The Impact of Internet Coverage of the March 2011 Japan Earthquake on Sleep and Posttraumatic Stress Symptoms: An International Perspective

To the Editor: On March 11, 2011, a 9.0 magnitude earthquake and tsunami struck Japan, killing 14,000 people and damaging nuclear and petrochemical plants. The immediate and intense media coverage exposed viewers across the world to disturbing images. While television coverage of a nearby disaster has been found to increase the risk for subsequent psychological distress (1), little is known about the effects of Internet coverage of a distant disaster.

We conducted an online survey simultaneously in France, Canada, and the United States within 2 weeks of the event. Adult participants (N=698; 42.7% from Canada, 44.6% from France, 8.2% from the United States, and 4.5% from "other/unspecified" countries) were contacted through online mailing lists and snowballing procedures (i.e., participants were also asked to circulate the link to the survey among their contacts) and asked to report 1) the time they spent viewing television and Internet coverage of the event during the first week afterward; 2) their immediate reactions after learning the news (i.e., peritraumatic distress [2] and dissociation [3]); and 3) any disruptive nocturnal behavior (i.e., trauma-related sleep disturbances such as nightmares [4]) they experienced during the first 10 days after the event.

After the earthquake and tsunami, most participants (64.9%) had increased their media consumption. The amount of television and Internet viewing correlated both with symptoms of peritraumatic distress and dissociation ($r>0.22$ and $p<0.001$ in all cases) and with disruptive nocturnal behavior ($r>0.17$ and $p<0.001$ in all cases), while 45% of the participants reported at least one disruptive nocturnal behavior. Being female, knowing someone in Japan, and figuring in the amount of time spent on the Internet each predicted at least one disruptive nocturnal behavior in a logistic regression (Table 1). In the second step, peritraumatic dissociation and distress significantly predicted disruptive nocturnal behavior; however, the time spent on the Internet became nonsignificant, suggesting a mediating effect of peritraumatic reactions. This was confirmed by a multiple mediator analysis revealing that the direct effect of Internet viewing on disruptive nocturnal behavior was not significant, while indirect effects through both peritraumatic distress and dissociation were significant ($p<0.05$), suggesting that peritraumatic reactions might explain the relationship between Internet exposure and disruptive nocturnal behavior. Replicating the analyses separately by

TABLE 1. Predictors of the Presence of at Least One Disruptive Nocturnal Behavior in the First Week After the March 2011 Japan Earthquake and of Posttraumatic Stress Symptoms 2 Months Later[a]

					Step 1		Step 2		Step 3	
Variable	Mean	SD	N	%	B	SE	B	SE	B	SE
Predicted at least one disruptive nocturnal behavior in the first 10 days[b]			314	45						
Step 1										
Age (years)	39.4	12.8			–0.01	0.01	–0.01	0.01	–0.01	0.01
Female			190	27.5	0.77***	0.18	0.92**	0.19	0.58**	0.21
Living alone			265	38.2	–0.10	0.16	–0.17	0.17	–0.19	0.18
Knowing someone in Japan at the time			181	26.2	0.40*	0.18	0.24	0.19	0.06	0.21
Step 2										
Time watching television in first week (hours/day)	0.65	1.01					0.11	0.1	0.01	0.11
Time on Internet in first week (hours/day)	0.52	1.01					0.38**	0.11	0.15	0.13
Step 3										
Peritraumatic distress[c]	10.6	6.0							0.12***	0.02
Peritraumatic dissociation[d]	12.2	4.1							0.13***	0.04
Predicted 2-month PTSD symptoms[e]	7.5	11.6								
Step 1										
Age	39.2	12.1			0.16	0.09	0.17	0.08*	0.20**	0.60
Female			27	25.0	2.65	2.5	4.04	2.28	1.32	1.73
Living alone			38	35.2	1.73	2.3	1.09	2.09	1.04	1.54
Knowing someone in Japan at the time			34	31.2	2.19	2.54	0.10	2.34	–1.33	1.73
Step 2										
Time watching television in first week (hours/day)	0.57	0.75					0.52	1.21	–0.185	0.89
Time on Internet in first week (hours/day)	0.52	1.10					4.82***	1.22	1.86	0.95
Step 3										
Peritraumatic distress[c]	10.8	6.40							0.58**	0.15
Peritraumatic dissociation[d]	12.1	4.10							1.36**	0.22

[a] N=698 assessed in the first week; some data are missing. N=109 assessed 2 months later; some data are missing. There were no differences on any variables between those who were reassessed at 2 months and those who were not.
[b] At least one item on the Pittsburgh Sleep Quality Index Addendum for PTSD.
[c] Peritraumatic Distress Inventory score (range 0–52).
[d] Peritraumatic Dissociative Experiences Questionnaire score (range 10–50).
[e] Impact of Event Scale–Revised total score (range 0–88).
*p<0.05. **p<0.01. ***p<0.001.

country yielded similar results. As a follow-up, in a subsample reassessed 2 months later (109 individuals who provided their e-mail addresses), similar analyses examining predictors of posttraumatic stress symptoms (5) revealed a significant effect of Internet viewing that was mediated by peritraumatic reaction.

Limitations of this study include the convenience sample, the lack of an assessment of disruptive nocturnal behavior and posttraumatic stress symptoms before the event, an expectedly low level of symptoms, and the possible response bias. However, the results suggest that Internet coverage of a distant disaster may induce sleep disturbances and subclinical psychological symptoms of posttraumatic stress in the general population. Such effects, consistent with previous reports (6, 7) of posttraumatic stress disorder symptoms after trauma exposure, were mediated by peritraumatic reactions.

References

1. Ahern J, Galea S, Resnick H, Kilpatrick D, Bucuvalas M, Gold J, Vlahov D: Television images and psychological symptoms after the September 11 terrorist attacks. Psychiatry 2002; 65:289–300
2. Brunet A, Weiss DS, Metzler TJ, Best SR, Neylan TC, Rogers C, Fagan J, Marmar CR: The Peritraumatic Distress Inventory: a proposed measure of PTSD criterion A2. Am J Psychiatry 2001; 158:1480–1485
3. Marmar CR, Weiss DS, Metzler TJ: The Peritraumatic Dissociative Experiences Questionnaire: assessing psychological trauma and PTSD, in Assessing Psychological Trauma and PTSD. Edited by Wilson JP, Keane TM. New York, Guilford, 1997, pp 412–428
4. Germain A, Hall M, Krakow B, Katherine Shear M, Buysse DJ: A brief sleep scale for posttraumatic stress disorder: Pittsburgh Sleep Quality Index Addendum for PTSD. J Anx Disord 2005; 19:233–244
5. Creamer M, Bell R, Failla S: Psychometric properties of the Impact of Event Scale–Revised. Behav Res Ther 2003; 41:1489–1496
6. Allenou C, Olliac B, Bourdet-Loubère S, Brunet A, David AC, Claudet I, Lecoules N, Roullet P, Bui E, Birmes P: Symptoms of traumatic stress in mothers of children victims of a motor vehicle accident. Depress Anxiety 2010; 27:652–657
7. Bui E, Brunet A, Allenou C, Camassel C, Raynaud JP, Claudet I, Fries F, Cahuzac JP, Grandjean H, Schmitt L, Birmes P: Peritraumatic reactions and posttraumatic stress symptoms in school-aged children victims of road traffic accident. Gen Hosp Psychiatry 2010; 32:330–333

ERIC BUI, M.D., PH.D.
Toulouse, France
RACHEL F. RODGERS, PH.D.
Boston
CHRISTOPHE HERBERT, M.A.
Montreal, Quebec
DEBRA L. FRANKO, PH.D.
Boston
NAOMI M. SIMON, M.D., M.SC.
Boston
PHILIPPE BIRMES, M.D., PH.D.
Toulouse, France
ALAIN BRUNET, PH.D.
Montreal, Quebec

Dr. Bui is supported by a fellowship from Association Traumapsy and Toulouse University Hospital. Dr. Rodgers is supported by a Fulbright fellowship. Dr. Simon has received research grants from American Foundation for Suicide Prevention, Eli Lilly, Forest Laboratories, GlaxoSmithKline, NARSAD, NIMH, Pfizer, Sepracor, and the U.S. Department of Defense and speaking, CME, or consulting fees from Massachusetts General Hospital Psychiatry Academy, Pfizer, and the U.S. Department of Defense. Dr. Brunet has received a salary award from the Fonds de la recherche en santé du Québec. All other authors report no financial relationships with commercial interests.

This letter (doi: 10.1176/appi.ajp.2011.11081281) was accepted for publication in November 2011.

Conceptual Issues in Psychiatric Gene-Environment Interaction Research

TO THE EDITOR: In their critical review of candidate gene-by-environment (cG×E) interaction research, published in the October 2011 issue of the *Journal*, Duncan and Keller (1) raise several methodological issues that cast serious doubt on many published G×E findings. While informative in many respects, their virtually exclusive methodological perspective does not address an important conceptual issue that has emerged in recent years concerning cG×E interaction: putative risk alleles often operate as plasticity alleles (2). Duncan and Keller appear to maintain the tradition of viewing all G×E inquiry from a diathesis-stress perspective, which stipulates that individuals carrying risk alleles will be more likely to develop psychopathology in the face of adversity relative to individuals without risk alleles under the same conditions. However, as it turns out, ever more cG×E findings appear consistent with an alternative—and more evolutionarily plausible—conceptual framework: differential susceptibility. According to this theory, some individuals are, for genetic reasons, more responsive to both negative and positive environmental influences (3).

This theory raises the possibility that one reason cG×E findings often do not replicate is the misconceptualization of candidate genes as risk genes. If individuals carrying certain plasticity alleles are disproportionately susceptible to a wide range of developmental experiences and contextual exposures, not just adverse ones, then the failure to include propitious factors in cG×E research could increase false negative findings. Consider in this regard Kilpatrick and colleagues' study (4) of the role of the serotonin-transporter-linked polymorphic region (5-HTTLPR) in moderating the effect of hurricane exposure on posttraumatic stress disorder (PTSD). Had the investigators not been in a position to detect the disproportionate benefit that those hurricane-exposed individuals with short—putative "risk"—alleles accrued from high social support, then it seems likely that their G×E study would also have yielded null results. And this is because, as it turned out, it was only those especially susceptible to positive and negative environmental influences (i.e., short allele carriers) but exposed only to the latter—hurricane and low social support—who proved especially likely to develop PTSD.

Given that samples will usually vary in both environmental risk exposure and availability of protective resources and that candidate genes may be associated with elevated susceptibility to both, the failure to explicitly measure and include positive supportive aspects of the environment in cG×E studies may be one important reason why G×E findings fail to replicate. These practices, we contend, derive from the embracing of vulnerability-only rather than plasticity models.

References

1. Duncan LE, Keller MC: A critical review of the first 10 years of candidate gene-by-environment interaction research in psychiatry. Am J Psychiatry 2011; 168:1041–1049