Cognitive Dysfunctions in Posttraumatic Stress Disorder

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SUMMARY

Posttraumatic stress disorder (PTSD) is a condition that occurs after a traumatic event, and its diagnostic criteria include attention and memory deficits as well as symptoms of anxiety. We aimed to review the literature related to attention, memory, and executive functions in PTSD. Although studies on the subject are limited (in that there is no uniformity in terms of trauma type, selection of the control groups or types of neuropsychological tests used), most reported similar deficits in PTSD subjects in terms of memory and executive functions including attention. Since the presence of psychiatric comorbidities may disrupt neuropsychological functions, results of studies that have not controlled comorbidity may be questionable. However, studies that excluded the comorbid conditions reported similar deficits in cognitive functions in PTSD patients. The relationship between recovery from PTSD symptoms and change in cognitive functions has been examined in only a few studies, and most have reported an improvement for both memory and executive functions in remitted patients. The improvement in executive functions, however, has been limited by the difficulty of task. Cognitive deficits have been among the major causes of disabilities in PTSD patients. Therefore, the amount of improvement in cognitive dysfunctions by current treatments of PTSD deserves more attention.

Key words: PTSD, attention, memory, executive functions, cognitive dysfunction

INTRODUCTION

Posttraumatic stress disorder (PTSD) is a chronic condition characterized by reexperiencing trauma; avoidance of trauma-related stimuli, thoughts or feelings; negative alterations in cognition and mood, and hyperarousal symptoms. The hyperarousal symptoms include sleep difficulties, irritability, exaggerated startle response, and problems in concentration in the aftermath of traumatic life events (assault, rape, accidents, and disasters). The disorder appears shortly after a traumatic event, but the onset may be delayed for months or even years. In DSM5, PTSD has been re-classified under the "trauma- and stressor-related disorders" category, due to its different features from anxiety disorders. The diagnostic criteria for PTSD include attention and memory deficits (such as inability to recall key features of the trauma), difficulty concentrating, and hypervigilance. The cognitive deficits in PTSD are not, however, limited to attention and memory. Since most studies that investigate cognitive functions in PTSD are cross-sectional, it has been difficult to state whether the cognitive functions are a cause or result of PTSD. The aim of this review was to summarize the study findings relating to cognitive dysfunctions and their possible causes in PTSD. All research and review articles were searched in Pubmed by the following keywords:"PTSD/ cognitive functions/ memory/ executive functions/ treatment". We also included the relevant research or review papers in the reference lists of articles that we reached through the initial Pubmed search. The search was limited to articles published in English and to those that had our keywords in the title/abstract.

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ATTENTION and MEMORY

Studies investigating the change in cognitive functions in PTSD, especially the early studies, focused on attention and memory functions. Findings on attention and memory functions were the most replicated findings reported in PTSD. The reason that researchers have focused on these cognitive functions may be related to their inclusion in the diagnostic criteria for PTSD. Most of these studies have reported deficits in attention and verbal memory for PTSD (Beers ve De Bellis 2002, Bremner et al. 1995a, Bremner et al. 1995b, Bremner et al. 1997, Bremner et al. 1993, Gilbertson et al. 2001, Golier et al. 2002, Gurvits et al. 1993, Jenkins et al. 1998, Semple et al. 1996, Uddo et al. 1993, Vasterling et al. 1998, Yehuda et al. 1995). However, there have been studies that did not report a specific deficit in attention and memory in PTSD (Barrett et al. 1996, Dalton et al. 1989, Gil et al. 1990, Gurvits et al. 1996, Stein et al. 1997, Zalewski et al. 1994). Studies that used trauma specific stimuli to evaluate cognitive functions found better attention and memory functions than controls (Golier et al. 2003, Paunovi et al. 2002, Vrana 1995, Wessa et al. 2006). The findings of these studies have been summarized below and categorized by the study design.

Two Group Comparisons

Under this topic, we covered studies in which the control groups consisted of either trauma survivors that did not develop PTSD, or healthy individuals that did not experience the trauma. The impossibility to distinguish whether the reported differences were due to the presence of trauma or PTSD has been an important limitation to these studies.

One of the earliest studies that investigated attention and memory functions in PTSD was conducted by Dalton et al. (1989). In this study, attention and memory functions in 100 Vietnam veterans with PTSD were not found to be different than the population norm. Multiple reports have been designed to evaluate memory; however, that reported deficits in attention and verbal memory in PTSD subjects (Bremner et al. 1993, Bremner et al. 1995b, Golier et al. 2002, Jenkins et al. 1998, Uddo et al. 1993, Vasterling et al. 1998, Yehuda et al. 1995). Both childhood maltreatment and combat related PTSD was shown to be associated with worsening in short- and long-term delayed verbal memory performance when compared to healthy controls (Bremner et al. 1993, Bremner et al. 1995b). In addition, trauma intensity was found to be positively correlated with short-term memory deficits (Bremner et al. 1995b). Authors concluded that their findings point to a wide-based deficit in the different steps of information processing including gathering, storage, and the recall of information. Another study, which applied a different neuropsychological battery to 16 Vietnam veterans with PTSD, reported a deficit only in the short-term verbal and visual memory but not in the long-term memory when compared to 15 healthy controls (Uddo et al. 1993). This finding was replicated in another study where the control group consisted of Veterans without PTSD (Gurvits et al. 1993). However, there have been studies that showed a decrease in delayed free recall performance (Bremner et al. 1995b, Bremner et al. 1993, Yehuda et al. 1995). The deficits in attention and memory were also shown in younger veterans with PTSD with fewer medical, neurological, and psychiatric comorbidities (Vasterling et al. 1998).

Gilbertson et al (2001) reported that the neuropsychological functions that best predict PTSD symptom severity were attention and memory. The difference observed in attention and memory remained significant when trauma severity, alcohol use history, current depression, IQ score, and learning problems were controlled. PTSD patients (n=19) however, were reported to have higher rates of learning problems (like failing class) than controls (n=13) in this study. Authors' report concluded that individuals whose explicit memory was better before the trauma may be more resilient to developing PTSD (Gilbertson et al. 2001).

There are findings in the literature that suggest PTSD patients process threat or trauma-related stimuli differently than neutral stimuli (McNally 1997, Minshew ve D'Andrea 2015). For example, both explicit and implicit trauma related words were recalled more efficiently than neutral words in women victims of partner violence (Minshew ve D'Andrea 2015). Studies comparing memory functions of PTSD patients with that of controls also reported that PTSD patients recalled neutral stimuli less than controls. However, they recalled trauma or threat-related stimuli better than controls (Paunovi et al. 2002, Vrana 1995). Therefore, changes in memory functions reported in PTSD patients were found to be related with the content of the stimuli.

Three Group Comparisons

Under this topic, we reviewed studies where the control groups included trauma survivors that did not develop PTSD as well as healthy individuals who were not exposed to the trauma. This design enables the clarification of whether the observed deficits were related to trauma or PTSD. Deficits found in PTSD patients (but not in either of the control groups) may be attributed to PTSD diagnosis. On the other hand, deficits found in trauma survivors (regardless of their PTSD diagnosis), but not in trauma-unexposed groups, may be better explained by the effects of trauma and not PTSD.

Jenkins et al (1998) compared 15 rape victims that developed PTSD with 16 rape victims that did not develop PTSD, and 16 controls who were not rape victims using California Verbal Learning Test (Jenkins et al. 1998). The 'number of words learned' and a '3-minute delayed free recall' were found to

be similar across groups, whereas a '20-minute delayed free recall' in PTSD patients was found to be worse than both control groups. Golier et al (2002) compared implicit and explicit memory performances of 31 Holocaust survivors with PTSD to that of 16 Holocaust survivors without PTSD and to 35 trauma-unexposed individuals. Explicit memory performance of the PTSD group, who were reported to have lower IQ scores and lower education, was found to be worse than the other groups; whereas, implicit memory performance was similar across groups. The findings of both studies suggest that there may be a problem in medial temporal lobe structures, for both showed presence of disturbance in delayed free recall and explicit memory, even though immediate free recall and implicit memory were preserved.

Stein et al. (2002) compared attention and memory performances of 39 women victims of partner violence (17 of whom have current PTSD), 22 women partner violence victims without PTSD, and 22 women controls who did not experience similar trauma. Verbal learning and verbal memory were assessed with California Verbal Learning Test and Wechsler Memory Scale, whereas visual construction and memory were assessed with Continous Visual Memory Test and Rey Osterrieth Complex Figure Test. Auditory attention and working memory were evaluated with Wechler Adult Intelligence Scale (WAIS) subtests, while speeded sustained attention was evaluated with digit vigilance test and paced auditory serial addition test. While verbal learning and memory were found to be similar in all groups, visual construction, visual memory, and attention were reported to be better in non-trauma exposed groups than that of trauma survivors (Stein et al. 2002). This finding was in contrast with the previous studies, which reported deficits in verbal learning and memory in PTSD patients. Authors offered three possible explanations for this inconsistency: 1) different types of traumas may lead to different neuropsychological abnormalities; 2) duration, intensity and severity of trauma differs among studies; and 3) personal characteristics like IQ may affect trauma response.

Studies that used trauma-specific stimuli reported worse general explicit memory but similar or better trauma-related memory functions in PTSD patients than trauma exposed and trauma non-exposed control subjects (Golier et al. 2003, Wessa et al. 2006).

Effects of Psychiatric Comorbidities

Other psychiatric disorders like major depression and substance use disorders are frequently comorbid with PTSD (Keane ve Wolfe 1990). Therefore, it is hard to tell whether the observed deficits in cognitive functions can be attributed to PTSD, to other psychiatric disorders, or associated with the comorbidity between them. The deficits reported in studies using a population norm as control may be attributed to either PTSD or having other disorders. Whether the cognitive deficits found in PTSD are specific to PTSD or can be observed in all psychiatric disorders should be clarified. In this section, we will review studies that address this question.

Gil and colleagues (1990) compared cognitive functions of patients with PTSD, major depression, generalized anxiety disorder, obsessive compulsive disorder, or phobia patients to those of a healthy control group (12 people from each group). They reported that attention as well as visual and verbal memories of all patient groups were worse than those of the healthy control group. The authors concluded that the cognitive deficits found in PTSD were not specific to PTSD. Moreover, they were similar to those seen in other psychiatric disorders, and may have developed secondary to psychiatric symptoms (Gil et al. 1990). In a similar study, Zalewski et al (1994) compared attention and memory performance of 241 PTSD, 241 generalized anxiety disorder patients, and healthy controls using a National Vietnam experience database of the Center for Disease Control. In contrast to Gil et al's (1990) findings, they did not find any difference between groups in terms of verbal or visual attention or memory (Zalewski et al. 1994). The discrepancy between these two studies may be attributed to larger sample size of the latter study, which better represents the population. Another study that used the same database (Barrett et al. 1996) investigated attention and memory functions in 2,441 Vietnam Veterans. They reported that neither PTSD (n = 236) nor other psychiatric disorders (depression, anxiety disorders, or substance use disorder, n = 242) was related with deficits in attention or memory. The verbal memory performance of PTSD patients with comorbid current depression, anxiety disorder, or substance use disorder (n = 128), however, was found to be impaired. The authors concluded that the memory deficit in PTSD patients may be related to comorbidity, and that this is of great importance for the treatment of these patients. Similarly, another study comparing 8 PTSD patients, comorbid war-related substance use disorder, and 8 healthy controls showed that false alarms were higher in the comorbid group than controls in continuous performance test. It was not clear, however, whether this result was attributable to PTSD, substance use disorder, or comorbidity of two disorders, which reflects a limitation of the study design (Semple et al. 1996).

Does recovery from PTSD result in improvement in memory functions?

Identification of a memory deficit, which is present during the course of PTSD, and recovers after treatment of PTSD, suggests that such a deficit is both specific to PTSD and is not a trait marker (i.e. is temporary in nature). Few studies investigated whether cognitive dysfunction in PTSD patients improve after recovery from PTSD. Two of these studies investigated the effect of paroxetine, a selective serotonin

reuptake inhibitor, on cognitive functions. Vermetten et al (2003) studied memory functions before and 9-12 months after paroxetine treatment and reported improvement for both the verbal and visual memory performances in PTSD patients. The lack of a control group, however, was a major limitation of this study. The same group studied the effects of paroxetine on memory performance of PTSD patients in a double-blind placebo-controlled study design (Fani et al. 2009). Although there was a 24% improvement in the verbal memory performance relative to the first evaluation, they found no difference between the paroxetine and placebo groups in terms of improvement in memory. Furthermore, there were no differences between the paroxetine and placebo groups in terms of recovery from PTSD. Since the responders and non-responders to paroxetine were not compared in a separate analysis, it is impossible to state whether the improvement in memory performance was related to recovery from PTSD symptoms. The small sample size in each group (n=18) was a major limitation of this study.

Another study, designed to answer whether cognitive dysfunction in PTSD patients improve after recovery from PTSD evaluated 53 earthquake survivors with a structured clinical interview (Eren-Koçak et al. 2009). Memory and prefrontal functions were examined in current PTSD patients (n=16), recovered PTSD patients (n=15), and earthquake survivors who never developed PTSD (n=22). Both immediate and delayed verbal free recall (defined as the number of words recalled immediately and 20-minutes after the second list of Rey Auditory Verbal Learning Test), were found to be impaired only in current PTSD patients but not in past PTSD patients nor in controls. Immediate and delayed verbal free recall of recovered PTSD patients were reported to be similar to that of control earthquake survivors. The immediate and delayed visual free recall, on the other hand, were reported to be similar in all three groups. The superiority of the design of this study to previous ones (Vermetten et al. 2003, Fani et al. 2009) was its ability to reveal the relationship of cognitive findings with current or past PTSD diagnosis. This study has shown that immediate and delayed verbal memory deficits that were present in current PTSD, improved after recovery from PTSD.

Summary of Memory Findings

In summary, most of the neuropsychological studies on trauma exposed individuals that developed PTSD reported memory and attention deficits when compared to those who did not develop PTSD after the trauma or who were not exposed to trauma (Beers ve De Bellis 2002, Bremner et al. 1995a, Bremner et al. 1995b, Bremner et al. 1997, Bremner et al. 1993, Gilbertson et al. 2001, Golier et al. 2002, Gurvits et al. 1993, Jenkins et al. 1998, Semple et al. 1996, Uddo et al. 1993, Vasterling et al. 1998, Yehuda et al. 1995, Eren-Koçak et al. 2009). On the other hand, the attention and memory performances related with trauma specific stimuli of these patients were found to be similar or even better than those of control groups (Golier et al. 2003, Paunovi et al. 2002, Vrana 1995, Wessa et al. 2006). There are studies arguing that attention and memory performances of PTSD patients were similar to those of patients with other psychiatric disorders (Gil et al. 1990) or those of controls (Barrett et al. 1996, Dalton et al. 1989, Gurvits et al. 1993, Stein et al. 1997, Zalewski et al. 1994). None of these studies investigated whether the cognitive deficits last after recovery from PTSD. Few studies that examined this issue have suggested that verbal memory deficits improve with recovery from PTSD (Eren-Koçak et al. 2009, Fani et al. 2009, Vermetten et al. 2003).

EXECUTIVE FUNCTIONS

Executive functions other than attention have not been as well studied as attention and memory functions in PTSD. The consistency among these study findings in this area has been low. Since most studies (especially the early ones that were focused on attention and memory) were not designed to evaluate executive functions, some inferences can be drawn from some tests administered as part of a neuropsychological battery. Studies investigating executive functions in PTSD patients increased in recent years with the increasing awareness of the role of frontal-subcortical structures in PTSD.

Two-Group Comparisons

Findings of studies that compared executive functions of PTSD patients with that of trauma-exposed or trauma non-exposed control groups, will be summarized under this topic. When evaluating the findings of these studies, it should be kept in mind that different tests were used to evaluate executive functions.

One of the first studies that provided information regarding executive functions of PTSD patients was carried out by Dalton et al. (1989). Executive functions of 100 PTSD patients evaluated with Stroop and Trail Making Tests in the neuropsychological battery were not different than the population norms. In contrast, Gurvits et al. (1993) reported lower scores in Trail Making Test Part B in PTSD veterans (n=27) than non-PTSD veterans (n=15). Gilbertson et al. (2001) also reported lower performance in Trail Making Part B in Vietnam veterans with PTSD (n=19) than veterans without PTSD (n=13), which is consistent with Gurvits et al.'s findings. Emotional Stroop test, which was developed by modification of the Stroop Test to include words with positive and negative emotional content, was used in studies to more specifically assess trauma-related cognitive processing. These studies found that PTSD patients were more prone to interference when processing trauma-related words compared to

controls (McNally et al. 1990, Paunovi et al. 2002). Verbal fluency, another test sensitive to prefrontal functions, was also lowered for animal names in PTSD patients than healthy controls (Uddo et al. 1993).

Perseverations (repetition of the same word during free recall), intrusions (recall of words that were not in the list), proactive interference (intrusion of the words from List A during recall of List B), and retroactive interference (intrusion of the words from List B during recall of List A) in memory tests were all reported to be higher in PTSD patients than those of trauma exposed non-PTSD subjects and non-traumatized controls (Uddo et al. 1993, Vasterling et al. 1998, Yehuda et al. 1995, Eren-Koçak et al. 2009). These findings point to a disturbance in the regulation and monitorization of memory processes by prefrontal cortex.

In addition to standard neuropsychological tests, Koenen et al. (2001) used delayed response, delayed alternation, and object alternation tests to evaluate prefrontal functions in PTSD patients. In delayed response test, the examiner placed a nickel under one of the two wells. Subsequently, a curtain was lowered to block the sight of the wells by the participant for a certain amount of time. Then, the curtain was raised and the participant was asked to locate the nickel. In delayed alternation test, both wells were baited with the nickel in the first trial, and the well not chosen by the participant was then baited with nickel in the second trial. Its place was changed every time the participant located the nickel correctly. The number of trials to reach 12 consecutive correct responses was used as the dependent variable. In the object alternation test, the nickel was placed under a stimulus object and the participants were expected to learn that the nickel was located under the stimulus object that was not rewarded on the previous correct trial. They reported that PTSD patients were impaired on delayed response, which is sensitive to lesions of dorsolateral prefrontal network, and on object alternation test, which is sensitive to lesions in ventral (orbitofrontal) network that has intimate connections with limbic system (Koenen et al. 2001). Authors interpreted that their findings support studies showing prefrontal dysfunction in PTSD.

Three-Group Comparisons

Studies designed to overcome the limitations of the studies summarized above included control groups of both trauma-exposed non-PTSD subjects, and also non-traumatised subjects. Using such a study design, Jenkins et al. (1998) did not find any differences in verbal learning strategies between raped victims that developed PTSD (n=15), raped victims that did not develop PTSD (n=16), or women that were not raped (n=16). In contrast, Trail Making Test Part-B, Category Test, and Stroop Test scores were found to be worse in women victims of partner violence (17 current PTSD, 22 past PTSD) than non-traumatized control women (n=22) (Stein et al. 2002). This finding suggested that the deficit in executive functions was related with trauma exposure rather than PTSD diagnosis. Studies applying Emotional Stroop, on the other hand, reported an increase in latency to name the color of the trauma-related words in sexual trauma-related PTSD patients, when compared to the other two control groups (Foa et al. 1991, Martinson et al. 2013).

Twamley et al. (2004) compared executive functions of trauma-exposed PTSD subjects (n=38) with those of trauma-exposed controls (n=105 students) without PTSD, and healthy controls (n=87). There were no differences between groups in terms of premorbid intelligence and most other neuropsychological tests. The only difference found was that it took more trials to complete first category in Wisconsin Card Sorting Test in trauma-exposed non-PTSD subjects when compared to healthy controls. Their learning efficiency, however, was found to be better than the healthy controls in the subsequent trials (Twamley et al. 2004). Authors interpreted these findings by the "cognitive resilience" of young university students.

Effects of Psychiatric Comorbidities

As mentioned above, it is not possible to link the impairment to PTSD in studies where other psychiatric disorders are not assessed. Furthermore, psychiatric comorbidities may be directly responsible for the observed cognitive deficits, or may aggravate the effects of PTSD on cognitive functions. It is important to consider all such possibilities. This section focuses on findings of studies that investigate these possibilities.

Abstract thinking ability measured by a similarities subtest of WAIS was found to be worse, not only in PTSD group but also in major depressive disorder, generalized anxiety disorder, obsessive compulsive disorder (n=12/group), or phobia when compared to healthy controls (n=12) (Gil et al. 1990). Barrett et al. (1996) applied Wisconsin Card Sorting Test to 236 PTSD patients and compared their results with that of major depression, anxiety disorder, or substance use disorder patients. They reported that PTSD alone did not cause a deficit in executive functions. Rather, the comorbidity of PTSD with major depression, anxiety disorder, or substance use disorder resulted in dysfunction in visual organizational abilities, concept formation, problem solving, and other cognitive functions that require active use of feedback in decision-making (Barrett et al. 1996). This finding raises concern about the possibility that previous findings of impaired executive functioning in PTSD may not be solely attributed to PTSD. Many studies addressed this concern by either excluding comorbidity in the study design or controlling the comorbidity statistically. These studies reported that the cognitive disturbances in PTSD persisted after excluding psychiatric comorbidities or statistically controlling their effects (Eren-Koçak et al. 2009, Jenkins et al. 1998, Stein et al. 2002). For example, when the statistical analyses were repeated after exclusion of comorbid

major depression in earthquake survivors, similar results were found in the neuropsychological test performances of PTSD patients (Eren-Koçak et al. 2009). The differences in trauma type, the neuropsychological tests used and severity of PTSD or other psychiatric comorbidities may be responsible for the discrepancies in studies summarized above.

Does recovery from PTSD result in improvement in executive functions?

To our knowledge, there are only two studies that investigated the improvement in executive functions with recovery from PTSD. The first study compared executive functions of earthquake survivors that did not develop PTSD to those of earthquake survivors that recovered from PTSD, and survivors with current PTSD (Eren-Koçak et al. 2009). Executive functions were evaluated by Stroop Test, Short Category Test, Colored Trail Making Test and Verbal Fluency Test. Current PTSD patients were impaired on verbal fluency in both animal and human names, while recovered PTSD patients were impaired only on verbal fluency in animal names when compared to controls. Attention, another prefrontal lobe function, was evaluated by number of words recalled after the first trial of Lists A and B of Rey Auditory Verbal Learning Test. Current PTSD patients performed worse than controls in both Lists A and B, while recovered PTSD patients were impaired only on List B. Authors concluded that these findings were related to the susceptibility of recovered PTSD patients to proactive interference (i.e. interference of the words from List A into List B), which is an indicator of prefrontal dysfunction. Susceptibility to interference however, was not observed in Stroop and Trail Making Tests. Authors explained this inconsistency by the fact that the interfering stimuli were presented in different modalities in the tests being compared. Verbal stimuli was presented in Rey Auditory Verbal Learning Test, whereas visual stimuli were used in Stroop and Trail Making Tests. Findings of this study point to an impairment in the prefrontal organization and monitorization of verbally processed information. To summarize, current PTSD patients were impaired on most tests of prefrontal functions when compared to controls; the performance of recovered PTSD patients was similar to controls on most prefrontal functions, but worsened as the difficulty of the test was increased (Eren-Koçak et al. 2009).

Another study, which investigated whether executive function deficits observed in PTSD improve with recovery from PTSD, compared executive functions before and after trauma-focused personal psychotherapy. They found significant improvement in both PTSD and depression symptoms as well as in executive functions after therapy (Walter et al. 2010). However, they did not study whether the improvement in both domains were parallel with each other.

Summary of Findings in Prefrontal Functions

Although the findings are not as robust as those on attention and memory, it has been widely accepted that PTSD patients are impaired on tests that measure prefrontal lobe functions (Barrett et al. 1996, Beers ve De Bellis 2002, Eren-Koçak et al. 2009, Gurvits et al. 1993, Koenen et al. 2001, Sutker et al. 1995, Uddo et al. 1993, Vasterling et al. 1998, Yehuda et al. 1995, Zalewski et al. 1994). One reason for this could be due to lower number of studies designed to evaluate prefrontal functions in PTSD, or to the fact that different tests of prefrontal functions are used in different studies. A study that employed tests specific to prefrontal functions found worse performance in delayed response and object alternation tests in PTSD patients than controls (Koenen et al. 2001). Stein (2002) reported that the impairment in executive functions was due to the trauma experience rather than to PTSD diagnosis; whereas, Twamley (2004), from the same research team, did not found any deficits in executive functions of either trauma exposed non-PTSD or PTSD groups. Gil (1990) suggested that the cognitive dysfunctions in PTSD were not specific to PTSD, and could be observed in other psychiatric disorders as well. Many executive functions that have been shown to be impaired in PTSD patients return to normal levels after recovery from PTSD. On the other hand, the performance of recovered PTSD patients in executive tests decreased to a level similar to that of current PTSD patients as the difficulty of the task increased. This finding indicated that, unlike memory functions, the prefrontal functions did not show a full recovery after PTSD is treated (Eren-Koçak et al. 2009).

CONCLUSION

The designs of the studies evaluating cognitive functions in PTSD are not uniform. The findings of studies on the topic require caution. Since trauma types differed across studies, control groups were either chosen among trauma exposed non-PTSD subjects or untraumatized healthy controls or both, different neuropsychological tests were used, and comorbidities were not evaluated in all. Despite these limitations, most of the studies reviewed here reported impairment in memory, and executive functions including attention. Although some authors suggested that cognitive deficits were limited to comorbid PTSD patients only, it has also been shown that cognitive impairment lasts even after comorbid depression was controlled. Both memory and executive functions recovered after treatment of PTSD symptoms, but the improvement in executive functions was dependent on the difficulty of the task. Since cognitive impairment leads both to a decrease in quality of life and severe disability, it is important to know whether cognitive deficits improve with PTSD treatment. Findings from a handful of studies on the topic are promising (Eren-Koçak et al. 2009, Fani et al. 2009, Walter et al. 2010), and future studies should target recruiting control subjects and larger cohorts of patients.

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