It is illegal to post this copyrighted PDF on any website. Can Development of PTSD Be Prevented After Acute Trauma?

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Commentary

S halev et al¹ have conducted a solid randomized controlled 5-arm, 12-week trial of 2 forms of cognitivebehavioral therapy, prolonged exposure,² and cognitive therapy,³ in comparison with escitalopram 20 mg versus placebo, and a waiting list control who received delayed prolonged exposure at month 5. Researchers screened study patients on average within 10 days of their single traumatic event (mostly traffic accidents). Of the 324 patients who met *DSM-IV* criteria for acute posttraumatic stress disorder (PTSD) (except for the 1-month duration) following a single trauma in adulthood, 82 participants declined treatment and 242 started the assigned intervention less than a month after the trauma.

The study was designed to further assess the efficacy of prolonged exposure and cognitive therapy delivered to prevent development of DSM-IV PTSD among relatively uncomplicated single acute trauma patients. The study specifically focused on maintenance of the previously observed short-term effects (at 5 months after trauma, 2 months after treatment termination) by following up all groups, even those assigned to the placebo condition, at 3 years after trauma. Intervening nonstudy treatments were tracked and did not differ among groups. The interesting and important results demonstrated that although prolonged exposure and cognitive therapy outperformed the other conditions at 2 months after termination/5 months after trauma, indicating that the 2 psychotherapies had in fact acutely blunted the severity of PTSD symptoms on the Clinician-Administered PTSD Scale (CAPS),⁴ at 3 years after trauma, no differences existed between any groups on any outcome measures, including employment status. Thirtyone percent of patients were retraumatized over the 3-year course of the study, independent of randomized condition. Importantly, roughly 35% of these patients, independent of which intervention they were randomized to, met criteria for (chronic) PTSD 3 years after their traumatic event. Despite overall high CAPS scores in all groups at baseline, it is not clear what percentage of these patients actually met criteria for PTSD when they started treatment. The authors wisely emphasize that outcome results for single-trauma acute ASD patients cannot be generalized to the more typical multiply traumatized individuals who are seen in PTSD

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clinics, and who present from the military.⁵ To complicate matters, much as acute stress disorder and acute PTSD are related, they are not identical, and it is not clear how many of these study patients actually achieved the diagnosis of "acute PTSD" by duration.

Although most people experience trauma in the course of their lives (50%–90%⁶), only about 8% develop PTSD.⁷ The course of acute stress disorder, specifically its progression to PTSD, has mostly been studied by trauma type and varies.⁸ The rationale for introducing and targeting the diagnosis of acute stress disorder was to identify traumatized individuals at high risk of developing chronic PTSD. Although a significant proportion of these individuals do subsequently develop PTSD,9 most individuals are resilient and do not meet criteria for acute stress disorder.¹⁰ Thus, it is unclear whether the patients who improved with the study psychotherapies would have progressed to PTSD or not, even though their psychosocial function and apparent PTSD symptoms had improved both absolutely and in relation to the competing arms at 2-month follow-up. While this is clinically important, we cannot know whether these interventions prevented development of chronic PTSD. In any case, whether the 2-month treatment psychotherapy responders did or did not carry the risk of disability of subsequent chronic PTSD and actually received prophylaxis from their 12-week treatments, this study clarifies that 3 years after trauma, about 35% of single-event traumatized individuals have chronic PTSD, regardless of prevention attempts.

Who are these patients, and what makes them vulnerable? Shalev et al¹ venture a discussion of potential target groups among traumatized patients who might harbor greater risk for developing chronic, nonremitting PTSD: those with depressed urinary cortisol levels or who had experienced childhood trauma. The effects of childhood trauma, which are poorly understood, may reflect both predisposing traumatic experiences and core attachment problems.¹¹ Silove and colleagues,¹² in a World Health Organization epidemiologic study of separation anxiety across 18 countries, highlight a specific, reciprocal risk relationship between development of PTSD and separation anxiety. Core anxious attachments and separation anxiety may play a role in predisposition to and subsequent chronicity of PTSD, potentially at least partially mediated through difficulty in developing, or deriving comfort from, social supports.¹¹ This study highlights the importance of longitudinal follow-up of potentially exciting outcomes from timelimited interventions and illustrates the continued need for better, potentially differently focused interventions for patients with chronic PTSD.

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REFERENCES

- Shalev AY, Ankri Y, Gilad M, et al. Long-term outcome of early interventions to prevent posttraumatic stress disorder. J Clin Psychiatry. 2016;77(5):e580–e587.
- 2. Hembree EA, Foa EB, Dancu CV. *Prolonged Exposure (PE) Manual*. Revised Version. Philadelphia, PA: University of Pennsylvania; 1999.
- Marks I, Lovell K, Noshirvani H, et al. Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring: a controlled study. *Arch Gen Psychiatry*. 1998;55(4):317–325.
- Weathers FW, Keane TM, Davidson JR. Clinician-Administered PTSD Scale: a review of the first ten years of research. *Depress Anxiety*. 2001;13(3):132–156.
- 5. Blosnich JR, Dichter ME, Cerulli C, et al. Disparities in adverse childhood

- Psychiatry. 2014;71(9):1041–1048.
 Breslau N, Kessler RC, Chilcoat HD, et al. Trauma and posttraumatic stress disorder in the community: the 1996 Detroit Area Survey of Trauma. Arch Gen Psychiatry. 1998;55(7):626–632.
- Kessler RC, Sonnega A, Bromet E, et al. Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry. 1995;52(12):1048–1060.
- 8. Giannoni-Pastor A, Eiroa-Orosa FJ, Kinori SG, et al. Prevalence and predictors of posttraumatic stress symptomatology among burn survivors: a systematic review and meta-analysis. *J Burn Care Res.* 2016;37(1):e79–e89.
- Bryant RA. Acute stress disorder. In: Gabbard GO, ed. *Treatment of Psychiatric Disorders*. 5th ed. Washington, DC: American Psychiatric Press; 2013:505-518.
- Bryant RA. Acute stress disorder as a predictor of posttraumatic stress disorder: a systematic review. J Clin Psychiatry. 2011;72(2):233–239.
- 11. Markowitz JC, Milrod B, Bleiberg K, et al. Interpersonal factors in understanding and treating posttraumatic stress disorder. *J Psychiatr Pract*. 2009;15(2):133–140.
- Silove D, Alonso J, Bromet E, et al. Pediatric-onset and adult-onset separation anxiety disorder across countries in the World Mental Health Survey. Am J Psychiatry. 2015;172(7):647–656.