



Psychology & Neuroscience

ISSN: 1984-3054

landeira@puc-rio.br

Pontifícia Universidade Católica do Rio de
Janeiro
Brasil

Ponzi Holmer, Leslie; Rodrigues Wilhelm, Alice; Martins de Almeida, Rosa Maria
Cognitive function and posttraumatic stress disorder: A case study
Psychology & Neuroscience, vol. 7, núm. 4, 2014, pp. 577-582
Pontifícia Universidade Católica do Rio de Janeiro
Rio de Janeiro, Brasil

Available in: <http://www.redalyc.org/articulo.oa?id=207032913017>

- How to cite
- Complete issue
- More information about this article
- Journal's homepage in redalyc.org

redalyc.org

Scientific Information System
Network of Scientific Journals from Latin America, the Caribbean, Spain and Portugal
Non-profit academic project, developed under the open access initiative

Cognitive function and posttraumatic stress disorder: A case study

Leslie Ponzi Holmer¹, Alice Rodrigues Willhelm², and Rosa Maria Martins de Almeida²

1- Psychologist, Porto Alegre, RS, Brazil

2- Universidade Federal do Rio Grande do Sul, Porto Alegre, RS, Brazil

Abstract

Posttraumatic stress disorder (PTSD) in children can cause repetitive memories in which the theme or aspects of the traumatic event are expressed and can appear at any moment during the day and may occur while dreaming without recognizable content. We conducted a case study with a female child, 11 years 3 months of age. To analyze cognitive function, the Wechsler Intelligence Scale for Children (WISC-III) was used. The Child Stress Scale (Escala de Stress Infantil [ESI]) was used to assess psychological, physical, psychological, and psychophysiological symptoms with depressive components related to stress. The child was diagnosed with PTSD. After the first assessment, she underwent psychotherapy using Cognitive Behavior Therapy. The patient's symptomology related to stress symptoms improved, and she no longer presented PTSD.

Keywords: posttraumatic stress disorder, cognitive function, cognitive behavioral therapy.

Received 19 January 2014; received in revised form 23 June 2014; accepted 24 June 2014. Available online 16 December 2014.

Introduction

Posttraumatic stress disorder (PTSD) can be caused by real exposure or the threat of exposure to death, serious injury, and rape, among other stressful situations. Individuals with PTSD experience distress, with intrusive, spontaneous, recurrent, and involuntary memories of the traumatic event (American Psychiatric Association, 2013).

In children, repetitive memories in which the theme or aspects of the traumatic event are expressed can appear at any moment during the day and may occur while dreaming without recognizable content. The individual also persistently avoids stimuli associated with the traumatic event. Two or more of the following symptoms can be present in this disorder: irritability, aggressiveness, self-destructiveness, reckless behavior, hypervigilance, an exaggerated startle response, difficulty concentrating, and

sleep disturbances. For a diagnosis of PTSD, the duration of such disturbances must be greater than 1 month, and the disturbances must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (American Psychiatric Association, 2013).

Posttraumatic stress is considered a major environmental challenge that puts the physical and mental health of the individual at risk. The main factor related to the symptoms of PTSD is reliving the traumatic event, which has elements of reactivating psychophysiological and psychological distress (McFarlane, 2010).

The fight-or-flight response to a threatening stimulus is a reflexive nervous system phenomenon that provides a survival advantage in evolutionary terms. However, the systems that organize these reflexive survival behaviors can often become dysregulated. Chronic dysregulation of these systems can lead to disability in some individuals with PTSD (Brenner, 2011).

There has been longstanding interest in the effects of stress on health and the hypothalamic-pituitary-adrenal (HPA) axis because the autonomic nervous system has expenditure in the body in response to repeated stress cycles (McFarlane, 2010). Previous studies have reported structural and functional brain changes associated with PTSD (Borges & Dell'Aglio, 2008).

Environmental stress activates the limbic HPA system, which leads to an increase in cortisol secretion. Limbic and frontal regions that are rich in glucocorticoid receptors may be particularly sensitive to the effects of cortisol (Carrion, Weems, Richert, Hoffman, & Reiss, 2010).

Leslie Ponzi Holmer, Psychologist, Porto Alegre, RS. Alice Rodrigues Willhelm, Graduate Program in Psychology, Institute of Developmental Psychology and Personality, Universidade Federal do Rio Grande do Sul, Porto Alegre, RS, Brazil, and Laboratório de Psicologia Experimental, Neurociências e Comportamento, CNPq. Rosa Maria Martins de Almeida, Universidade Federal do Rio Grande do Sul, Porto Alegre and Instituto de Psicologia do Desenvolvimento e da Personalidade, Universidade Federal do Rio Grande do Sul, Porto Alegre, RS, Brazil, and Laboratório de Psicologia Experimental, Neurociências e Comportamento, Pesquisadora de Produtividade CNPq 1D. Correspondence regarding this article should be directed to: Rosa Maria M. de Almeida, Rua Ramiro Barcelos, 2600, Porto Alegre, Brazil. E-mail: rosa_almeida@yahoo.com or rosa.almeida@ufrgs.br

PTSD is associated with long-term neurobiological changes in which neurohormonal systems including glucocorticoids and norepinephrine act on brain areas to modulate the symptoms of PTSD and memory (Bremner, Elzinga, Shmahl, & Vermetten, 2008).

Traumatic stress has a wide range of effects on the brain, and the areas involved in these responses include the amygdala, hippocampus, and medial prefrontal cortex (Bremner, 2006a). Similarly, the brain regions that are related to PTSD include the prefrontal cortex, hippocampus, hypothalamus, and amygdala. These regions generate endocrine stress responses, including activation of the hypothalamic-pituitary-adrenal axis, with plastic changes at the neuronal intracellular level (Porte, 2007).

The prefrontal cortex, hippocampus, and amygdala are related to the HPA axis, which is highly sensitive to external changes and thus a major focus of PTSD research. Patients with PTSD exhibit an increase in glucocorticoid receptor responsiveness, suggesting that negative feedback inhibition plays an important role in the pathophysiology of this disorder (Ruiz, Neto, Schoedl, & Mello, 2007).

The amygdala is a limbic structure that is involved in emotional processing, and it is fundamental to the acquisition of fear responses (Sherin & Nemeroff, 2011). The amygdala has been shown to be involved in the acquisition and expression of conditioned fear and enhancement of emotional memory (Koenigs & Grafman, 2009). Greater reactivity of the amygdala may represent a biological risk factor for developing PTSD (Sherin & Nemeroff, 2011).

Previous studies have shown that stress is associated with changes in the structure of the hippocampus, an area of the brain that plays a critical role in learning and memory (Uno, Tarara, Else, Suleman, & Sapolsky, 1989; Sapolsky, Uno, Rebert, & Finch, 1990). People with PTSD present a wide range of memory problems (Bremner, 2006b). The hippocampus is involved in the control of stress responses, declarative memory, and contextual aspects of fear conditioning. The reduction of hippocampal volume has been studied in cases of PTSD (Sherin & Nemeroff, 2011).

Changes in hippocampal volume are associated with PTSD (Bremner *et al.*, 1995). Furthermore, several studies have shown consistent changes in cognition and brain structure associated with PTSD including the loss of declarative memory (Elzinga & Bremner, 2002; Bremner, 2006b). Thus, PTSD patients exhibit deficits in hippocampal activation while performing a verbal declarative memory task (Bremner *et al.*, 2003; Shin *et al.*, 2004; Bremner, 2006b).

Studies have shown that PTSD is associated with changes in brain areas and neurobiological systems that mediate responses to stress and cognition (Bremner, 2006a; Stein, Koverola, Hanna, Torchia, & McClarty, 1997). Learning difficulties and memory loss are frequently reported in patients with PTSD (Vasterling & Brailey, 2005; Sachinvala *et al.*, 2000). Moreover,

deficits related to attention and executive function have also been observed in individuals with this disorder (Fani *et al.*, 2011). Research has investigated differences in neuropsychological functioning in individuals with PTSD. Numerous studies have observed decreases in verbal memory performance and learning in subjects with PTSD (Elzinga & Bremner, 2002; Gil, Calev, Greenberg, Kugelmass, & Lerer, 1990; Golier & Yehuda, 2002; Horner & Hamner, 2002; Koenen, Moffitt, Poulton, Martin, & Caspi, 2001; Uddo, Vasterling, Brailey, & Sutker, 1993; Yehuda *et al.*, 1995).

Diagnosing PTSD is difficult because these patients are often reluctant to talk about their traumatic experience (Yehuda, 2002). The traumatic events are not necessarily rare or extreme, but individuals who are predisposed to the development of PTSD may be more sensitive than other individuals to certain biopsychosocial factors (Yehuda, 2002; Figueira & Mendlowicz, 2003). Therefore, the study of individual cases is important to analyze and determine the factors that cause PTSD in different people and the factors that are related to improvements in PTSD symptoms. Patients with PTSD have difficulties in concentration and work performance (Horner & Hamner, 2002; Koenen *et al.*, 2001). In real life, determining the source of stress can be a challenge. Another difficulty is recognizing which activities are more difficult to perform.

The aim of the present study was to analyze an individual case of a child with symptoms of anxiety who was diagnosed with PTSD. This study may be beneficial to other psychotherapists who seek to recognize trends in the symptomatology of PTSD.

Material and methods

The present case study involved a female child, 11 years 3 months of age and from a high socioeconomic status. She was in 5th grade in a private school in Porto Alegre, Brazil. The patient lived with her parents and a younger brother. The school referred the girl for the assessment of cognitive function because her academic performance fell. Moreover, she presented irritable behavior, exaggerated vigilance, difficulty concentrating and focusing attention, sleep difficulties, a persistent negative emotional state, body aches, and chronic fatigue. The traumatic event experienced by the child was ascertained to be witnessing her mother suffer from cancer and undergo surgery and chemotherapy.

To analyze cognitive function, we used the Wechsler Intelligence Scale for Children (WISC-III). The Child Stress Scale (Escala de Stress Infantil [ESI]) was used to assess psychological, physical, psychological, and psychophysiological symptoms with depressive components related to stress. The first assessment was conducted in January 2012. The child was diagnosed with PTSD based on symptomatology reflected by the scale and interviews with the patient and her family. The interview with the family aided in diagnosis because they provided more details about

the child's symptomology. After this assessment, the girl underwent psychotherapy using cognitive behavior therapy. The reevaluation after treatment occurred in January 2013 when the girl presented total remission of stress symptoms. The evaluation was conducted in three sessions (1 h each).

The WISC-III (Wechsler, 1991) is a standardized measure of intellectual functioning for children between 6 and 16 years of age. The WISC-III consists of 12 subtests that assess verbal and execution areas. It also assesses verbal comprehension, perceptual organization, resistance to distraction, and processing speed. Simões (2002) studied that the WISC-III was present in neuropsychological assessment instruments and was an integral part of the best neuropsychological assessments of children and adolescents. This author also suggested that all neuropsychological examinations should include assessments of verbal and nonverbal intellectual function.

The ESI (Lipp & Lucarelli, 2005) is composed of 35 questions that investigate psychological and psychophysiological symptoms related to stress reactions in children. Between the first and second assessment, the treatment and intervention for PTSD was conducted using Cognitive Behavioral Therapy (CBT) twice per week for a period of 12 months. Cognitive Behavioral Therapy has been considered a resource of choice for the treatment of PTSD (Gonçalves et al., 2011).

The interventions that were used for psychotherapy included relaxation techniques, psychoeducation about PTSD for the child and family, gradual desensitization of the memories of the traumatic event, and cognitive restructuring. The participation of the family and school was critical for the success of the interventions.

Anxiety-related behavior in parents has been recognized as a crucial factor in the development and maintenance of anxiety disorders in childhood (Cobham, Dadds, & Spence, 1998; Kenardy, Cobham, Nixon, McDermott, & March, 2010).

Results and discussion

PTSD may be associated with deficits in specific indices of verbal intelligence (Saigh, Yasik, Oberfield, Halamandaris, & Bremner, 2006). The results of the WISC-III (Figure 1 and 2) showed that the child's score of 5 on the Comprehension subtest was very low in 2012 when she presented stress symptoms and was diagnosed with PTSD. However, according to Saigh et al. (2006), young people with PTSD also have low scores on the Similarities and Vocabulary subtests. After treatment, the child's Comprehension subtest score increased to 11, which was clinically significant and reflected by important improvements in performance.

In the same study (Saigh et al., 2006), the authors found no significant differences on the Execution IQ or Perceptual Organization Index subtests, suggesting no relationship between PTSD and these subtests. However, different results were found in the present case study. The child had low scores on the Codification, Arrangement of Figures, and Cubes subtests in 2012 (Figure 1) when she presented stress symptoms and was diagnosed with PTSD. These discrepant results may be attributable to differences in the sociocultural contexts, age groups, and methodological designs between the previous study and present case study.

In 2013, after the child achieved complete remission of PTSD symptoms, her scores showed clinically significant improvement mainly on the Vocabulary,

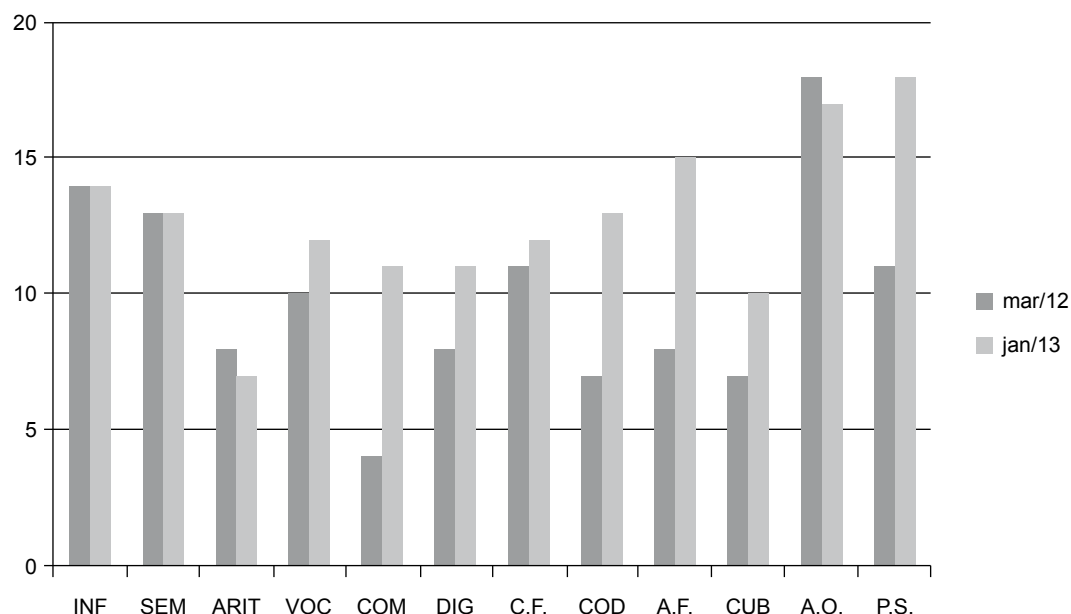


Figure 1. Scores on the WISC-III pre- and posttreatment. WISC-III Subtests: Inf, Information; SEM, Semantic; ARIT, Arithmetic; VOC, Vocabulary; COM, Comprehension; DIG, Digits; C.F., Complete with Figures; COD, Codification; A.F., Arrangement of Figures; CUB, Cubes; A.O., Assembly of Objects; P.S., Search Symbols.

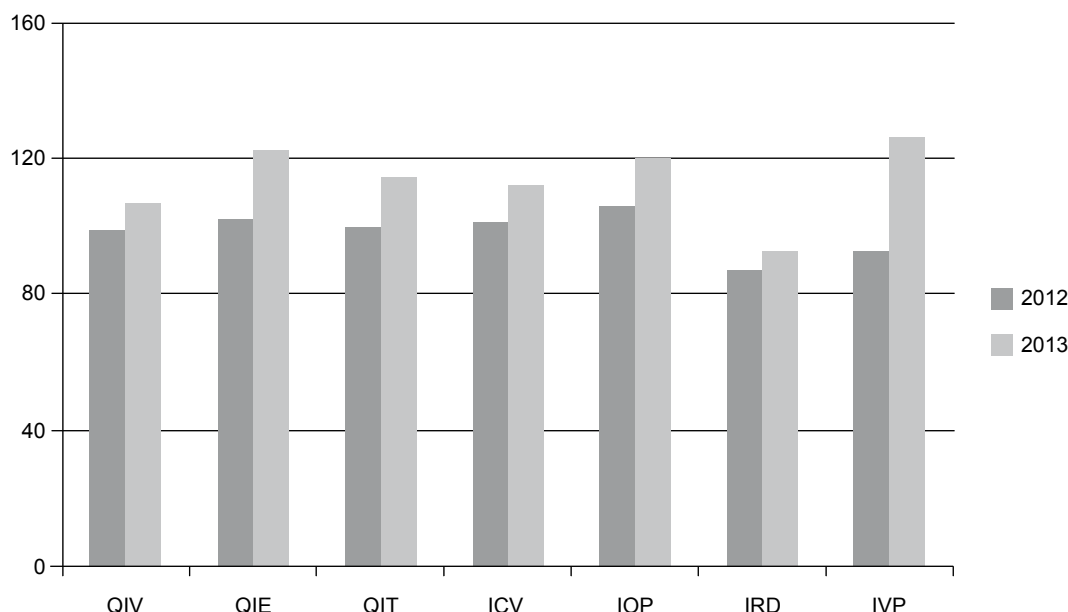


Figure 2. Scores on the WISC-III pre- and posttreatment. Factorial IQ indices: QIV, Verbal IQ; QIE, Execution IQ; QIT, total IQ; ICV, Verbal Comparison Index; IOP, Perceptual Organization Index; IRD, Resistance Distractibility Index; IVP, Processing Speed Index.

Comprehension, Digits, Codification, Arrangement of Figures, Cubes, and Search Symbols subtests (Figure 1). Neuropsychological testing has revealed that cognitive impairment occurs in PTSD. The main functions that are impaired include memory, attention, and executive function (Horner & Hamner, 2002).

A previous study used the ESI to evaluate children, with the objective of verifying the different manifestations of PTSD. The researchers concluded that the children who had a higher incidence of PTSD initially presented with psychological reactions, followed by depressive, psychophysiological, and eventually physical symptoms (Oliveira & Santos, 2006). These psychological reactions correspond to scores on the psychological symptoms of ESI, including feelings of exhaustion or fatigue, frustration, tension, anxiety, anger, depression, panic, chronic irritability, and restlessness, among others (Table 1). Excessive psychological stress has been associated with neuronal changes in the hippocampus, a brain area involved in learning and memory (Bremner, 2006b).

The child's scores on the ESI were clinically significant, reflected by changes in sleep patterns and appetite. When the child was exposed to a stressful event, she entered into a state of alertness, which

triggers activation of the sympathetic nervous system and the pituitary gland via the hypothalamus (Sherin & Nemoroff, 2011). Similarly, activation of the adrenal glands releases epinephrine, which inhibits vegetative activities and may cause changes in eating and sleeping habits (Lipp & Lucarelli, 2005).

The clinically significant differences between the assessments of the child conducted in 2012 and 2013 on both the WISC-III and ESI likely indicate total remission of PTSD symptoms as a result of CBT. Behavioral and cognitive changes that occur after CBT appear to lead to changes in the activation of brain circuits that involve the prefrontal cortex. Such changes also suggest the possibility of modifying neural associations (Mocaiber *et al.*, 2008). Some studies have shown that modifying behavior and beliefs is an effective psychological treatment approach for PTSD (Passarela & Mendes, 2010).

In the present case study, the results showed significant improvement in cognitive function after full remission of PTSD symptoms. The ESI results indicated that the child initially presented significant stress symptoms that were not manifest in the revaluation in 2013. PTSD is a chronic and debilitating disorder that is associated with significant impairments in both social and academic functioning (Kenardy *et al.*, 2010).

Table 1. Child Stress Scale (Escala de Stress Infantil [ESI]).

	Total of Items Scale	Score in 2012	Score in 2013
Total score	140	91	8
Physical reactions	36	15	2
Psychophysiological reactions	32	20	1
Psychological reactions with depressive component	36	26	2
Psychological reactions	36	30	3

Between the first and second evaluations, the child attended psychotherapy sessions that used a cognitive-behavioral approach, to which she responded positively. School performance and family and social relationships significantly improved. The child exhibited complete remission of irritable behavior, exaggerated startle responses, difficulty concentrating, sleep difficulties, a persistent negative emotional state, body aches, and chronic fatigue.

Final comments

After therapy, the child's stress symptoms improved, and she no longer had a diagnosis of PTSD. The questionnaires and WISC-III indicated improvement in other non-verbal and verbal abilities. PTSD is associated with multiple problems in different contexts of life, and CBT can assist in alleviating its symptomatology. The present case presented a detailed analysis of a child with dynamic and pathological symptoms, which may benefit other researchers and clinicians who study similar cases and aid professionals' understanding of PTSD symptoms and treatments in other patients.

References

- American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition. Washington DC: American Psychiatric Press.
- Borges, J. L., & Dell'Aglio, D. D. (2008). Relações entre abuso sexual na infância, transtorno de estresse pós-traumático (TEPT) e prejuízos cognitivos. *Psicologia em Estudo*, 13(2), 371-379.
- Bremner, J. D. (2006a). The relationship between cognitive and brain changes in posttraumatic stress disorder. *Annals of the New York Academy of Sciences*, 1071, 80-86.
- Bremner, J. D. (2006b). Stress and brain atrophy. *CNS and Neurological Disorders Drug Targets*, 5, 503-512.
- Bremner, J. D., Elzinga, B., Schmahl, C., & Vermetten, E. (2008). Structural and functional plasticity of the human brain in posttraumatic stress disorder. *Progress in Brain Research*, 167, 171-186.
- Bremner, J. D., Randall, P. R., Scott, T. M., Bronen, R. A., Seibyl, J. P., Southwick, S. M., ... Innis, R. B. (1995). MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *American Journal of Psychiatry*, 152(7), 973-981.
- Bremner, J. D., Vythilingam, M., Vermetten, E., Southwick, S. M., McGlashan, T., Nazeer, A., ... Charney, D. S. (2003). MRI and PET study of deficits in hippocampal structure and function in women with childhood sexual abuse and posttraumatic stress disorder. *American Journal of Psychiatry*, 160(5), 924-932.
- Brenner, L. A. (2011). Neuropsychological and neuroimaging findings in traumatic brain injury and post-traumatic stress disorder. *Dialogues in Clinical Neuroscience*, 13(3), 311-323.
- Carrion, V. G., Weems, C. F., Richert, K., Hoffman, B. C., & Reiss, A. L. (2010). Decreased prefrontal cortical volume associated with increased bedtime cortisol in traumatized youth. *Biological Psychiatry*, 68(5), 491-493.
- Cobham, V. E., Dadds, M. R., & Spence, S. H. (1998). The role of parental anxiety in the treatment of childhood anxiety. *Journal of Consulting and Clinical Psychology*, 66, 893-905.
- Elzinga, B. M., & Bremner, J. D. (2002). Are the neural substrates of memory the final common pathway in PTSD? *Journal of Affective Disorders*, 70(1), 1-17.
- Fani, N., Kitayama, N., Ashraf, A., Reed, L., Afzal, N., Jawed, F., & Bremner, J. D. (2009). Neuropsychological functioning in patients with posttraumatic stress disorder following short-term paroxetine treatment. *Psychopharmacology Bulletin*, 42(1), 53-68.
- Figueira, I., & Mendlowicz, M. (2003). Diagnóstico do transtorno de estresse pós-traumático. *Revista Brasileira de Psiquiatria*, 25(Suppl. 1), 12-16.
- Gil, T., Calev, A., Greenberg, D., Kugelmass, S., Lerer, B. (1990). Cognitive functioning in post-traumatic stress disorder. *Journal of Traumatic Stress*, 3, 29-45.
- Golier, J., & Yehuda, R. (2002). Neuropsychological processes in post-traumatic stress disorder. *Psychiatric Clinics of North America*, 25(2), 295-315.
- Gonçalves, R., Lages, A. C., Rodrigues, H., Pedrozo, A. L., Coutinho, E. S. F., Neylan, T., Figueira, I., & Ventura, P. (2011). Potenciais biomarcadores da terapia cognitivo-comportamental para o transtorno de estresse pós-traumático: uma revisão sistemática. *Revista Brasileira de Psiquiatria*, 38(4), 155-160.
- Horner, M. D., & Hamner, M. B. (2002). Neurocognitive functioning in posttraumatic stress disorder. *Neuropsychology Review*, 12, 15-30.
- Institute of Medicine (US) Committee on Health and Behavior: Research, Practice, and Policy. (2001). *Health and Behavior: The Interplay of Biological, Behavioral, and Societal Influences*. (pp. 39-86). Washington (DC): National Academies Press (US). Available from: <https://www.ncbi.nlm.nih.gov/books/NBK43743/>
- Kenardy, J., Cobham, V., Nixon, R. D. V., McDermott, B., & March, S. (2010). Protocol for a randomised controlled trial of risk screening and early intervention comparing child- and family-focused cognitive-behavioural therapy for PTSD in children following accidental injury. *BMC Psychiatry*, 11, 15.
- Koenen, K. C., Moffitt, T. E., Poulton, R., Martin, J., & Caspi, A. (2001). Early childhood factors associated with the development of post-traumatic stress disorder: results from a longitudinal birth cohort. *Psychological Medicine*, 37(2), 181-192.
- Koenigs, M., & Grafman, J. (2009). Posttraumatic stress disorder: the role of medial prefrontal cortex and amygdala. *Neuroscientist*, 15(5), 540-548.
- Lipp, M. E. N., & Lucarelli, M. D. M. (2005). *Escala de Stress Infantil: ESI: Manual*. São Paulo: Casa do Psicólogo.
- McFarlane, A. C. (2010). The long-term costs of traumatic stress: intertwined physical and psychological consequences. *World Psychiatry*, 9(1), 3-10.
- Mocaiber, I., Oliveira, L., Pereira, M. G., Machado-Pinheiro, W., Ventura, P. R., Figueira, I. V., & Volchan, E. (2008). Neurobiologia da regulação emocional: implicações para a terapia cognitivo-comportamental. *Psicologia em Estudo*, 13(3), 531-553.
- Oliveira, L. H., & Santos, C. S. S. (2006). As diferentes manifestações do transtorno de estresse pós traumático (TEPT) em crianças vítimas de abuso sexual. *Revista da SBPH*, 9(1), 31-53.
- Passarela, C. M., & Mendes, D. D. (2010). A systematic review to study efficacy of cognitive behavior therapy for sexually abused children and adolescents with posttraumatic stress disorder. *Revista de Psiquiatria Clínica*, 37(2), 60-65.
- Porte, P. R. (2007). *Estudo de revisão sistemática de terapia cognitivo-comportamental e neuroimagem nos transtornos de ansiedade*. Masters dissertation. Rio de Janeiro: UFRJ.
- Ruiz, J. E., Neto, J. B., Schoedl, A. F., & Mello, M. F. (2007). Psiconeuroendocrinologia do transtorno de estresse pós-traumático. *Revista Brasileira de Psiquiatria*, 29(1), 7-12.
- Sachinvala, N., Von Scotti, H., McGuire, M., Fairbanks, L., Bakst, K., McGuire, M., ... Brown, N. (2000). Memory, attention, function, and mood among patients with chronic posttraumatic stress disorder. *Journal of Nervous and Mental Disease*, 188(12), 818-823.
- Saigh, P. A., Yasik, A. E., Oberfield, R. A., Halamandaris, P. V., & Bremner, J. D. (2006). The intellectual performance of traumatized children and adolescents with or without posttraumatic stress disorder. *Journal of Abnormal Psychology*, 115(2), 332-340.
- Sapolsky, R. M., Uno, H., Rebert, C. S., & Finch, C. E. (1990). Hippocampal damage associated with prolonged glucocorticoid exposure in primates. *Journal of Neuroscience*, 10(9), 2897-2902.
- Sherin, J. E., & Nemeroff, C. B. (2011). Post-traumatic stress disorder: the neurobiological impact of psychological trauma. *Dialogues in Clinical Neuroscience*, 13(3), 263-278.
- Shin, L. M., Shin, P. S., Heckers, S., Krangel, T. S., Macklin, M. L., Orr, S. P., ... Rauch, S. L. (2004). Hippocampal function in posttraumatic stress disorder. *Hippocampus*, 14(3), 292-300.
- Simões, M. R. (2002). Utilizações da Wisc-III na avaliação neuropsicológica de crianças e adolescentes. *Paidéia*, 12(23), 113-132.
- Stein, M. B., Koverola, C., Hanna, C., Torchia, M. G., & McClarty, B. (1997). Hippocampal volume in women victimized by childhood sexual abuse. *Psychological Medicine*, 27(4), 951-959.
- Uddo, M., Vasterling, J., Brailey, K., Sutker, P. (1993). Memory and attention in combat-related post-traumatic stress disorder. *Journal of Psychopathology and Behavioural Assessment*, 15, 43-52.

- Uno, H., Tarara, R., Else, J. G., Suleman, M. A., & Sapolsky, R. M. (1989). Hippocampal damage associated with prolonged and fatal stress in primates. *Journal of Neuroscience*, 9(5), 1705-1711.
- Vasterling, J. J., & Brailey, K. (2005). Neuropsychological findings in adults with PTSD. In: J. J. Vasterling, & C. Brewin (Eds.), *Neuropsychology of PTSD: biological, cognitive, and clinical perspectives* (pp. 178-207). New York: Guilford Press.
- Wechsler, D. (1991). *The Wechsler intelligence scale for children—third edition*. San Antonio, TX: The Psychological Corporation.
- Yehuda, R., Keefe, R., Harvey, P., Levengood, R., Gerber, D., Geni, J., & Siever, L. (1995). Learning and memory in combat veterans with posttraumatic stress disorder. *American Journal of Psychiatry*, 152, 137-139.
- Yehuda, R. (2002). Post-traumatic stress disorder. *New England Journal of Medicine*, 346, 108-114.