Psychology Research and Behavior Management

Dovepress
Taylor & Francis Group

Open Access Full Text Article

REVIEW

Brain Fog and Cognitive Dysfunction in Posttraumatic Stress Disorder: An Evidence-Based Review

Brahm D Sanger[1,2,*], Arij Alarachi[1,2,*], Heather E McNeely[2,3], Margaret C McKinnon[2–4], Randi E McCabe[2,3]

[1]Department of Psychology, Neuroscience, and Behaviour, McMaster University, Hamilton, ON, Canada; [2]St Joseph's Healthcare Hamilton, Hamilton, ON, Canada; [3]Department of Psychiatry and Behavioural Neurosciences, McMaster University, Hamilton, ON, Canada; [4]Homewood Research Institute, Homewood Health Centre, Guelph, ON, Canada

*These authors contributed equally to this work

Correspondence: Randi E McCabe, Anxiety Treatment and Research Clinic, St Joseph's Healthcare Hamilton, 100 West 5th Street, Hamilton, ON, L9C 0E3, Canada, Email mccabr@mcmaster.ca

Abstract: The term "brain fog" has long been used both colloquially and in research literature in reference to various neurocognitive phenomenon that detract from cognitive efficiency. We define "brain fog" as the subjective experience of cognitive difficulties, in keeping with the most common colloquial and research use of the term. While a recent increase in use of this term has largely been in the context of the post-coronavirus-19 condition known as long COVID, "brain fog" has also been discussed in relation to several other conditions including mental health conditions such as post-traumatic stress disorder (PTSD). PTSD is associated with both subjective cognitive complaints and relative deficits on cognitive testing, but the phenomenology and mechanisms contributing to "brain fog" in this population are poorly understood. PTSD psychopathology across cognitive, affective and physiological symptom domains have been tied to "brain fog". Furthermore, dissociative symptoms common in PTSD also contribute to the experience of "brain fog". Comorbid physical and mental health conditions may also increase the risk of experiencing "brain fog" among individuals with PTSD. Considerations for the assessment of "brain fog" in PTSD as part of psychodiagnostic assessment are discussed. While standard psychological intervention for PTSD is associated with a reduction in subjective cognitive deficits, other cognitive interventions may be valuable when "brain fog" persists following PTSD remission or when "brain fog" interferes with treatment. Limitations of current research on "brain fog" in PTSD include a lack of consistent definition and operationalization of "brain fog" in the literature, as well as limited tools for measurement. Future research should address these limitations, as well as further evaluate the use of cognitive remediation as an intervention for "brain fog".

Keywords: subjective cognition, cognitive complaints, mental fatigue, trauma

Introduction

Post-traumatic stress disorder (PTSD) is a mental health condition that can develop following exposure to actual or threatened death, serious injury, or sexual violence through direct experience, witnessing it occurring to others, learning about it occurring to a close family member or friend, or repeatedly being exposed to aversive details of traumatic events.[1] While 70% of individuals will experience such exposure in their lifetime,[2] only approximately 6% of them will develop PTSD.[3] Estimates of PTSD prevalence range from 2–9% of the population, and risk of developing PTSD may be greater for individuals who have experienced childhood, prolonged, or interpersonal traumatic events.[4] PTSD symptomology is characterized by four distinct clusters: re-experiencing of traumatic experiences, avoidance of potentially triggering stimuli, negative alterations to mood and cognition, and alterations to arousal and reactivity.[1]

Alterations to cognitive processes in PTSD include difficulty concentrating or maintaining attention, difficulty recalling important aspects of traumatic events, involuntary recollections of traumatic events, as well as perceptual alterations during dissociative experiences such as derealization (eg, feeling as if the world is unreal or dreamlike) and depersonalization (eg, feeling as if one is outside their own body).[1] Beyond this, individuals with the disorder also report subjective cognitive failures in daily living, including attentional slips leading to mistakes in tasks, forgetting tasks, and losing objects.[5] Such cognitive failures

Received: 7 November 2024
Accepted: 21 February 2025
Published: 12 March 2025

have been described as symptoms of "brain fog",[6] which is neither a diagnostic symptom of PTSD, nor a formally used medical term. What constitutes the phenomenology of "brain fog" in PTSD is unclear and currently, there is no unified definition of it in the literature, particularly in the PTSD clinical population. This lack of conceptualization and understanding of brain fog may reduce accuracy of assessment processes and ultimately lead to poor clinical outcomes. Importantly, 30-50% of those with PTSD have poor responses to first-line interventions;[7] given this, it is essential to consider associated complaints of the disorder that may act as markers of treatment non-response.

Conceptualizations of "Brain Fog" in Other Clinical Populations

Notably, "brain fog" has been described as a consequence of PTSD following coronavirus exposure (ie, severe acute respiratory syndrome [SARS] and Middle East respiratory syndrome [MERS]).[8] PTSD may be a differential diagnosis for COVID-19 survivors,[8] where "brain fog" has been described as one of the major symptoms associated with the post-COVID condition known as long COVID.[9–11] In long COVID, the main symptom indicators of 'brain fog' appear to be subjective memory impairment, word-finding difficulties, fatigue, non-orthostatic dizziness, and muscle pain.[12]

The symptom of "brain fog" has also been reported by those with chronic fatigue syndrome, postural tachycardia syndrome (POTS), fibromyalgia, mild and moderate-to-severe traumatic brain injury.[13–17] Beyond medical conditions, "brain fog" is also a common complaint for those experiencing menopause[18,19] and those with mental health conditions,[20] although the latter area is highly under-researched. Moreover, many of the chronic medical conditions associated with symptoms of "brain fog" are highly comorbid with clinical anxiety and depression, which also involve cognitive symptoms.[21,22] McWhirter et al[20] examined over 1500 Reddit posts on "brain fog" and found that individuals described forgetfulness, concentration issues, dissociation, cognitive "slowness", brain "fuzziness", and fatigue as manifestations of "brain fog". Corroborating this are the results of a study on individuals with POTS in which the most endorsed descriptors of "brain fog" were "forgetfulness", "cloudiness", and "difficulty focusing, thinking and communicating".[16] Paralleling heterogeneity in its colloquial description, the definition of 'brain fog' has also varied tremendously in the literature. While some describe it as a subjective experience of cognitive deficits or impairment,[12] it is elsewhere described as a subclinical or preclinical form of a cognitive disorder, used interchangeably with cognitive dysfunction or impairment,[23,24] functional cognitive disorder,[25] or as a symptom of chronic neuroinflammation.[26]

Measuring "Brain Fog"

Unsurprisingly, given its nebulous nature, there is considerable variability in how "brain fog" is measured. For example, over the last few years, four different scales have been developed to operationalize "brain fog". These scales were validated across heterogeneous populations, including samples of individuals with and without traumatic brain injury,[27] (Elliott et al, 2023), Polish university students,[28] the general population in Turkey,[29] and individuals with coeliac-related gastrointestinal disease.[30] Moreover, the factor structure between these four scales demonstrates significant variability with respect to number of factors (two or three) and factor groupings identified (eg, fatigue and cognition; cognitive and affective or somatic for the two-factor scales and cognitive, psychological, and physiological; mental fatigue, cognitive acuity and confusion for the three factor scales). Given that the operationalization of "brain fog" for measurement has only begun recently, these scales were unsurprisingly not assessed for convergence with one another. Excepting Knowles et al,[30] these scales also lacked any comparison to existing scales of subjective cognitive deficits. Finally, only Knowles et al[30] evaluated their measure of "brain fog" for convergence with neuropsychological test performance, with weak and non-significant results. Problems with operationalization and variance in psychometric validation methodology may be tied to the inconsistent definition of "brain fog" within the literature, as selection of measures for comparison with new scales is tied to construct conceptualization. Clearly, the lack of a unifying definition for "brain fog" is a significant limiting factor for its study across various conditions, PTSD included.

Kaseda and Levine[8] have argued that the stressors experienced by individuals undergoing hospitalization may fulfill the first diagnostic criteria for PTSD, leading to symptoms of "brain fog". Thus, PTSD symptoms may, in part, explain the experience of "brain fog" among individuals with chronic conditions. However, no studies have directly explored the phenomenology or associated symptoms of "brain fog" in those with PTSD. Thus, it is unclear how and which aspects of PTSD may lead to "brain fog". Moreover, the lack of existing literature about "brain fog" in PTSD may be due to a lack of consistent definition, both in the literature and in its colloquial use.

Definition

Despite the lack of a consistent definition of "brain fog" in the research literature, and limited validated measures of "brain fog", it is valuable to critically review how PTSD may support "brain fog". This review will use the following definition of "brain fog": *a subjective experience of cognitive impairment or diminished mental capacity.*[15,19] While other studies have included objective cognitive deficits in their conceptualization of "brain fog", we have elected not to do so as a growing body of evidence includes self-report of "brain fog" among individuals with a lack of objective cognitive impairment, and subjective cognitive deficits may not be associated with worse cognitive test performance in PTSD and trauma-exposed individuals,[31–35] although some studies have shown this association.[36,37] Given that "brain fog" has been mentioned across several physical and mental health conditions, it also may not be appropriate to tie it to a specific preclinical or subclinical cognitive disorder, or as a symptom of neuroinflammation. Nonetheless, cognitive performance on testing will be considered within this review insofar as it may corroborate subjectively reported cognitive deficits.

Purpose of the Review and Objectives

Individuals with PTSD are increasingly reporting "brain fog" as a complaint, yet the phenomenology and associated clinical implications are poorly understood. To date, no studies have directly attempted to review the literature on "brain fog" in individuals with PTSD, using an explicit definition of it. Moreover, the available literature on "brain fog" focuses on other clinical populations, with fragmented definitions and conceptualizations of the phenomenon. Thus, the current review will examine the literature on "brain fog" and its component parts (eg, subjective cognitive impairment, difficulties with mental acuity, and fatigue) in individuals with a diagnosis of PTSD. First, the available evidence on the experience of "brain fog" in PTSD populations will be described to elucidate its phenomenology. Within this section, the different origins of "brain fog" symptomatology (ie, within PTSD, comorbid conditions such as depression and anxiety, and physiological or organic causes of "brain fog") will be explored. Next, the implications of "brain fog" in those with PTSD on assessment and intervention outcomes will be discussed, and preliminary recommendations for mental health professionals will be provided. Lastly, research gaps and future directions will be identified with the goal of enriching current understandings of "brain fog" in individuals with PTSD and other mental health disorders.

Methods

This study employed a narrative review approach to examine existing research on "brain fog" among individuals with PTSD. This approach was selected to allow for a comprehensive and flexible evaluation of the topic, which was crucial given the dearth of literature specifically addressing "brain fog" in PTSD. Given the terminology related to "brain fog" has been highly inconsistent across the literature, a narrative review allowed for a more detailed examination and comparison of findings across studies meeting the present conceptualization of "brain fog". Use of a narrative review style also allowed for an iterative approach to provide a general overview of "brain fog" in PTSD as further information was gathered and common themes in the research were identified.

An initial search was conducted using the Ovid Medline and APA PsycINFO databases for English-language papers from 2004-present with key words to represent the concepts of "brain fog" and "PTSD". A total of 2944 papers were initially identified, and search strings are provided in Supplementary Table 1. As this was a narrative review, we selected the literature we considered most relevant to our focus, and in keeping with our working definition of "brain fog": the subjective experience of cognitive impairment or diminished mental capacity. Following identification of papers relevant to our review, reference lists were reviewed to identify more relevant literature. Additional papers were identified through author suggestions. As additional key concepts were identified, searches were re-run to include them.

Overview of Trauma & "Brain Fog" Literature

While PTSD is associated with both self-reported cognitive difficulties and objective deficits on neuropsychological measures, there is mixed evidence regarding their relation. Mattson et al[37] reported a significant association between subjective and objective cognitive deficits among combat-exposed veterans, and that this relationship was mediated by PTSD symptom severity. Multiple other studies, however, have not found a significant relationship between subjective

and objective cognitive deficits in PTSD.[32,33,38] Interestingly, Samuelson et al[39] found that among military veterans the association between PTSD and functional outcomes was mediated by subjective rather than objective cognitive deficits. Objective cognitive deficits, however, may also have a significant relationship with outcomes in PTSD. In their review of studies of attention deficits among adults with PTSD, Punski-Hoogervorst et al[40] found that 61.2% (n = 30) of studies found a significant correlation between PTSD symptoms and attention deficits, and 20.4% (n = 10) of studies found that attention deficits were predictive of worse PTSD symptomology. Furthermore, cognitive deficits in verbal memory and executive functions are associated with worse treatment outcomes among individuals with PTSD receiving psychotherapy.[41–45] Similar effects have been found among individuals with depressive disorders, as deficits to executive functions have been associated with worse outcomes of pharmacological therapy (see Groves et al,[46] for a review). Thus, understanding factors contributing to the development of both "brain fog" and objective cognitive deficits in PTSD is highly relevant to improve functioning. These factors include additional PTSD symptoms and comorbid mental health and physiological conditions.

Cognitive complaints in PTSD are not only common, but they also represent a core diagnostic domain of the disorder. For example, DSM-5-TR criteria E5, "problems with concentration",[1] may capture the report of "brain fog" among individuals with PTSD as attentional difficulties underpin its colloquial descriptors.[16,20] Moreover, poor self-reported attentional control, mediated by rumination, is related to PTSD symptoms.[47] In addition to self-reported attentional difficulties, individuals with PTSD consistently perform worse on tests of attention and executive functions, such as attentional control, working memory, inhibition, and switching.[48–51] Critically, on several clinical trials the most prominent deficits in PTSD (ie, executive functions, verbal memory) are associated with worse response to psychological interventions.[41–45]

Alterations to arousal in PTSD, which include hyper- and hypo-arousal are also associated with concentration difficulties. Both of these states can have significant implications for perceived cognitive challenges; hyperarousal, characterized by heightened physiological and emotional reactivity, may overwhelm cognitive resources and contribute to perceived cognitive deficits, while hypo-arousal, characterized by emotional numbing, detachment, and reduced physiological reactivity, may lead to disengagement from the environment and reduced goal-directed cognitive activity.[52,53] In a study of Chinese breast cancer participants (n = 204), PTSD symptoms and fatigue independently accounted for subjective cognitive impairment, and among PTSD symptom categories, only symptoms related to hyper-arousal significantly contributed to subjective cognitive impairment.[54]

Dissociative symptoms are associated with cognitive dysfunction across neuropsychiatric disorders (see McKinnon et al,[52] for a review), as well as with other negative outcomes in PTSD. For example, Boyd et al[55] found that among military members, veterans, and first responders, dissociative symptoms mediated the relationship between PTSD symptoms and functional impairment. Interestingly, Park et al[56] found that dissociative symptoms were a significant predictor of impairment on objective neuropsychological test performance but not subjective cognitive impairment, while PTSD symptom severity predicted subjective cognitive impairment but not test performance. Conversely, Gold et al[57] reported three case studies of women with lifetime trauma exposure and dissociative symptoms, who were referred for neuropsychological assessment of memory decline complaints. The neuropsychological test results did not support objective cognitive deficits among the women despite their subjective reports of poor daily functioning. The authors proposed that dissociative symptoms interfered with cognitive processes supporting learning in daily living, leading to lapses in memory. Indeed, dissociative symptoms are associated with worse attention, executive functions, and memory in PTSD.[52] As such, dissociation and hypo-arousal may explain distress related to altered cognitive functioning among individuals with "brain fog". Moreover, dissociation is associated with a diminished sense of agency among adults,[56] which may contribute to the perception of "brain fog". The authors evaluated the role of post-traumatic stress and poor sleep quality on dissociative symptoms and impaired sense of agency and found that they represented independent pathways to both constructs.[58]

Research also suggests that specific DSM-5 PTSD symptoms criteria may be more related to subjective cognitive complaints than others. For example, Gunak et al[59] used network models to explore the associations between PTSD symptoms and subjective cognitive functioning among a large sample of military veterans (n = 1484) and found robust relations between subjective cognitive functioning and the diagnostic criteria "trouble experiencing positive feelings";

and "difficulty concentrating". While the findings were limited by observational design, the results were replicated in a three-year follow-up (n = 713).[59]

Individuals with PTSD reexperience traumatic events in mentally distressing ways (eg, intrusive memories, flashbacks, dreams), and these symptoms and perceived inability to control them may also contribute to reports of "brain fog" in PTSD. In their review of the role of attentional deficits in the symptomatology of PTSD, Punski-Hoogervorst et al[40] suggested that the disruptive interface between affective re-experiencing symptoms and attentional skills is a primary driver of any correlation between PTSD symptom severity and attentional functions. To reduce intense negative emotions, individuals with PTSD avoid thoughts, feelings and reminders of trauma memories; this avoidance depletes necessary attentional resources for other tasks. Avoidance behaviors can include use of maladaptive emotion regulation strategies such as emotion suppression, which individuals with PTSD use more frequently than individuals without PTSD.[60] Use of emotional suppression is associated with worse memory for information among healthy controls,[61–63] as well as individuals with PTSD.[64] Given increased use of emotion suppression among individuals with PTSD, the associated memory deficits may contribute to perceived "brain fog", but no evaluations of this potential relation were found in the literature. Difficulties with emotion regulation may also contribute to alterations in arousal and reactivity, as Park et al[65] found that among military member and veterans as well as public safety personnel (n = 61), difficulties with emotion regulation along with PTSD symptom severity explained variance in self-reported cognitive functioning. Furthermore, the consequences of difficulties with emotion regulation may include alterations to arousal and reactivity in PTSD, leading to the "brain fog" associated with hypo- and hyper-arousal.

Comorbid Conditions and "Brain Fog"

PTSD is frequently comorbid with a myriad of mental health conditions, with approximately 80% of the clinical population meeting diagnostic criteria for an additional mental health condition. The most commonly comorbid mental health conditions are depressive disorders (eg, 54%) and anxiety and related disorders (eg, 36% for social anxiety disorder; 28% for obsessive-compulsive disorder).[66] The presence of these comorbid mental health disorders may contribute to the experience of "brain fog" in those with experiences of trauma or PTSD, either directly due to experience of cognitive impairment associated with the comorbid disorder (as cognitive impairment is also common among other mood and anxiety disorders) mimicking or adding to symptoms associated with "brain fog", or indirectly due to the comorbid conditions' aggravation of PTSD symptom severity.

Approximately 30–50% of individuals with PTSD also have comorbid clinical depression.[66,67] Even amongst those who experience a traumatic event but do not meet diagnostic criteria for PTSD, the risk of developing depression is high.[68] Moreover, there are higher rates of trauma exposure, particularly childhood abuse and neglect amongst those with clinical depression than healthy controls.[69] The mechanism underlying the high co-occurrence between trauma or PTSD and depression is still under investigation; however, one hypothesis is that the two disorders share risk factors, such as negative affectivity and experiences of childhood adversity.[70,71] Interestingly, although depressive disorders are best characterized by symptoms of low mood and anhedonia, research suggests that cognitive impairment is also core feature of clinical depression.[72] Specifically, a meta-analysis using 24 studies found that those with depression show more deficits than healthy controls in domains of executive functioning, memory, and attention.[72] Furthermore, even those with remitted depression were found to have persistently impaired cognitive abilities.[72] Moreover, the diagnostic criteria for depressive disorders include subjective reports of concentration or decision-making difficulties, and fatigue.[73] Thus, even for those without objectively impaired cognitive abilities, the clinical presentation and psychopathology of PTSD with comorbid depression may further contribute to the risk of experiencing "brain fog". Lastly, research suggests that experiences of childhood abuse and neglect in those with clinical depression are associated with poorer cognitive functioning.[69]

Research also suggests that melancholic depression (eg, a more severe subtype of depression that tends to be less responsive to psychosocial interventions)[74] may be associated with a higher risk for "brain fog" than non-melancholic depression.[75,76] In one study of combat veterans, over two-thirds endorsed symptoms related to melancholic depression and the remaining sub-group endorsed symptoms related to catatonic, atypical, or no particular depression group subtype.[77] Here, it is possible that those with PTSD and comorbid melancholic-type depression may be at the highest risk of experiencing "brain fog" symptoms.

Similarly, anxiety and related disorders are highly comorbid with PTSD,[66] with research also suggesting this cooccurrence may be related to shared vulnerability factors.[71,78] Even when studied in isolation, clinical anxiety is associated with impaired performance on neuropsychological tests of executive functioning.[78,79] Moreover, some clinical features such as distressing emotional states, anticipation of perceived threat, and avoidance behaviors heavily overlap between anxiety disorders and PTSD.[80] The presence of symptoms of both disorders likely exacerbates symptoms of hyperarousal, increasing the overall burden on cognitive capacity. Eysenck et al[81] specify that some neurocognitive functions may worsen due to anxiety, while others improve (eg, due to enhanced efforts). Regardless of the potential benefit of the presence of clinical anxiety for cognition, the authors report that overall, anxiety reduces processing efficiency,[81] which may be experienced as taxing, or a symptom of "brain fog". This hypothesis was corroborated by a recent study that found that the presence of repeated negative thinking (eg, worry, rumination) explained symptoms of fatigue in a clinical anxiety sample, even when anxiety-related sleep disturbance was controlled for.[82] Thus, even for those with trauma, PTSD, and comorbid clinical anxiety without objective cognitive deficits, there may be subjective cognitive complaints that are consistent with reports of "brain fog". Research even suggests that for some, but certainly not all, patients with psychiatric conditions, discrepancy between objective and subjective impairment may be driven and/or exacerbated by perfectionism and unrealistic expectations for cognitive performance.[83,84]

Additionally, some diagnostic criteria for anxiety disorders specifically refer to subjective reports of cognitive difficulties. For example, a study found that in a sample of 175 adults with generalized anxiety disorder, 90% endorsed the diagnostic criteria of "difficulty concentrating or mind going blank".[85] Another study found that the high rate of endorsement of concentration difficulties persisted, even when depression was controlled for.[86] In panic disorder, some individuals experience dissociative symptoms,[80] and dissociation within this clinical population is positively correlated with the number of traumatic events experienced;[87] how this relationship interfaces with cognitive performance was not investigated. Nonetheless, taken together, this research suggests that the source of "brain fog" complaints in those with co-occurring PTSD and clinical anxiety depends on the specific symptoms reported, as well as the type of comorbid anxiety disorder present.

Approximately 30% of those with PTSD report difficulties with alcohol use, and 25–50% report difficulties with substance use,[88,89] which may further increase risk of experiencing "brain fog" symptoms. Problematic substance and/or alcohol use is known to be directly associated with cognitive impairment.[90] Risk of cognitive impairment is further exacerbated by polysubstance use; a study with 753 individuals with polysubstance use found that 70% of the sample reported experiencing cognitive deficits (ie, subjective cognitive impairment), 50% showed objective cognitive deficits, and approximately 30% of the sample met criteria for substance-induced neurocognitive impairment.[91] Research suggests there is a bidirectional relationship between PTSD symptom severity and alcohol and/or/ substance use difficulties.[92] However, a study with 334 individuals participating in a specialized PTSD treatment program found that dissociative symptom severity mediated this relationship, such that higher PTSD symptom severity was associated with higher dissociative symptom severity, which was associated with greater levels of alcohol use difficulties.[93] Given that dissociative symptom severity in PTSD is linked to poorer functional outcomes,[55] it is likely that alcohol and/or substances are used in an attempt to cope with symptoms. Unfortunately, cognitive difficulties in both PTSD and alcohol and/or substance use are likely exacerbated by their co-occurrence which may contribute to client complaints of "brain fog".

It is also worth noting that individuals with PTSD are frequently prescribed medications associated with subjective and objective cognitive changes, including anticholinergic,[94–96] sedative,[97] and anti-psychotic medications.[98–100] Estimates of use of these medications are high. For example, in a cohort study of American veterans with PTSD (n = 1073183) found 34.4% were prescribed selective-serotonin reuptake inhibitors, 13.6% serotonin–norepinephrine reuptake inhibitors, 17.1% trazodone, 7.5% mirtazapine, 8.7% benzodiazepines, 6.0% non-benzodiazepine hypnotics, and 7.0% atypical antipsychotics.[101] Similarly, in a sample of inpatients (n = 1044) with PTSD in German-speaking countries, 72.0% were found to use antidepressants, 58.4% antipsychotics drugs, and 29.3% tranquilizing medications (29.3%).[102]

In addition to mental health conditions, PTSD is often comorbid with several physical health conditions, including cardiovascular disease, musculoskeletal conditions, sleep apnea and other sleep disorders, chronic pain, migraine, and traumatic brain injury,[103–107] even when confounders such as body mass index, age, substance use, and physical exercise

are controlled for.[108] Importantly, these conditions are also associated with experiences of subjective cognitive complaints,[109–114] and multimorbidity significantly increases likelihood of subjective cognitive complaints in the general population.[115] Among 224,842 adults from low- and middle-income countries, subjective cognitive complaints increased with each chronic condition; interestingly, 30% of the association between multimorbidity and subjective cognitive complaints was explained by psychological factors,[115] suggesting a role of mental health conditions in exacerbating the experience of cognitive impairment associated with physical health conditions.

While there is less available literature comparing associations between these physical health conditions and cognitive complaints in the context of co-morbid PTSD, Martindale et al[116] found that sleep quality affected cognitive test performance independently of and beyond the effects of PTSD symptoms among military combat veterans (n = 135). The mechanism underlying the association between these various conditions and the experience of subjective cognitive impairment is poorly understood. However, "brain fog" has been previously described as a consequence of chronic neuroinflammation damaging healthy cells,[26] and a growing body of evidence consisting of post-mortem and in-vivo studies in humans as well as experimental studies in animals has indicated significant microglial activation associated with psychiatric conditions.[117] In a recent review of neuroinflammation in PTSD, Lee et al[118] summarized evidence indicating that psychological stress in PTSD mediates the relationship between the immune system and the brain to produce neuroinflammation.

Discussion

Overall, the experience of "brain fog" or subjective cognitive complaints is frequently reported among individuals with trauma, PTSD, and other related mental health concerns. It is unclear whether symptoms of "brain fog" always converge with objective measurements of cognitive deficits, however they nonetheless represent a form of functional impairment and suffering that may interfere with treatment. Given this significance, there are several clinical implications and considerations when clients with PTSD or trauma present reporting "brain fog".

Clinical Implications

Assessment

The most critical step of effective care is a comprehensive assessment, including for individuals with PTSD who report "brain fog" symptoms. The colloquial language used by individuals to describe the experience of subjective cognitive complaints (eg, "fuzziness", "slowness", "forgetfulness") does not always clearly correspond to the diagnostic criteria for PTSD or comorbid conditions. Moreover, subjective cognitive complaints reported by those with psychiatric conditions, PTSD included, do not always converge with objective measurements[32,33,38] that may be used in neuropsychological assessments, despite their reflection of the everyday experience of sufferers in uncontrolled real-world conditions that may trigger disease symptoms and affective dysregulation associated with poor cognitive performance. As such, individuals reporting "brain fog" that screen negative on clinical or neuropsychological assessments may be overlooked. Nonetheless, those reporting "brain fog" can often identify functional impairment and distress associated with their symptoms.[119] Research also suggests that experiences of subjective cognitive complaints in those with PTSD may signal a higher risk of suicidality, further necessitating robust screening for "brain fog".[120] Therefore, these complaints represent a form of suffering, mirrored for some in functional impairment in real-world circumstances (as opposed to intact performance that might be obtained in the highly controlled and structured neuropsychological testing environment) that warrants clinical attention. It is further notable that for individuals who performed above average on neuropsychological testing prior to disease onset (as also indexed in measures of pre-morbid intellectual functioning and occupational and educational attainment), neuropsychological performance in the average or low average range is nonetheless a performance decrement and likely to be experienced as such.

Screening

PTSD-related cognitive symptoms may improve following treatment of PTSD, particularly for memory;[121] therefore, screening of cognition may suffice when clients report some symptoms of "brain fog". Screening can be completed by trained mental health professionals and includes interviewing to assess psychiatric and cognitive symptoms, and

screening-based standardized measurement of cognition and subjective cognition. Clinicians are encouraged to conduct an evidence-based standardized diagnostic interview with clients with PTSD symptoms to screen for clinically significant problems with concentration, memory, processing speed, and executive functioning (DSM-5-TR criterion E5 for PTSD[1]) as well as other PTSD symptoms and co-occurring symptoms. During these psychodiagnostic assessments, clinicians should assess for and document-specific factors that place clients at higher risk of experiencing subjective cognitive complaints are present, including: levels of dissociation,[57] anxiety symptoms,[82,85] comorbid melancholic depression,[75,76] and the use of emotional suppression.[61,63] Clients who report a diminished sense of agency or self-efficacy may also be considered higher risk.[58] Notably, in emerging research among individuals with PTSD, higher levels of self-compassion are associated with lower PTSD symptoms, and lower fear of self-compassion is associated with lower PTSD symptomatology (see Winders et al,[122] for a review).

Also, critical to consider during the psychodiagnostic assessment are factors related to the nature of the traumatic events also affect cognitive complaints, as dissociation is more common among individuals with more frequent and earlier onset of traumatization.[123,124] Severity of traumatic exposure may also be predictive of cognitive complaints, as Singh et al[125] and Stein et al[126] found that among responders to the World Trade Center attacks, higher intensity exposure was associated with greater cognitive concerns. While Singh et al[125] found that the relationship between high-intensity traumatic exposure was predictive of self-reported cognitive change even after controlling for PTSD, depression, and alcohol abuse, Stein et al[126] found that the relationship between the highest high-intensity traumatic exposure and cognitive concerns was attenuated when accounting for depression, anxiety, PTSD and psychotropic medication use. Clinicians are also encouraged to query the onset of "brain fog" symptoms, as well as how they are precipitated in day-to-day life (eg, did the brain fog start before the traumatic event, after, or was it associated with a physical health change, such as menopause? Do the symptoms get precipitated by intrusion-type symptoms, such as a flashback of the traumatic event, or by experiences of anhedonia?) to parse out the various contributing factors of the symptoms and determine if "brain fog" may be greater than typical of PTSD.

Existing standardizing screening interviews for cognitive complaints (eg, Cognitive Complaints Toolkit [California Alzheimer's Disease Centers]; Cognitive Complaint Interview)[127] have largely centered on evaluating for cognitive complaints in the context of dementia, which may not be appropriate for cognitive complaints seen in "brain fog" for PTSD. Another standardized interview, the Cognitive Assessment Interview (CAI),[128] was developed to assess daily functioning among individuals with schizophrenia, who also experience significant cognitive deficits but typically have a higher level of cognitive functioning than individuals with dementia. While the pattern and severity of cognitive deficits and complaints in schizophrenia and PTSD may not be the same, use of this tool may present an opportunity to screen for "brain fog" with standardized interviewing developed for a primary mental health population.

Some researchers have also evaluated the use of cognitive testing to screen for "brain fog" in long COVID. Lynch et al[129] used the Montreal Cognitive Assessment (MoCA),[130] a cognitive screening tool designed to be sensitive to mild cognitive impairment, along with a neuropsychological test battery to validate objective cognitive impairment in 60 participants with long COVID seeking care for "brain fog". They found that only approximately 37% of participants reporting "brain fog" scored below the MoCA cutoff for mild cognitive impairment, and that the MoCA was approximately 63% accurate at detecting diminished performance on the neuropsychological test battery.[129] Overall, the authors concluded that the MoCA was not suitable at detecting cognitive impairment among participants presenting with "brain fog" and long COVID,[129] a finding that may be related to ceiling effects on this measure and its inability to capture more subtle cognitive performance decrements in this population.

Administration of a self-report measure of subjective cognitive concerns may be helpful to determine whether neuropsychological investigation is required. Although there are some self-report questionnaires that have been recently developed to measure "brain fog",[27–29] there are other, more validated ones that assess general subjective cognitive complaints. A systematic review examining the utility of measures of subjective cognitive complaints in a variety of psychiatric populations identified 35 studies, with 11 different questionnaires used.[131] Groenman et al[131] found that the Behavior Rating Inventory of Executive Function (BRIEF),[132] followed by the Cognitive Failures Questionnaire (CFQ),[133] was most frequently used across different psychiatric diagnoses. Both the BRIEF[134–136] and the CFQ[36] have been used to capture subjective cognitive complaints in samples with PTSD and trauma, however the research

on how well these measures correlate with objective cognitive impairment is limited. Although further research is needed to fully understand these screening tools' psychometric performance in those with PTSD and trauma, they may provide clinical utility for determining severity of "brain fog" symptoms.

Neuropsychological Assessment

For a subset of patients who report severe cognitive symptoms beyond what is typical of PTSD and co-morbidities, or whose cognitive symptoms interfere with treatment, or whose cognitive symptoms persist even after other PTSD symptoms improve (eg, with psychotherapy or pharmacological intervention),[137] formal neuropsychological evaluation is recommended. Neuropsychological assessment using standardized measures with greater levels of sensitivity to mild to moderate cognitive deficits may be required in this condition. Given that there may be a separation between reported "brain fog" and objective cognitive deficits in PTSD (as in other mental health conditions), it remains unclear whether objective neuropsychological testing would consistently produce findings consistent with subjective cognitive complaints. Nonetheless, neuropsychological assessment may be beneficial in characterizing the cognitive profile of individuals with "brain fog". Objective neuropsychological assessment is conducted in neutral, distraction-free, quiet, standardized settings intended to allow individuals to perform at their best. Skilled examiners attend to behavioral indicators of emotional distress, fatigue, and even dissociation, to support clients to perform at their best by offering breaks and non-specific support. The results can then be used to extrapolate how well an individual might perform in less optimal, real-world settings characterized by distractions, fatigue, and emotional distress, in which cognitive failures may be more likely to occur. In addition to obtaining detailed information on objective cognitive performance, comprehensive neuropsychological assessment also includes a review of medical, neurological, and psychological factors to investigate the probable contributors to "brain fog" complaints and yield recommendations for lifestyle modifications or other medical interventions that might improve cognitive functioning (eg, sleep study, cognitive-behavioral therapy for insomnia, etc.) as well as individualized compensatory strategies or accommodations based on the individual's profile of cognitive strengths and weaknesses.

Feedback

An additional critical assessment consideration is the provision of psychoeducation related to "brain fog" symptoms during feedback. Clients may not be aware that some "brain fog" symptoms correspond directly to diagnostic criteria of PTSD and other mental health conditions, or associated features and that various malleable lifestyle factors such as poor sleep, poor diet, lack of activity, and alcohol and substance use may also contribute to a subjective experience of "brain fog". Also, the self-labelling of symptoms as "brain fog" may cause distress about the cause of symptoms as well as whether it is even a treatable condition, or possibly a degenerative condition. As such, providing psychoeducation around what individual "brain fog" symptoms represent by referring to them as symptoms of other conditions (eg, "PTSD-related concentration difficulties" or "subjective cognitive complaints due to depression") is recommended. Using these more appropriate labels may reduce feelings of alarm and lack of control associated with the poorly understood experience of "brain fog". The importance of psychoeducation was highlighted in a study by Rosada et al,[138] wherein they found that in comparison to a no-feedback control condition, neuropsychological assessment feedback was associated with improved quality of life, insight into condition, as well as ability to cope with its effects at follow-up (n = 218). Taken together, demystifying the confusing experience of "brain fog" by validating the associated distress and impairment, while shifting to a more specific explanation of the symptoms (eg, subjective cognitive difficulties or PTSD symptoms) and strategies for remediating and compensating for difficulties after assessment is highly recommended.

Intervention

First-line psychological interventions for PTSD include cognitive-behavioral therapy, cognitive-processing therapy, and prolonged exposure therapy.[139] For individuals that report "brain fog" or subjective cognitive complaints, an augmented treatment approach may be most appropriate. The treatment plan used to target "brain fog" is dependent on the underlying mechanisms contributing to symptoms (eg, is it due to PTSD, comorbid mental health conditions, or health issues). Research suggests that "brain fog" symptoms that appear to be rooted in psychopathology tend to improve following psychological treatments (see Krysta et al,[140] for a review). Based on the results of the current review,

specifically targeting dissociation, emotion dysregulation, low self-efficacy, and melancholic depression in therapy may mediate clinically significant improvement. Alternatively, "brain fog" symptoms rooted in comorbid physical health conditions, hormone-related changes, or medication side-effects may necessitate a multidisciplinary approach. For example, Krishnan et al[141] recommend a multidisciplinary approach for treating "brain fog" in long COVID, involving improving sleep hygiene, addressing psychological factors, and reducing neuroinflammation through better nutrition.

Critically, on several clinical trials, the most prominent cognitive deficits in PTSD (ie, executive functions, verbal memory) are associated with worse response to psychological interventions.[41–45] While subjective cognitive complaints may not always be linked with objective deficits on standardized cognitive testing, it is important to recognize that PTSD is associated with significant subjective cognitive deficits that may nevertheless be functionally impairing. Clients with severe subjective cognitive complaints (eg, higher levels of associated distress or impairment) may benefit less from psychological interventions, given the cognitive demand of structured psychotherapy. Indeed, Samuelson et al[39] found that self-reported cognitive problems, and not performance on cognitive tests, mediated the relationship between PTSD diagnosis and functional outcomes. As such, targeting "brain fog" symptoms and subjective cognitive failures as part of intervention for PTSD may not only be appropriate, but essential for some individuals.

While there may be some disassociation between the measurement of objective and subjective cognitive symptoms among individuals with PTSD, the probable underlying domain of these deficits appears similar. Namely, attentional and executive deficits often contribute to deficits across other cognitive domains and are also likely to underlie the subjective symptoms cited in the experience of "brain fog" (eg, concentration issues, "slowness"). Thus, targeting attention and executive cognitive functioning may be particularly beneficial. Cognitive remediation therapies target cognitive deficits in varying domains (eg, attention, executive functioning, metacognition) to improve functioning and lead to long-lasting, generalizable improvements in these areas (as defined at the Cognitive Remediation Experts Workshop, 2010). While cognitive remediation therapies have largely been developed for brain injury populations, they have, in more recent years, been effectively applied for mental disorders (see Kim et al[142] for a review).

Metacognitive strategy training has emerged as the practice standard for targeting executive deficits (including impairment in emotion regulation) in acquired brain injury.[143] These training programs are typically structured, short-term, and include components such as psychoeducation on cognitive deficits associated with PTSD, strategies for self-monitoring, mindfulness activities, and skills to improve various executive functions such as planning, memory, and more.[144] Administration involves a clinician skilled in PTSD and its associated cognitive deficits, instructional materials, and worksheets for homework tasks between sessions.[145] Emerging evidence on the application of metacognitive strategy training for executive deficits in PTSD has promising results for subjective cognition and objective test performance. In a pilot randomized control trial (n = 40) of Goal Management Training (GMT)[146] a metacognitive strategy training program in an outpatient PTSD clinic, Protopopescu et al,[147] found that GMT was uniquely associated with improvements in measures of subjective cognition and functional outcomes, as well as cognitive test performance, particularly in the domain of executive functions. Boyd et al[148] found that in an inpatient PTSD context, GMT with standard treatment but not standard treatment alone was associated with improvements on cognitive testing, while both conditions were associated with improvements of subjective cognition. Another metacognitive training program, goal-oriented attentional self-regulation (GOALS) has also been associated with improvement in executive functions as well as complex functional task performance among veterans with comorbid PTSD and TBI.[149] In addition to metacognitive strategy training, other cognitive remediation strategies have been applied in PTSD with significant improvements in objective measurements of cognitive skills and PTSD symptoms.[150–152]

Larger-scale clinical trials are required to fully realize the benefits of cognitive remediation therapy for PTSD and determine the treatment suitability requirements (eg, which target populations and conditions are most appropriate). Moreover, referral to and administration of these interventions may not be feasible in resource-limited settings or by non-specialists. There is preliminary evidence that some cognitive remediation strategies, including GMT, may be adapted for single-session administration with improvement in subjective cognitive function (Caseletto et al, 2016; Carstens, 2016), but it is unclear whether this approach would be efficacious for "brain fog" in PTSD. However, it remains a promising intervention for the cognitive symptoms that may be referred to as 'brain fog' in this clinical population.

Limitations

The current study is the first to our knowledge to provide a descriptive summary of the current state of the literature on "brain fog", a complex, poorly understood phenomenon, particularly in individuals with PTSD. However, given the dearth of research in this area, there are some limitations of the current study to be noted. Firstly, the lack of research on "brain fog" in those with PTSD necessitated the methodological approach (eg, narrative review rather than a systematic review). Despite the authors' efforts to follow a semi-structured approach, the narrative review process is iterative and semi-structured; therefore, it is possible that researcher bias influenced the selection of databases used or inclusion of literature. Moreover, we centered our review of "brain fog" associated with PTSD on subjective cognitive complaints, however, it remains that the various "brain fog" definitions in the larger literature have yet to be validated. As such, the findings of the study correspond to our definition, but may not generalize to divergent understandings of the phenomenon. Lastly, given the construct validity of 'brain fog' associated with PTSD is yet to be established, it is important to note that we cited empirical findings on subjective cognitive complaints to inform the clinical recommendations. In addition, these recommendations are preliminary and there remain several research gaps that necessitate further empirical investigation.

Future Research Directions

Cognitive complaints are common in those with PTSD, but it is unclear which cognitive complaints would fall within the concept of "brain fog", and which would not. Furthermore, given the variation in the colloquial use of "brain fog",[20] it is also apparent that the patients reporting it may not be referring to the same phenomenon. To better elucidate "brain fog" in individuals with PTSD, employing a phenomenological qualitative approach (eg, with semi-structured in-depth interviews, focus groups) may provide an opportunity to initially understand the component parts of this construct and the features that differentiate it from subjective cognitive complaints in other conditions. Furthermore, learning about the lived experiences of individuals with PTSD and "brain fog" symptoms may provide insight into functional correlates and clinical areas to target.

A key finding of this review was that several factors may support "brain fog" among individuals with PTSD, including PTSD symptoms such as intrusions, avoidance, alterations to arousal and reactivity, and negative changes in thinking and mood, frequently comorbid mental and physical health conditions such as mood and anxiety disorders, sleep disorders, substance use disorders, cardiovascular disease, musculoskeletal conditions, chronic pain, migraine, and traumatic brain injury. Moreover, medications often prescribed for PTSD are also associated with "brain fog". It is unclear, however, whether these factors contribute to the same facets of "brain fog" and the mechanisms by which they may contribute to subjective cognitive complaints. Key targets for assessment and intervention may also vary with the factors contributing to "brain fog". To that end, future studies will require multiple modalities of inquiry. Given that there may be significant variability between individuals in the aspects of PTSD and related conditions contributing to "brain fog", case-analyses are likely to be an effective method of presenting key findings in the assessment of and intervention for "brain fog", which may be studied in larger follow-up investigations.

An important area of future inquiry is the relationship between objective cognitive deficits (as reflected by worse performance on cognitive test scores) and "brain fog". It is unclear why some studies have found a disassociation between objective and subjective cognitive function among individuals with PTSD,[32,33,38] while others have found the two to be correlated.[36,37] Building off qualitative approaches to better understand the component parts of "brain fog" in PTSD, future studies may use quantitative methods to understand how these parts relate to other factors influencing cognitive function. Linear model frameworks (eg, structural equation modeling) may be used to evaluate how objective cognitive deficits interact with factors influencing cognitive function in daily living (eg, dissociation, difficulties in emotion regulation), and how this relates to the experience of "brain fog". Understanding how different factors relate to both objective and subjective cognitive deficits may elucidate key targets for intervention approaches.

Validation of the scales and tools to screen for "brain fog" symptoms in PTSD may also be useful. Evaluating existing scales first rather than development of new, standalone tools may be a more pragmatic approach given content overlap with other scales, and recently developed scales explicitly centering on "brain fog" have not been validated alongside existing scales of subjective cognitive function. It will also be valuable to conduct factor analyses informed by insight gained from qualitative work to identify key factors thought to inform the subjective report of "brain fog", as there has been significant variation in the factor structure between recently developed scales. Inclusion of objective cognitive tests

in psychometric evaluation may be valuable to better characterize "brain fog", however based on findings to date, it is likely that the subjective reports of cognitive deficits may not correlate with cognitive test performance. This may reflect ceiling effects of certain tests, suggest that cognitive changes may be very subtle, or may only appear under "real world" situations in which greater cognitive flexibility, inhibition of distraction, or coping with negative self-appraisals or other sources of emotional distress may be layered on top of cognitive performance. As such, selecting tests that are more reflective of daily tasks would be important. Complex functional tasks (eg, Complex Task Performance Assessment)[153] emulating activities of daily living may be more ecologically valid and consistent with subjective cognitive complaints.

Recognizing that "brain fog" among individuals with PTSD reflects a subjective experience supported by several factors highlights the need for multidisciplinary collaboration by experts across fields in its investigation. Particularly, neuroimaging techniques (eg, functional magnetic resonance imaging) may be used to study the neuroscience of "brain fog" and alterations in regions supporting attentional and executive deficits likely to underlie subjective cognitive complaints. Alterations to large-scale brain networks have been posited to underly PTSD symptomology, including the default mode network (DMN involved in self-referential processing and memory, the salience network (SN) involved in detecting salient external and internal stimuli, and the central executive network (CEN) involved in executive functions including attentional control, planning, and decision making.[154–156] Among individuals with PTSD, DMN dysfunction is associated with an altered sense of self, SN dysfunction is associated with hypervigilance and hyperarousal, and CEN dysfunction is associated with cognitive dysfunction, including memory and executive function deficits.[156] As each of these alterations has been associated with "brain fog", future studies should employ neuroimaging techniques in conjunction with task-based measures as well as self-report measures to evaluate which alterations may support subjective cognitive deficits.

It is unclear whether certain conditions associated with PTSD and "brain fog" develop prior to or after the onset of PTSD. Inclusion of self-report measures of "brain fog" in longitudinal population studies will also be valuable in understanding in its development. An existing longitudinal study has shown that multimorbidity significantly increases likelihood of subjective cognitive complaints in the general population, partially explained by psychological factors.[113] Future longitudinal studies may further our understanding on the development of "brain fog" over time with the inclusion and analysis of measures of factors thought to support "brain fog" among individuals with PTSD.

Use of the term "brain fog" has little specificity to inform assessment or intervention currently, but it does provide sensitivity for further evaluation to define more specific complaints, cognitive challenges, and any clinical implications of these. Given that perceived cognitive problems, rather than test performance, mediate the relationship between PTSD and functional outcomes,[39] further exploration of how reported "brain fog" contributes to functional outcomes may provide insights into factors to consider during assessment and intervention.

Regarding assessment, evaluating the use of complex functional tasks for individuals with reported "brain fog" would assist in identifying whether "brain fog" uniquely contributes to failures in activities of daily living rather than comparatively simple tasks. Feedback following assessment is another valuable area of future study because, while feedback is associated with therapeutic benefits, this may not be the case for individuals with "brain fog" whose subjective cognitive functioning may not be captured on testing. That said, neuropsychologists experienced in working with mental health populations may nevertheless be able to use the feedback session to provide psychoeducation related to the impact of psychological factors in daily life that interfere with an individual's intact objective function as determined based on the assessment results. Future studies may evaluate the effect of feedback in these cases, as well as investigate whether there are any components of feedback that are especially valuable in improving outcomes (eg, psychoeducation on "brain fog", motivational interviewing, highlighting any strengths in cognitive profile).

Future directions for the treatment of "brain fog" is linked to its understanding and assessment, but large-scale evaluations of cognitive remediation therapies, particularly metacognitive training strategies will be of benefit as preliminary results indicate improvements to both subjective and objective measures of cognition, among other therapeutic effects in PTSD samples. As metacognitive cognitive training strategies such as GMT contain multiple components, such as psychoeducation, strategy training, and group participation, follow-up studies should investigate effective interventions for the most potent active ingredients. Abbreviated interventions containing the most potent components of these interventions should also be developed and evaluated for use in resource-limited settings. Another important consideration for evaluation of intervention for "brain fog" in PTSD will be the temporal order of cognitive

remediation in relation to other treatments. While standard interventions for PTSD are frequently associated with improvement in subjective cognition,[137] it is also the case that cognitive deficits in PTSD can reduce the effectiveness of PTSD interventions,[41–45] and problems with concentration can persist even after PTSD is in remission.[137]

Conclusions

"Brain fog" is a term used colloquially by clients with various health conditions, trauma and PTSD included, to refer to subjective experiences of deficits in cognitive functioning. There is a lack of consistent definition and operationalization of "brain fog" in the research literature, limiting current understandings of its phenomenology and implications for assessment and intervention. Given this, the current review synthesized available research on "brain fog" in those with PTSD and identified several themes including the association of cognitive complaints with PTSD symptoms, the role of physical and mental health comorbidities, considerations for assessment and intervention, as well as important areas of future investigation.

Acknowledgment

We would like to thank St. Joseph's Healthcare Hamilton medical librarian Kaitryn Campbell for her assistance in our literature search.

Disclosure

The author(s) report no conflicts of interest in this work.

References

1. APAP. Trauma- and Stressor-Related Disorders. In: *Diagnostic and Statistical Manual of Mental Disorders*. DSM Library. American Psychiatric Association Publishing; 2022.
2. Kessler RC, Aguilar-Gaxiola S, Alonso J, et al. Trauma and PTSD in the WHO World Mental Health Surveys. *Eur J Psychotraumatol*. 2017;8 (suppl 5):1353383. doi:10.1080/20008198.2017.1353383
3. Koenen KC, Ratanatharathorn A, Ng L, et al. Posttraumatic stress disorder in the World Mental Health Surveys. *Psychol Med*. 2017;47 (13):2260–2274. doi:10.1017/S0033291717000708
4. 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(5):748–766. doi:10.1037//0022-006x.68.5.748
5. Pineau H, Marchand A, Guay S. Specificity of Cognitive and Behavioral Complaints in Post-Traumatic Stress Disorder and Mild Traumatic Brain Injury. *Behav Sci*. 2015;5(1):43–58. doi:10.3390/bs5010043
6. Shin LM. Looking Through a Fog: what Persistent Derealization Can Teach Us About PTSD. *AJP*. 2022;179(9):599–600. doi:10.1176/appi.ajp.20220573
7. Steenkamp MM, Litz BT, Hoge CW, Marmar CR. Psychotherapy for military-related PTSD: a review of randomized clinical trials. *JAMA*. 2015;314(5):489–500. PMID: 26241600. doi:10.1001/jama.2015.8370
8. Kaseda ET, Levine AJ. Post-traumatic stress disorder: a differential diagnostic consideration for COVID-19 survivors. *Clini Neuropsychol*. 2020;34(7–8):1498–1514. doi:10.1080/13854046.2020.1811894
9. Asadi-Pooya AA, Akbari A, Emami A, et al. Long COVID syndrome-associated brain fog. *J Med Virol*. 2022;94(3):979–984. doi:10.1002/jmv.27404
10. Theoharides TC, Cholevas C, Polyzoidis K, Politis A. Long-COVID syndrome-associated brain fog and chemofog: luteolin to the rescue. *BioFactors*. 2021;47(2):232–241. doi:10.1002/biof.1726
11. Orfei MD, Porcari DE, D'Arcangelo S, Maggi F, Russignaga D, Ricciardi E. A New Look on Long-COVID Effects: the Functional Brain Fog Syndrome. *J Clin Med*. 2022;11(19):5529. doi:10.3390/jcm11195529
12. Jennings G, Monaghan A, Xue F, Duggan E, Romero-Ortuño R. Comprehensive Clinical Characterisation of Brain Fog in Adults Reporting Long COVID Symptoms. *J Clin Med*. 2022;11(12):3440. doi:10.3390/jcm11123440
13. Bell T, Crowe M, Novack T, Davis RD, Stavrinos D. Severity and correlates of brain fog in people with traumatic brain injury. *Research in Nursing & Health*. 2023;46(1):136–147. doi:10.1002/nur.22280
14. Dass R, Kalia M, Harris J, Packham T. Understanding the Experience and Impacts of Brain Fog in Chronic Pain: a Scoping Review. *Can J Pain*. 2023;7(1):2217865. doi:10.1080/24740527.2023.2217865
15. Ocon AJ. Caught in the thickness of brain fog: exploring the cognitive symptoms of Chronic Fatigue Syndrome. *Front Physiol*. 2013;4:63. doi:10.3389/fphys.2013.00063
16. Ross AJ, Medow MS, Rowe PC, Stewart JM. What is brain fog? An evaluation of the symptom in postural tachycardia syndrome. *Clin Auton Res*. 2013;23(6):305–311. doi:10.1007/s10286-013-0212-z
17. Teodoro T, Edwards MJ, Isaacs JD. A unifying theory for cognitive abnormalities in functional neurological disorders, fibromyalgia and chronic fatigue syndrome: systematic review. *J Neurol Neurosurg Psychiatry*. 2018;89(12):1308–1319. doi:10.1136/jnnp-2017-317823
18. Metcalf CA, Duffy KA, Page CE, Novick AM. Cognitive Problems in Perimenopause: a Review of Recent Evidence. *Curr Psychiatry Rep*. 2023;25(10):501–511. doi:10.1007/s11920-023-01447-3
19. Shrividya S, Joy M. Brain Fog among Perimenopausal Women: a Comparative Study. *J Int Women's Stud*. 2021;22(6):11–21.

20. McWhirter L, Smyth H, Hoeritzauer I, Couturier A, Stone J, Carson AJ. What is brain fog? *J Neurol Neurosurg Psychiatry*. 2023;94(4):321–325. doi:10.1136/jnnp-2022-329683
21. Raj V, Opie M, Arnold AC. Cognitive and Psychological Issues in Postural Tachycardia Syndrome. *Auton Neurosci*. 2018;215:46–55. doi:10.1016/j.autneu.2018.03.004
22. Premraj L, Kannapadi NV, Briggs J, et al. Mid and long-term neurological and neuropsychiatric manifestations of post-COVID-19 syndrome: a meta-analysis. *J Neurol Sci*. 2022;434:120162. doi:10.1016/j.jns.2022.120162
23. Gorenshtein A, Liba T, Leibovitch L, Stern S, Stern Y. Intervention modalities for brain fog caused by long-COVID: systematic review of the literature. *Neurol Sci*. 2024;45(7):2951–2968. doi:10.1007/s10072-024-07566-w
24. Markousis-Mavrogenis G, Bacopoulou F, Kolovou G, et al. Pathophysiology of cognitive dysfunction and the role of combined brain/heart magnetic resonance imaging (Review). *Exp Ther Med*. 2022;24(3):1–10. doi:10.3892/etm.2022.11506
25. McWhirter L, Ritchie C, Stone J, Carson A. Functional cognitive disorders: a systematic review. *Lancet Psychiatry*. 2020;7(2):191–207. doi:10.1016/S2215-0366(19)30405-5
26. Kverno K. Brain Fog: a Bit of Clarity Regarding Etiology, Prognosis, and Treatment. *J Psychosoc Nurs Ment Health Serv*. 2021;59(11):9–13. doi:10.3928/02793695-20211013-01
27. Elliott TR, Hsiao YY, Randolph K, et al. Efficient assessment of brain fog and fatigue: development of the Fatigue and Altered Cognition Scale (FACs). *PLoS One*. 2023;18(12):e0295593. doi:10.1371/journal.pone.0295593
28. Debowska A, Boduszek D, Ochman M, et al. Brain Fog Scale (BFS): scale development and validation. *Pers Individ Dif*. 2024;216:112427. doi:10.1016/j.paid.2023.112427
29. Atik D, Inel Manav A. A Scale Development Study: brain Fog Scale. *Psychiatry Danub*. 2023;35(1):73–79. doi:10.24869/psyd.2023.73
30. Knowles SR, Apputhurai P, Tye-Din JA. Development and validation of a brain fog scale for coeliac disease. *Aliment Pharmacol Ther*. 2024;59(10):1260–1270. doi:10.1111/apt.17942
31. Brück E, Larsson JW, Lasselin J, et al. Lack of clinically relevant correlation between subjective and objective cognitive function in ICU survivors: a prospective 12-month follow-up study. *Crit Care*. 2019;23(1):253. doi:10.1186/s13054-019-2527-1
32. Ord AS, Martindale SL, Jenks ER, Rowland JA. Subjective cognitive complaints and objective cognitive functioning in combat veterans: effects of PTSD and deployment mild TBI. *Appl Neuropsychol Adult*. 2023;1–7. doi:10.1080/23279095.2023.2280807
33. O'Neil ME, Laman-Maharg B, Schnurr PP, et al. Objective cognitive impairment and subjective cognitive problems in veterans initiating psychotherapy for posttraumatic stress disorder: an exploratory study. *Appl Neuropsychol Adult*. 2019;26(3):247–254. doi:10.1080/23279095.2017.1395334
34. Donnelly K, Donnelly JP, Warner GC, Kittleson CJ, King PR. Longitudinal study of objective and subjective cognitive performance and psychological distress in OEF/OIF Veterans with and without traumatic brain injury. *Clini Neuropsychol*. 2018;32(3):436–455. doi:10.1080/13854046.2017.1390163
35. Soble JR, Spanierman LB, Fitzgerald Smith J. Neuropsychological functioning of combat veterans with posttraumatic stress disorder and mild traumatic brain injury. *J Clin Experiment Neuropsychol*. 2013;35(5):551–561. doi:10.1080/13803395.2013.798398
36. Koso M, Sarač-Hadžihalilović A, Hansen S. Neuropsychological performance, psychiatric symptoms, and everyday cognitive failures in Bosnian ex-servicemen with posttraumatic stress disorder. *Rev Psychol*. 2012;19(2):131–139.
37. Mattson EK, Nelson NW, Sponheim SR, Disner SG. The impact of PTSD and mTBI on the relationship between subjective and objective cognitive deficits in combat-exposed veterans. *Neuropsychology*. 2019;33(7):913–921. doi:10.1037/neu0000560
38. Carlozzi NE, Reese-Melancon C, Thomas DG. Memory functioning in post-traumatic stress disorder: objective findings versus subjective complaints. *Stress Health*. 2011;27(3). doi:10.1002/smi.1355
39. Samuelson KW, Abadjian L, Jordan JT, Bartel A, Vasterling J, Seal K. The Association Between PTSD and Functional Outcome Is Mediated by Perception of Cognitive Problems Rather Than Objective Neuropsychological Test Performance. *J Trauma Stress*. 2017;30(5):521–530. doi:10.1002/jts.22223
40. Punski-Hoogervorst JL, Engel-Yeger B, Avital A. Attention deficits as a key player in the symptomatology of posttraumatic stress disorder: a review. *J Neurosci Res*. 2023;101(7):1068–1085. doi:10.1002/jnr.25177
41. Scott JC, Lynch KG, Cenkner DP, et al. Neurocognitive predictors of treatment outcomes in psychotherapy for comorbid PTSD and substance use disorders. *J Consulting Clin Psychol*. 2021;89(11):937–946. doi:10.1037/ccp0000693
42. Haaland KY, Sadek JR, Keller JE, Castillo DT. Neurocognitive Correlates of Successful Treatment of PTSD in Female Veterans. *J Int Neuropsychol Soc*. 2016;22(6):643–651. doi:10.1017/S1355617716000424
43. Nijdam MJ, de Vries GJ, Gersons BPR, Olff M. Response to Psychotherapy for Posttraumatic Stress Disorder: the Role of Pretreatment Verbal Memory Performance. *J Clin Psychiatry*. 2015;76(8):18178. doi:10.4088/JCP.14m09438
44. Scott JC, Harb G, Brownlow JA, Greene J, Gur RC, Ross RJ. Verbal memory functioning moderates psychotherapy treatment response for PTSD-Related nightmares. *Behav Res Ther*. 2017;91:24–32. doi:10.1016/j.brat.2017.01.004
45. Wild J, Gur RC. Verbal memory and treatment response in post-traumatic stress disorder. *Br J Psychiatry*. 2008;193(3):254–255. doi:10.1192/bjp.bp.107.045922
46. Groves SJ, Douglas KM, Porter RJ. A Systematic Review of Cognitive Predictors of Treatment Outcome in Major Depression. *Front Psychiatry*. 2018;9. doi:10.3389/fpsyt.2018.00382
47. Cox RC, Olatunji BO. Linking attentional control and PTSD symptom severity: the role of rumination. *Cognitive Behaviour Therapy*. 2017;46(5):421–431. doi:10.1080/16506073.2017.1286517
48. Scott JC, Matt GE, Wrocklage KM, et al. A quantitative meta-analysis of neurocognitive functioning in posttraumatic stress disorder. *Psychol Bull*. 2015;141(1):105–140. doi:10.1037/a0038039
49. Aupperle RL, Allard CB, Grimes EM, et al. Dorsolateral Prefrontal Cortex Activation During Emotional Anticipation and Neuropsychological Performance in Posttraumatic Stress Disorder. *Arch Gen Psychiatry*. 2012;69(4):360–371. doi:10.1001/archgenpsychiatry.2011.1539
50. Rehman Y, Zhang C, Ye H, et al. The extent of the neurocognitive impairment in elderly survivors of war suffering from PTSD: meta-analysis and literature review. *AIMS Neurosci*. 2020;8(1):47–73. doi:10.3934/Neuroscience.2021003
51. Schuitevoerder S, Rosen JW, Twamley EW, et al. A meta-analysis of cognitive functioning in older adults with PTSD. *J Anxiet Disorders*. 2013;27(6):550–558. doi:10.1016/j.janxdis.2013.01.001

52. McKinnon MC, Boyd JE, Frewen PA, et al. A review of the relation between dissociation, memory, executive functioning and social cognition in military members and civilians with neuropsychiatric conditions. *Neuropsychologia*. 2016;90:210–234. doi:10.1016/j.neuropsychologia.2016.07.017
53. Hayes JP, Hayes SM, Mikedis AM. Quantitative meta-analysis of neural activity in posttraumatic stress disorder. *Biol Mood Anxiety Disord*. 2012;2(1):9. doi:10.1186/2045-5380-2-9
54. Li J, Yu L, Long Z, Li Y, Cao F. Perceived cognitive impairment in Chinese patients with breast cancer and its relationship with post-traumatic stress disorder symptoms and fatigue: perceived cognitive impairment in Chinese patients with breast cancer. *Psycho-Oncology*. 2015;24(6):676–682. doi:10.1002/pon.3710
55. Boyd JE, Protopopescu A, O'Connor C, et al. Dissociative symptoms mediate the relation between PTSD symptoms and functional impairment in a sample of military members, veterans, and first responders with PTSD. *Eur J Psychotraumatol*. 2018;9(1):1463794. doi:10.1080/20008198.2018.1463794
56. Park AH, Patel H, Mirabelli J, et al. Machine learning models predict PTSD severity and functional impairment: a personalized medicine approach for uncovering complex associations among heterogeneous symptom profiles. *Psychological Trauma: Theory Res Pract Pol*. 2023;17(2):372–386. doi:10.1037/tra0001602
57. Gold AI, Meyerowitz BE, Thames AD. The influence of posttraumatic stress on memory complaints in neuropsychological settings. *Psychological Trauma: Theory Res Pract Pol*. 2021;13(2):240–248. doi:10.1037/tra0000993
58. Bregman-Hai N, Soffer-Dudek N. Posttraumatic symptoms and poor sleep are independent pathways to agency disruptions and dissociation: a longitudinal study with objective sleep assessment. *JmPsychopathol Clin Sci*. 2024;133(2):192–207. doi:10.1037/abn0000885
59. Günak MM, Ebrahimi OV, Pietrzak RH, Fried EI. Using network models to explore the associations between posttraumatic stress disorder symptoms and subjective cognitive functioning. *J Anxiet Disorders*. 2023;99:102768. doi:10.1016/j.janxdis.2023.102768
60. Roemer L, Litz BT, Orsillo SM, Wagner AW. A Preliminary Investigation of the Role of Strategic Withholding of Emotions in PTSD. *J Trauma Stress*. 2001;14(1):149–156. doi:10.1023/A:1007895817502
61. Dillon DG, Ritchey M, Johnson BD, LaBar KS. Dissociable effects of conscious emotion regulation strategies on explicit and implicit memory. *Emotion*. 2007;7(2):354–365. doi:10.1037/1528-3542.7.2.354
62. Hayes JP, VanElzakker MB, Shin LM. Emotion and cognition interactions in PTSD: a review of neurocognitive and neuroimaging studies. *Front Integr Neurosci*. 2012;6. doi:10.3389/fnint.2012.00089.
63. Kim SH, Hamann S. The effect of cognitive reappraisal on physiological reactivity and emotional memory. *Int J Psychophysiol*. 2012;83(3):348–356. doi:10.1016/j.ijpsycho.2011.12.001
64. Moore SA, Zoellner LA. The Effects of Expressive and Experiential Suppression on Memory Accuracy and Memory Distortion in Women with and without PTSD. *J Experiment Psychopathol*. 2012;3(3):368–392. doi:10.5127/jep.024411
65. Park AH, Protopopescu A, Pogue ME, et al. Dissociative symptoms and emotion dysregulation contribute to alterations in cognitive performance among military members, Veterans, and public safety personnel with a presumptive diagnosis of post-traumatic stress disorder. 2024. https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=&cad=rja&uact=8&ved=2ahUKEwjo0LPgtvaLAxXpga8BHcSeK1kQFnoECBYQAQ&url=https%3A%2F%2Fmacsphere.mcmaster.ca%2Fbitstream%2F11375%2F30202%2F2%2FPark_Anna_H_2024August_PhD.pdf&usg=AOvVaw3osjjdDNSOw2YMvcS0ayVM&cshid=1741297587489092&opi=89978449
66. Qassem T, Aly-ElGabry D, Alzarouni A, Abdel-Aziz K, Arnone D. Psychiatric Co-Morbidities in Post-Traumatic Stress Disorder: detailed Findings from the Adult Psychiatric Morbidity Survey in the English Population. *Psychiatr Q*. 2021;92(1):321–330. doi:10.1007/s11126-020-09797-4
67. 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(6):593–602. doi:10.1001/archpsyc.62.6.593
68. Shih RA, Schell TL, Hambarsoomian K, Belzberg H, Marshall GN. Prevalence of posttraumatic stress disorder and major depression after trauma center hospitalization. *J Trauma*. 2010;69(6):1560–1566. doi:10.1097/TA.0b013e3181e59c05
69. Dannehl K, Rief W, Euteneuer F. Childhood adversity and cognitive functioning in patients with major depression. *Child Abuse Negl*. 2017;70:247–254. doi:10.1016/j.chiabu.2017.06.013
70. Miller MW, Wolf EJ, Reardon A, Greene A, Ofrat S, McInerney S. Personality and the latent structure of PTSD comorbidity. *J Anxiety Disord*. 2012;26(5):599–607. doi:10.1016/j.janxdis.2012.02.016
71. Spinhoven P, Penninx BW, van Hemert AM, de Rooij M, Elzinga BM. Comorbidity of PTSD in anxiety and depressive disorders: prevalence and shared risk factors. *Child Abuse Negl*. 2014;38(8):1320–1330. doi:10.1016/j.chiabu.2014.01.017
72. Rock PL, Roiser JP, Riedel WJ, Blackwell AD. Cognitive impairment in depression: a systematic review and meta-analysis. *Psychological Medicine*. 2014;44(10):2029–2040. doi:10.1017/S0033291713002535
73. APAP. Depressive Disorders. In: *Diagnostic and Statistical Manual of Mental Disorders*. DSM Library. American Psychiatric Association Publishing; 2022.
74. Brown WA. Treatment response in melancholia. *Acta Psychiatr Scand*. 2007;115(s433):125–129. doi:10.1111/j.1600-0447.2007.00970.x
75. Parker G. Ask depressed patients about brain fog to ensure melancholia is not mist. *Australas Psychiatry*. 2022;30(5):612–614. doi:10.1177/10398562221104402
76. Zaninotto L, Solmi M, Veronese N, et al. A meta-analysis of cognitive performance in melancholic versus non-melancholic unipolar depression. *J Affective Disorders*. 2016;201:15–24. doi:10.1016/j.jad.2016.04.039
77. Constans JI, Lenhoff K, McCarthy M. Depression Subtyping in PTSD Patients. *Ann Clin Psychiatry*. 1997;9(4):235–240. doi:10.1023/A:1022304410404
78. Dittrich WH, Johansen T. Cognitive deficits of executive functions and decision-making in obsessive-compulsive disorder. *Scandinavian J Psychology*. 2013;54(5):393–400. doi:10.1111/sjop.12066
79. Fujii Y, Kitagawa N, Shimizu Y, et al. Severity of generalized social anxiety disorder correlates with low executive functioning. *Neurosci Lett*. 2013;543:42–46. doi:10.1016/j.neulet.2013.02.059
80. APAP. Anxiety Disorders. In: *Diagnostic and Statistical Manual of Mental Disorders*. DSM Library. American Psychiatric Association Publishing; 2022.
81. Eysenck MW, Derakshan N, Santos R, Calvo MG. Anxiety and cognitive performance: attentional control theory. *Emotion*. 2007;7(2):336–353. doi:10.1037/1528-3542.7.2.336

82. Leung P, Li SH, Graham BM. The relationship between repetitive negative thinking, sleep disturbance, and subjective fatigue in women with Generalized Anxiety Disorder. *British J Clinic Psychol*. 2022;61(3):666–679. doi:10.1111/bjc.12356
83. Barkley RA, Knouse LE, Murphy KR. Correspondence and disparity in the self- and other ratings of current and childhood ADHD symptoms and impairment in adults with ADHD. *Psychol Assess*. 2011;23(2):437–446. doi:10.1037/a0022172
84. Sibley MH. Empirically-informed guidelines for first-time adult ADHD diagnosis. *J Clin Exp Neuropsychol*. 2021;43(4):340–351. doi:10.1080/13803395.2021.1923665
85. Hallion LS, Steinman SA, Kusmierski SN. Difficulty concentrating in generalized anxiety disorder: an evaluation of incremental utility and relationship to worry. *J Anxiet Disorders*. 2018;53:39–45. doi:10.1016/j.janxdis.2017.10.007
86. Comer JS, Pincus DB, Hofmann SG. Generalized anxiety disorder and the proposed associated symptoms criterion change for DSM-5 in a treatment-seeking sample of anxious youth: research Article: DSM-5 GAD Associated Symptoms. *Depress Anxiety*. 2012;29(12):994–1003. doi:10.1002/da.21999
87. Michelson L, June K, Vives A, Testa S, Marchione N. The role of trauma and dissociation in cognitive-behavioral psychotherapy outcome and maintenance for panic disorder with agoraphobia. *Behav Res Ther*. 1998;36(11):1011–1050. doi:10.1016/S0005-7967(98)00073-4
88. Van Ameringen M, Mancini C, Patterson B, Boyle MH. Post-Traumatic Stress Disorder in Canada. *CNS Neurosci. Ther.* 2008;14(3):171–181. doi:10.1111/j.1755-5949.2008.00049.x
89. Pietrzak RH, Goldstein RB, Southwick SM, Grant BF. Prevalence and Axis I comorbidity of full and partial posttraumatic stress disorder in the United States: results from Wave 2 of the National Epidemiologic Survey on Alcohol and Related Conditions. *J Anxiet Disorders*. 2011;25(3):456–465. doi:10.1016/j.janxdis.2010.11.010
90. Bruijnen CJWH, Dijkstra BAG, Walvoort SJW, et al. Prevalence of cognitive impairment in patients with substance use disorder. *Drug Alcohol Rev*. 2019;38(4):435–442. doi:10.1111/dar.12922
91. Toledo-Fernández A, Marín-Navarrete R, Villalobos-Gallegos L, Salvador-Cruz J, Benjet C, Roncero C. Exploring the prevalence of substance-induced neurocognitive disorder among polysubstance users, adding subjective and objective evidence of cognitive impairment. *Psychiatry Res*. 2020;288:112944. doi:10.1016/j.psychres.2020.112944
92. Tripp JC, Worley MJ, Straus E, Angkaw AC, Trim RS, Norman SB. Bidirectional relationship of posttraumatic stress disorder (PTSD) symptom severity and alcohol use over the course of integrated treatment. *Psychol Addict Behav*. 2020;34(4):506–511. doi:10.1037/adb0000564
93. Patel H, O'Connor C, Andrews K, Amlung M, Lanius R, McKinnon MC. Dissociative symptomatology mediates the relation between posttraumatic stress disorder severity and alcohol-related problems. *Alcoholism Clin Exp Res*. 2022;46(2):289–299. doi:10.1111/acer.14764
94. Campbell N, Boustani M, Limbil T, et al. The cognitive impact of anticholinergics: a clinical review. *Clin Interventions Aging*. 2009;4:225–233. doi:10.2147/cia.s5358
95. Reallon E, Gervais F, Moutet C, et al. Impact of cumulative exposure to anticholinergic and sedative drugs on cognition in older adults: a memory clinic cohort study. *Alzheimer's Res Ther*. 2024;16(1):163. doi:10.1186/s13195-024-01530-8
96. Risacher SL, McDonald BC, Tallman EF, et al. Association Between Anticholinergic Medication Use and Cognition, Brain Metabolism, and Brain Atrophy in Cognitively Normal Older Adults. *JAMA Neurol*. 2016;73(6):721–732. doi:10.1001/jamaneurol.2016.0580
97. Stewart SA. The effects of benzodiazepines on cognition. *J Clin Psychiatry*. 2005;66(2):9–13.
98. Allott K, Yuen HP, Baldwin L, et al. Effects of risperidone/paliperidone versus placebo on cognitive functioning over the first 6 months of treatment for psychotic disorder: secondary analysis of a triple-blind randomised clinical trial. *Transl Psychiatry*. 2023;13(1):1–9. doi:10.1038/s41398-023-02501-7
99. Husa AP, Moilanen J, Murray GK, et al. Lifetime antipsychotic medication and cognitive performance in schizophrenia at age 43 years in a general population birth cohort. *Psychiatry Res*. 2017;247:130–138. doi:10.1016/j.psychres.2016.10.085
100. Moncrieff J, Cohen D, Mason JP. The subjective experience of taking antipsychotic medication: a content analysis of Internet data. *Acta Psychiatrica Scandinavica*. 2009;120(2):102–111. doi:10.1111/j.1600-0447.2009.01356.x
101. Holder N, Woods A, Neylan TC, et al. Trends in Medication Prescribing in Patients With PTSD From 2009 to 2018: a National Veterans Administration Study. *J Clin Psychiatry*. 2021;82(3):32806. doi:10.4088/JCP.20m13522
102. Reinhard MA, Seifert J, Greiner T, Toto S, Bleich S, Grohmann R. Pharmacotherapy of 1,044 inpatients with posttraumatic stress disorder: current status and trends in German-speaking countries. *Eur Arch Psychiatry Clin Neurosci*. 2021;271(6):1065–1076. doi:10.1007/s00406-020-01223-x
103. El-Gabalawy R, Blaney C, Tsai J, Sumner JA, Pietrzak RH. Physical health conditions associated with full and subthreshold PTSD in U.S. military veterans: results from the National Health and Resilience in Veterans Study. *J Affective Disorders*. 2018;227:849–853. doi:10.1016/j.jad.2017.11.058
104. Loignon A, Ouellet MC, Belleville G. A Systematic Review and Meta-analysis on PTSD Following TBI Among Military/Veteran and Civilian Populations. *J Head Trauma Rehab*. 2020;35(1):E21. doi:10.1097/HTR.0000000000000514
105. Ryder AL, Azcarate PM, Cohen BE. PTSD and Physical Health. *Curr Psychiatry Rep*. 2018;20(12):116. doi:10.1007/s11920-018-0977-9
106. Siqveland J, Hussain A, Lindstrøm JC, Ruud T, Hauff E. Prevalence of Posttraumatic Stress Disorder in Persons with Chronic Pain: a Meta-analysis. *Front Psychiatry*. 2017;8. doi:10.3389/fpsyt.2017.00164.
107. Sommer JL, El-Gabalawy R, Mota N. Understanding the association between posttraumatic stress disorder characteristics and physical health conditions: a population-based study. *J Psychosomatic Res*. 2019;126:109776. doi:10.1016/j.jpsychores.2019.109776
108. O'Toole BI, Catts SV. Trauma, PTSD, and physical health: an epidemiological study of Australian Vietnam veterans. *J Psychosom Res*. 2008;64(1):33–40. doi:10.1016/j.jpsychores.2007.07.006
109. Chu HT, Liang CS, Lee JT, et al. Subjective cognitive complaints and migraine characteristics: a cross-sectional study. *Acta Neurol Scand*. 2020;141(4):319–327. doi:10.1111/ane.13204
110. Hill NL, Bhargava S, Brown MJ, et al. Cognitive complaints in age-related chronic conditions: a systematic review. *PLoS One*. 2021;16(7):e0253795. doi:10.1371/journal.pone.0253795
111. Stillman AM, Madigan N, Torres K, Swan N, Alexander MP. Subjective Cognitive Complaints in Concussion. *J Neurotrauma*. 2020;37(2):305–311. doi:10.1089/neu.2018.5925
112. Pidal-Miranda M, González-Villar AJ, Carrillo-de-la-Peña MT, Andrade E, Rodríguez-Salgado D. Broad cognitive complaints but subtle objective working memory impairment in fibromyalgia patients. *PeerJ*. 2018;6:e5907. doi:10.7717/peerj.5907

113. Tardy M, Gonthier R, Barthelemy JC, Roche F, Crawford-Achour E. Subjective sleep and cognitive complaints in 65 year old subjects: a significant association. The PROOF cohort. *J Nutr Health Aging*. 2015;19(4):424–430. doi:10.1007/s12603-014-0547-8
114. Wolfe F, Rasker JJ, ten Klooster P, Häuser W. Subjective Cognitive Dysfunction in Patients With and Without Fibromyalgia: prevalence, Predictors, Correlates, and Consequences. *Cureus*. 2021;13(12):e20351. doi:10.7759/cureus.20351
115. Koyanagi A, Smith L, Shin JI, et al. Multimorbidity and subjective cognitive complaints: findings from 48 low- and middle-income countries of the world health survey 2002–2004. *J Alzheimers Dis*. 2021;81(4):1737–1747. doi:10.3233/JAD-201592
116. Martindale SL, Morissette SB, Rowland JA, Dolan SL. Sleep quality affects cognitive functioning in returning combat veterans beyond combat exposure, PTSD, and mild TBI history. *Neuropsychology*. 2017;31(1):93–104. doi:10.1037/neu0000312
117. Mondelli V, Vernon AC, Turkheimer F, Dazzan P, Pariante CM. Brain microglia in psychiatric disorders. *Lancet Psychiatry*. 2017;4(7):563–572. doi:10.1016/S2215-0366(17)30101-3
118. Lee DH, Lee JY, Hong DY, et al. Neuroinflammation in Post-Traumatic Stress Disorder. *Biomedicines*. 2022;10(5):953. doi:10.3390/biomedicines10050953
119. Van Patten R, Nguyen TT, Mahmood Z, et al. Physical and Mental Health Characteristics of 2,962 Adults With Subjective Cognitive Complaints. *Int J Aging Hum Dev*. 2022;94(4):459–477. doi:10.1177/00914150211026548
120. Cations M, Cook JM, Nichter B, Esterlis I, Pietrzak RH. Subjective cognitive difficulties and posttraumatic stress disorder interact to increase suicide risk among middle-aged and older US military veterans. *Int Psychogeriatrics*. 2023;2023:1–9. doi:10.1017/S1041610222001053
121. Susanty E, Sijbrandij M, Srisayekti W, Suparman Y, Huizink AC. The Effectiveness of Eye Movement Desensitization for Post-traumatic Stress Disorder in Indonesia: a Randomized Controlled Trial. *Front Psychol*. 2022;13:845520. doi:10.3389/fpsyg.2022.845520
122. Winders SJ, Murphy O, Looney K, O'Reilly G. Self-compassion, trauma, and posttraumatic stress disorder: a systematic review. *Clin Psychol Psychother*. 2020;27(3):300–329. doi:10.1002/cpp.2429
123. Chiu CD, Tseng MCM, Chien YL, et al. Cumulative traumatization associated with pathological dissociation in acute psychiatric inpatients. *Psychiatry Res*. 2015;230(2):406–412. doi:10.1016/j.psychres.2015.09.028
124. Schimmenti A. The developmental roots of dissociation: a multiple mediation analysis. *Psychoanal Psychol*. 2017;34(1):96–105. doi:10.1037/pap0000084
125. Singh A, Zeig-Owens R, Rabin L, et al. PTSD and Depressive Symptoms as Potential Mediators of the Association between World Trade Center Exposure and Subjective Cognitive Concerns in Rescue/Recovery Workers. *Int J Environ Res Public Health*. 2020;17(16):5683. doi:10.3390/ijerph17165683
126. Stein CR, Cooney ML, Frank B, Bender HA, Winkel G, Lucchini RG. Mental health mediators of subjective cognitive concerns among World Trade Center responders. *J Psychiatr Res*. 2021;140:187–196. doi:10.1016/j.jpsychires.2021.05.081
127. Thomas-Antérion C, Honoré-Masson S, Laurent B. The cognitive complaint interview (CCI). *Psychogeriatrics*. 2006;6(s1). doi:10.1111/j.1479-8301.2006.00129.x
128. Ventura J, Reise SP, Keefe RSE, et al. The Cognitive Assessment Interview (CAI): development and Validation of an Empirically Derived, Brief Interview-Based Measure of Cognition. *Schizophr Res*. 2010;121(1–3):24–31. doi:10.1016/j.schres.2010.04.016
129. Lynch S, Ferrando SJ, Dornbush R, Shahar S, Smiley A, Klepacz L. Screening for brain fog: is the Montreal cognitive assessment an effective screening tool for neurocognitive complaints post-COVID-19? *Gen Hosp Psychiatry*. 2022;78:80–86. doi:10.1016/j.genhosppsych.2022.07.013
130. Nasreddine ZS, Phillips NA, Bédirian V, et al. The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. *J Am Geriatr Soc*. 2005;53(4):695–699. doi:10.1111/j.1532-5415.2005.53221.x
131. Groenman AP, van der Werf S, Geurts HM. Subjective cognition in adults with common psychiatric classifications; a systematic review. *Psychiatry Res*. 2022;308:114374. doi:10.1016/j.psychres.2021.114374
132. Gioia GA, Isquith PK, Guy SC, Kenworthy L. TEST REVIEW Behavior Rating Inventory of Executive Function. *Child Neuropsychol*. 2000;6(3):235–238. doi:10.1076/chin.6.3.235.3152
133. Broadbent DE, Cooper PF, FitzGerald P, Parkes KR. The Cognitive Failures Questionnaire (CFQ) and its correlates. *Br J Clin Psychol*. 1982;21(1):1–16. doi:10.1111/j.2044-8260.1982.tb01421.x
134. Li Y, Dong F, Cao F, Cui N, Li J, Long Z. Poly-victimization and executive functions in junior college students. *Scandinavian J Psychology*. 2013;54(6):485–492. doi:10.1111/sjop.12083
135. Yang R, Xiang YT, Shuai L, et al. Executive function in children and adolescents with posttraumatic stress disorder 4 and 12 months after the Sichuan earthquake in China. *J Child Psychol Psychiatr*. 2014;55(1):31–38. doi:10.1111/jcpp.12089
136. Lantrip C, Szabo YZ, Pazienza S, Benge J. Associations of childhood trauma and executive functioning in everyday life of those with subjective cognitive complaints. *Appl Neuropsychol Adult*. 2023;30(1):101–109. doi:10.1080/23279095.2021.1913738
137. Larsen SE, Fleming CJE, Resick PA. Residual symptoms following empirically supported treatment for PTSD. *Psychol Trauma*. 2019;11(2):207–215. doi:10.1037/tra0000384
138. Rosado DL, Buehler S, Botbol-Berman E, et al. Neuropsychological feedback services improve quality of life and social adjustment. *Clini Neuropsychol*. 2018;32(3):422–435. doi:10.1080/13854046.2017.1400105
139. Katzman MA, Bleau P, Blier P, Chokka P, Kjernisted K, Van Ameringen M. Canadian clinical practice guidelines for the management of anxiety, posttraumatic stress and obsessive-compulsive disorders. *BMC Psychiatry*. 2014;14(Suppl 1):S1. doi:10.1186/1471-244X-14-S1-S1
140. Krysta K, Krzystanek M, Janas-Kozik M, Klasik A, Krupka-Matuszczyk I. Impact of pharmacological and psychological treatment methods of depressive and anxiety disorders on cognitive functioning. *J Neural Transm*. 2015;122(1):101–110. doi:10.1007/s00702-014-1282-3
141. Krishnan K, Lin Y, Prewitt KRM, Potter DA. Multidisciplinary approach to brain fog and related persisting symptoms post COVID-19. *J Health Service Psychol*. 2022;48(1):31–38. doi:10.1007/s42843-022-00056-7
142. Kim EJ, Bahk YC, Oh H, Lee WH, Lee JS, Choi KH. Current Status of Cognitive Remediation for Psychiatric Disorders: a Review. *Front Psychiatry*. 2018;9:461. doi:10.3389/fpsyt.2018.00461
143. Cicerone KD, Goldin Y, Ganci K, et al. Evidence-Based Cognitive Rehabilitation: systematic Review of the Literature From 2009 Through 2014. *Arch Phys Med Rehabil*. 2019;100(8):1515–1533. doi:10.1016/j.apmr.2019.02.011
144. Levine B, Schweizer TA, O'Connor C, et al. Rehabilitation of executive functioning in patients with frontal lobe brain damage with goal management training. *Front Hum Neurosci*. 2011;5:9. doi:10.3389/fnhum.2011.00009

145. Boyd JE, Sanger BD, Cameron DH, et al. A Pilot Study Assessing the Effects of Goal Management Training on Cognitive Functions among Individuals with Major Depressive Disorder and the Effect of Post-Traumatic Symptoms on Response to Intervention. *Brain Sci*. 2022;12(7):864. doi:10.3390/brainsci12070864
146. Levine B, Robertson IH, Clare L, et al. Rehabilitation of executive functioning: an experimental-clinical validation of goal management training. *J Int Neuropsychol Soc*. 2000;6(3):299–312. doi:10.1017/s1355617700633052
147. Protopopescu A, O'Connor C, Cameron D, Boyd JE, Lanius RA, McKinnon MC. A Pilot Randomized Controlled Trial of Goal Management Training in Canadian Military Members, Veterans, and Public Safety Personnel Experiencing Post-Traumatic Stress Symptoms. *Brain Sciences*. 2022;12(3):377. doi:10.3390/brainsci12030377
148. Boyd JE, O'Connor C, Protopopescu A, et al. An Open-Label Feasibility Trial Examining the Effectiveness of a Cognitive Training Program, Goal Management Training, in Individuals With Posttraumatic Stress Disorder. *Chronic Stress*. 2019;3:2470547019841599. doi:10.1177/2470547019841599
149. Novakovic-Agopian T, Posecion L, Kornblith E, et al. Goal-Oriented Attention Self-Regulation Training Improves Executive Functioning in Veterans with Post-Traumatic Stress Disorder and Mild Traumatic Brain Injury. *J Neurotrauma*. 2021;38(5):582–592. doi:10.1089/neu.2019.6806
150. Bomyea J, Stein MB, Lang AJ. Interference control training for PTSD: a randomized controlled trial of a novel computer-based intervention. *J Anxiet Disorders*. 2015;34:33–42. doi:10.1016/j.janxdis.2015.05.010
151. Fonzo GA, Fine NB, Wright RN, et al. Internet-delivered computerized cognitive & affective remediation training for the treatment of acute and chronic posttraumatic stress disorder: two randomized clinical trials. *J Psychiatr Res*. 2019;115:82–89. doi:10.1016/j.jpsychires.2019.05.007
152. Saunders N, Downham R, Turman B, et al. Working memory training with tDCS improves behavioral and neurophysiological symptoms in pilot group with post-traumatic stress disorder (PTSD) and with poor working memory. *Neurocase*. 2015;21(3):271–278. doi:10.1080/13554794.2014.890727
153. Wolf TJ, Morrison T, Matheson L. Initial development of a work-related assessment of dysexecutive syndrome: the Complex Task Performance Assessment. *Work*. 2008;31(2):221–228.
154. Menon V. Large-scale brain networks and psychopathology: a unifying triple network model. *Trends Cognit Sci*. 2011;15(10):483–506. doi:10.1016/j.tics.2011.08.003
155. Pankey BS, Riedel MC, Cowan I, et al. Extended functional connectivity of convergent structural alterations among individuals with PTSD: a neuroimaging meta-analysis. *Behav Brain Funct*. 2022;18(1):9. doi:10.1186/s12993-022-00196-2
156. Lanius RA, Frewen PA, Tursich M, Jetly R, McKinnon MC. Restoring large-scale brain networks in PTSD and related disorders: a proposal for neuroscientifically-informed treatment interventions. *Eur J Psychotraumatol*. 2015;6(1):27313. doi:10.3402/ejpt.v6.27313