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Contrasting Models of Posttraumatic Stress Disorder: Reply to Monroe and Mineka (2008)

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Abstract

We address the four main points in Monroe and Mineka (2008)'s Comment. First, we first show that the DSM PTSD diagnosis includes an etiology and that it is based on a theoretical model with a distinguished history in psychology and psychiatry. Two tenets of this theoretical model are that voluntary (strategic) recollections of the trauma are fragmented and incomplete while involuntary (spontaneous) recollections are vivid and persistent and yield privileged access to traumatic material. Second, we describe differences between our model and other cognitive models of PTSD. We argue that these other models share the same two tenets as the diagnosis and we show that these two tenets are largely unsupported by empirical evidence. Third, we counter arguments about the strength of the evidence favoring the mnemonic model, and fourth, we show that concerns about the causal role of memory in PTSD are based on views of causality that are generally inappropriate for the explanation of PTSD in the social and biological sciences.

Monroe and Mineka (2008)'s Comment has four main points, which structure our response. First, they argue that the DSM PTSD diagnosis does not imply a theoretical model, thus, we should not have contrasted our model with the DSM. Second, they criticize us for not evaluating our theoretical model against other cognitive models of PTSD. Third, they argue that some of the evidence that we presented in support of the mnemonic model is weak and may even go against the model. Fourth, they challenge the role of the traumatic memory as a causal mechanism.

We counter all of these points. We first show that the DSM PTSD diagnosis implies a theoretical model, following standard definitions of what qualifies as a theoretical model, and we document that this model has a long history in psychology and psychiatry. We next describe differences between our model and other cognitive models of PTSD. Although Monroe and Mineka find these alternative cognitive models to be "much richer and more comprehensive," we show that they are largely unsupported by empirical evidence and argue that they are rooted in the same theoretical model as the diagnosis. We then clarify the evidence that Monroe and Mineka call into question, and address the notion of causality in relation to the mnemonic model

Space limitations do not allow us to address the numerous more minor points raised by Monroe and Mineka, depicting aspects of our article as (in alphabetical order) inappropriate, irrelevant,

misleading, spurious, unconvincing, uninformative and containing dubious inferences, fundamental misunderstandings, and inconsistencies. Focusing on the main points, on the other hand, allows us to help advance research on PTSD by discussing central issues in the understanding of the disorder.

Why the DSM PTSD Diagnosis Implies a Theoretical Model

Monroe and Mineka challenge our argument that there is a causal model inherent in the DSM diagnostic criteria for PTSD. Surprisingly, they argue that “in PTSD, the event is part of a complex of criteria required for the diagnosis, *not a cause in any formally proposed sense*.” Thus, there simply is no “DSM model” in any theoretical sense of the term (emphasis added).” Although it is the case that the DSM from the third edition and onwards adopts a descriptive, data-driven approach with no reference to etiology, most scholars would claim that the PTSD diagnosis forms an exception. One pertinent example is Spitzer and colleagues recently arguing that: “a key distinguishing feature of PTSD is that it is not agnostic to etiology. Unlike virtually all diagnoses in the *Diagnostic and Statistical Manual of Mental Disorders (DSM)*, PTSD rests on the assumption of a specific etiology, whereby a distinct set of events (criterion A) is assumed to be the uniformly most potent contributor to outcome” (Spitzer, Rosen & Lilienfeld, 2008, p. 319). Many other scholars have made similar claims, but Spitzer was the chief editor of the DSM-III in which PTSD was introduced, thus, his assessment deserves special attention (see also Rosen & Lilienfeld, 2008; Rosen, Spitzer, & McHugh, 2008). Similarly, the Institute of Medicine’s (2006) report on PTSD notes that “the necessary cause of PTSD is by definition a traumatic event” (p. 23). In short, Monroe and Mineka’s statement that PTSD does not imply a causal model is in direct conflict with the assessment of major authorities in the field.

Furthermore, the position taken by Monroe and Mineka is logically contradicted by the diagnosis. All five reexperiencing symptoms and three avoidance symptoms explicitly refer to “the traumatic event” or “the trauma” – e.g., the person may have “recurrent and intrusive distressing recollections of the event” and “efforts to avoid thoughts, feelings, or conversations associated with the trauma”. Thus, without the event, by definition eight of the seventeen PTSD symptoms in the current diagnosis would not be possible. As pointed out by Young (1995): “The DSM theory of PTSD is simple. . . it is simply taken for granted that time and causality move *from* the event *to* the other critical symptoms. Because the traumatic event is the cause of the syndromal feelings and behaviors, it is logical to say that it precedes them. If this were not true, if it were acceptable for syndromal features to occur before the traumatic event, then the term “reexperience” would lose its accepted meaning.” (pp. 117–116). Monroe and Mineka seem to partly acknowledge this, when they state that the traumatic event (although not a cause in their view) is “a necessary environmental precursor to the disorder”. The distinction between a causal factor and “a necessary environmental precursor” is strained.

How does this relate to the DSM PTSD diagnosis implying a theoretical model? Causality is central to most theoretical models (Woodward, 2003). Following common definitions, a theoretical model constitutes an explanation of a given phenomenon. It specifies which factors (e.g., variables) should be considered as part of the explanation and how these factors are related. Explanation and prediction are typically the goal of a theoretical model. Both are possible because the theoretical model presents some causal explanation of a given phenomenon, which may be testable (e.g., Klein & Zedeck, 2004; Whetten, 1989; Woodward, 2003). Under this description, the DSM PTSD diagnosis implies a theoretical model. It specifies the key factors of the disorder in terms of the A stressor criteria for the event and the ensuing reexperiencing, avoidance, and arousal symptoms and it describes how at least some of them are related (i.e., a stressful event fulfilling the A criteria is necessary for the development of the symptoms). In addition, a causal relation is implied between some of the reexperiencing symptoms (e.g., “recurrent and intrusive distressing recollections of the event”)

and some of the avoidance symptoms (e.g., “efforts to avoid activities, places or people that arouse recollections of the trauma”) because the latter appears to be a consequence of the former.

Next we address the history of this theoretical model. We dwell on this question not only because it is relevant to our claim that the diagnosis rests on a theoretical model but also because it permits us to evaluate the cognitive theories mentioned by Monroe and Mineka. As we will point out, these cognitive theories are largely rooted the same underlying model. However, as we will also show, this model finds little empirical support.

Where Does the DSM Model Come From?

The diagnosis of PTSD was introduced in the DSM III in 1980. Numerous historical accounts have reviewed the political, scientific and practical motivations for this inclusion, notably the central role played by Vietnam war veterans and their supporters (e.g., Jones & Wessely, 2007; Scott, 1990; Young, 1995). As pointed out in such accounts, the introduction of the diagnosis represented a shift in the way post-traumatic stress was conceptualized. Before PTSD, mental distress caused by traumatic events was viewed as a transient phenomenon that in part depended on the patient’s ability to adapt. Prolonged stress reactions were considered abnormal and were assumed to reflect some pretrauma vulnerability of the person. After the introduction of the PTSD diagnosis, however, posttraumatic stress reactions were viewed as a general stress response that could develop in anyone who encountered a sufficiently powerful environmental stressor. Thus, the occurrence of PTSD depended on the severity of the event rather than on individual vulnerability. Obviously, neither of the two positions is atheoretical or politically neutral. As summarized by Horowitz (1999): “The diagnosis of PTSD emerged officially in 1980, after a controversy that involved difficult forensic issues as well as scientific observations: A polarization of causation was argued: Did the traumatic event in and of itself cause a psychiatric problem? Were pre-existing features of the victim’s personality and vulnerability heavily involved? If trauma were the main cause of the symptoms, the institutions or people responsible for causing or not preventing the traumatic events could be held legally responsible for damage to victims. If prior personality or other predispositions were the cause, and the traumatic event was only a precipitant, then individual or institutional responsibility would be less” (pp. 1–2; see also Yehuda & McFarlane, 1995).

Considering the traumatic event – and not individual vulnerability – as the most powerful etiological agent can thus be seen as a marked theoretical and ideological position. The necessary integration of clinical observations, experiments, and prior theories into a coherent and detailed theoretical model for PTSD was carried out largely by Horowitz several years before the introduction of the diagnosis in the DSM. In the middle of the 1970s a working group formed by Vietnam veterans and supporters was struggling to convince the chief editor of DSM III to include a diagnosis for combat-related disorders. Among others with whom the working group corresponded was Horowitz, who joined the group in 1976 (Scott, 1990). At this time, Horowitz had just finished a comprehensive research project on psychological reactions to emotional stress, culminating in the publication of his groundbreaking monograph *Stress Response Syndromes*. Readers of this book and the work it summarizes can hardly overlook the impressive overlaps between the stress reactions identified by Horowitz (1976) and the symptoms of PTSD that were listed in the DSM III four years later, as well as in subsequent revisions.

The influence of Horowitz (1976) can be summarized in terms of three main points. First, there are *general* response tendencies to stressful events that are not dependent on individual predisposing factors. Although there are individual differences with regard to level of stress response, all individuals may show some stress response if they are subjected to enough stress.

This emphasis on the environmental stressor agrees with the importance of the A-criteria in the PTSD diagnosis. Second, Horowitz (1976) identified most of the reexperiencing, avoidance and arousal symptoms of PTSD. In his conception, a typical stress response involves a fluctuation between phases with intrusive repetitions of the event in thoughts, emotion and behavior, on the one hand, and phases with efforts at avoiding such repetitions. This corresponds to the reexperiencing and avoidance symptoms in the PTSD diagnosis. In addition, the intrusion phase in Horowitz's conception was associated with hypervigilance, startle reactions, and sleep and dream disturbances and the avoidance phase included numbness and total or partial amnesia, both corresponding to symptoms in the PTSD diagnosis. Third, Horowitz (1975, 1976) classified involuntary (spontaneously arising) recollections as a typical stress response mechanism contingent upon a special '*active memory storage*' with an inherent tendency at automatically repeating its own contents until the processing of the stressful material had been completed – that is, until the stressful material had become integrated into preexisting cognitive schemata. By dedicating a special memory system to the processing of stressful material and by assuming a close link between involuntary remembering and the processing of traumatic/stressful material, Horowitz (1975, 1976) laid the ground work for PTSD theories that explains PTSD in terms of cognitive mechanisms that are assumed specific to stressful and traumatic events (for a more detailed historical review, see Berntsen, in press). These are the cognitive theories that Monroe and Mineka criticize us for not discussing in our article. We therefore turn to them next.

Our Model Compared to Other Cognitive Models of PTSD?

Having established that the DSM PTSD diagnosis indeed implies a theoretical model with a traceable history, we now examine a group of PTSD theories that agree with the basic tenet of our mnemonic model--that is, that the memory of the trauma is central to the understanding of PTSD--but that differ from us by arguing that special memory mechanisms are needed. We did not go into a detailed discussion of these theories in our article because we considered this as the next level of analysis – a level that was not needed for the points we wanted to make about the mnemonic model in relation to the PTSD diagnosis – and because of space limitations. We have, however (as Monroe and Mineka note) discussed these theories elsewhere and refer to this work in our article. Nonetheless, we are happy to have the opportunity to describe how our memory based approach to PTSD differs from other cognitive models of the disorder.

Other cognitive models and their background

Horowitz's (1975, 1976) model for stress responses has two main tenets that were adopted by cognitive theories of PTSD and that are expanded upon by Monroe and Mineka. One is that the memory of the stressful event tends to repeat itself in an involuntary and uncontrollable fashion. The other point is that voluntary (strategic and controlled) remembering of the event is considerably reduced by such psychological mechanisms as denial and repression. Periods with intrusive images may be paralleled by partial or complete amnesia. The notion of *flashback* developed in Horowitz's (1969b) work on LSD patients was incorporated into the literature on traumatic stress to designate instances of involuntary trauma memories with extremely high levels of emotional and behavioral reliving (Berntsen, in press; Frankel, 1994). According to Horowitz, the underlying cause of both the enhanced involuntary remembering and the impaired voluntary access is incomplete cognitive processing of the traumatic event along with the activation of defense mechanisms (e.g., repression and denial). Instead of a normal integration into the cognitive schemata of the person, the event is subsumed to an *active memory storage* – a hypothesized memory system that tends to repeat its own content until its processing has been completed. This memory system constitutes a direct explanation for the enhanced involuntary remembering. The impairment of voluntary memory, on the other hand, is attributed to the activation of defense mechanisms that serve to protect

the person against reliving the emotional stress as well as to the poor cognitive match between the trauma and preexisting schema-structures.

We designate this view of stress response *the special mechanism view* because it relies on hypothesized memory mechanisms (e.g., the active memory storage) that have particular relevance to stressful or traumatic material but are supported by very little evidence. The cluster of cognitive PTSD theories that Monroe and Mineka cite differ on details but almost all of them share this view. In other words, they all assume that the encoding of the traumatic event is faulty and that voluntary memory access is impaired while involuntary remembering is enhanced. This view is summarized by Halligan, Clark, and Ehlers (2002) as a “pattern of poor intentional recall and easy triggering of involuntary memories” (p. 74). Historically, this idea can be traced back to Breuer and Freud’s (1895) theory of hysteria. In Horowitz’s (1976) interpretation of an often quoted sentence from this work: “ ‘Hysterics suffer mainly from reminiscences’ because they cannot remember and they cannot *not* remember (pp. 83–84).

Following the special mechanisms view, Monroe and Mineka expand on two empirically testable claims. First, voluntary and involuntary recall are argued to reflect the operations of two distinct memory systems or fundamentally different kinds of processes, with involuntary remembering privileged for accessing and reliving traumatic events. Second, voluntary memories of the trauma are disorganized and fragmented. Contrary to what Monroe and Mineka claim, these assumptions are indeed constrained by the current diagnosis. The assumption of disorganized and fragmented voluntary memory is reflected in the C3 symptom of “inability to recall an important aspect of the trauma”, addressed at length in our article. The assumption of involuntary remembering having privileged access to traumatic and stressful material agrees with the listing of involuntary (intrusive) recollection as a symptom of PTSD with no mention of enhanced voluntary remembering. It should be noted that recurrent and intrusive recollections were not part of the diagnoses preceding the PTSD diagnosis, such as gross stress reaction in DSM I (Jones & Wessely, 2001). They appear to have entered the PTSD diagnosis in 1980 largely due to the influence of Horowitz’s (1976) model for stress response syndromes. We are not here arguing against the relevance of intrusive involuntary remembering as a symptom of PTSD. We do believe, however, that the hypothesized dissociation between involuntary and voluntary remembering in relation to the disorder is wrong, and we review the evidence shortly.

The alternative view, which we advocate, is a *basic mechanisms view*. It is based on what is generally known about the relation between emotion and memory and neither posits nor requires a special memory mechanism to explain responses to stressful events. There is considerable evidence that emotional stress enhances memory encoding and consolidation and thus enhances (rather than impairs) subsequent access to the memory (e.g., McGaugh, 2003, 2004). Based on research on involuntary autobiographical memories (e.g., Berntsen, in press), we assume that, in cases of emotional stress, access to the memory is enhanced for both voluntary and involuntary recall. However, the latter is experienced as particularly bothersome for the trauma victim for two reasons: first because it is uncontrollable, and second because involuntary remembering *in general* comes with more emotional reliving (Berntsen & Hall, 2004; Berntsen, in press; Rubin, Boals, & Berntsen, in press). We next review the evidence for Horowitz’s two tenets and thus the two competing theoretical views.

Does involuntary remembering have privileged access to traumatic/stressful material?

Over the last two decades, evidence has accumulated showing that involuntary remembering is not specific to traumatic or stressful events. On the contrary, it is a common phenomenon in everyday life with a predominantly positive content (Berntsen, in press, for a review). The claim that involuntary (compared to voluntary) remembering has privileged access to stressful/traumatic material has little empirical support. Instead the data suggest that the memory

enhancement associated with stressful/traumatic material concerns *both* involuntary and voluntary memory (e.g., Berntsen & Rubin, 2008, Hall & Berntsen, 2008; Rubin, Boals, & Berntsen, in press). The following is a brief review of relevant empirical work (for more details, see Berntsen, in press).

In a number of experiments conducted in the late 1960's and 70's, Horowitz and colleagues had their participants watch either an emotionally upsetting film or a neutral film. Afterwards, the participants took part in a trivial signal detection task, which was frequently interrupted and the subjects were requested to report their immediately preceding thoughts. Participants who had seen a stressful film reported significantly more intrusive film related thoughts than subjects who had been watching a neutral film. Horowitz (1969a, 1975a, 1976) interpreted these findings as supporting his view that intrusive and repetitive thought is a response to emotional stress. However, because the experiments did not include a voluntary memory condition, they did not rule out that emotional stress during encoding would have a similar enhancing effect on subsequent voluntary recall. If so, involuntary remembering would be no more a stress response than voluntary remembering. This possibility was recently examined by Hall and Berntsen (2008) in a diary study of involuntary and voluntary memories of emotionally upsetting pictures. As predicted from the basic mechanisms view, voluntary and involuntary recall both correlated positively with measures of emotional stress reported during encoding. No differences were observed between the two modes concerning the level of emotional stress associated with the remembered pictures.

A number of studies have examined the prediction derived from the special mechanisms view that incomplete cognitive processing during the encoding of stressful stimuli is followed by an increased occurrence of intrusive memories in a subsequent diary study (e.g., Holmes, Brewin & Hennesy, 2004; Brewin & Saunders, 2001). Counter to this prediction, these studies have generally found that divided attention during encoding decreases (rather than increases) the amount of subsequent intrusive memories. Although counter to the special mechanisms view, this result is exactly what would be expected based on research on attention and memory in general (Craik et al., 1996; Mulligan, 1998). To date only one published study has indicated that an unrelated cognitive task during encoding – a particular verbal distraction task – is followed by a subsequent increase in the amount of conscious intrusive memories (Holmes et al., 2004, Experiment 3). However, there have been difficulties with consistently replicating this finding (Holmes & Bourne, 2008).

In order to systematically examine the points at issue here, one would ideally have to compare the eight conditions that occur when the following three distinctions are intersected: Traumatic versus non-traumatic event by involuntary versus voluntary recall by PTSD versus non-PTSD population. So far, only one study has come close to satisfying this requirement (Rubin, Boals, & Berntsen, in press). In this study, undergraduates with high versus low scores on a PTSD scale participated in a diary study on involuntary and voluntary memories. For each involuntary memory, participants recorded a voluntary memory from the same period of their life. The participants rated both types of memories on scales measuring characteristics of the autobiographical memories. According to the special mechanisms view, we should expect interactions between low versus high PTSD symptom levels and involuntary versus voluntary memory characteristics. For example, the involuntary memories would be expected to be more trauma-related and emotionally negative than the voluntary memories and this effect would be more pronounced for the high than the low PTSD symptom group. However, consistent with the basic mechanisms view, no such effects were found. For none of the memory measures was an interaction seen between the involuntary versus voluntary condition and the high versus low PTSD symptom groups. Consistent with previous studies, (Berntsen & Hall, 2004) the involuntary memories had more mood impact and more physical reaction than the voluntary memories and they were seen as less central to the life story than their voluntary counterparts.

These differences were equally pronounced for the high versus low PTSD symptom groups. The high PTSD symptom group reported less positive memories and more mood impact and physical reaction than the low PTSD symptom group, and their memories were more trauma-related. These group differences were equally pronounced for the involuntary and voluntary memories. In short, high versus low levels of PTSD did not have differential effects on involuntary versus voluntary memory qualities. This finding contradicts the special mechanisms view but is consistent with the basic mechanisms view.

Are trauma memories fragmented?

As Monroe and Mineka note (see their footnote 4), we repeatedly find that people rate their memories of trauma as, or more, coherent than other memories. Given that the PTSD diagnosis is based on a self-report in a clinical interview, it seems odd to us that Monroe and Mineka reject these data and ask for the judgments of a neutral observer, citing Foa, Molnar, and Cashman (1995). What is the evidence of fragmentation in trauma memories as judged by a neutral observer? Foa et al. (1995) did not address the fragmentation issue. All their narratives were trauma narratives from patients with PTSD, so there were no data available for possible comparisons to non-trauma memories or to people without PTSD.

In order to address the key questions, we need studies comparing trauma and non-trauma ‘control’ memories from people varying on PTSD symptoms. We could not find such studies. This is not a minor problem. Level of education is both a risk factor for PTSD, as noted in our article, and a correlate of being able to construct a well-formed narrative. To establish a relation between narrative coherence and PTSD, we need to eliminate educational differences. Therefore, either ‘control’ narratives or measures of narrative skills are needed. For instance, Gray and Lombardo (2001) replicated a study by Amir, Stafford, Freshman, and Foa (1998), which showed that the trauma narratives of participants with, as opposed to without, PTSD were less coherent. Gray and Lombardo, however, also included ‘control’ narratives and these showed similar effects. Moreover, differences in writing skills and cognitive abilities between the two groups accounted for the main effect of PTSD on coherence, even though the participants were clinically diagnosed college students and thus had a restricted range on these variables compared to most samples in the literature. The need for controls in establishing a causal relation is clear. The same criticism—a lack of crucial relevant controls—could be leveled at many other studies (e.g., Halligan, Micheal, Clark, & Ehlers, 2003; Harvey & Bryant, 1999; Jones, Harvey, & Brewin, 2007; Murray, Ehlers & Mayou, 2002).

Leaving aside the question of proper controls, we may ask if there is any evidence of fragmentation in recorded narratives. Jones et al. (2007) found significant differences between people with and without PTSD for memories of their trauma on their measure of global coherence. However, there was no evidence that the trauma memories of participants with PTSD were incoherent. Jones et al.’s scale went from 0 = extremely coherent to 10 = extremely incoherent with a mean over all times for the PTSD patients of 2.02, thus, far from the level one would need to have to use fragmentation as an explanatory mechanism. Moreover, all their participants were victims of road traffic accidents, and half had traumatic brain injury; so some of the lack of extremely coherent narratives might be due to subclinical or observed brain damage. For more detailed analyses of these problems, see Gray and Lombardo (2001), McNally (2003), Porter and Birt (2001), and Shobe and Kihlstrom (1997).

Memory impairment can also refer to the memory being poorly integrated into the cognitive structures supporting the person’s life story and identity. According to the special mechanisms view, such lack of integration (sometimes called dissociation) would be positively related to level of PTSD symptoms. In order to examine the validity of this claim, Berntsen and Rubin (2006a) developed the Centrality of Event Scale (CES), which measures the extent to which a traumatic event is perceived as central to the person’s life story and identity, thus the opposite

of a lack of integration. The CES contains such questions as “I feel that this event has become part of my identity.” “This event has become a reference point for the way I understand myself and the world.” “I often think about the effects this event will have on my future.” We have demonstrated in several studies that the CES correlates positively with the level of PTSD symptoms (Berntsen & Rubin, 2006a, 2006b, 2007, 2008).

To summarize: the findings reviewed here contradict the special-mechanisms view that involuntary remembering yields privileged access to traumatic events and that, in PTSD, the trauma memory is fragmented and poorly integrated into the person’s autobiographical knowledge base. Instead, the findings support the basic-mechanisms view that involuntary and voluntary remembering follow the same pattern and that both are enhanced by emotional stress during encoding. Horowitz and colleagues would have done better if they could have based their explanation of stress response syndromes not on cognitive theories of completion tendencies (and the ensuing assumption of incomplete processing of traumatic events) but on modern research regarding the relation between emotion and memory and involuntary autobiographical memories in daily life. However, most of this work was not available at the time, partly because systematic research on autobiographical memory was almost non-existent in the mid seventies. We should use the data that have accumulated since then to correct past mistakes. This is one important reason why the mnemonic model is needed.

Having addressed the two most elaborate points in Monroe and Mineka’s Comment, namely, the existence of a theoretical model for PTSD and the relation between our mnemonic model and other cognitive models, we now turn to their critique of our evidence and their concerns about causality.

The Evidence for the Mnemonic Model is Robust

Our article uses four headings to review evidence showing that level of PTSD symptoms varies systematically with the availability of the traumatic memory. Monroe and Mineka mention only the two of these headings of which they are critical: *Pharmacologically Induced Amnesia* and *Organic Amnesia*. They make no mention of the evidence under the other two headings: *Childhood Amnesia* and *Self-Relevance Memory Enhancement* or, for example, our review of predisposing factors or the evidence challenging the C3 symptom, also predicted by our model. We refer the reader back to these for a more balanced view of the evidence.

With respect to *Pharmacologically Induced Amnesia*, the propranolol pharmacological intervention we considered is a creative attempt to reduce PTSD symptoms without fully removing the memory of the traumatic event and thus causing numerous ethical and legal issues. The pharmacological interventions are based on a biological theory of memory, which, like our behavioral model, assumes that a stressful event ‘overconsolidates’ and thereby increases the availability of the stressful memory rather than causing it to be more fragmented and less available. Moreover, like our view of memory (e.g., Rubin, 2006), the biological theory considers memory as the integration of basic neural systems including emotion. Put most simply, the biological theory holds that stress increases the activity of the emotional systems of the brain and increases consolidation to the point of ‘overconsolidation’; interfering with ‘overconsolidation’ will affect both the emotional aspects of the memory and also its high availability (Pitman & Delahanty, 2005). The initial exploratory studies with small samples are feasibility tests. Monroe and Mineka’s evaluation of these as full clinical trials is inappropriate both in general and for our theoretical purposes. The reported differences in symptom levels support our model.

Further support for our view and for the biological theory on which these interventions rely comes from recent neuropsychological lesion studies. The amygdala and the ventromedial prefrontal cortex are important components of the emotion circuitry that modulates the

formation and later availability of memories: the amygdala increasing and the ventromedial prefrontal cortex usually decreasing memory formation and retrieval. Koenigs et al. (2008) found that unilateral focal brain damage in these two areas, and only these brain areas, reduced the frequency of PTSD in combat veterans. Full amnesia caused by damage in areas near the amygdala, including the hippocampus, would require bilateral damage and given the nature of the insults, this would likely be fatal. Like the recent pharmacological work mentioned above, this is an exciting novel area of research, suggesting a key role of highly available memories in PTSD.

With respect to *Organic Amnesia*, a main point of our model is that the memory of a stressful event, rather than the event itself, is the key to PTSD. As we reviewed in our article, the Traumatic Brain Injury (TBI) literature supports this claim. When TBI reduces the memory of the trauma, PTSD symptoms diminish. When a memory reduced by TBI is augmented by the later implanting of a memory for the original event, delayed onset PTSD can occur. This delayed onset PTSD is similar to cases mentioned in our article, where memories implanted (de Rivera, 1997) or a reinterpreted as traumatic can result in increased PTSD symptoms (McNally, Perlman, Ristuccia, & Clancy, 2006). Again we stress two points. First, TBI studies that do not measure the memory itself are of relevance only if one assumes that the loss of a brief period of the memory immediately surrounding the trauma, which is the requirement for TBI in most studies, indicates a more extended, general autobiographical memory loss for the trauma that persists for at least a month, and thus until PTSD can be assessed. Second, none of the studies demonstrated a total loss of the memory for the traumatic event and so we do not need a total loss of PTSD to fit our theory. Of note is the observation that as time passes and the period of amnesia from TBI shrinks, the protective effects of TBI on PTSD are reduced (Jones, Harvey, & Brewin, 2005).

In their Comment, Monroe and Mineka fault studies measuring the loss of autobiographical memory in TBI on two counts. First, they ask, “If patients don’t recall the event, how can they reexperience the event?” and they refer to Harvey, Brewin, Jones, and Kopelman (2003), “for a related discussion.” However, Harvey et al. (2003, p. 670) note that a diagnosis of PTSD requires only one of five reexperiencing symptoms. They list four --“intrusive thoughts, dreams, and distress or physiological arousal on exposure to trauma related cues” -- noting that “none of these symptoms are dependent on having a conscious memory of the traumatic incident itself.” Second, Monroe and Mineka note as an objection to our theory that the main difference in the TBI studies that actually measure the autobiographical memory and the reexperiencing, avoidance, and arousal symptoms individually, find significant differences only in the reexperiencing symptoms and not in the avoidance and arousal symptoms. But the equal levels in the latter are not a problem for our model, because one should expect much higher levels of avoidance and arousal symptoms in the TBI groups. There are two reasons for this. First, on the average, the traumas resulting in TBI are more severe. Second, there is an overlap in the avoidance and arousal symptoms of PTSD and TBI. Bryant (2001) notes that four of the seven avoidance symptoms of PTSD and three of the five arousal symptoms are present in TBI. Thus, an imaginary TBI group with absolutely no PTSD would have more avoidance and arousal symptoms than a non-TBI group with absolutely no PTSD.

Causality and Levels of Explanation

By the end of their Comment, Monroe and Mineka raise a number of questions concerning causality. Notably, they suggest that “current perceptions and memories may not ‘cause’ PTSD, as claimed by the mnemonic model, but rather simply be consequences or correlates of the core psychopathology”. Unfortunately, they are not clear about what they consider as the core psychopathology of PTSD, which renders their suggestion vague and hard to evaluate. The possibility which they suggest as an example – that the core psychopathology might be found

at the neurobiological level of the person – is problematic for a number of reasons. First, it would require consistent markers of PTSD to be identified at the neurobiological level. However, in spite of extensive research, no such markers have been found (see Rosen et al., 2008). Second, Monroe and Mineka’s suggestion seems to assume that a behavioral level and a neurobiological level of explanation are mutually exclusive, whereas most scientists would regard the two explanatory levels as complementary. We too consider the two levels as complementary, and many of our arguments about the role of the memory in PTSD are bolstered by neurobiological findings.

One further issue raised by Monroe and Mineka concerns the relation between causality and time. They ask: “How could current memory have ‘caused’ the PTSD of yesterday or yesteryear?” They also suggest a clear distinction between a factor initiating a process and a factor maintaining that process, with only the first qualifying as a cause. But this assumes that it would be possible to identify a particular point in time when the ‘cause’ ends and ‘maintenance’ begins. We do not think that it is possible to arrive at such temporal marker. Indeed, we do not accept the premises for this line of argument, which appears to rely on a simplistic understanding of causality that is generally not applicable to explanations in the social and biological sciences (e.g., Oyama, 2000). Expressed in the standard terminology of developmental science, the type of relationship we are proposing between the current state of the person and his or her way of remembering the event is not a matter of mechanistic linear causality but of dynamic interaction. Many, if not most, researchers on psychological phenomena that develop and change over time would advocate such an interactionistic view. In the view of most researchers, a mechanistic linear and static causal relation between memory and symptoms would be a flaw in any model of PTSD.

Concluding Remarks

We of course agree with Monroe and Mineka’s statement that those suffering from PTSD “deserve the best collective efforts from scientists and clinicians alike to advance useful knowledge of its causes and clinical course.” The mnemonic model and the basic mechanisms view that we have described here reflect our efforts in this regard. We appreciate the opportunity Monroe and Mineka’s Comment has given us to clarify and extend our work on this disorder and to place it in its proper historical and theoretical context.

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