Working Memory Impairment in Military Personnel

Affected Brain Areas, Cortical and Subcortical Regions, and Cognitive Functions

RICCARDO MAIRA

OCTOBER 2025 | riccardomaira.contact@gmail.com

Contents

Abstract

Working memory (WM) impairment has been known to affect cognitive function and impact the quality of life of affected individuals. Among the most prevalent causes for WM impairment affecting military personnel, TBI and mTBI have been the most pervasive and consistently on the rise worldwide. Hence, current research efforts have been made to investigate relevant brain areas, cortical and subcortical regions, and cognitive functions related to WM impairment in military personnel. The aim of this review is to identify brain areas, cortical and subcortical regions, and cognitive functions related to WM impairment in military personnel from a selection of recent studies in order to inform novel clinical applications, including cognitive rehabilitation methodologies, for the benefit of individuals suffering from WM impairment. Affected brain areas included the VLPFC, DLPFC, VMPFC, OFC, FP, hippocampus, dorsolateral and orbitofrontal striatal circuits, and caudate nucleus. Identified disruptions to cognitive functions included slower reaction times, lower WM performance and accuracy in delayed visual and verbal abilities, and worse neuropsychological performance. Future research should focus on the cross-examination of underexplored WM impairment causes and affected brain areas and cognitive functions.

Keywords: military personnel, working memory, impairment, traumatic brain injury, cognitive impairment, prefrontal cortex, hippocampus, caudate nucleus, striatal circuits

Introduction

Background

Working memory (WM) is defined as the system of retention in which transient information is stored to aid complex cognitive processes (e.g., learning, reasoning, problem-solving, planning, information processing; Baddeley, 1992; Cowan, 2014). WM impairments have been known to affect cognitive processes (Christodoulou et al., 2001), which can significantly impact quality of life of affected individuals and are often caused by brain trauma. The most common causes for WM impairment in military personnel include traumatic brain injury (TBI), defined by the National Institute of Neurological Disorders and Stroke (NINDS) as a type of brain injury caused by abrupt damage to the brain, and mild traumatic brain injury (mTBI), defined as a subset of TBI in which the survivor may or may not retain consciousness for a few minutes immediately after the traumatic episode, possibly experiencing a set of neurological symptoms common to TBI and mTBI (e.g., confusion, fatigue, mood and behavioral changes, issues related to attention, thinking, concentration, and/or memory; National Academies of Sciences, Engineering, and Medicine et al., 2019).

The Defense Health Agency (DHA) and the Department of Defense (DOD) provide further indications for mTBI, specifically in the context and experience of military personnel, including the likelihood to experience disorientation, confusion, and/or memory loss lasting less than 24 hours and unconsciousness lasting up to 30 minutes (DOD Worldwide TBI Numbers, 2024b). In the same report, DHA further indicates that computed tomography (CT) is not recommended for patients with mTBI (also see Smith, 2012), primarily due to its inability to represent injury-related functional changes in the brain. Therefore, functional magnetic resonance imaging (fMRI), a neuroimaging method capable of recording functional changes, has been deemed the method of choice for evaluating patients with mTBI (Eierud et al., 2014), TBI (O'Neill et al., 2017), and other WM-related conditions (e.g., schizophrenia; Ding et al., 2024).

With the development of advanced neuroimaging techniques (e.g., high resolution structural MRI, dynamic susceptibility contrast MRI; Koerte et al., 2016), the diagnosis of mTBI and investigation of mTBI-affected WM cortical regions and networks have become more sophisticated (Smith et al., 2019), particularly as they pertain to military-related mTBI. Such developments have allowed the identification of microstructural aberrations after mTBI, including pathological ones pertaining to glymphatic functions which may lead to neurodegeneration (Kim et al., 2023). However, such techniques may not yet be widely accessible; therefore, fMRI is still considered the neuroimaging method of choice for the diagnosis and study of TBI and mTBI (Medaglia, 2017). Magnetoencephalography (MEG) has

also been particularly useful in the detection of neural alterations related to cognitive dysfunction (Da Costa et al., 2015), relevant biomarkers (Allen et al., 2021; Dimitriadis et al., 2015; Huang et al., 2020), and network variations in patients with mTBI (Alhourani et al., 2016).

Brain areas, cortical and subcortical regions, and neural networks which contribute to the functioning of WM—and hence key areas of neuroimaging investigation concerning WM impairment, TBI, and mTBI—include the frontoparietal network (Wallis et al., 2015), dorsolateral prefrontal cortex (DLPFC; Balconi, 2013; Barbey et al., 2013), frontal pole (FP), ventromedial prefrontal cortex (VMPFC; Yin et al., 2021), ventrolateral prefrontal cortex (VLPFC; Segal & Elkana, 2023), thalamus (Essad et al., 2016; Sours et al., 2015), orbitofrontal cortex (OFC; Barbey et al., 2011; Johnson et al., 2022), hippocampus (Hung et al., 2022; Leszczynski, 2011), amygdala (Levens et al., 2011), basal ganglia (Baier et al., 2010; Voytek & Knight, 2010; Witt, 2021), cerebellum (Chai et al., 2018), caudate nucleus (Grahn et al., 2009; Lewis et al., 2004), and left anterior prefrontal cortex (Mallas et al., 2020). Overall, the prefrontal cortex (PFC) has been deemed critical to the functioning of WM (Eriksson et al., 2015; Hoskison et al., 2009). White matter changes, as they relate to alterations in the WM functional network, have also been identified as consequential to WM impairment and mTBI (Chung et al., 2019; S. Huang et al., 2022; Palacios et al., 2012).

Rationale

In active-duty military personnel worldwide, mTBI has been reported as the most prevalent form of TBI. According to the most recent DHA report, as of February 2024, mTBI has accounted for 82.2% of total TBIs worldwide between the years 2000 and 2023, amounting to 410,798 cases across army, navy, air force, and marines divisions (DOD Worldwide TBI Numbers, 2024a). The same agency also released another report, disclosing that mTBIs accounted for 83.1% of all TBIs in the year 2023, further indicating a rise in mTBI.

TBI and mTBI have been known to particularly affect WM (Chen et al., 2012) and related areas of executive functioning (Arciniega et al., 2021; Kumar et al., 2013). This impacts cognitive functions, often contributing to the onset of behavioral and mental health disorders (e.g., suicidality, substance use disorder, post-traumatic stress disorder [PTSD], anxiety, depression, sleep disorders; Howlett et al., 2022; McAllister, 2022; Meier & Savitz, 2022; Olsen & Corrigan, 2022; Piantino et al., 2022) and increasing the risk for neurodegenerative disorders (e.g, dementia, Parkinson's disease; Brett et al., 2022). Hence, identifying key brain areas, cortical and subcortical regions, and cognitive functions to further investigate and define their relationship with WM impairment and its most prominent causes (e.g., TBI, mTBI) may be fundamental to the development of effective treatments and informed surgical interventions to improve the quality of life of WM impairment survivors.

Objectives

Four empirical research studies have been selected for review, namely Cheng et al. (2019), Huang et al. (2019), Newsome et al. (2015), and Runyan et al. (2022). The selection criteria for reviewed studies include the following: (1) research must be original and empirical, (2) studies must have been published in the last 10 years, (3) studies must make use of brain imaging techniques, and (4) studies must include human participants only. The only selection macro-criterion consisted in the number of papers to be selected, namely four.

Cheng et al. (2019) and Runyan et al. (2022), in particular, explored the use of resting-state functional connectivity (rsFC; i.e., the correlation of signal activity between related brain regions occurring in a state of privation from stimuli and action, assessed using neuroimaging techniques; Biswal, 2015; Husain & Schmidt, 2014) as it relates to mTBI and investigated areas such as (1) dysfunctions of the hippocampus and WM impairment in military pilots caused by aircraft noise and (2) the neural and cortical relationships between mTBI and PTSD as caused by WM impairments in U.S. military personnel. Huang et al. (2019) examined WM functional abnormalities of combat-related mTBI, and Newsome et al. (2015) investigated WM activation severance as it pertains to chronic blast-related TBI.

The aim of this review is to examine current research and findings concerning WM impairment (with a particular focus on TBI and mTBI, given their prevalence, as previously discussed) and affected brain areas and cognitive functions in military personnel, in order to encourage further research and inform novel clinical applications, including cognitive rehabilitation methodologies, which may benefit both civilians and the military population.

Literature Review

Reduced Connectivity in VLPFC Affects WM Performance

Runyan et al. (2022) explored the associations between WM impairments and mTBI and PTSD in 127 U.S. military personnel between the ages of 18 and 59 years old. Three main groups were featured in this study, namely military personnel with mTBI only, military personnel with PTSD only, and a control group suffering from orthopedic injury. Study aims included the examination of variations in WM network connectivity in mTBI and a comparison with PTSD, the investigation of the relationship between resting state functional connectivity (rsFC) of WM regions and WM performance, and the analysis of differences in rsFC as they apply to WM frontal and parietal cortex regions in the military population.

Resting-state fMRI was used to measure rsFC in WM networks and regions, and multivariate modeling was used to examine discrepancies in seed-based voxelwise rsFC of WM regions. Functional images were taken while subjects were lying awake, staying still with their eyes closed. Data processing was executed thoroughly and included steps such as despiking, motion correction, temporal filtering, and spatial smoothing. Thirteen key regions were selected from Owen et al. (2005), a meta-analysis of fMRI studies of WM. Newer meta-analyses could have been taken into consideration when selecting key regions. Group differences were investigated using post hoc unpaired two-sample t tests, with family-wise error (FWE) adjustments at cluster level being used to correct for multiple comparisons. Multiple linear regression was used to examine the relationship between the study's significant rsFC findings and WM performance.

Findings suggest that there may be common neural substrates for WM impairments, with both mTBI and PTSD groups showing reduced rsFC in WM brain regions when compared to the control group. However, definite patterns of connectivity in WM areas were found in the PTSD group. Areas indicating reduced rsFC included WM regions located in the lateral premotor cortex, DLPFC, and VLPFC as well as the dorsal attention and somatomotor networks. The VLPFC's rsFC was significantly correlated with WM performance, and reduced VLPFC connectivity was significantly associated with lower WM performance. The suggested association between VLPFC and WM can be found in other neuroimaging studies (e.g., Segal & Elkana, 2023), reinforcing the prominent and mediating role of the PFC in regard to mTBI- and PTSD-caused WM impairment.

Overall, Runyan et al. (2022)'s suggested association between VLPFC and WM provides the basis for further investigations of the prominent role of VLPFC in the context of mTBI- and PTSD-induced WM impairments in military personnel. However, further explorations focused on comparing key WM areas with other subtypes of mTBI, the employment of more generalizable sets of WM tasks, and stricter criteria for PTSD group selection (e.g., making sure other categories of trauma are not concurrent in study participants) should be pursued in future research.

Aberrant Activations in Prefrontal Cortex Regions

Huang et al. (2019) examined WM functional abnormalities caused by combat-related mTBI. The two groups featured in this study were a group of 25 affected military individuals (either active-duty personnel or veterans) and a control group of 20 healthy individuals with matched combat backgrounds. Study aims included the exploration of combat-related mTBI and WM abnormalities, as well as their neuronal mechanisms, using an N-back WM task and MEG, the analysis of the relationship between MEG-recorded WM

activations and executive functioning, and the examination of abnormal MEG WM signals' characteristics as they relate to combat-related mTBI WM cognitive deficits.

MEG was employed to assess the underlying neuronal systems in WM functional abnormalities caused by combat-related mTBI, with data sampled at 1000 Hz and source-magnitude images procured for alpha, beta, gamma, and low-frequency bands. Pre-processing involved band-pass filtering and voxel-wise group statistical analysis. The night before the MEG scans were taken, all participants (combat mTBI-affected and control group) were encouraged to avoid substances and alcohol. A three-load conditions N-back WM task, comprised of online monitoring, updating, and manipulation of recalled information, was adopted, and MEG recordings were taken during the task. Neuropsychological tests were used to assess processing speed (Symbol Search and Digit Symbol Coding subsets from the Wechsler Adult Intelligence Scale [WAIS]) and executive functions (Kaplan Executive Function System [D-KEFS] Trail Making Test and Verbal Fluency Test).

Findings indicated that hyperactivation in regions of the prefrontal cortex (namely DLPFC, VMPFC, OFC, and FP) and hypoactivation in the anterior cingulate cortex (ACC) were detected in combat-related mTBI participants. WM neuropsychological tests' poorer performance and slower reaction times were associated with hyperactivation in the anterior DLPF, FP, and OFC. WM-related cognitive deficits in mTBI-affected participants were associated with functionally significant aberrant neuronal activity in the prefrontal cortex. Limitations include MEG spatial resolution and localization accuracy constraints for subcortical areas, lack of selective control for past concussions not related to blast, and potential post-preprocessing lingering effects of residual artifacts from eye-movement, heartbeats, and eye blinks.

Overall, Huang et al. (2019) provided evidence for the relationship between aberrant activations and WM impairment, using MEG to document the presence of the former in combat-related mTBI participants during the execution of a N-back WM task. In line with the reviewed literature, PFC areas such as the VMPFC, DLPFC , OFC, and FP were revealed to be central to WM functioning (e.g., Barbey et al., 2013; Eriksson et al., 2015; Hoskison et al., 2009), and aberrant activations in such areas were associated with slower reaction times and worse neuropsychological test performance. Future applications following this study may involve the research and implementation of innovative therapies and surgical interventions targeting relevant PFC areas to aid WM impairment caused by combat-related mTBI.

Hippocampal Dysfunctions from Aircraft Noise Exposure

Cheng et al. (2019) investigated the relationship between neural areas of WM impairment in military pilots and long-term aircraft noise exposure. Compared groups included 30 randomly selected Chinese male military pilots and a control group of Chinese military officers. Age, education, and employment were matched between groups, and exclusion criteria (e.g., neuropsychological diseases, noise trauma) were effectively applied. Study aims included the exploration of the relationship between WM impairment and variations in brain structure and function of military pilots caused by excessive aircraft noise; the assessment of the pilots' WM and neurobehavioral performances against correspondent alterations in neuroimaging data; the measurement and comparison between pilots and control group of cerebral grey matter volumes (GMV), amplitude of low-frequency fluctuation (ALFF), fractional ALFF (fALFF), and regional homogeneity (ReHo); and analysis of the fundamental underlying principles of neuropsychological impairment caused by aircraft noise exposure.

Structural MRI and resting-state fMRI results were pre-processed, analyzed using voxel-based morphometry (VBM), and used to assess GMV, ALFF, fALFF, and ReHo variations between groups. A neurobehavioral test battery featuring both immediate and delayed computerized verbal/visual memory tests was used to assess WM performance, continuous cockpit noise levels were recorded during the latest flight, participants' total flight hours were obtained via questionnaire and personal archives, correlation analyses were conducted to evaluate differences between neurobehavioral performance and neuroimaging changes, a student t test was used to determine distinctions between neurobehavioral and demographic parameters, and Spearman correlation coefficients were independently produced to assess correlations between ReHo/GMV and neuropsychological parameters.

Findings propounded that WM deficits were detected in military pilots exposed to long-term aircraft noise, who were presenting significantly lower accuracy in delayed visual and verbal memory tests. The following areas exhibited reduced ReHo, suggesting disruption in local neural activity: left thalamus, left amygdala, right superior and middle frontal gyrus, and left superior temporal gyrus. Neuronal dysfunction was indicated by decreased ReHo, GMV, and ALFF activity in the left hippocampus from resting-state fMRI and structural MRI data. A significant association between WM accuracy and both ReHo and GMV of the left hippocampus was found through Spearman correlation analysis, suggesting that dysfunctions of the hippocampus may be directly linked to aircraft noise exposure-caused WM impairment and that this area may be critical for WM function. Limitations included the absence of randomization in the study design, differences in environments and individual

circumstances, and lack of control for (1) dose-effect relationship between imaging anomalies and neurobehavioral impairment and (2) possible influential effects of concurrent exposure to toxicants and pollutants.

Overall, Cheng et al. (2019) contributed evidence for the association between hippocampus dysfunctions and WM impairment (also presented by Hung et al., 2022), further solidifying the role of the hippocampus as it relates to WM function (as evidenced by Leszczynski, 2011) to the benefit of future WM impairment prevention programs and health interventions of military pilots.

Blast TBI-Induced Caudate Activation Disruption

Newsome et al. (2015) examined fMRI-recorded WM activation severance in chronic blast-related TBI. Four main groups, namely veterans without TBI, veterans with blast-related TBI, civilians with blunt-force TBI, and a civilian control group, were featured in this study, and twenty-five participants were included in each group. Study aims included the comparison of brain activation mediated by a WM task between blunt force and blast TBIs, the assessment of whether brain activation during a WM task would differ between civilians with blunt force TBI and veterans with blast TBI, and the analysis of extended neuropsychological outcomes in TBI groups.

High-resolution structural MRI and fMRI were used to document alterations in brain areas and activations in participants. Pre-screening of participants was conducted via telephone to make sure they met predetermined inclusion and exclusion criteria. Exclusion criteria included history of severe psychiatric disorder (except for PTSD), absence of English fluency, history of cerebral and/or cognitive neurologic disorders, and unsuitability to undertake MRI. The Sternberg Item Recognition Task (SIRT) was used to measure WM performance, and neuropsychological tests, such as the Controlled Oral Word Association Test (COWAT), the California Verbal Learning Test-II (CVLT-II), the Trail Making Test (parts A and B; TMT), and the Symbol Digit Modalities Test (SDMT), were administered to evaluate cognitive deficits. ANOVA, FDR correction, and deconvolution analysis were employed for data processing. Study approval for recruitment and procedures was obtained from various institutional review boards (e.g., U.S. DOD, Louis Stokes Veterans Affairs Medical Center [VAMC], Cleveland Clinic).

Findings evinced that poor SIRT performance was associated with blast TBI, following comparison with other groups. It was determined that during encoding, the monotonic relationship between WM set size and fMRI-recorded caudate activation was disrupted by TBI. Researchers also concluded that dorsolateral and orbitofrontal striatal circuits engaging the caudate nucleus were particularly affected by chronic blast-specific brain alterations,

suggesting that the caudate may be especially sensitive to blast injury and that caudate activation patterns alterations could be used as biomarkers for blast TBI. Limitations included lack of control for participants' multiple blasts exposure (relevant since about half of participants were previously exposed to multiple blasts, which may have increased symptoms), unaccounted for secondary blast effects which may have caused further pathologies, and absence of WM non-verbal tasks, reducing the generalizability of results.

Overall, Newsome et al. (2015) provided evidence for blast TBI-induced caudate activation disruption amidst WM tasks, highlighting the role of the caudate nucleus (also see Grahn et al., 2009) and striatal circuits in the WM encoding process, and documented the severing effects of blast TBI-caused WM impairment. Subcortical structures engaging the caudate nucleus, such as the dorsolateral and orbitofrontal striatal circuits (the disruption of which was also associated with neurodegenerative disorders, such as Parkinson's disease; reviewed by Lewis et al., 2004), have been deemed particularly affected by chronic blast-specific brain alterations. A rationale was offered for future clinical research, which may be focused on the development of more accurate biomarker detection systems, improved interventions for increasing blast TBI-affected caudate activation, and long-term treatment options for military personnel affected by blast TBI and WM impairment.

Conclusion

WM impairment has been known to affect cognitive function (Chai et al., 2018; Christodoulou et al., 2001) and impact the quality of life of affected individuals (Irfani Fitri et al., 2020; Lanfranchi & Carretti, 2016). Among the most recurrent WM impairment causes in military personnel, the prevalence of TBI and mTBI has been reported by DHA and DOD (DOD Worldwide TBI Numbers, 2024a; DOD Worldwide TBI Numbers, 2024b) and has been addressed in other studies (e.g., Chen et al., 2012), therefore occupying a prominent role in the associated research field and in this review.

While novel advanced neuroimaging techniques (e.g., dynamic susceptibility contrast MRI; Koerte et al., 2016) may provide cutting-edge solutions to today's most pressing clinical research questions and valuable insights related to WM impairment, approval for use and availability in clinical research settings may take time and require further economic resources. Hence, it is fundamental for researchers to continue the study of key affected brain areas and cortical and subcortical regions, as well as cognitive and structural functions, using available imaging techniques and tools (e.g., fMRI, structural MRI, MEG). fMRI in particular has been deemed the method of choice for TBI and mTBI research (Medaglia, 2017), followed by MEG (Alhourani et al., 2016; Da Costa et al., 2015). Both were selectively employed in the studies included in this review, the aim of which is the

examination of current research and findings concerning brain areas, cortical and subcortical regions, and cognitive and structural functions affected by WM impairment in military personnel.

The four selected studies provided diverse contributions to the current understanding of WM impairment in military personnel and were focused respectively on (1) resting-state connectivity of WM regions and the association between WM impairment in mTBI and PTSD (Runyan et al., 2022), (2) WM functional abnormalities caused by combat-related mTBI (Huang et al., 2019), (3) the relationship between neural areas of WM impairment in military pilots and long-term aircraft noise exposure (Cheng et al., 2019), and (4) WM activation severance in chronic blast-related TBI (Newsome et al., 2015).

Runyan et al. (2022) evidenced the role of the VLPFC as it relates to mTBI- and PTSD-induced WM impairments in military personnel, pointing at the significant correlation between reduced VLPFC rsFC and lower WM performance. Huang et al. (2019) highlighted the contributions towards WM functioning of PFC areas, such as the DLPFC, VMPFC, OFC, and FP, revealing an association between aberrant activations in such areas, worse neuropsychological tests' performance, and slower reaction times. Cheng et al. (2019) identified the association between WM impairment and hippocampal dysfunctions, evidencing the direct role the hippocampus occupies in the functioning of visual and verbal WM. Newsome et al. (2015) denotated the high susceptibility to chronic blast-specific brain alterations of the dorsolateral and orbitofrontal striatal circuits and evidenced the substantial contributions of the caudate nucleus and striatal circuits in the WM encoding process.

In conclusion, the review of this selection of four studies has identified brain areas, cortical and subcortical regions, and cognitive functions related to WM impairment in military personnel. The most-affected brain areas and cortical and subcortical regions include the VLPFC, DLPFC, VMPFC, OFC, FP, hippocampus, dorsolateral and orbitofrontal striatal circuits, and caudate nucleus. Identified disruptions to cognitive functions include slower reaction times, lower WM performance and accuracy in delayed visual and verbal abilities, and worse neuropsychological performance. Future research should focus on the cross-examination of underexplored potential WM impairment causes (e.g., repeated long-term aircraft G-force exposure), as well as further investigations on affected brain areas and cognitive functions. Furthermore, review and application into future research of methods of cognitive rehabilitation and other rehabilitative methods should be integrated to assist the development of effective clinical applications for the benefit of military personnel and civilian patients suffering from WM impairment.

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