

The role of memory in posttraumatic stress disorder: implications for clinical practice

O papel da memória no transtorno de estresse pós-traumático: implicações para a prática clínica

Marcelo Montagner Rigoli,¹ Gustavo Ramos Silva,¹ Fernando Rainho de Oliveira,¹ Giovanni Kuckartz Pergher,²
Christian Haag Kristensen¹

Abstract

Introduction: Posttraumatic stress disorder (PTSD) is a highly prevalent disorder with important social consequences. Several models have been developed with the aim of understanding the mechanisms underlying its symptoms. Intrusions are idiosyncratic symptoms that commonly take the form of involuntary recollection of images or flashbacks about the traumatic event. **Objective:** To review how memory is conceptualized in each of these models and the implications for clinical practice.

Methods: A narrative review of the literature was conducted through analysis of the perspectives of memory in theoretical models of PTSD.

Results: Two main perspectives were identified: 1) models in which specific mechanisms of memory for processing traumatic events are proposed, especially those based on clinical studies, and 2) models in which common mnemonic mechanisms are utilized to explain the phenomenon, primarily based on basic experimental research studies investigating memory. The different theories based on these approaches have led to distinct psychotherapy interventions.

Conclusion: In order to clarify these discrepancies, future research should aim for the methodological rigor of experimental studies, while maintaining the ecological applicability of findings. Cognitive experimental psychopathology is therefore an area on which research funding should be focused. Such studies could elucidate the role of mnemonic aspects in PTSD and how they impact psychological treatments.

Keywords: Posttraumatic stress disorder, memory, experimental psychopathology, cognitive therapy.

Resumo

Introdução: O transtorno de estresse pós-traumático (TEPT) é altamente prevalente e traz consequências sociais importantes. Diversos modelos foram desenvolvidos com o objetivo de compreender os mecanismos subjacentes aos seus sintomas. Intrusões constituem sintomas idiossincráticos, que regularmente tomam a forma de lembranças involuntárias de imagens ou flashbacks do evento traumático.

Objetivo: Revisar como a memória é definida conceitualmente nos modelos teóricos propostos e as implicações para a prática clínica. **Métodos:** Uma revisão narrativa da literatura foi conduzida através da análise das perspectivas de memória em modelos teóricos de TEPT. **Resultados:** Duas perspectivas principais foram identificadas: 1) modelos com a proposição de mecanismos de memória específicos ao processamento de eventos traumáticos, especialmente apoiados em estudos clínicos, e 2) modelos em que mecanismos mnemônicos comuns são utilizados para explicar o fenômeno, primariamente baseados em estudos experimentais de base investigando memória. As diferentes teorias baseadas nessas perspectivas levaram a distintas abordagens psicoterapêuticas.

Conclusões: Para clarificar essas discrepâncias, sugere-se que pesquisas futuras busquem maior rigor metodológico de estudos experimentais, mantendo a aplicabilidade ecológica dos achados. A psicopatologia cognitiva experimental é uma área na qual devem ser focados os financiamentos de pesquisa. Tais estudos podem elucidar o papel de aspectos mnemônicos no TEPT e como impactam tratamentos psicológicos.

Descritores: Transtorno de estresse pós-traumático, memória, psicopatologia experimental, terapia cognitiva.

¹ Programa de Pós-Graduação em Psicologia, Escola de Humanidades, Pontifícia Universidade Católica do Rio Grande do Sul (PUCRS), Porto Alegre, RS, Brazil. ² Faculdades Integradas de Taquara (FACCAT), Taquara, RS, Brazil.

Financial support: Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES) and Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPa).

Submitted Jan 06 2015, accepted for publication Sep 05 2015. No conflicts of interest declared concerning the publication of this article.

Suggested citation: Rigoli MM, Silva GR, de Oliveira FR, Pergher GK, Kristensen CH. The role of memory in posttraumatic stress disorder: implications for clinical practice. Trends Psychiatry Psychother. 2016;38(3):119-127. http://dx.doi.org/10.1590/2237-6089-2014-0063

Introduction

Until recently, posttraumatic stress disorder (PTSD) was listed as one of the anxiety disorders in the Diagnostic and Statistical Manual of Mental Disorders (DSM).1 However, a new category was introduced in the DSM 5 to encompass several disorders, including PTSD: the trauma and stressor-related disorders.² The explicit presence of a traumatic or stressful event is a core etiological factor and diagnostic criterion for disorders in this category. This assumption differentiates the category from others in a unique way since it is the only one in which an event in the past is listed as a diagnostic criterion for something that is happening in the present. What then is the connection between experiencing a traumatic or stressful event and the symptoms of these disorders? The answer is memory. Memory is the cognitive function that allows us to connect with the past, accumulate knowledge and learn from our experiences, providing us with a notion of self.3,4 More specifically, it has been widely argued that PTSD is mainly a memory disorder, and that intrusions are among its hallmark symptoms2 (cluster B).

There is a longstanding debate on how memory works when it comes to traumatic events and PTSD. One of two main approaches assumes that there must be mnemonic mechanisms that are idiosyncratic to PTSD.^{3,5-7} In contrast, researchers who uphold the other perspective state that there is no specific mechanism involved,⁸ and that individual differences in emotional processing and autobiographical memory account for development of the disorder.^{4,9} Our aim in this article is to summarize the different perspectives on memory that underlie theoretical models of PTSD and their relationships with clinical practice.

Posttraumatic stress disorder (PTSD)

Stressful situations that threaten physical and psychological wellbeing are part of the overall experience of life. Nonetheless, they can become traumatic depending on their intensity and frequency and on the response of people who experience them. 10-11 Approximately 60 to 90% of people go through a potentially traumatic event during their lifetimes 12-13 and around 29% of people exposed to trauma are diagnosed with PTSD, 14 which is the mental disorder most commonly triggered after traumatic events. 15-17

The American Psychiatric Association (APA)² defines PTSD as a symptomatic response to experiences involving death, threatened death, actual or threatened serious injury, or actual or threatened sexual violence

(criterion A), either by direct exposure, witnessing in person, learning that it happened to a close relative or close friend or by repeated or extreme indirect exposure to aversive details of the event. This response must fulfill at least one criteria from the symptom clusters of intrusive memories, dreams or flashbacks (criterion B) and persistent effortful avoidance of distressing traumarelated stimuli (criterion C), as well as two criteria from negative alterations to cognitions and mood that began or worsened after the traumatic event (criterion D) and trauma-related alterations in arousal and reactivity (criterion E). These symptoms must cause distress or functional impairment (criterion G) for at least a month (criterion F).

Lifetime PTSD prevalence has been estimated at around 6.8%,¹⁶⁻¹⁸ making it the fifth most prevalent disorder in the United States. Moreover, the disorder is responsible for significant social and economic consequences, such as medical care costs, work absenteeism, detachment from family and friends and others.¹⁹⁻²³ Risk factors for PTSD are: 1) pre-traumatic factors, such as the existence of previous traumas, adjustment problems, depressive symptoms and mental illness in the family; 2) peritraumatic factors, especially perceived risk of death, maladaptive emotional response and dissociation; and 3) posttraumatic factors, such as the lack of social support.²⁴⁻²⁵

In short, PTSD is a highly prevalent mental disorder¹⁸ with important socioeconomic consequences²⁰ and a wide range of risk factors.²⁴ Its symptoms manifest in behavioral, cognitive and physiological changes, with intrusive symptoms playing a central role.² This symptom cluster is intimately connected to how traumarelated information is processed. However, there is no consensus among researchers regarding how such processing occurs and several explanatory models have attempted to explain this phenomenon.

Theoretical models of PTSD and memory

Explanatory models for PTSD have existed since its formalization as a diagnostic category in the DSM-III.²⁶ Before the diagnosis was structured, however, a two-factor model proposed by Mowrer²⁷ accounted for learning and maintenance of fear by classical and operant conditioning, leaving the cognitive portion of the phenomenon unaccounted for. Subsequently, new models aiming at greater clinical applicability emerged along with the emotional processing theory proposed by Foa & Kozak.⁷ In the cognitive model developed by Ehlers & Clark,⁶ memory becomes a more central aspect of the disorder and it is the central feature of dual

representation theory⁵ and the memory-based model.^{9,14} Many more psychological theories of PTSD exist, such as stress response theory,²⁸ shattered assumptions theory²⁹ and the anxious apprehension model.^{30,31} However,

since they do not focus on the mnemonic aspect of the disorder, they are not reviewed here (Table 1). For further information, we recommend the review by Brewin & Holmes.³²

Table 1 - Main characteristics of PTSD models

Model	Main authors	Main characteristics
Conditioning model	Mowrer ²⁷	Classic and operant conditioning: fear conditioning due to the traumatic experience, pairing previous neutral stimuli with the aversive and threatening stimuli.
Emotional processing theory	Foa & Kozak ⁷	The fear structure concept: stimuli, responses and semantic attributes stored in memory form a maladaptive network, identifying non-threatening stimuli as dangerous and generating inadequate responses and attributions.
Cognitive model	Ehlers & Clark ⁶	The trauma is processed in a way that creates a sense of serious current threat because of two main features: individual differences in the evaluation of trauma and/or its consequences; individual differences in the nature of memory of the event and its relationship with other autobiographical memories.
Dual representation theory	Brewin ³	Two memory systems: verbally accessible memories, including context-bound trauma material that can be voluntarily recalled and described; situationally accessible memories, limited to sensory-bound material recalled through involuntary cues. During a traumatic experience, the lack of contextual processing compared to sensory information results in difficulty narrating the trauma and integrating it with autobiographical memories.
Memory based model	Rubin ³³	Basic mechanisms of emotion, autobiographical memory and personality are sufficient to account for development of PTSD. A series of independent systems (sensory, visuospatial, language, emotion, narrative, motor, explicit memory, search and retrieval) interact to produce autobiographical memories and each part of this network has a specific and important role in the recall of events.

Conditioning model

One of the first attempts to explain PTSD was mainly based on classic and operant conditioning and took Mowrer's two factor model²⁷ as its core inspiration. The model is based on the concept that a traumatic experience would create a fear conditioning scenario, pairing previously neutral stimuli with the aversive and threatening stimuli. Several previously neutral stimuli then start to elicit a fear response. The person may learn that avoiding the conditioned stimuli can prevent the fear responses from being elicited. This safety-seeking behavior works as negative reinforcement, which will not allow the fear conditioning to dissipate by the expected extinction.³⁴

Evidence for this model has been found in both animal models and clinical research. It is possible to draw several parallels between manifestations of fear conditioning in animals and posttraumatic reactions. In rats, analgesia and avoidance are very similar to PTSD symptoms of persistent arousal, numbing and avoidance.³⁵ There is also evidence from the field of epigenetics supporting the importance of fear

conditioning in PTSD.³⁶ Furthermore, this understanding of these mechanisms led to all exposure-based treatments for PTSD and these have been shown to be effective.³⁷

In this model, memory processes are limited to storing and retrieving data regarding stimuli and associations involved in the conditioning processes and no explanations of underling mechanisms are offered. Later, Rescorla³⁸ proposed changes to the classical conditioning theory, suggesting that learned associations would be stored in long term memory. Additionally, events associated with fear would then work as predictors of future threatening situations.²² Although these concepts did not immediately lead to acknowledged interventions, they were the precursors of effective exposure treatments such as prolonged exposure therapy.³⁹

Emotional processing theory

The emotional processing theory developed by Foa & Kozak 7 is mainly based on the concept of fear structures, originally proposed by Lang. 40

These structures are arrangements of propositions about stimuli (e.g., a firearm), responses (e.g., to hyperventilate) and semantic attributes (e.g., "I am going to die") stored in memory. The theory suggests that these structures malfunction in PTSD, identifying non-threatening stimuli as dangerous and therefore generating inadequate responses and attributions. Foa & Rothbaum⁴¹ cite a series of behavioral and cognitive mechanisms, including mnemonic processes, to explain these structural failures. As in the conditioning model, avoidance plays an important role in maintaining PTSD. This avoidance is not limited to behavioral safety seeking, but also includes cognitive avoidance (i.e., trying not to think about things that may trigger trauma memories) and emotional numbing, both of which momentarily reduce fear structure activation, while preserving it over the long term.⁴²

In this model, memory starts to occupy a more central position in the explanatory framework of PTSD. The aforementioned fear structures are thought to be represented in memory. It is argued that peritraumatic dissociative states (e.g., derealization and depersonalization) result in a disjointed and fragmented memory, and therefore in a malfunctioning fear structure, which, in turn, would lead to future incorrect attributions of environmental stimuli and to further generalization of the fear structure.

Emotional processing theory is the cornerstone of prolonged exposure therapy, which is a widely employed and effective treatment for PTSD.⁴³ This procedure exposes the patient to "corrective information" by confronting the fear structures, in contrast to avoidance behaviors. This is achieved through imaginal exposure or situational exposure. In the first case, the patient is requested to narrate trauma memories several times, inducing the fear responses in therapy until the connections between the emotional reaction and the trauma related stimuli are diminished or become extinct. In the second case, situational or in vivo exposure requires the patient to face distressing trauma-related stimuli in a safe context. This procedure aims to break or decrease the association between the fear response and the traumarelated stimuli.41 In both interventions, the goal is to modify the existing memories of the trauma and of its consequences to more adaptive ones.

Cognitive model

The cognitive model of PTSD developed by Ehlers & Clark⁶ proposes that people develop the disorder by processing the event in a way that creates a sense of serious current threat. The authors highlight two

main features of trauma processing: 1) individual differences in the evaluation of trauma and/or its consequences; and 2) individual differences in the nature of memories of the event and their relationship with other autobiographical memories.

With relation to the first feature, Ehlers & Clark⁶ point out that different appraisals of the trauma may have a role in the development of PTSD. Firstly, the traumatic event itself may be overgeneralized, broadening the scope of threatening situations (e.g., situations that had previously been neutral are now associated with fear responses). In terms of trauma sequelae, a variety of negative appraisals of the consequences of the event may be developed, creating a sense of current threat (e.g., not seeing the symptoms as a natural part of the recovery process, but as an irreversible situation). These appraisals lead to maladaptive emotional and behavioral responses.

As for the nature of trauma memory, Ehlers & Clark⁶ mainly base their view on the dual representation model proposed by Brewin et al.5 (explained in detail in the next section). These memories are comprised largely of sensory impressions experienced as if they are occurring in the present (i.e., re-experience symptoms), as opposed to thoughts about a past event. Besides their content, these memories: a) are poorly elaborated and lack incorporation into autobiographical memory (i.e., the event is dissociated from the rest of the individual's personal history; b) have a strong stimulus-stimulus (S-S) and stimulusresponse (S-R) relationship for trauma-related content (i.e., trauma memories are easily triggered by a wide range of stimuli); and c) have strong perceptual priming, meaning that a reduced threshold exists for triggering trauma related memories through trauma cues. Differences in appraisals and the nature of the trauma memory create a circular relationship between them. When the individual retrieves information from the traumatic event, the recall is biased by appraisals. In other words, only mnemonic information coherent with these appraisals is recalled.

The cognitive model is the theoretical basis for cognitive therapy protocols for PTSD (CT-PTSD).⁴⁴ The CT-PTSD method is an individualized application of the cognitive model aiming to achieve three main goals: goal 1 is to adjust the excessively negative appraisals about the trauma and its consequences; goal 2 is to reduce re-experience symptoms by reprocessing trauma-related memories and identifying trauma cues; and goal 3 is to replace dysfunctional behaviors and cognitive strategies with more adaptive ones. These goals are primarily achieved through a series of cognitive and behavioral interventions, such

as identifying safety behaviors and "hot spots" in the patients' recollection of the trauma, reclaiming areas in the patients' life that have been left behind due to the traumatic event, cognitive restructuring and behavioral experiments. For an overview of the treatment see the study by Ehlers et al.,⁴⁵ in which the intervention is shown to be effective for early-onset PTSD.

Dual representation theory

The dual representation theory developed by Brewin et al.5 explores memory functioning and its central role in the development of re-experiencing symptoms. The existence of two memory systems is proposed: verbally accessible memories (VAM) and situationally accessible memories (SAM). These two systems operate in parallel, although one can overlap the other at certain times. The VAM system is part of oral and written reports of traumatic situations, generating an integrated notion of autobiographical content, which can be voluntarily retrieved. As such, VAM memories are represented in a coherent context, including the notion of past, present and future, aggregating prior information, during and after the trauma for storing in long term memory through conscious processing. Therefore, despite their availability for verbal recollection, these memories are limited regarding the amount of information that can be encoded consciously.

At the same time, during the traumatic experience, conscious processing is impaired by the attention given to immediate threat and the high level of emotional reactivity. This disruption is related to the development of intrusive memories and flashbacks, since these are predominately situationally accessible memories that are retrieved when triggered involuntarily by external trauma cues (e.g., the sound of a motor vehicle) or internal ones (e.g., a specific emotional state). Situationally accessible memories contain information encoded and registered with a low degree of conscious processing of the traumatic event and are focused on perceptual elements, such as sounds and images.

The SAM system is also responsible for registering physiological reactions to trauma, such as heart rate, sweating, temperature variations and pain. This results in memories with higher intensity, the feeling of "here and now" and in greater sensory involvement of intrusive memories when compared to voluntarily retrieved memories. Another important aspect of SAM is that they are not encoded verbally, therefore it is difficult to narrate them, process them and integrate them with autobiographical memories. Situationally accessible memories turn out to be very hard to control, since people are likely to encounter trauma mnemonic

cues that trigger these memories independently of voluntary intent.³² The relevance of this model lies in the fact that re-experience symptoms are among the most important traits of PTSD.^{3,46}

Clinical implications of this model mainly relate to early interventions, 47 which happen during early stages of memory consolidation. The idea is that, with the correct intervention, it might be possible to achieve a substantial reduction in PTSD symptoms, by favoring VAM processing instead of SAM, 48 particularly of symptoms that are part of the intrusive cluster. In recent studies, researchers tested this model by conducting concurrent tasks during memory consolidation of aversive stimuli. Playing the videogame TETRIS, for example, successfully reduced the number of intrusions of persons exposed to videos of traumatic events. According to the dual representation theory, this is due to the fact that the visuospatial task (i.e., the videogame) competes with sensory-based processing of the aversive video, which is in turn processed in a context-bound manner, through the VAM system.^{3,47,48}

Memory based model

Rubin et al.4 have classified all of the theories presented so far, which are largely derived from the clinical research context, as adopting a "special mechanisms view", since they consider the mnemonic phenomena of PTSD to be idiosyncratic. In contrast, Rubin et al.9 propose that development of PTSD can be accounted for by the basic mechanisms of emotion, autobiographical memory and personality, which they call a "basic mechanisms view". The memory-based model9 suggests that people experience negative events and then alter them. Memory is not constant; it changes over time due to factors that affect all memories of all people and are related to individual differences such as personality traits and gender. The interaction of these factors will determine the incidence of PTSD.

Autobiographical memories are those related to events experienced by the individual and they play an important role in many psychological disorders, such as mood disorders. ⁴⁹⁻⁵¹ According to Beck's cognitive theory, psychological disorders can be explained by the cognitive triad, consisting of the view that people have of themselves, the world and the future, of which the vision of self is the most determinant of psychopathological symptoms.²²

According to Rubin,⁵² the computational metaphor of cognition does not take into account the specific properties of each particular system that influences memory. A computer has only one way of processing

data, independent of other base systems. Human memory processing can only be understood using an approach that considers each base system involved and their respective properties: cognitive, neural and behavioral. Each system must be comprehended individually and along with each aspect related to it.^{33,52}

The systems that integrate this proposed model are the sensory system (e.g., sight, hearing and smell); the visuospatial imagery system, related to the spatial location of objects and people; language; emotion; narrative; the motor system; explicit memory; and the search and retrieval system, which coordinates and links information with other systems.33,53 The autobiographical memory model proposed by Rubin,³³ known as the basic-systems model (BSM), consists of a series of independent systems that interact with each other. According to the author, each system comprises a network containing its specificities (processes and forms of organization that are typical of each system). The interaction of this network produces autobiographical memories and each part of this network has a specific and important role in the recall of events.

Given that a relationship between autobiographical memories and PTSD exists, the model suggests that the mnemonic system as a whole is associated with the disorder, rather than only memories directly related to the traumatic event. This view contradicts the idea of memories processed by a specific mechanism characteristic of PTSD. Even though exposure to a certain type of event (i.e., life-threatening or traumatic) is required for a PTSD diagnosis, it is unlikely that memories related to trauma have a different set of mechanisms than those involved in general autobiographical memories.⁵⁴

The autobiographical memory theory of PTSD proposed by Rubin takes into consideration three factors that predict responses related to the memory of a potentially traumatic event: 1) the emotional intensity of the memory, 2) when and how often it was recalled in the past and 3) how central it was considered to be in the individual's life and identity. The higher the recall frequency, voluntary or not, the greater will be the tendency for future recollection and for perceiving the memory as central.⁵⁴ Centrality and repetition are therefore associated with memory maintenance, as well as higher intensity and emotional valence.⁴

Autobiographical memory does not remain static once encoded, rather it is subject to changes in the representational meaning of memory content due to new events – including potential traumas. Interaction between the characteristics of an event and the processes of encoding, storage and recall may

contribute to the occurrence of more frequent and intense PTSD symptoms.⁴

Experimental psychopathology

Albeit recent, there is increasing interest in the cognitive mechanisms involved in PTSD.55 While many of these studies are ex post facto, they offer a better understanding of the processes involved, such as the selective processing of trauma-related stimuli, the greater capacity to access trauma-related memories, increased rumination of memories related to the event, and difficulty in recalling specific trauma-related autobiographical events.⁵⁵ Nevertheless, a practical difficulty with addressing PTSD exists, since the disorder is frequently studied in isolated parts, such as attentional changes⁵⁶⁻⁵⁷ or physiological changes.⁵⁸⁻⁶¹ This difficulty may relate to an ethical and methodological concern: the infeasibility of generating trauma in research participants. One possible answer to this problem is to perform systematic assessments with people who will carry out activities with a high probability of encountering potentially traumatic events, such as firefighters⁶² or in military extreme survival training.63 While such studies solve the aforementioned problem and show great ecological validity, the lack of control over peritraumatic variables remains an issue.

Experimental studies attempting to understand different psychopathological phenomena have increased over recent decades. 64,65 Experimental psychopathology research is conducted in a controlled environment, with sampling of human and nonhuman animals, in order to investigate the etiology, development and maintenance of mental disorders, which could contribute to strategies for prevention and intervention.65 This approach is at the threshold of two other major areas of Psychology, 1) basic research and 2) clinical psychopathology, but it also has some crucial differences in terms of its object of study and final aim. The first studies specific and fundamental phenomena⁶⁵ and takes great care over methodological control and falsifiability of its findings, 66,67 but has reduced clinical applicability. The second area aims to achieve better understanding of the phenomena with more immediate answers and direct impacts on health, and is very much concerned with measuring and responding to ecological demands. 65 In turn, the experimental study of psychopathological processes tries to fill the gap between these two approaches, preserving and maintaining the experimental rigor, while proposing secondary prophylactic interventions.64

Many studies have utilized the trauma analogue paradigm along with tasks that stimulate or consume

verbal or visuospatial processing for the experimental study of PTSD phenomena. The trauma analogue paradigm consists of the use of videos of stressful content, usually with scenes involving threat to physical integrity, with participants without a diagnosis of mental disorders. Variables of interest are manipulated and subsequently measured.⁶⁸ Attention and memory are among the psychological processes involved in development and maintenance of PTSD that have received greatest emphasis,⁶⁹ along with their impact on the incidence of symptoms of reliving.^{46,70,71} To achieve this, manipulations are performed during and immediately after the trauma analogue, usually involving tasks aiming to increase verbal encoding of information, or reduce it through concurrent verbal tasks.⁶⁸

Conclusions

In response to the social impact of PTSD, several explanatory models have been developed to explain the disorder. Novel models have focused on the cognitive processes involved, especially on memory and its functioning during trauma. In summary, current models of PTSD consider the mechanisms involved in cognitive processing and intrusion symptoms as fundamental to understanding the disorder. Theories based on clinical studies hypothesize the existence of specific memory mechanisms involved in PTSD,5,6 but it has also been hypothesized that the disorder is triggered by mechanisms of regular autobiographical memory when interacting with individual characteristics in the context of stressful events.9 There is increasing investment in experimental studies designed to achieve better understanding of these phenomena.

It is hypothesized that these mechanisms are involved in development and maintenance of intrusion symptoms, which are a distinctive manifestation of the disorder. Posttraumatic stress disorder also presents us with an apparent paradox: while patients have great difficulty reporting the event accurately and voluntarily (criterion D1), information about the trauma invades consciousness intensely and involuntarily (criterion B1) and is often experienced as though the events were happening here and now (criterion B3).²

Mowrer's conditioning model presents an elegant and parsimonious solution for development and maintenance of PTSD. However, it fails to account for individual differences in conditioning and dysfunctional beliefs.²² Furthermore, the model is not clear about etiological differences regarding other anxiety disorders and several posttraumatic symptoms.³² Furthermore, memory is not an important aspect of this model and its importance lies

in the fact that it laid the foundations for other theories and treatments, such as prolonged exposure therapy.³⁹

The emotional processing model⁷ is one of the most widely used by clinicians and is well articulated with the interventions that derive from it.²² In this model memory starts to play a more central role in understanding PTSD, mainly through fear structures. In spite of its great explanatory power and clinical applicability,³² this model presents several conceptual problems, such as lack of specificity of the elements and mechanisms involved.⁷²

The cognitive model⁶ brought together several aspects from previous theoretical frameworks, such as conditioning processes and the role of fear and exposure in treatment. However, this new proposal is focused on how each person interprets each event and their consequences. These interpretations relate mainly to how the individual processes trauma memories. It is argued that the attempt to encompass all of these aspects leads to a less parsimonious and even speculative view.²² Cognitive therapy for PTSD has shown promising results, including in a comprehensive framework.⁷³

The dual processing theory⁵ is not intended to be a full explanatory model for PTSD. It focuses on how memory is processed and on what might be the origin of one of the most central symptoms of PTSD, intrusive thoughts. According to Rubin's basic mechanisms proposal: 1) both voluntary and involuntary access to trauma memories are increased; 2) the level of posttraumatic symptoms will be positively related to how available the memory of trauma is for voluntary and involuntary retrieval, and its centrality in the life and identity of the individual; 3) involuntary memories will not be exclusively about negative or trauma-related content; 4) involuntary trauma memories do not involve more sensory content than general content.^{4,33} These implications are amplified to generate a major controversy in the field of PTSD treatment: are memories of traumatic events inherently different from other autobiographical memories? Should the clinician understand processing of trauma memories differently from that of non-trauma memories?74

Finally, we propose that experimental investigation of cognitive mechanisms underlying psychopathology can contribute to the issue. Both sides of the argument have raised methodological criticisms of the other, one being that basic research into memory cannot encompass all the aspects of PTSD and the other that clinical study designs impede assumptions about mechanisms. Therefore, application of experimental rigor to psychopathology should not only help to clear up issues related to the mechanisms debated, but also opens a new path to devising new interventions.

Acknowledgements

The authors are grateful for the support provided by Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES), Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq), and Pontifícia Universidade Católica do Rio Grande do Sul (PUCRS).

References

- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR). Arlington: American Psychiatric Publishing; 2000.
- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5). Arlington: American Psychiatric Publishing; 2013.
- Brewin CR. Episodic memory, perceptual memory, and their interaction: foundations for a theory of posttraumatic stress disorder. Psychol Bull. 2014;140:69-97.
- Rubin DC, Boals A, Berntsen D. Memory in posttraumatic stress disorder: properties of voluntary and involuntary, traumatic and nontraumatic autobiographical memories in people with and without posttraumatic stress disorder symptoms. J Exp Psychol Gen. 2008;137:591-614.
- Brewin CR, Dalgleish T, Joseph S. A dual representation theory of posttraumatic stress disorder. Psychol Rev. 1996;103:670-86.
- Ehlers A, Clark DM. A cognitive model of posttraumatic stress disorder. Behav Res Ther. 2000;38:319-45.
- Foa EB, Kozak MJ. Emotional processing of fear: exposure to corrective information. Psychol Bull. 1986;99:20-35.
- Shobe KK, Kihlstrom JF. Is traumatic memory special? Curr Dir Psychol Sci. 1997;6:70-4.
- Rubin DC, Berntsen D, Bohni MK. A memory-based model of posttraumatic stress disorder: evaluating basic assumptions underlying the PTSD diagnosis. Psychol Rev. 2008;115:985-1011.
- Gunnar M, Quevedo K. The neurobiology of stress and development. Annu Rev Psychol. 2007;58:145-73.
- Yehuda R, LeDoux J. Response variation following trauma: a translational neuroscience approach to understanding PTSD. Neuron. 2007;56:19-32.
- Breslau N. Epidemiologic studies of trauma, posttraumatic stress disorder, and other psychiatric disorders. Can J Psychiatry. 2002;47:923-9.
- Ogle CM, Rubin DC, Berntsen D, Siegler IC. The frequency and impact of exposure to potentially traumatic events over the life course. Clin Psychol Sci. 2013;1:426-34.
- Santiago PN, Ursano RJ, Gray CL, Pynoos RS, Spiegel D, Lewis-Fernandez R, et al. A systematic review of PTSD prevalence and trajectories in DSM-5 defined trauma exposed populations: intentional and non-intentional traumatic events. PLoS One. 2013;8:e59236.
- Keane TM, Marshall AD, Taft CT. Posttraumatic stress disorder: etiology, epidemiology, and treatment outcome. Annu Rev Clin Psychol. 2006;2:161-97.
- Kessler RC. The national comorbidity survey: preliminary results and future directions. Int J Methods Psychiatr Res. 1995;5:139-51.
- Kessler RC, Chiu WT, Demler O, Merikangas KR, Walters EE. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry. 2005;62:617-27.
- Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry. 2005;62:593-602.
 Kessler RC. Gender differences in major depression:
- Kessler RC. Gender differences in major depression: epidemiological findings. In: Frank E, editor. Gender and its effects on psychopathology. American Psychopathological Association Series. Arlington: American Psychiatric Publsihing; 2000. p. 61-84.
- Olatunji BO, Cisler JM, Tolin DF. Quality of life in the anxiety disorders: a meta-analytic review. Clin Psychol Rev. 2007;27:572-

- 81
- 21. Schaefer LS, Lobo B de OM, Kristensen CH. Transtorno de estresse pós-traumático decorrente de acidente de trabalho: implicações psicológicas, socioeconômicas e jurídicas. Estud Psicol (Natal). 2012;17:329-36.
- Taylor S. Clinician's guide to PTSD: a cognitive-behavioral approach. New York: Guilford Press; 2006.
- Walker EA, Katon W, Russo J, Ciechanowski P, Newman E, Wagner AW. Health care costs associated with posttraumatic stress disorder symptoms in women. Arch Gen Psychiatry. 2003;60:369-74.
- Brewin CR, Andrews B, Valentine JD. Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. J Consult Clin Psychol. 2000;68:748-66.
- 25. Ozer EJ, Best SR, Lipsey TL, Weiss DS. Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis. Psychol Bull. 2003;129:52-73.
- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Third Revised Edition (DSM-III-R). Washington: American Psychiatric Association; 1987.
- 27. Mowrer OH. On the dual nature of learning—a re-interpretation of "conditioning" and "problem-solving." Harv Educ Rev. 1947;17:102-48.
- Horowitz MJ. Stress response syndromes: PTSD, grief, and adjustment disorders. 3rd edition. New York: Jason Aronson; 1997.
- 29. Janoff-Bulman R. Shattered assumptions. New York: Simon and Schuster; 2010.
- 30. Jones JC, Barlow DH. The etiology of posttraumatic stress disorder. Clin Psychol Rev. 1990;10:299-328.
- Jones JC, Barlow DH. A new model of posttraumatic stress disorder: implications for the future. In: Saigh PA, editor. Posttraumatic stress disorder: a behavioral approach to assessment and treatment. New York: Macmillan; 1992. p. 147-65
- 32. Brewin CR, Holmes EA. Psychological theories of posttraumatic stress disorder. Clin Psychol Rev. 2003;23:339-76.
- 33. Rubin DC. A basic-systems approach to autobiographical memory. Curr Dir Psychol Sci. 2005;14:79-83.
- Keane TM, Fairbank JA, Caddell JM, Zimering RT, Bender ME. A behavioral approach to assessing and treating post-traumatic stress disorder in Vietnam veterans. In: Figley CR, editor. Trauma and its wake: the study and treatment of post-traumatic stress disorder. New York: Brunner/Mazel; 1985. p. 257-94.
 Foa EB, Zinbarg R, Rothbaum BO. Uncontrollability and
- Foa EB, Zinbarg R, Rothbaum BO. Uncontrollability and unpredictability in post-traumatic stress disorder: an animal model. Psychol Bull. 1992;112:218-38.
- Zovkic IB, Sweatt JD. Epigenetic mechanisms in learned fear: implications for PTSD. Neuropsychopharmacology. 2013;38:77-93.
- Taylor S, Thordarson DS, Maxfield L, Fedoroff IC, Lovell K, Ogrodniczuk J. Comparative efficacy, speed, and adverse effects of three PTSD treatments: exposure therapy, EMDR, and relaxation training. J Consult Clin Psychol. 2003;71:330-8.
- 38. Rescorla RA. Pavlovian conditioning. It's not what you think it is. Am Psychol. 1988;43:151-60.
- Foa E, Hembree E, Rothbaum BO. Prolonged exposure therapy for PTSD: emotional processing of traumatic experiences therapist guide. New Tork: Oxford University; 2007.
- Lang PJ. Imagery in therapy: an information processing analysis of fear. Behav Ther. 1977;8:862-86.
- 41. Foa EB, Rothbaum BO. Treating the trauma of rape: cognitive-behavioral therapy for PTSD. New York: Guilford Press; 2001.
- Dunmore E, Clark DM, Ehlers A. Cognitive factors involved in the onset and maintenance of posttraumatic stress disorder (PTSD) after physical or sexual assault. Behav Res Ther. 1999;37:809-20
- 43. Jayawickreme N, Cahill SP, Riggs DS, Rauch SA, Resick PA, Rothbaum BO, et al. Primum non nocere (first do no harm): symptom worsening and improvement in female assault victims after prolonged exposure for PTSD. Depress Anxiety. 2014;31:412-9.
- Ehlers A, Clark DM, Hackmann A, McManus F, Fennell M. Cognitive therapy for post-traumatic stress disorder: development and evaluation. Behav Res Ther. 2005;43:413-31.
- 45. Ehlers A, Clark DM, Hackmann A, McManus F, Fennell M, Herbert C, et al. A randomized controlled trial of cognitive therapy, a self-help booklet, and repeated assessments as early interventions for posttraumatic stress disorder. Arch Gen Psychiatry. 2003;60:1024-32.

- Laposa JM, Alden LE. The effect of pre-existing vulnerability factors on a laboratory analogue trauma experience. J Behav Ther Exp Psychiatry. 2008;39:424-35.
- Holmes EA, James EL, Kilford EJ, Deeprose C. Key steps in developing a cognitive vaccine against traumatic flashbacks: visuospatial Tetris versus verbal Pub Quiz. PLoS One. 2010;5:e13706.
- Holmes EA, James EL, Coode-Bate T, Deeprose C. Can playing the computer game "Tetris" reduce the build-up of flashbacks for trauma? A proposal from cognitive science. PLoS One. 2009;4:e4153.
- Nandrino JL, Pezard L, Posté A, Réveillère C, Beaune D. Autobiographical memory in major depression: a comparison between first-episode and recurrent patients. Psychopathology. 2002:35:335-40.
- Scott J, Stanton B, Garland A, Ferrier IN. Cognitive vulnerability in patients with bipolar disorder. Psychol Med. 2000;30:467-72.
- 51. Williams JM, Broadbent K. Autobiographical memory in suicide attempters. J Abnorm Psychol. 1986;95:144-9.
- 52. Rubin DC. The basic-systems model of episodic memory. Perspect Psychol Sci. 2006;1:277-311.
- Larsson M, Willander J. Autobiographical odor memory. Ann N Y Acad Sci. 2009;1170:318-23.
- Rubin DC, Dennis MF, Beckham JC. Autobiographical memory for stressful events: the role of autobiographical memory in posttraumatic stress disorder. Conscious Cogn. 2011;20:840-56.
- McNally RJ. Experimental approaches to cognitive abnormality in posttraumatic stress disorder. Clin Psychol Rev. 1998;18:971-82.
- 56. Bryant RA, Harvey AG. Attentional bias in posttraumatic stress disorder. J Trauma Stress. 1997;10:635-44.
- Pineles SL, Shipherd JC, Welch LP, Yovel I. The role of attentional biases in PTSD: is it interference or facilitation? Behav Res Ther. 2007;45:1903-13.
- 58. Cohen H, Benjamin J, Geva A, Matar M, Kaplan Z, Kotler M. Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: application of power spectrum analysis of heart rate variability at rest and in response. Psychiatry Res. 2000;96:1-13.
- Lindauer RT, van Meijel EP, Jalink M, Olff M, Carlier IV, Gersons BP. Heart rate responsivity to script-driven imagery in posttraumatic stress disorder: specificity of response and effects of psychotherapy. Psychosom Med. 2006;68:33-40.
- Sack M, Hopper JW, Lamprecht F. Low respiratory sinus arrhythmia and prolonged psychophysiological arousal in posttraumatic stress disorder: Heart rate dynamics and individual differences in arousal regulation. Biol Psychiatry. 2004;55:284-90.
- Tan G, Dao TK, Farmer L, Sutherland RJ, Gevirtz R. Heart rate variability (HRV) and posttraumatic stress disorder (PTSD): a pilot study. Appl Psychophysiol Biofeedback. 2011;36:27-35.

- 62. Bryant RA, Guthrie RM. Maladaptive self-appraisals before trauma exposure predict posttraumatic stress disorder. J Consult Clin Psychol. 2007;75:812-5.
- 63. Morgan CA 3rd, Hazlett G, Doran A, Garrett S, Hoyt G, Thomas P, et al. Accuracy of eyewitness memory for persons encountered during exposure to highly intense stress. Int J Law Psychiatry. 2004;27:265-79.
- 64. Forsyth JP, Zvolensky MJ. Experimental psychopathology, clinical science, and practice: an irrelevant or indispensable alliance? Appl Prev Psychol. 2002;10:243-64.
- Zvolensky MJ, Lejuez CW, Stuart GL, Curtin J. Experimental psychopathology in psychological science. Rev Gen Psychol. 2001;5:371-81.
- Lakatos I. Falsification and the methodology of scientific research programmes. In: Harding SG, editor. Can theories be refuted? Berlin: Springer; 1975. p. 205-59.
- 67. Popper KR. The logic of scientific discovery. London: Hutchinson; 1959.
- Holmes EA, Brewin CR, Hennessy RG. Trauma films, information processing, and intrusive memory development. J Exp Psychol Gen. 2004;133:3-22.
- 69. Ehring T, Szeimies AK, Schaffrick C. An experimental analogue study into the role of abstract thinking in trauma-related rumination. Behav Res Ther. 2009;47:285-93.
- Bourne C, Frasquilho F, Roth AD, Holmes EA. Is it mere distraction? Peri-traumatic verbal tasks can increase analogue flashbacks but reduce voluntary memory performance. J Behav Ther Exp Psychiatry. 2010;41:316-24.
- Holmes EA, Bourne C. Inducing and modulating intrusive emotional memories: a review of the trauma film paradigm. Acta Psychol (Amst). 2008;127:553-66.
- 72. McNally RJ. Progress and controversy in the study of posttraumatic stress disorder. Annu Rev Psychol. 2003;54:229-52.
- Ehlers A, Clark DM, Hackmann A, Grey N, Liness S, Wild J, et al. Intensive cognitive therapy for PTSD: a feasibility study. Behav Cogn Psychother. 2010;38:383-98.
- Brewin CR. Autobiographical memory for trauma: update on four controversies. Memory. 2007;15:227-48.

Correspondence:

Christian Haag Kristensen Pontifícia Universidade Católica do Rio Grande do Sul Programa de Pós-Graduação em Psicologia Av. Ipiranga, 6681, prédio 11 90619-900 - Porto Alegre, RS - Brazil E-mail: christian.kristensen@pucrs.br