



Published in final edited form as:

JAMA. 2011 September 28; 306(12): 1374–1375. doi:10.1001/jama.2011.1358.

Understanding the Mental Health Effects of Indirect Exposure to Mass Trauma Through the Media

Yuval Neria, PhD and Gregory M. Sullivan, MD

Department of Psychiatry, College of Physicians and Surgeons (Drs Neria and Sullivan) and Department of Epidemiology, Mailman School of Public Health (Dr Neria), Columbia University, New York City, New York; and Divisions of Clinical Therapeutics (Dr Neria) and Molecular Imaging and Neuropathology (Dr Sullivan), New York State Psychiatric Institute, New York City

Exposure to mass trauma is common. In the United States, 15% of women and 19% of men have reported lifetime exposure to natural disasters alone.¹ Since the advent of 24-hour television news, exposure to mass violence and natural disasters through the media is even more widespread. Although exposure to trauma has a wide range of psychopathological consequences, posttraumatic stress disorder (PTSD) has been shown to be the most common.²

PTSD is unique among psychiatric disorders because it requires exposure and fearful response to traumatic events as the proximate precipitants of the syndrome. The clinical presentation of PTSD includes a diverse array of distressing and disabling ongoing symptoms in the realms of reexperiencing phenomena (eg, nightmares), hyperarousal (eg, startle response), avoidance of reminders of the exposure, and general affective numbing. Yet community surveys have consistently demonstrated that most adults exhibit resilience following exposure to trauma, even among those who develop symptoms consistent with PTSD in the immediate aftermath, with many recovering over time without clinical intervention. A significant minority, however, will develop PTSD, which is the posttrauma clinical trajectory associated with a considerable health burden, including psychiatric comorbidities, severe psychosocial dysfunction, and increased rates of suicidal behaviors.

The neuroscience model of pavlovian fear conditioning and extinction has been proposed to have direct relevance to PTSD pathophysiology. This heuristic model explains how fear learning may be involved in the emergence of PTSD symptoms and how the persistence of symptoms over time potentially involves a failure of basic extinction processes of learned fear memory.³ In a neurobiological study, a fear learning and extinction task with psychophysiological and functional brain imaging assessments revealed significant biological differences between patients exposed to trauma who had developed PTSD and controls exposed to trauma who had not.³ Moreover, increased efforts to develop and test extinction-based psychotherapeutic approaches for treatment of PTSD, such as prolonged exposure (a type of therapy that encourages reexperience of trauma through recalling and reengaging with it), have proven these interventions to be efficacious for many, and these approaches have become first-line interventions for PTSD.⁴ However, the fear-learning and

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Corresponding Author: Yuval Neria, PhD, Department of Psychiatry, College of Physicians and Surgeons, Columbia University, 1051 Riverside Dr, Unit 69, New York, NY 10032 (ny126@columbia.edu).

Conflict of Interest Disclosures: The authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest.

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extinction model has yet to be fully applied for understanding the trajectories of PTSD over time among populations indirectly exposed to mass trauma, disasters, and terrorism.

The attacks of September 11, 2001, unprecedented in the United States in their magnitude and aftermath, were followed by a surge of research on their mental health consequences over the last decade. Longitudinal studies suggest that the mental health burden among directly exposed populations, such as evacuees, first responders, and bereaved persons, is substantial and frequently enduring. For example, a 2-wave study among individuals directly exposed to the World Trade Center attacks found prevalence of PTSD symptoms among rescue/recovery workers to increase from 12.1% at 2 to 3 years after the attacks to 19.5% at 5 to 6 years after the attacks.⁵ In contrast, the burden of PTSD symptoms in the general population, who were for the most part indirectly exposed to the trauma through media, was found to be lower in severity and substantially diminished over time. In New York City, the initial estimates of prevalence of probable PTSD declined from 7.5% at 1 month after the September 11 attacks to 1.7% and 0.6% at 4 and 6 months, respectively, after the attacks.⁶

Some of the general population studies following September 11 have also examined the association between exposure to the attacks via television and development of PTSD. In a national survey, exposure to televised live broadcasting during the attacks was found to be associated with increased risk for short-term PTSD symptoms 1 to 2 months after the attacks.⁷ Those findings raised the question of whether indirect exposure in general, and via the media in particular, is indeed traumatic, with effects similar to those of direct exposure, and whether the PTSD symptoms related to indirect exposure persist over a long period.

The New York City data regarding persons indirectly exposed to the September 11 attacks are comparable to data from the 2008–2009 military conflict in southern Israel. A 3-wave, longitudinal study of young adults evacuated from the Israel-Gaza border to distant areas with minimal exposure to missile attacks found that probable PTSD symptoms spiked to 20% during the conflict, followed by a rapid and sharp decline to 3.0% and 2.0%, 2 and 4 months, respectively, after the conflict had ceased.⁸

The findings in these 2 populations, characterized by acute emergence of PTSD-like symptoms that rapidly decline over several months to a lower, more stable rate with ongoing PTSD, raise a number of possible explanations about the effects of indirect exposure to mass violence. For example, the more transient symptoms in the majority of persons with PTSD symptoms who present initially may be due to activation of the fear/stress response system in a setting of actual ongoing threat of war or terrorism; once the immediacy of the actual threat abates, appropriate deactivation occurs, with learned fear responses mostly extinguished when the situation is “safe” and “aversive” events no longer appear imminent. Another possibility is that indirect exposure to mass trauma acts as a low-impact trauma that may result in rapidly resolving symptoms of PTSD in low-vulnerability populations and that only a small portion of individuals exposed—those with preexisting vulnerability mediated through genetic factors, prior exposure to trauma, or preexposure psychiatric history—remain with a clinical diagnosis of PTSD months to years after indirect exposure.⁹ An analysis of a subset of New York City residents indirectly exposed to trauma during the September 11 attacks (those not in the vicinity of the World Trade Center, experiencing no September 11 loss, or not taking part in recovery and cleaning efforts) from a sample of primary care patients studied after the attacks supports this hypothesis. At 1 year after the attacks, indirect exposure alone was not associated with PTSD among individuals without a history of pre-September 11 trauma, family psychiatric history, or both.¹⁰

These findings, if supported by future studies, may suggest that even though exposure severity is similar among persons indirectly exposed to terrorist attacks or disasters (and

might be considered a lower-severity exposure despite exposure to graphic images on television, because the danger is not imminent), development of longer-term PTSD to such exposure may have more to do with the preexisting vulnerability than with the indirect exposure severity per se. Thus, such lower-severity exposure may be expected to result in low rates of PTSD over time, such as 1% to 2% after September 11 in New York City⁶ or 2% to 3% after war in the Israel study.⁸ Moreover, the initial elevated rates of PTSD symptoms may be an artifact of measurement too soon after the exposures, when extinction mechanisms have not had sufficient time to aid in recovery of the majority of indirectly exposed individuals. Future research evaluating the putative role of fear conditioning in the development of PTSD and fear extinction in recovery processes, whether spontaneous or attributable to treatment, should address these questions and could aid in the triage of individuals exposed to mass trauma to identify those most likely to develop ongoing psychopathology.

In sum, evidence from epidemiologic studies indicates initial but not extended increases in rates of PTSD symptoms among individuals indirectly exposed to mass trauma through sources such as mass media. This raises questions about the trajectory of clinical course in these individuals and the possibility of preexisting vulnerability among those with enduring symptoms. Importantly, these questions may be directly addressed by research using fear conditioning and extinction models, ultimately aiding in the identification of high-risk individuals for triage of clinical interventions that prevent development of, or enhance recovery from, PTSD through enhancement of basic extinction processes.

Acknowledgments

Dr Neria reported receiving research funding from the National Institute of Mental Health (NIMH), the Spunk Fund Inc, and the National Alliance for Research on Schizophrenia and Depression (NARSAD) and receiving royalties for published books from Cambridge University Press. Dr Sullivan reported receiving research funding from NIMH, NARSAD, the American Foundation for Suicide Prevention, and the Dana Foundation; serving as a member of the Scientific Advisory Board of TONIX Pharmaceuticals Inc; and serving as a consultant for Ono Pharma USA Inc.

Funding/Support: This writing of this Commentary was supported by NIMH grant 2RO1-MHO72833 (Dr Neria).

Role of the Sponsors: The funding organizations had no role in the preparation, review, or approval of the manuscript.

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