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Trauma processing and the development of posttraumatic stress disorder

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1. Introduction

Most people are exposed to at least one violent or life-threatening situation during the course of their lives (Ozer, Best, Lipsey, & Weiss, 2003). As people progress through their life cycle, they are also increasingly confronted with the death of close friends or relatives. The prognosis varies by the type and intensity of trauma, with physical attack and witnessing someone hurt or killed at the highest risk for chronic symptoms (Breslau, 1998). However, there are great differences in how people deal with these highly aversive experiences. Although most trauma victims report PTSD symptoms immediately after experiencing a trauma, only one-third of the trauma victims show persistent symptoms and develop chronic PTSD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). One of the puzzles surrounding PTSD is which factors determine the maintenance of PTSD symptoms (see also Ehlers & Steil, 1995). Prospective studies have shown that early PTSD symptoms such as the frequency of initial intrusive recollections have little predictive power for chronic PTSD (Shalev, Freedman, Brandes, & Peri, 1997). Among the best candidates to explain the maintenance of PTSD symptoms are information-processing theories contending that the way the traumatic event is processed resulted in chronic

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symptomatology (Foa & Riggs, 1993; Foa & Rothbaum, 1998; Horowitz, 1976, 1986; Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000).

During the last decade research on PTSD received growing interest resulting in a plethora of studies aimed at discovering the psychological nucleus of PTSD. In the eighties, most research on PTSD focused predominantly on Vietnam veterans and used cross-sectional designs. Recently, several longitudinal studies have been done in the immediate aftermath of diverging traumatic events such as motor vehicle accidents, train crashes, physical assaults etc. But still, most studies on PTSD are cross-sectional showing correlates of the disorder that are supposed to constitute plausible risk factors, even though these variables were measured after individuals had developed PTSD. As a starting point, such studies may be highly informative, especially when PTSD patients are compared with trauma victims who did not develop PTSD. Fortunately, researchers do not shrink away from the extensive work of running a longitudinal study on trauma victims. In the past 10 years, number of longitudinal studies sprung up like mushrooms. They may entail either pre-trauma, i.e., prospective, or post-trauma assessments. From the handful of prospective studies, cognitive ability and neuroticism may be considered as the best predictors of PTSD (see for a review McNally, Bryant, & Ehleres, 2003). For example, lower pre-trauma IQ scores predicted greater severity of PTSD symptoms in Vietnam veterans (Kaplan et al., 2002; Macklin et al., 1998) and higher neuroticism was related to higher PTSD scores after miscarriage (Engelhard, van den Hout, & Kindt, 2003). Recent developments in cognitive psychology, such as memory research, are also indispensable for a better understanding of the psychological nucleus of PTSD. These general cognitive theories often form the basis for analogue studies about PTSD-like symptoms. The crucial issue now is, how these predisposing vulnerability factors interact with factors that are supposed to play a role in the development of PTSD. With this special issue, we intended to present studies using diverging methods aimed at understanding the development of chronic PTSD from a cognitive perspective. Although, we are far from a clear model on the development of PTSD, the present articles like dozens of other studies in this field contribute to the understanding of the development of PTSD.

2. The origin of intrusive memories

Intrusive re-experiencing is a core symptom of PTSD. It can take various forms, including intrusive images, flashbacks, nightmares, and distress and physiological reactions, when confronted with reminders (American Psychiatric Association, 1994). Although several studies focused on the quality of intrusive memories, showing that intrusive recollections may be characterized by sensory impressions of the traumatic event (Steil & Ehlers, 2000), relatively little is known about the content of intrusive memories. Holmes et al. performed a qualitative study showing that the worst moments or the "hotspots" of explicit trauma recall correspond with the content of intrusive memories. These results are conceptualized in terms of

aberrations in information processing of the traumatic event. That is, at moments of peak distress (hotspots), arousal will be highest, causing the most disruption to emotional processing. It is suggested that these parts of the trauma are most likely to be processed in a sensory-perceptual manner, rendering them vulnerable to involuntary intrusions in the presence of trauma-related cues (Brewin, 2001; Ehlers & Clark, 2000; Ehlers, Hackmann, & Michael, 2004).

The contention that the arousal level during the traumatic event is responsible for the highly vivid intrusive memories is also indirectly supported by emotional memory research. Superior emotional memory has been frequently observed in laboratory studies in which explicit memory performance is assessed for emotional or neutral stimuli. Evidence indicates that arousal is the crucial factor that is responsible for this memory superiority effect (Bradley, Greenwald, Petry, & Lang (1992), modulated by noradrenergic activation at encoding and consolidation (Cahill & Alkire, 2003; Cahill & McGaugh, 1998; Cahill, Prins, Weber, & McGaugh, 1994; McGaugh, 2000). In a typical explicit memory experiment, participants intentionally search memory for words or perceptual details they saw earlier. If successful, they have a conscious recollection of having seen the stimuli before. Implicit memory, on the other hand, refers to unintentional and unconscious recollections. Although intrusive memories are often referred to as implicit memory, they may best be described as involuntary explicit memory (McNally, 2003; Schacter, 1987). That is, intrusions are involuntarily activated but trauma victims are aware of them. It may, therefore, be questioned whether observations about explicit memory for emotional stimuli being reduced by blocking noradrenergic activation can be extrapolated to intrusive memories. Arntz and colleagues argue that the implicit–explicit distinction fails to explain the highly sensory or perceptual quality of intrusive memories. Inspired by the work of Roediger (1990), who proposed a framework in which the implicit–explicit memory dimension is completely distinguished from the perceptual–conceptual dimension, they investigated which of the four combinations would show the superior emotional memory. Although Ehlers and Clark (2000) and Brewin (2001) propose that perceptual encoding is crucial for the etiology of intrusive memories, both theories tend to associate the perceptual quality with the implicit pathway of retrieval. In contrast with the predictions that follow from these theories, Arntz et al. observed evidence for a superior emotional memory for perceptual details irrespective of the implicit–explicit character of the memory test. Interestingly, the paradigm used by Arntz et al. was the same as that used by Cahill et al. (1994), who showed that the superior emotional memory was the result of noradrenergic activation. Hence, the contention that arousal is responsible for the etiology of intrusive memories does not only apply to subjective reports of distress as was observed by Holmes et al., but seems to be also applicable to physiological levels of arousal. In sum, intrusive memories, which are considered the core symptom of PTSD may originate from perceptual processing of the most arousing moments of the traumatic event. However, as is known from prospective studies, intrusive memories in the immediate aftermath of a trauma are a common symptom in trauma victims. The next question that arises is what mechanisms explain the maintenance of intrusive memories.

3. Prediction of PTSD and its mechanisms

The work of Engelhard and Arntz shows how research on PTSD may be designed in order to elucidate its psychological nucleus. Ex-consequentia reasoning, i.e., inferring danger from the presence of anxiety or intrusions, was shown in Vietnam veterans with PTSD but not in Vietnam veterans without PTSD. The authors suggest that ex-consequentia reasoning may start as a vicious circle in which subjective fear responses or intrusive recollections are used to erroneously validate thoughts of impending doom, which amplifies anxiety responses. However, as noted by them, the cross-sectional nature of the study does not warrant any causal inference with respect to the ex-consequentia reasoning in the development of chronic PTSD. As a next step, they performed a longitudinal study, showing that ex-consequentia reasoning was reliably related to acute and chronic symptoms of PTSD in victims who were exposed to a disastrous train crash. They are currently conducting a study of Dutch soldiers on a peacekeeping mission. Although causal relations can still not be inferred from these predictive observations, it does at least instigate to perform the next step in this series to test whether ex-consequentia reasoning is indeed causally related to the maintenance of PTSD symptoms. However, this is difficult to design, which is often the case with testing causality issues, and restrains researchers from designing the crucial experiments to test vicious circles or causality issues. The same was observed in the field of information processing biases and anxiety. For at least two decades, dozens of cross-sectional studies were performed showing an anxiety-related bias for threatening information. Only recently, studies appear to show evidence for a causal relation of processing bias and anxiety, by inducing attentional or interpretation bias for threat in normal controls (e.g. MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002; Mathews & Mackintosh, 2000). With respect to ex-consequentia reasoning, the causality issue may be tested by inducing ex-consequentia reasoning in normal controls. This is a challenging endeavor and the authors are working on this.

From a recent meta-analysis on predictors of PTSD, peri-traumatic dissociation was the single best predictor of PTSD (Ozer et al., 2003). Note, however, that only 12% of the variance of PTSD symptoms is explained by this concept ($r = 0.35$). From other studies, catastrophic interpretations of both the traumatic event and the individual's responses are shown to be predictive of chronic PTSD (see Ehlers & Clark, 2000). It is unclear whether disturbed reasoning styles, catastrophic interpretation, and dissociation add independently to the development of PTSD or to what degree they interact. Future research should focus on these different predictors in order to obtain a more transparent model of the development of PTSD. An important finding in the studies by Engelhard et al. is that neither neuroticism nor intelligence appears to account for the relation between disturbed reasoning styles and PTSD. Hence, with respect to the general vulnerability factors, ex-consequentia reasoning seems to add to the predictive power of PTSD without conceptual overlap with the predisposing vulnerability factors.

Although prospective studies may shed a light on the predictors of PTSD, which are clinically of great interest, it may not always be clear on a theoretical level why a

certain predictor is related to the development of chronic symptoms. There is indeed convincing evidence that dissociation at the time of the trauma and during the first month after the trauma is one of the best predictors of PTSD. However, the pathogenic mechanism of dissociation remains unclear. The prevailing contention is that dissociation disturbs the processing of traumatic events resulting in typical memory disturbances observed in PTSD, such as memory fragmentation. The paper by Kindt et al. addresses this hypothesis by testing whether state dissociation is related to objectively assessed memory disturbances. The authors argue that most evidence for the memory disturbances in PTSD is based on subjective reports obtained from clinical reports without experimental control of the stimulus material. From cognitive psychology it is known that the subjective experience of memory does not necessarily overlap with the memory performance itself (Shimamura & Squire, 1986). If the supposed memory disturbances result from disturbances in information processing, not only subjectively assessed memory disturbances, should be observed, but also objectively memory disturbances. In two analogue laboratory studies they indeed showed a relation between state dissociation and memory fragmentation. However, this was confined to the subjective experience of memory fragmentation. These results question the viability of the hypothesis that the detrimental effects of dissociation are due to disturbances in information processing of the traumatic event. A remarkable finding was that the presence of intrusions explained half of the variance of the relation between dissociation and subjective memory fragmentation. Note that intrusions are normal characteristics after trauma experiences and that they are characterized by their snapshot or fragmented character (Hackmann, Ehlers, Speckens, & Clark, 2004). This may suggest that the presence of intrusions themselves results in perceived memory fragmentation of the traumatic event. The experience of fragmentation may just be a reflection of PTSD. Alternatively, the influence of dissociation on subsequent PTSD may be mediated by the way individuals appraise their dissociative reactions (see McNally et al., 2003). Future studies should focus on the interrelation between these concepts in order to understand the theoretical basis of the predictive relation of dissociation and subsequent PTSD.

The work by Brewin and Smart is relevant with respect to the understanding of the relation between cognitive ability and the development of PTSD, although this was not the purpose of the study. The pathogenic mechanism that explains why lower cognitive ability is a risk factor for the development of PTSD remains unresolved. In fact, it is striking that whereas numerous studies focus on predictors of PTSD, relatively few studies investigate the underlying pathogenic mechanisms. For practical and clinical reasons, knowledge of predictors may be sufficient to select the most vulnerable individuals. On the other hand, knowledge of the pathogenic mechanism may have consequences for intervention, even if the vulnerability factor itself cannot be changed. An important finding of the study by Brewin et al. is that the relation between working memory capacity is related to the ability to intentionally suppress personally relevant intrusive thoughts. Based on the observation that the effect could not be explained by differences in negative mood, the authors suggest that there are relatively stable individual differences in the ability

to inhibit unwanted material from entering consciousness. Given that working memory capacity and IQ are strongly related, the protective influence of higher IQ scores may be due to the capacity to inhibit successfully intrusive memories of the trauma. When intrusions are not successfully inhibited, a catastrophic interpretation may be inferred. In other words, catastrophic interpretations of intrusions and ex-consequentia reasoning may not only lead to suppression of intrusions and hence to the development of PTSD, but unsuccessful inhibition may in itself also instigate ex-consequential reasoning or other catastrophic interpretations and may therefore smooth the way for the development of chronic PTSD.

4. Conclusion

The articles in this Special Issue contribute, among dozens of other studies, to the understanding of the relations between predisposing vulnerability factors and cognitive factors that influence the processing of traumatic events. A restriction of this Issue was that the papers focus on cognitive processing theories and ignore, for instance, the neurobiological theories of PTSD. The main problem, however, is that we are far from a clear cognitive model with respect to the development of PTSD. Research on the interplay between neurobiological factors and cognitive processes is only interesting when highly specific predictions with respect to the etiology of PTSD can be inferred from an interdisciplinary theory (see also Dalgleish, 2004). Even in the highly specific domain of cognitive theories on PTSD, an enormous divergence in paradigms is employed. This Special Issue presents only a handful of them.

Much of psychology's theories about trauma processing have come from individuals who sought treatment. Even in case treatment studies for PTSD give insight in the mechanisms of change, it would, strictly taken, be unwarranted to extrapolate these studies to the aetiology of chronic PTSD. That is, mechanisms of change in PTSD patients do not necessarily mirror the causal mechanism in the aetiology of chronic PTSD. For example, data-driven or perceptual processing is supposed to be dysfunctional in that it strengthens the intrusive quality of trauma memories. However, we would also suggest that perceptual processing is required in treatment of PTSD patients. That is, perceptual processing may help to promote conceptual processing, allowing for a transformation of the meaning of the trauma and its effects. For example, focusing on perceptual aspects of the traumatic event may help trauma victims to reappraise their own behaviour during the trauma, or to reappraise their own emotional reactions to thinking back of the trauma. The function of perceptual processing during treatment may be to obtain a realistic database such that functional conceptual processing may follow (see also Ehlers et al., 2004). Hence, perceptual processing may be dysfunctional in case it is promoted in all trauma victims, whereas it may contribute to recovery in PTSD patients. The same holds for suppression of trauma memories. That is, avoidance or suppression of trauma memories may only be dysfunctional in patients suffering from PTSD who endorse irrational beliefs with respect to the experienced trauma and its consequences. Psychological theories on PTSD should therefore distinguish

between recovery of PTSD patients and resilience or vulnerability factors in the development of PTSD. In order to reveal the psychological nucleus of PTSD, we hope that future research will focus on the clarification of these divergent processes.

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