

Do sudden gains occur in Prolonged Exposure therapy for PTSD?

Lisa R. Stines, Ph.D., Norah C. Feeny, Ph.D., & Lori A. Zoellner, Ph.D.

Case Western Reserve University and University of Washington

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INTRODUCTION

A growing body of research in recent years has examined the phenomenon of rapid, large decreases in symptoms during treatment, and these reductions have been labeled "sudden gains" (Tang & DeRubeis, 1999). Among those with depression and social phobia, sudden gains do occur in cognitive behavioral treatment, and these gains are related to overall symptom reduction. No studies to date have examined this occurrence in CBT for posttraumatic stress disorder (PTSD). The purpose of the current study was to examine sudden gains in those received treatment for PTSD and whether these early gains are related to overall symptom reduction.

METHOD

Participants

- Female assault survivors with chronic PTSD were enrolled in a treatment outcome study ($N=31$) in which they could choose prolonged exposure (PE) or sertraline. Only those who chose PE were examined in the current study ($N=23$).
- Exclusion criteria included no PTSD diagnosis or PTSD not primary, current psychosis or suicidality, unstable bipolar disorder, pregnancy, and current substance or alcohol dependence.

Measures

- Posttraumatic Stress Diagnostic Scale** (PDS; Foa, Cashman, Jaycox, & Perry, 1997).

Procedure

- PE consisted of (1) breathing retraining (a form of relaxation); (2) psychoeducation about common reactions to trauma; (3) imaginal exposure, or repeated reliving of the trauma memory; and (4) in-vivo exposure, or real-life confrontation with feared, avoided trauma-related situations or activities.
- Women completed self-report measures at each of the ten treatment sessions.

ANALYTIC PLAN

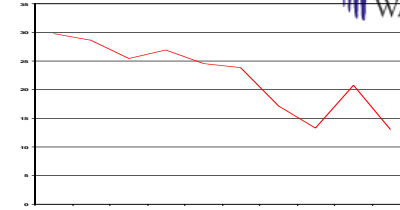
- Sudden gains were defined as clinically significant reductions in PTSD symptoms between any session N and session $N+1$ (see Tang & DeRubeis, 1999).
- Clinical significance was computed using the 2- to 3-week test-retest reliability (.83) and standard deviation (9.96) originally reported for participants with PTSD on the PDS, such that the standard error of measurement for the scale is computed as $SE = 9.96 \sqrt{1 - .83}$, yielding 4.41. The standard error of the difference between two administrations of the measure would be calculated as $\sqrt{2(SE^2)} = \sqrt{2(4.41^2)}$ or 6.24. See Devilly & Foa, 2001.
- Thus, operationally defined for the current study, sudden gains occurred when PTSD scores were reduced by a minimum of 6.24 between sessions N and $N+1$ on the PDS.

RESULTS

Demographics

	<i>M</i>	<i>SD</i>
Age	29.5	9.09
	7	
	%	
Ethnicity (% African-American)	21.7	
Income (% <\$20,000)	50	
Employment (% Full-time)	34.8	
Education (% College)	39.1	
Primary Trauma (% Sexual Assault)	72.7	

PTSD: Mean PDS scores across treatment sessions



DISCUSSION

Based on the above findings, anxiety sensitivity looks to play a role in mediating the relationship between trait anxiety and PTSD symptomatology in a sample of undergraduate women. This relationship, however, did not hold within the community sample. Post hoc t-tests showed that the community samples' scores on the STAI-T, ASI, and PDS were all significantly higher than those for the undergraduate sample ($p < .0001$). Thus, general levels of anxiety may be a more powerful correlate of PTSD symptomatology than anxiety sensitivity in more clinically impaired populations. In response to the debate surrounding the relative specificity (or non-specificity) of anxiety sensitivity (see Lilienfeld, Jacob, & Turner, 1989; see McNally, 1989), these findings suggest that sample characteristics, specifically levels of psychopathology, may play a role in diminishing the distinctiveness of anxiety sensitivity as it is related to PTSD symptomatology. The present study is limited by the fact that it is cross-sectional and does not assess changes in psychopathology over time. In addition, the outcome measure (PDS) for PTSD is based on self-report; these findings may look different among individuals diagnosed with PTSD by a trained clinician.

REFERENCES

Correspondence concerning this poster should be addressed to:
lisa.stines@case.edu