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### Postscript: Evidence and Counterevidence

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We welcome the chance to respond to the five main critiques in Monroe and Mineka's (2008) postscript to their comment. First, they claimed in their postscript that they never denied that the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev. [DSM-IV-TR]; American Psychiatric Association, 2000) posttraumatic stress disorder (PTSD) diagnosis implies a causal model. Thus, our careful demonstration that it does was "entirely irrelevant" (Monroe & Mineka, 2008, p. 1096). However, in their comment, Monroe and Mineka clearly rejected the DSM-IV-TR PTSD diagnosis as a causal model:

Rubin and colleagues emphatically stated, "there is a causal model inherent in the DSM-IV-TR diagnostic criteria of PTSD". . . . Although we acknowledge that DSM-IV-TR can be (and has been) misunderstood on this issue regarding PTSD, there is in fact no formal causal model inherent in the DSM-IV-TR diagnostic criteria. (Monroe & Mineka, 2008, p. 1085)

Second, Monroe and Mineka (2008) faulted us for reviewing only what they considered to be an outdated cognitive theory of PTSD. However, we spent 12 paragraphs reviewing current cognitive theories in our reply (Berntsen, Rubin, & Bohni, 2008). We

showed how these theories were conceptually related to Horowitz's original theoretical work and that empirical findings go against these theories' main tenets of fragmented voluntary memory and enhanced involuntary remembering.

Third, Monroe and Mineka (2008) repeated their objections to two studies we cited as evidence that a pharmacologically reduced trauma memory can reduce PTSD. The critical attention devoted to a one-paragraph section in our article in both their comment and postscript is surprising. We clearly stated in our article that these studies "have small numbers of participants and are in the early stages of what could be a very exciting area of research, but for our current theoretical purposes, though promising, they must be taken with caution" (Rubin, Berntsen, & Bohni, 2008, p. 1002).

Fourth, their postscript was still concerned that arousal and avoidance symptoms are equal in traumatic brain injury (TBI) and non-TBI groups even though we gave concrete arguments that the TBI group should be higher if there were no memory effects. Also, the postscript still was concerned that some studies have not shown a decrease in PTSD symptoms with TBI, even though we noted that our claim is about memory loss, which was not measured in these studies and could have been a loss of memory that lasted only for minutes. Decrease in PTSD symptoms does occur where substantial memory loss for the event is noted.

Fifth, Monroe and Mineka (2008, p. 1098) claimed we "deflected attention from questions about the mnemonic model" by not giving a "more respectful" treatment of the questions they raised about causality. We did not mean to show disrespect, but their distinction between cause and maintenance is not helpful in our view because we see no objective way to separate these phases in time. Thus, Monroe and Mineka's comment and postscript

focused on how we presented our model: points about what model we used for comparison, whether the comparison was to a causal model, and how strong evidence had to be to be included. Why did they not instead consider counterevidence to our easy-to-falsify claims, if their goal were to “provide plausible alternative perspectives” (Monroe & Mineka, 2008, p. 1098)?

The basic tenet of the mnemonic model is that the current memory of the traumatic event, not the event per se, determines the level of PTSD symptoms. The memory is not fixed. It changes over time due to factors that characterize autobiographical memory in general, autobiographical memory of emotional events in particular, individual differences, and the current concerns of the person. We invoke no trauma-specific mechanisms to explain these effects. We focus on conscious (explicit) autobiographical memory because it appears to play a key role for PTSD and because this focus provides a testable model. This position led us to generate falsifiable claims, which we listed as hypotheses in our article and summarize here. Events that do not satisfy the A1 event criterion and A2 emotions criterion of the current diagnosis can be followed by PTSD symptoms. Symptom severity varies systematically with the availability of the traumatic memory; in the extreme, if the memory could be removed, PTSD could be prevented from developing or continuing. According to what is known about tunnel memory in stressful events, important aspects of the trauma would be better recalled; therefore, the C3 symptom of the diagnosis—referring to faulty memory for central parts of the event—should be poorly correlated with the remaining PTSD symptoms. Individual differences that are known to have specific effects on autobiographical memory for emotional events, such as gender and neuroticism, should have specific theoretically expected effects on PTSD. In our article (Rubin et al., 2008), we reviewed evidence in support of all these hypotheses. In their comment, Monroe and Mineka (2008) presented no direct counterevidence to any of them. Instead, they critiqued some of the evidence presented in relation to the second-mentioned claim. On the basis of a request in their comment that we include a discussion of current cognitive theories, we included two additional claims from our other work in our reply (Berntsen, Rubin, & Bohni, 2008). First, involuntary recall has no privileged access to traumatic events, and second, memories of traumatic events are not fragmented. Evidence for both

claims were reviewed in our reply. Monroe and Mineka presented no counterevidence.

The reader may ask why we did not say more about current cognitive theories in our original article. We have a program of research in which theories of autobiographical memory (e.g., Rubin, 2006) and involuntary memories (e.g., Berntsen, in press) have been developed that have direct implications for PTSD, implications that were formalized in our article. Detailed theoretical and empirical contrasts to current cognitive theories have appeared elsewhere (e.g., Berntsen, in press, chapter 7; Berntsen & Rubin, 2008; Rubin, Boals, & Berntsen, in press), as briefly reviewed in our reply (Berntsen et al., 2008).

We do not deny that our model has limitations. No scientific theory is complete and correct; aspects of the mnemonic model are likely to be shown to be wrong. However, we believe that a clear, testable exposition is preferred to one with limitations and hedges impeding its falsification. We look forward to seeing how our ideas hold up to the data and competing theories.

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