (4.52)	
Total	658	529	487	
Post-Ike trauma				152.83***
None	602 (91.49)	428 (80.91)	308 (63.24)	
One	51 (7.75)	81 (15.31)	123 (25.26)	
Two or more	5 (0.76)	20 (3.78)	56 (11.5)	
Total	658	529	487	
Income decline				2.16
No	534 (81.16)	427 (80.72)	409 (83.98)	
Yes	124 (18.84)	102 (19.28)	78 (16.02)	
Total	658	529	487	
Mental health service				13.67***
No	605 (91.95)	469 (88.66)	414 (85.01)	
Yes	53 (8.05)	60 (11.341)	73 (14.99)	
Total	658	529	487	
Post-Ike stress[d]				
M	0.3	0.91	1.39	10.31***[e]
SD	0.67	1.309	1.69	14.83***[f]
Social support[d]				
M	27.77	25.86	26.74	−6.94***[e]
SD	7.578	7.318	7.701	−3.41***[f]

Note. PTSD = posttraumatic stress disorder.
[a] Pearson χ^2 goodness-of-fit tests were employed to test for possible dependencies between values in Waves 1, 2, and 3 for each time-varying categorical variable. [b] For the time-varying dichotomous variable (PTSD), McNemar's test was used to test for significant differences in PTSD frequencies between Waves 2 and 1 ($p = .002$), and between Waves 3 and 1 ($p = .014$), respectively. [c] *** $p < .01$. [d] For the time-varying continuous variables (post-Ike stress and social support), paired t tests were used to test for significant differences between mean values in Waves 1 and 2 and between mean values in Waves 1 and 3, respectively. [e] Paired T statistic for the mean difference between Wave 2 and Wave 1. [f] Paired T statistic for the mean difference between Wave 3 and Wave 1.

not true of population average models which typically require that such be missing completely at random (Gibbons et al., 2010; Hu et al., 1998). In our context, for example, generalized estimation equation is known to produce inconsistent estimates if the probabilities of survivor responses in any wave depend on their responses in other waves (Rabe-Hesketh & Skrondal, 2008, p. 561).

Within this analytical framework, we construct a sequence of four regression models, where the first two models versus the last two models roughly reflect the dichotomy between the main model and buffering model of social support discussed in the introductory part. Here we treat these models statistically (following the original work of Cohen & Wills, 1985) as "main effects" (MEs) and "interaction effects," respectively. In particular, the first ME model focuses on possible direct effects of survivor attributes on PTSD risk, including our key Ike-related stress and received social support variables. The second model, main effects over time (MET), allows possible variations in these direct effects over time (i.e., time interaction effects). The third BEs model allows the social support received by survivors to moderate (buffer) the relation between their levels of Ike-related stress and PTSD risk. Finally, our fourth buffering effects over time (BET) model allows this possible moderating (buffering) effect of social support to vary over time. This involves a higher order (three-way) interaction effect that requires special treatment from a statistical viewpoint, as detailed in the Results section below.

Results

Our key results are summarized in terms of the three hypotheses formulated in the introductory part. Because these hypotheses were implicitly motivated in terms of the dramatic decline in PTSD prevalence (Pietrzak et al., 2012), it is appropriate to begin by noting that our results provide additional support for this finding. In particular, Table 2 shows that PTSD prevalence rates decreased from 9.12% in Wave 1 to 4.52% in Wave 3. In addition, McNemar's pairwise χ^2 test for this dichotomous variable (Footnote b in Table 2) shows that differences in PTSD frequencies between Wave 1 and Wave 3 were quite significant ($p = .014$). In addition, the results of the ME model show that even after controlling for our other key variables, the direct effect of time on PTSD risk is significantly negative ($\beta = -0.95$, $p < .01$).

Turning first to H1a, our results for the ME model show that there is indeed a significant positive relationship between Ike-related stress and PTSD risk ($\beta = 0.62$, $p < .01$). However, with respect to H1b, the MET model shows that there is no significant change in this relationship over time, and in particular, no significant decrease. This is further supported by the ME of Ike-related stress in this MET model, which is virtually identical to the ME model both in value and significance.

Turning next to H2a, our results for the ME model show that received social support has no significant direct effect on PTSD risk, and in particular, no negative effect. Similarly, with respect to

H2b, our results for the MET model show that there is no significant change in the relation between received social support and PTSD risk over time. So while our pairwise comparisons in Table 2 do show that levels of received social support significantly decreased between Wave 1 and Wave 3 ($p < .01$), this decrease appears to have had no effect on the relation between received social support and PTSD risk.

Finally, we turn to our main hypothesis, H3. With respect to H3a, our results for the BE model show that the interaction between received social support and Ike-related stress is significantly negative ($\beta = -0.03$, $p < .01$). Hence, the positive effects of Ike-related stress on PTSD risk are indeed being buffered by increased levels of received social support. With respect to H3b, the results of our BET model show that the three-way interaction between Ike-related stress, received social support, and time is significantly positive ($\beta = 0.03$, $p < .05$). This interaction coefficient can be interpreted as the amount by which the two-way interaction coefficient between received social support and Ike-related stress changes when time is increased by one unit (20). More specifically, assuming that the two-way interaction is negative, so that received social support buffers the effect of Ike-related stress on PTSD risk, this positive three-way interaction implies that the BE of received social support declines over time. However, since this interpretation depends on the signs of “low-order” effects (and in particular on the presence of buffering), it is our view that such relations require more careful analysis. Following Dawson and Richter (2006), this can be accomplished by analyzing these relations simultaneously.

In particular, if in expression (Equation 1) of the article we now denote PTSD risk (conditioned on given unobserved individual attributes) by Y and let Irs = Ike-related stress, Rss = received social support, and T = Time, then Equation 1 can be written more explicitly for purposes of analysis as follows:

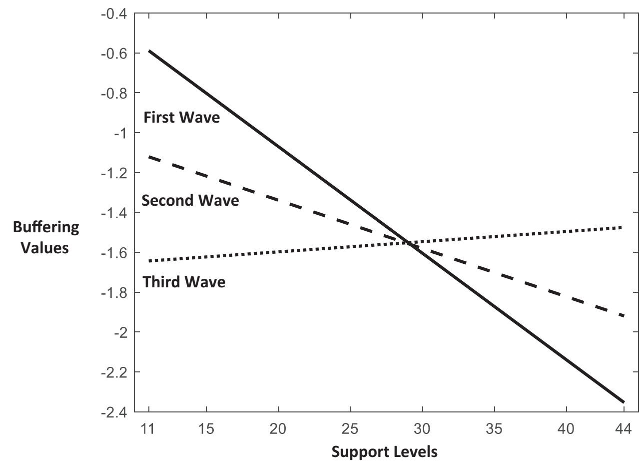
$$\begin{aligned} Y &= \beta_0 + \beta_1 \mathrm{Irs} + \beta_2 \mathrm{Rss} + \beta_3 T + \beta_{12}(\mathrm{Irs})(\mathrm{Rss}) \\ &\quad + \beta_{13}(\mathrm{Irs} \times T) + \beta_{23}(\mathrm{Rss} \times T) + \beta_{123}(\mathrm{Irs} \times \mathrm{Rss} \times T) \\ &= [\beta_0 + \beta_2 \mathrm{Rss} + \beta_3 T + \beta_{23}(\mathrm{Rss} \times T)] + [\beta_1 + \beta_{12} \mathrm{Rss} \\ &\quad + \beta_{13} T + \beta_{123}(\mathrm{Rss} \times \mathrm{T})] \times \mathrm{Irs}. \end{aligned} \tag{2}$$

where all other variables (including observed individual risk factors) are evaluated at fixed values and are implicitly taken to be part of constant β_0. In these terms, it follows that for each fixed value of Rss and T, the second line in Equation 2 represents Y as a linear function of Irs with intercept given by the first bracketed expression and slope given by the second bracketed expression. Here all terms other than the ME of Irs on PTSD risk ($\beta_1 = 2.59$***) represent possible sources for buffering this effect, which we now designate as the (potential) buffering function,

$$\begin{aligned} B(\mathrm{Rss}, T) &= \beta_{12} \mathrm{Rss} + \beta_{13} T + \beta_{123}(\mathrm{Rss} \times T), \\ &= \beta_{13} T + (\beta_{12} + \beta_{123} T) \times \mathrm{Rss}. \end{aligned} \tag{3}$$

More specifically, buffering is present at levels Rss and T if and only if $B(\mathrm{Rss}, T) < 0$. More generally, all conditions required for H3b can similarly be expressed as linear inequalities involving $(\beta_{12}, \beta_{13}, \beta_{123})$. To see this graphically, we first note from the results of the BET model that the specific values of these coefficients are given by $\beta_{12} = -0.05$***, $\beta_{13} = 0.86$**, and $\beta_{123} = 0.03$**. So if the buffering function in Equation 3 is evaluated over the range of received social support values, Rss = 11, …, 44, and time value, $T = 0, 1, 2$ (corresponding to Waves 1, 2, and 3, respectively) then we obtain the plot shown in Figure 1. Here it is evident that the highest value of B over the observable ranges of both Rss and T, namely -0.60, is achieved in Wave 1 ($T = 0$) at the minimum support level, Rss = 11, so that global buffering holds. More generally, to ensure global buffering it suffices to require that:

Figure 1
Stress-BET



Note. BET = buffering effects over time.

$$B(11, 2) < B(11, 1) < B(11, 0) < 0. \tag{4}$$

Before testing this assertion, it is necessary to complete H3b by observing that the degree of buffering in successive time periods is represented by the steepness of their respective line segments (each denoting buffering values at all levels of Rss). The decline in steepness of these lines shows that as time progresses, additional received social support levels, Rss, yield less additional BE (which is essentially flat in the final time period, $T = 2$). To characterize this decline in steepness for testing H3b, it suffices to require that the differences, $\Delta(T) = B(11, T) - B(44, T)$, be decreasing in T, that is,

$$\Delta(2) < \Delta(1) < \Delta(0). \tag{5}$$

By using Equation 3 to expand conditions Equation 4 and Equation 5 in terms of $(\beta_{12}, \beta_{13}, \beta_{123})$, it can be shown that following system of inequalities is sufficient for both Equation 4 and Equation 5 to hold:

$$[\beta_{12} < 0, \beta_{123} > 0, \beta_{13} + (11)\beta_{123} < 0]. \tag{6}$$

The first two conditions are obvious from the discussion above, and the third condition is needed to ensure global buffering (Equation 4). So the appropriate test of H3b amounts to a simultaneous test of these three inequality conditions. p values, p_1 and p_2, for one-sided tests of the first two hypotheses are obtainable directly from the results of the BET model, and are given respectively by $p_1 = .001$ and $p_2 = .023$. The p value, p_3, for a one-sided test of the third hypothesis can also be obtained from the BET model by an application of the Stata command, lincom, which yields $p_3 = .045$. Finally, to correct for multiple testing, we employ Holm's method (Holm, 1979) to obtain an upper bound on the familywise error rate, that is, the probability that at least one of the above hypotheses is falsely rejected. In particular,

Table 3

Using Random Intercept Logistic Regression Models to Predict PTSD Over Time (N = 658)

Variable	ME model		MET model		BE model		BET model	
	β[a] (*SE*)	*OR* (*SE*)	β (*SE*)	*OR* (*SE*)	β (*SE*)	*OR* (*SE*)	β (*SE*)	*OR* (*SE*)
Time	−0.95***[b] (0.225)	0.39*** (0.0871)	−0.25 (0.810)	0.78 (0.632)	−0.11 (0.870)	0.90 (0.783)	3.79* (2.071)	44.29* (91.74)
Race/ethnicity								
White[c]								
Black	0.74 (0.500)	2.09 (1.044)	0.76 (0.506)	2.15 (1.086)	0.76 (0.494)	2.14 (1.057)	0.75 (0.507)	2.11 (1.070)
Hispanic	0.58 (0.462)	1.78 (0.821)	0.54 (0.468)	1.72 (0.804)	0.59 (0.456)	1.81 (0.825)	0.60 (0.466)	1.82 (0.849)
Other	0.67 (0.754)	1.96 (1.477)	0.74 (0.762)	2.10 (1.604)	0.68 (0.754)	1.97 (1.482)	0.66 (0.773)	1.93 (1.488)
Marital status								
Married[c]								
Living with a partner	−0.13 (0.815)	0.88 (0.717)	−0.09 (0.823)	0.92 (0.756)	0.05 (0.798)	1.05 (0.839)	−0.01 (0.822)	0.99 (0.817)
Separated	−0.01 (0.726)	0.99 (0.721)	0.00 (0.733)	1.00 (0.733)	0.07 (0.717)	1.07 (0.766)	0.10 (0.729)	1.11 (0.807)
Divorced	−0.40 (0.573)	0.67 (0.384)	−0.42 (0.581)	0.66 (0.382)	−0.35 (0.565)	0.70 (0.398)	−0.37 (0.577)	0.69 (0.400)
Widowed	0.75 (0.542)	2.11 (1.143)	0.76 (0.547)	2.13 (1.165)	0.73 (0.533)	2.07 (1.101)	0.74 (0.543)	2.09 (1.135)
Never married	−0.49 (0.547)	0.61 (0.336)	−0.50 (0.555)	0.61 (0.336)	−0.45 (0.540)	0.64 (0.346)	−0.43 (0.553)	0.65 (0.359)
Gender								
Male[c]								
Female	1.42*** (0.429)	4.12*** (1.766)	1.40*** (0.434)	4.05*** (1.756)	1.42*** (0.427)	4.15*** (1.774)	1.46*** (0.438)	4.30*** (1.885)
Income below $40,000								
No[c]								
Yes	0.09 (0.424)	1.09 (0.463)	0.10 (0.428)	1.10 (0.471)	0.04 (0.419)	1.04 (0.434)	0.04 (0.428)	1.04 (0.443)
Educational attainment								
<High school[c]								
High school	−0.38 (0.557)	0.68 (0.381)	−0.43 (0.564)	0.65 (0.368)	−0.38 (0.553)	0.69 (0.380)	−0.37 (0.565)	0.69 (0.388)
Some college	−0.17 (0.566)	0.85 (0.479)	−0.21 (0.572)	0.81 (0.465)	−0.11 (0.558)	0.90 (0.502)	−0.15 (0.571)	0.86 (0.493)
College degree	−0.45 (0.633)	0.64 (0.404)	−0.49 (0.640)	0.61 (0.393)	−0.43 (0.622)	0.65 (0.406)	−0.46 (0.636)	0.63 (0.401)
Graduate work	−0.69 (0.813)	0.50 (0.410)	−0.74 (0.820)	0.48 (0.392)	−0.67 (0.806)	0.51 (0.414)	−0.73 (0.824)	0.48 (0.399)
Employed before Ike								
Yes[c]								
No	0.00 (0.368)	1.00 (0.368)	0.00 (0.372)	1.00 (0.371)	−0.02 (0.363)	0.99 (0.357)	−0.02 (0.370)	0.98 (0.364)
PTSD prior to Ike								
No[c]								
Yes	1.77*** (0.432)	5.85*** (2.526)	1.79*** (0.437)	6.01*** (2.627)	1.79*** (0.427)	6.01*** (2.565)	1.85*** (0.441)	6.36*** (2.805)
Ike-related trauma								
None[c]								
One	0.80 (0.484)	2.22 (1.073)	0.79 (0.488)	2.21 (1.079)	0.792* (0.471)	2.207* (1.039)	0.80* (0.481)	2.22* (1.068)
Two or more	2.18** (0.885)	8.87** (7.847)	2.10** (0.897)	8.19** (7.344)	1.82** (0.876)	6.17** (5.402)	1.82** (0.895)	6.18** (5.532)
Ike-related stress	0.62*** (0.117)	1.86*** (0.219)	0.61*** (0.139)	1.83*** (0.254)	1.55*** (0.402)	4.70*** (1.888)	2.19*** (0.545)	8.94*** (4.872)
Post-Ike trauma								
None[c]								
One	0.10 (0.400)	1.10 (0.440)	0.13 (0.402)	1.13 (0.456)	0.07 (0.398)	1.07 (0.425)	0.04 (0.403)	1.04 (0.418)
Two or more	0.202 (0.662)	1.223 (0.810)	0.255 (0.664)	1.29 (0.856)	0.204 (0.653)	1.227 (0.801)	0.185 (0.661)	1.203 (0.796)
Post-Ike stress	0.22** (0.113)	1.25** (0.142)	0.24** (0.118)	1.27** (0.150)	0.23** (0.115)	1.26** (0.145)	0.23** (0.117)	1.26** (0.147)
Income declined								
No[c]								
Yes	0.37 (0.349)	1.45 (0.505)	0.36 (0.354)	1.43 (0.507)	0.43 (0.351)	1.53 (0.537)	0.43 (0.355)	1.54 (0.548)
Mental health services								
No[c]								
Yes	0.72* (0.379)	2.06* (0.781)	1.14** (0.533)	3.14** (1.671)	1.10** (0.524)	2.99** (1.568)	1.09** (0.533)	3.00** (1.591)

(*table continues*)

Table 3 (*continued*)

Variable	ME model β[a] (*SE*)	ME model *OR* (*SE*)	MET model β (*SE*)	MET model *OR* (*SE*)	BE model β (*SE*)	BE model *OR* (*SE*)	BET model β (*SE*)	BET model *OR* (*SE*)
Social support	0.02 (0.021)	1.02 (0.021)	0.04 (0.026)	1.04 (0.027)	0.18*** (0.062)	1.19*** (0.074)	0.28*** (0.083)	1.32*** (0.110)
Time × Ike-Related Stress			0.02 (0.109)	1.02 (0.111)	−0.01 (0.108)	0.99 (0.106)	−0.86** (0.428)	0.43** (0.182)
Time × Mental Service			−0.48 (0.470)	0.62 (0.292)	−0.43 (0.458)	0.65 (0.300)	−0.41 (0.468)	0.66 (0.311)
Time × Social Support			−0.02 (0.023)	0.98 (0.023)	−0.03 (0.023)	0.98 (0.022)	−0.16** (0.071)	0.85** (0.060)
Social Support × Ike-Related Stress					−0.03*** (0.013)	0.97*** (0.012)	−0.05*** (0.017)	0.95*** (0.016)
Time × Social Support × Ike-Related Stress							0.03** (0.015)	1.03** (0.015)
Constant	−7.94*** (1.160)	0.000*** (0.000)	−8.474*** (1.290)	0.000*** (0.000)	−12.55*** (2.192)	0.00*** (0.000)	−15.65*** (2.892)	0.00*** (0.000)
Observations	1,674	1,674	1,674	1,674	1,674	1,674	1,674	1,674
Number of groups	658	658	658	658	658	658	658	658
Random effects parameters var (_cons)[d]	2.829034 (1.08841)		2.910329 (1.124371)		2.558158 (1.036813)		2.739964 (1.11193)	
LR test versus logistic model: chibar2 (01)[e]	21.83***		22.18***		18.62***		19.50***	
LR test (*df*)[f]	ME model nested in MET model 2 (333)							
LR test (*df*)[g]	ME model nested in BE model 9.20* (5)							
LR test (*df*)[h]	ME model nested in BET model 13.38** (5)							

Note. PTSD = posttraumatic stress disorder; ME = main effect; MET = main effect over time; BE = buffering effect; BET = buffering effect over time; LR = log likelihood ratio.
[a] β stands for log (odds) coefficients. [b] * $p < .1$. ** $p < .05$. *** $p < .01$. [c] Denotes a reference group. [d] The estimated variance in the intercept on the logit scale and standard errors are reported here. [e] Compared to conventional logistic regressions, the random-effect logistic regressions employed for the ME, MET, BE, and BET in our study provide significantly better fits to the data. [f] Compared to the ME model, the MET model is not significantly different. [g] Compared to the ME model, the BE model provides a weakly significant better fit to the data. [h] Compared to the ME model, the MET model provides a highly significant better fit to the data.

if these three p values are ranked by size (.045, .023, .001) and multiplied by their ranks to yield the "corrected" p values (.045, .046, .003), then it can be shown (Holm, 1979) that the largest of these yields an upper bound on familywise error rate. So the probability that at least one of the three hypotheses in Equation 6 is falsely rejected is no greater than .046. In this sense, we may conclude that our three-way interaction hypothesis, H3b, is significant at the .046 level.

In addition to these main hypotheses, our four logistic models yield a number of other results worthy of mention. First, while Table 2 shows that both post-Ike trauma and post-Ike stress increased over time ($p < .01$ for each), only post-Ike stress contributed significantly to Ike-related PTSD. Moreover, the constancy of this significance across all four models ($\beta = 0.23$, $p = .052$) suggests that in each successive wave, post-Ike stress events (since the last interview) acted as significant triggers of Ike-related PTSD in that wave. It is also of interest to note from Table 2 that while the use of mental health services increased over time ($p < .01$) and exhibited a significant positive association with Ike-related PTSD ($p < .05$), auxiliary regressions (not reported) show that the use of mental health services was more significantly related to levels of post-Ike trauma and post-Ike stress over time ($p < .001$ for each). Finally, as suggested by the uniform results of all four models, Ike-related PTSD appears to be significantly more prevalent among females ($\beta = 1.42$, $p = .001$) and among those survivors experiencing PTSD prior to Ike ($\beta = 1.79$, $p < .001$).

Discussion

In this study, we have examined the interrelations between (a) PTSD suffered by a sample of Hurricane Ike survivors, (b) the stressful events experienced by these survivors, and (c) the levels of social support they received over time. Our main findings were that receiving social support buffered the effects of Ike-related stress on the likelihood of PTSD, but that this BE diminished over time. Before discussing these results in more detail, it is appropriate to relate some of our specific results to previous work. First, our finding that post-Ike stress may trigger additional occurrences of Ike-related PTSD is consistent with similar findings of Lowe et al. (2013) that long-term stressors may influence PTS/depressive symptoms over time. Second, our findings that Ike-related PTSD is significantly more prevalent both among females and among survivors experiencing prior PTSD are consistent with those of McLaughlin et al. (2011) for Hurricane Katrina, and more generally, are consistent with the findings of many other disaster studies as systematically reviewed by Galea et al. (2005).

But as articulated in the three hypotheses developed in the introductory part, our main results focus on the relation between Ike-related stress and Ike-related PTSD, and on the buffering of this relation by received social support over time. Turning first to stress itself, our results suggest that the influence of Ike-related stress on Ike-related PTSD persists over time, even when post-Ike stressors are controlled for. This closely parallels the results of Felix and Afifi (2015) with respect to the effects of fire-related stress on the subsequent mental health status of fire victims. However, our present results sharpen their single-interview findings by tracing effects over three waves of interviews. Turning next to social support, most previous disaster-related studies have focused on the relationship between mental health outcomes and levels of social support as perceived by survivors (Felix & Afifi, 2015; Fleming et al., 1982; Kaniasty & Norris, 2009). But following Kaniasty and Norris (2009), we believe there is a need for more studies of social support actually received by victims. In this light, our findings are closely related to those of Platt et al. (2016), who also studied relations between PTS symptoms suffered by Hurricane Ike survivors and three distinct types of social support that they may have received over time. But rather than carrying out separate analyses for distinct types of social support, we here take a more holistic approach in which both emotional and material forms of received social support are (implicitly) allowed to interact. In addition, our present results focus more explicitly on pathways in which received social support acts to moderate (buffer) the effects of Ike-related stress on the likelihood of PTSD over time. As detailed in Kaniasty and Norris (2009), such support may increase survivors' resilience to stressors resulting from disasters. In this setting, our results confirm that received social support does indeed serve to buffer the effect of Ike-related stressors on PTSD likelihood. Moreover, while this buffering relation persists throughout all three interview periods, its effects are shown to diminish over time (Table 3).

Limitations

As with all secondary data studies, these findings can only be interpreted by explicitly recognizing the limitations of the data itself. First, there is the question of possible selection bias in terms of those survivors omitted from the study. Fortunately, unlike Hurricane Katrina (Fussell, et al., 2010), there is no evidence of substantial population exodus following Hurricane Ike. However, as noted in Platt et al. (2016), it is still possible that survivors suffering from severe Ike-related PTSD and/or Ike-related stress were more likely to decline participation in this study altogether. In addition, there is the question of missing data for those who participated at least partially. Here it must be recognized that while multiple data imputations are reasonably robust, there may still be systematic effects violating the MAR assumption, such as early withdrawal from the interview process triggered by PTSD itself.

Conclusion

The present study makes substantial contributions to both theory and implementation around how PTSD risk can be decreased following a natural disaster. For instance, a key finding of this study is that the effects of social support received by Hurricane Ike survivors are consistent with the more general buffering effects theory of social support (Cohen & Wills, 1985). Additionally, and perhaps of equal importance, we found that the ME model that received social support exhibited no significant direct effect on PTSD risk. Additional tests (not reported) show that even when Ike-related stress and/or time are removed from this model, there continues to be no significant direct effect. So, for these survivors, we find no evidence for the main effects theory of social support (Cohen & Wills, 1985; Fleming et al., 1982; Kaniasty & Norris, 2009; Kawachi & Berkman, 2001).

In terms of how implementation practices can lead to reduced PTSD risk, the stress-BE identified in this study suggests survivors who were exposed to higher levels of stress during a given disaster be prioritized in receiving social support. Given the social supports referred to in this study encompass three dimensions of support: emotional, informational, and tangible support, policy and related social service interventions should address these dimensions respectively. For example, expanding eligibility rules for the Individuals and Households Program could allow many housing loss victims to receive financial support from the Federal Emergency Management Agency for rebuilding or repairing their damaged homes (García, 2022). Furthermore, the authorities and media should focus on providing accurate information to reduce unnecessary relocations and evacuations in disaster-prone areas (Kaniasty & Norris, 2004). In addition, providing spiritual and emotional care in response to a disaster may lower the PTSD risk among the survivors who experienced resource losses (Aten et al., 2015). Finally, perhaps our most important finding is that such stress-BEs diminish over time. This provides evidence that the timing of social support interventions is critical and that early interventions may well prove to be most effective for PTSD remission.

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