
COMMENT

Complex PTSD is on the Trauma Spectrum: Comment on Resick et al. (2012)

Marianne Goodman^{1,2}

¹Department of Psychiatry, Mount Sinai School of Medicine, New York, New York, USA

²Mental Illness Research, Education, and Clinical Center, James J. Peters VA Medical Center, Bronx, New York, USA

Conceptualizing posttraumatic stress disorder (PTSD) along a spectrum with complex and simple features is integrative and models an approach taken by disciplines in medicine outside of psychiatry. This perspective is offered in the Resick et al. (2012) review. To best delineate the nature and permeability of the border between Complex PTSD and PTSD, an emphasis on clarifying underlying biological processes is needed to move beyond our current reliance on symptomatic description.

This timely and important review of the construct validity of complex posttraumatic stress disorder (CPTSD) by Resick et al. (2012) analyzes currently available data and concludes that presently there is insufficient data to support the inclusion of CPTSD in the *DSM-5* as a separate disorder by highlighting limitations in the definition and instrumentation measuring the construct along with lack of differential response to treatment. These conclusions should not be taken to mean that CPTSD does not exist as a disorder, but rather should propel the field to refine the definition and means of measurement of CPTSD, and to focus on clarifying its precise relationship with PTSD. One of the most compelling contributions of the article was the authors' suggestion of viewing CPTSD as part of a spectrum of adaptation to traumatic exposure. This integrative conceptualization allows for overlap in clinical symptomatology between the two entities, but recognizes distinguishing features of CPTSD. It also sets the stage for productive advances in treatment development and clarifying the basic biology underlying each disorder.

Psychiatry's reliance on the *DSM* and its description of symptoms that cluster together provides a common language to describe clinical phenomenology. These diagnostic labels are not, however, based on treatment response nor do they inform us about underlying etiology. Psychiatry lags behind other

branches of medicine in its understanding of the basic pathological processes of mental disorders, and this is reflected in our descriptive diagnostic process.

Resick and colleagues, (2012) in asking the question of whether PTSD and CPTSD are distinct disorders, chose to focus on differences in treatment response and descriptive phenomenology. These choices may not have been the most informative. Mental health treatments, particularly psychotherapy, which is the primary modality reviewed, are not particularly sensitive, and we are in the early stages of understanding what creates change. In the article, much effort is expended in articulating the diverse traumas triggering each disorder and the different maladaptive responses to the trauma. A comparison of the basic biological processes underlying each disorder is conspicuously missing from the analysis.

What do we know about the overlap in biological processes between PTSD and CPTSD? Although there is considerable research describing neuroendocrine and brain imaging abnormalities in PTSD (e.g., Yehuda & LeDoux, 2007), such investigation in CPTSD is in its infancy. Recent data, however, are promising and focus on altered hippocampal activation to negative stimuli (Thomaes et al., 2009, 2011) and differences in ventral anterior cingulate activity as compared to healthy controls (Thomaes et al., 2010). Although this important research begins to describe declarative memory and emotional processing deficits in CPTSD, because all subjects met criteria for both PTSD and CPTSD, any ability to determine differences between these two groups was unavailable. Moreover, neuroimaging studies by Lanius and colleagues (2010) articulate differences in brain function pertaining to dissociative, self-dysregulation (Frewen & Lanius, 2006), and emotion modulation difficulties in PTSD. Though the research subjects included in these studies

Correspondence concerning this article should be addressed to Marianne Goodman, Department of Psychiatry, Mount Sinai School of Medicine, New York, NY, 10029. E-mail: marianne.goodman@va.gov

Published 2012. This article is a US Government work and is in the public domain in the USA. View this article online at wileyonlinelibrary.com
DOI: 10.1002/jts.21695

were not selected for CPTSD, problems with the self and severe dissociation are important elements of CPTSD that differentiate it from PTSD.

There is a growing appreciation for the brain changes and hormonal alterations that occur with traumatic experiences in childhood, and recognition of the subsequent impact on learning, memory, and emotional regulation processes (e.g., Ford, 2009). Prolonged traumas that occur in childhood, presumably the basis for a CPTSD diagnosis, might well have a differential impact on these biological systems as compared to traumas occurring later when key developmental phases have occurred (e.g., adulthood) or traumas of more limited exposure. Future demonstration of these differences will be important steps in delineating PTSD and CPTSD and whether these syndromes are part of a spectrum or discrete entities.

Resick et al. (2012) are correct in asserting that nowhere else in psychiatry is there a precedent for splitting off a more severe form of any disorder. Nevertheless, examples from other disciplines are worth considering. In neurology, simple versus complex seizures are described based on whether consciousness is lost. In orthopedics, an impact or stress can break a bone resulting in a simple or complex fracture differentiated by skin breakdown. Similarly, pathologic fractures occur if there are other features that predispose to a weakening of the bone such as osteoporosis. Similar ways of thinking may be applied to simple versus complex PTSD. Traumatic reactions that are confined to basic reexperiencing and avoidance symptoms may be delineated as simple, but those that extend to involvement of attachment and altered self-concept may be described as CPTSD. Similarly, pathologic parameters such as biological diathesis that constitute PTSD risk can be described. Such an approach will help target appropriate treatments for patients. Just as simple versus complex seizures dictate differential approaches to treatment, the selected criteria for simple versus complex PTSD should similarly inform treatment recommendations and approaches.

The model just described reflects my proposal of viewing CPTSD as part of a trauma spectrum disorder. Rather than seeing the Resick et al. (2012) review as discounting the notion of

CPTSD as a distinct disorder, reconceptualizing PTSD along a spectrum with simple and complex features is integrative and useful. An improved appreciation of the myriad of factors—including duration of the traumatic exposure, the developmental phase during which it occurred, genetic vulnerabilities, and other biological variables—in addition to the specifics of the traumatic antecedent, could very well help stratify patients according to prognosis and inform treatment selection and the development of new approaches. Similarly, a clearer appreciation of the underlying pathology will ultimately help to distinguish complex from simple presentations.

References

- Ford, J. D. (2009). Neurological and developmental research: Clinical implications. In C. A. Courtois & J. D. Ford (Eds.), *Treating complex traumatic stress disorders: An evidence based guide* (pp. 31–58). New York: Guilford Press.
- Frewen, P. A., & Lanius, R. A. (2006). Toward a psychobiology of posttraumatic self-dysregulation, reexperiencing, hyperarousal, dissociation, and emotional numbing. *Annals of the New York Academy of Sciences*, *1071*, 110–124. doi:10.1196/annals.1364.010
- Lanius, R. A., Vermetten, E., Loewenstein, R. J., Brand, B., Schmahl, C., Bremner, J. D., & Spiegel, D. (2010). Emotion modulation in PTSD: Clinical and neurobiological evidence for a dissociative subtype. *American Journal of Psychiatry*, *167*, 640–647. doi:10.1176/appi.ajp.2009.09081168
- Resick, P., Bovin, M. J., Calloway, A. L., Dick, A. M., King, M. W., Mitchell, K. S., . . . Wolf, E. J. (2012). A critical evaluation of the complex PTSD literature: Implications for *DSM-5*. *Journal of Traumatic Stress*, *25*, 239–249.
- Thomaes, K., Dorrepaal, E., Draijer, N., de Ruiter, B. M., Elzinga, M. B., Sjoerds, Z., . . . Veltman, D. J. (2011). Increased anterior cingulate cortex and hippocampus activation in complex PTSD during encoding of negative words. *Social Cognitive and Affective Neuroscience*, Advance online publication. doi:10.1093/scan/nsr084
- Thomaes, K., Dorrepaal, E., Draijer, N., de Ruiter, M. B., Elzinga, B. M., van Balkom, A. J., . . . Veltman, D. J. (2009). Increased activation of the left hippocampus region in Complex PTSD during encoding and recognition of emotional words: A pilot study. *Psychiatry Research: Neuroimaging*, *171*, 44–53. doi:10.1016/j.psychres.2008.03.003
- Thomaes, K., Dorrepaal, E., Draijer, N., de Ruiter, M. B., van Balkom, A. J., Smit, J. H., & Veltman, D. J. (2010). Reduced anterior cingulate and orbitofrontal volumes in child abuse-related complex PTSD. *Journal of Clinical Psychiatry*, *71*, 1636–1644. doi:10.4088/JCP.08m04754blu
- Yehuda, R., & LeDoux, J. (2007). Response variation following trauma: A translational neuroscience approach to understanding PTSD. *Neuron*, *56*, 19–32. doi:10.1016/j.neuron.2007.09.006