
Working Memory Impairment in Military Personnel: Affected Encephalic Areas and Cognitive Functions

Riccardo Maira

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, which can significantly impact quality of life and are often caused by brain trauma, have been known to affect cognitive functioning (Christodoulou et al., 2001). 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. TBIs range in severity, with 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 (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), more recently renamed as the Department of War (DoW), provide further indicators of 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 that 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). Magnetoencephalog-

raphy (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).

Encephalic areas, including 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 & Elkan, 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 Marine Corps Services (DoD Worldwide TBI Numbers, 2024a). The same agency released another report, disclosing that mTBIs accounted for 83.1% of all TBIs in the year 2023 (DoD Worldwide TBI Numbers, 2024b). TBIs 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). The impact of TBIs on cognitive functions has been associated with 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 encephalic areas (e.g., cortical and subcortical regions) and cognitive functions to further investigate and define their relationship with WM impairment and its most common causes (e.g., TBI, mTBI) may be fundamental to the development of effective treatments and informed interventions to improve the quality of life of individuals who experienced TBIs with continuing symptoms.

Objectives

The aim of this mini-review was 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 encephalic 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. Four empirical research studies have been selected for this 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) studies must investigate WM-impairment affected encephalic areas and cognitive functions, (2) research must be original and empirical, (3) studies must have been published in the last 10 years (2015-2025), (4) studies must make use of brain imaging techniques, (5) studies must include military personnel participants, and (6) the number of studies to be selected must be four. Databases searched included PubMed, ScienceDirect, and SSRN. Search phrases used included combinations of the following: “working memory”, “working memory impairment”, “neuroimaging”, “fMRI”, and “military”. Reasons for exclusion included article publication date was older than 10 years, study did not make use of brain imaging techniques, sample did not feature military personnel, and study was not focused on affected encephalic areas and cognitive functions.

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 US 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.

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 US 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 who experienced orthopedic injuries. Study aims included the examination of variations in WM network connectivity in military personnel with mTBI or PTSD, the investigation of the relationship between rsFC of WM regions and WM performance, and the analysis of differences in rsFC as they apply to WM frontal and parietal cortex regions.

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 participants were lying awake, staying still with their eyes closed. Data processing 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. Group differences were investigated using post hoc unpaired two-sample t-tests, with family-wise error 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 connectivity patterns 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; 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-related WM impairment. Overall, the association between VLPFC and WM suggested by Runyan et al. (2022) provides the basis for further investigations of the role of VLPFC in the context of mTBI- and PTSD-related 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 in the context of combat-related mTBI. The study included a group of 25 military personnel with a history of combat-related mTBI (either active-duty personnel or veterans) and a control group of 20 healthy individuals with matched combat experiences. 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 associated with 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) and executive functions (Kaplan Executive Function System 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 participants with combat-related mTBI. Per neuropsychological tests, poorer WM performance and slower reaction times were associated with hyperactivation in the anterior DLPFC, FP, and OFC. WM-related cognitive deficits in participants with mTBI were associated with aberrant neuronal activity in the PFC. Limitations included MEG spatial resolution and localization accuracy constraints for subcortical areas, lack of selective control for past concussions not related to blast exposure, 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 participants with a history of combat-related mTBI 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 promising innovative therapies and neuromodulation-based interventions, such as carefully targeted and integrated deep brain stimulation (DBS; i.e., a minimally invasive surgical procedure featuring neuromodulation; Khan et al., 2019; Salehi et al., 2024; Tan et al., 2020), targeting relevant PFC areas to aid WM impairment caused by combat-related mTBI.

Hippocampal Dysfunctions Associated with 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. The sample included two groups: 30 randomly selected Chinese male military fighter jet pilots and 30 Chinese military officers. Groups were matched based on age, education, and employment, and exclusion criteria (e.g., neuropsychological diseases, noise-related trauma history) 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 corresponding 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, 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 non-parametric correlation coefficients were independently produced to assess correlations between ReHo/GMV and neuropsychological parameters.

Findings indicated 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 than the control group. 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 non-parametric correlation analysis, suggesting that dysfunctions of the hippocampus may be directly linked to aircraft noise exposure-related 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-Related TBI Associated with 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 included in this study; 25 participants were in each group. Study aims included the comparison of brain activation during 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 encephalic areas and activations in participants. 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, the California Verbal Learning Test-II, the Trail Making Test (parts A and B), and the Symbol Digit Modalities Test, were administered to evaluate cognitive performance. 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, Cleveland Clinic).

Findings evinced that, following a comparison between blast TBI and blunt-force groups, worse SIRT performance was associated with blast TBI. It was determined that during encoding, the monotonic (i.e., consistently directional) 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-related TBI. Limitations included lack of control for participants' multiple blast exposures (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-related TBI associations with 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. 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 associated with chronic blast-specific brain alterations. Future clinical research 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

In conclusion, the review of this selection of four studies has identified relevant encephalic areas, including cortical and subcortical regions, and cognitive functions related to WM impairment in military personnel, providing diverse contributions to the current understanding of WM impairment in military personnel. 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 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](#)) denoted 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. Most of the identified encephalic regions associated with WM performance and impairment, such as PFC areas and the hippocampus, provided continuity with previous research. With TBIs retaining a position of prevalence in WM impairment causes affecting the military population, other adjacent and more subtle causes, such as PTSD and long-term aircraft noise exposure, may be underrepresented in current WM impairment research efforts. However, relevant distinctive challenges to the current neuropsychological understanding of complex interactions between alterations in key encephalic areas and cognitive changes in WM performance continue to be presented by military population-specific WM impairment causes and environments, such as potential WM disruptions related to repeated long-term aircraft G-force exposure in military pilots, as well as unique opportunities arising from the development and deployment of novel neuroimaging technologies integrated with innovative neurocognitive assessments and targeted surgical interventions and rehabilitation methods, such as high-resolution neuroimaging and DBS. Future research should focus on the cross-examination of underexplored potential WM impairment causes particularly related to specific military populations, as well as further related investigations on affected

encephalic areas and cognitive functions. Furthermore, review and application into future research of methods of neurocognitive rehabilitation and surgical intervention, such as novel applications of targeted DBS, can be integrated to assist the development of effective clinical treatments for the benefit of military personnel and civilian patients experiencing reduced WM performance.

The author of this article can be contacted at: ricar-domaira.contact@gmail.com

References

- Alhourani, A., Wozny, T. A., Krishnaswamy, D., Pathak, S., Walls, S. A., Ghuman, A. S., Krieger, D. N., Okonkwo, D. O., Richardson, R. M., & Niranjana, A. (2016). Magnetoencephalography-based identification of functional connectivity network disruption following mild traumatic brain injury. *Journal of Neurophysiology*, 116(4), 1840–1847. <https://doi.org/10.1152/jn.00513.2016>
- Allen, C. M., Halsey, L., Topcu, G., Rier, L., Gascoyne, L. E., Scadding, J. W., Furlong, P. L., Dunkley, B. T., Das Nair, R., Brookes, M. J., & Evangelou, N. (2021). Magnetoencephalography abnormalities in adult mild traumatic brain injury: A systematic review. *NeuroImage: Clinical*, 31, 102697. <https://doi.org/10.1016/j.nicl.2021.102697>
- Arciniega, H., Shires, J., Furlong, S., Kilgore-Gomez, A., Cerreta, A., Murray, N. G., & Berryhill, M. E. (2021). Impaired visual working memory and reduced connectivity in undergraduates with a history of mild traumatic brain injury. *Scientific Reports*, 11(1), 2789. <https://doi.org/10.1038/s41598-021-80995-1>
- Baddeley, A. (1992). Working memory: The interface between memory and cognition. *Journal of Cognitive Neuroscience*, 4(3), 281–288. <https://doi.org/10.1162/jocn.1992.4.3.281>
- Baier, B., Karnath, H.-O., Dieterich, M., Birklein, F., Heinze, C., & Müller, N. G. (2010). Keeping memory clear and stable—The contribution of human basal ganglia and prefrontal cortex to working memory. *Journal of Neuroscience*, 30(29), 9788–9792. <https://doi.org/10.1523/JNEUROSCI.1513-10.2010>
- Balconi, M. (2013). Dorsolateral prefrontal cortex, working memory and episodic memory processes: Insight through transcranial magnetic stimulation techniques. *Neuroscience Bulletin*, 29(3), 381–389. <https://doi.org/10.1007/s12264-013-1309-z>
- Barbey, A. K., Koenigs, M., & Grafman, J. (2011). Orbitofrontal contributions to human working memory. *Cerebral Cortex*, 21(4), 789–795. <https://doi.org/10.1093/cercor/bhq153>
- Barbey, A. K., Koenigs, M., & Grafman, J. (2013). Dorsolateral prefrontal contributions to human working memory. *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior*, 49(5), 1195–1205. <https://doi.org/10.1016/j.cortex.2012.05.022>
- Biswal, B. (2015). Resting-state functional connectivity. In *Brain Mapping* (pp. 581–585). Elsevier Science. <https://doi.org/10.1016/B978-0-12-397025-1.00335-3>
- Brett, B. L., Gardner, R. C., Godbout, J., Dams-O'Connor, K., & Keene, C. D. (2022). Traumatic brain injury and risk of neurodegenerative disorder. *Biological Psychiatry*, 91(5), 498–507. <https://doi.org/10.1016/j.biopsych.2021.05.025>
- Chai, W. J., Abd Hamid, A. I., & Abdullah, J. M. (2018). Working memory from the psychological and neurosciences perspectives: A review. *Frontiers in Psychology*, 9, 401. <https://doi.org/10.3389/fpsyg.2018.00401>
- Chen, C.-J., Wu, C.-H., Liao, Y.-P., Hsu, H.-L., Tseng, Y.-C., Liu, H.-L., & Chiu, W.-T. (2012). Working memory in patients with mild traumatic brain injury: Functional MR imaging analysis. *Radiology*, 264(3), 844–851. <https://doi.org/10.1148/radiol.12112154>
- Cheng, H., Sun, G., Li, M., Yin, M., & Chen, H. (2019). Neuron loss and dysfunctionality in hippocampus explain aircraft noise induced working memory impairment: A resting-state fMRI study on military pilots. *BioScience Trends*, 13(5), 430–440. <https://doi.org/10.5582/bst.2019.01190>
- Christodoulou, C., DeLuca, J., Ricker, J. H., Madigan, N. K., Bly, B. M., Lange, G., Kalnin, A. J., Liu, W.-C., Steffener, J., Diamond, B. J., & Ni, A. C. (2001). Functional magnetic resonance imaging of working memory impairment after traumatic brain injury. *Journal of Neurology, Neurosurgery & Psychiatry*, 71(2), 161–168. <https://doi.org/10.1136/jnnp.71.2.161>
- Chung, S., Wang, X., Fieremans, E., Rath, J. F., Amorapanth, P., Foo, F.-Y. A., Morton, C. J., Novikov, D. S., Flanagan, S. R., & Lui, Y. W. (2019). Altered relationship between working memory and brain microstructure after mild traumatic brain injury. *AJNR: American Journal of Neuroradiology*, 40(9), 1438–1444. <https://doi.org/10.3174/ajnr.A6146>
- Cowan, N. (2014). Working memory underpins cognitive development, learning, and education. *Educational Psychology Review*, 26(2), 197–223. <https://doi.org/10.1007/s10648-013-9246-y>
- Da Costa, L., Robertson, A., Bethune, A., MacDonald, M. J., Shek, P. N., Taylor, M. J., & Pang, E. W. (2015). Delayed and disorganized brain activation detected with magnetoencephalography after mild traumatic brain injury. *Journal of Neurology, Neurosurgery & Psychiatry*, 86(9), 1008–1015. <https://doi.org/10.1136/jnnp-2014-308571>
- Dimitriadis, S. I., Zouridakis, G., Rezaie, R., Babajani-Feremi, A., & Papanicolaou, A. C. (2015). Func-

- tional connectivity changes detected with magnetoencephalography after mild traumatic brain injury. *NeuroImage: Clinical*, 9, 519–531. <https://doi.org/10.1016/j.nicl.2015.09.011>
- Ding, X., Fu, H., Luo, L., & Wu, J. (2024). Working memory deficit in schizophrenia: A systematic review and meta-analysis of fMRI studies examining frontal and parietal brain activity. *Folia Neuropathologica*, 62(2), 136–146. <https://doi.org/10.5114/fn.2023.132329>
- DOD numbers for traumatic brain injury worldwide 2000–2023 (DOD Worldwide TBI Numbers). (2024a). Defense Health Agency. <https://health.mil/Reference-Center/Reports/2024/05/03/2000-2023-DOD-Worldwide-Numbers-for-TBI>
- DOD numbers for traumatic brain injury worldwide 2023. (2024b). Defense Health Agency. <https://health.mil/Reference-Center/Reports/2024/02/22/2023-Q4-DOD-Worldwide-Numbers-for-TBI>
- Eierud, C., Craddock, R. C., Fletcher, S., Aulakh, M., King-Casas, B., Kuehl, D., & LaConte, S. M. (2014). Neuroimaging after mild traumatic brain injury: Review and meta-analysis. *NeuroImage: Clinical*, 4, 283–294. <https://doi.org/10.1016/j.nicl.2013.12.009>
- Eriksson, J., Vogel, E. K., Lansner, A., Bergström, F., & Nyberg, L. (2015). Neurocognitive architecture of working memory. *Neuron*, 88(1), 33–46. <https://doi.org/10.1016/j.neuron.2015.09.020>
- Essad, K., Azmi, H., Feldman, J., Ogedegbe, C., & Wylie, G. (2016). Impacts of mTBI on working memory over time: The pilot longitudinal study (P3.313). *Neurology*, 86(16 supplement), P3.313. https://doi.org/10.1212/WNL.86.16_supplement.P3.313
- Grahn, J. A., Parkinson, J. A., & Owen, A. M. (2009). The role of the basal ganglia in learning and memory: Neuropsychological studies. *Behavioural Brain Research*, 199(1), 53–60. <https://doi.org/10.1016/j.bbr.2008.11.020>
- Hoskison, M. M., Moore, A. N., Hu, B., Orsi, S., Kobori, N., & Dash, P. K. (2009). Persistent working memory dysfunction following traumatic brain injury: Evidence for a time-dependent mechanism. *Neuroscience*, 159(2), 483–491. Crossref. <https://doi.org/10.1016/j.neuroscience.2008.12.050>
- Howlett, J. R., Nelson, L. D., & Stein, M. B. (2022). Mental health consequences of traumatic brain injury. *Biological Psychiatry*, 91(5), 413–420. <https://doi.org/10.1016/j.biopsych.2021.09.024>
- Huang, M., Lewine, J. D., & Lee, R. R. (2020). Magnetoencephalography for mild traumatic brain injury and posttraumatic stress disorder. *Neuroimaging Clinics of North America*, 30(2), 175–192. <https://doi.org/10.1016/j.nic.2020.02.003>
- Huang, M.-X., Nichols, S., Robb-Swan, A., Angeles-Quinto, A., Harrington, D. L., Drake, A., Huang, C. W., Song, T., Diwakar, M., Risbrough, V. B., Matthews, S., Clifford, R., Cheng, C.-K., Huang, J. W., Sinha, A., Yurgil, K. A., Ji, Z., Lerman, I., Lee, R. R., & Baker, D. G. (2019). MEG working memory n-back task reveals functional deficits in combat-related mild traumatic brain injury. *Cerebral Cortex*, 29(5), 1953–1968. <https://doi.org/10.1093/cercor/bhy075>
- Huang, S., Huang, C., Li, M., Zhang, H., & Liu, J. (2022). White matter abnormalities and cognitive deficit after mild traumatic brain injury: Comparing DTI, DKI, and NODDI. *Frontiers in Neurology*, 13. <https://doi.org/10.3389/fneur.2022.803066>
- Hung, Y., Vandewouw, M., Emami, Z., Bells, S., Rudberg, N., Da Costa, L., & Dunkley, B. T. (2022). Memory retrieval brain–behavior disconnection in mild traumatic brain injury: A magnetoencephalography and diffusion tensor imaging study. *Human Brain Mapping*, 43(17), 5296–5309. <https://doi.org/10.1002/hbm.26003>
- Husain, F. T., & Schmidt, S. A. (2014). Using resting state functional connectivity to unravel networks of tinnitus. *Hearing Research*, 307, 153–162. <https://doi.org/10.1016/j.heares.2013.07.010>
- Johnson, E. L., Chang, W. K., Dewar, C. D., Sorensen, D., Lin, J. J., Solbakk, A.-K., Endestad, T., Larsson, P. G., Ivanovic, J., Meling, T. R., Scabini, D., & Knight, R. T. (2022). Orbitofrontal cortex governs working memory for temporal order. *Current Biology*, 32(9), R410–R411. <https://doi.org/10.1016/j.cub.2022.03.074>
- Khan, I. S., D’Agostino, E. N., Calnan, D. R., Lee, J. E., & Aronson, J. P. (2019). Deep brain stimulation for memory modulation: A new frontier. *World Neurosurgery*, 126, 638–646. <https://doi.org/10.1016/j.wneu.2018.12.184>
- Kim, S. Y., Yeh, P.-H., Ollinger, J. M., Morris, H. D., Hood, M. N., Ho, V. B., & Choi, K. H. (2023). Military-related mild traumatic brain injury: Clinical characteristics, advanced neuroimaging, and molecular mechanisms. *Translational Psychiatry*, 13, 289. <https://doi.org/10.1038/s41398-023-02569-1>
- Koerte, I. K., Hufschmidt, J., Muehlmann, M., Lin, A. P., & Shenton, M. E. (2016). Advanced neuroimaging of mild traumatic brain injury. In D. Laskowitz & G. Grant (Eds), *Translational Research in Traumatic Brain Injury*. CRC Press/Taylor and Francis Group. <http://www.ncbi.nlm.nih.gov/books/NBK326714/>
- Kumar, S., Rao, S. L., Chandramouli, B. A., & Pillai, S. (2013). Reduced contribution of executive functions in impaired working memory performance in mild traumatic brain injury patients. *Clinical Neurology and Neurosurgery*, 115(8), 1326–1332. <https://doi.org/10.1016/j.clineuro.2012.12.038>
- Leszczynski, M. (2011). How does hippocampus contrib-

- ute to working memory processing? *Frontiers in Human Neuroscience*, 5, 1–2. <https://doi.org/10.3389/fnhum.2011.00168>
- Levens, S. M., Devinsky, O., & Phelps, E. A. (2011). Role of the left amygdala and right orbital frontal cortex in emotional interference resolution facilitation in working memory. *Neuropsychologia*, 49(12), 3201–3212. <https://doi.org/10.1016/j.neuropsychologia.2011.07.021>
- Lewis, S. J. G., Dove, A., Robbins, T. W., Barker, R. A., & Owen, A. M. (2004). Striatal contributions to working memory: A functional magnetic resonance imaging study in humans. *European Journal of Neuroscience*, 19(3), 755–760. <https://doi.org/10.1111/j.1460-9568.2004.03108.x>
- Mallas, E.-J., De Simoni, S., Scott, G., Jolly, A. E., Hampshire, A., Li, L. M., Bourke, N. J., Roberts, S. A. G., Gorgoraptis, N., & Sharp, D. J. (2020). Abnormal dorsal attention network activation in memory impairment after traumatic brain injury. *Brain*, 144(1), 114–127. Crossref. <https://doi.org/10.1093/brain/awaa380>
- McAllister, T. W. (2022). Chicken or egg? Mental illness as a risk factor and outcome of traumatic brain injury. *Biological Psychiatry*, 91(5), 402–404. <https://doi.org/10.1016/j.biopsych.2021.05.024>
- Medaglia, J. D. (2017). Functional neuroimaging in traumatic brain injury: From nodes to networks. *Frontiers in Neurology*, 8, 407. <https://doi.org/10.3389/fneur.2017.00407>
- Meier, T. B., & Savitz, J. (2022). The kynurenine pathway in traumatic brain injury: Implications for psychiatric outcomes. *Biological Psychiatry*, 91(5), 449–458. <https://doi.org/10.1016/j.biopsych.2021.05.021>
- National Academies of Sciences, Engineering, and Medicine, Health and Medicine Division, Board on Health Care Services, & Committee on the Review of the Department of Veterans Affairs Examinations for Traumatic Brain Injury. (2019). Definitions of traumatic brain injury. In *Evaluation of the disability determination process for traumatic brain injury in veterans*. National Academies Press (US). <https://www.ncbi.nlm.nih.gov/books/NBK542588/>
- Newsome, M. R., Durgerian, S., Mourany, L., Scheibel, R. S., Lowe, M. J., Beall, E. B., Koenig, K. A., Parsons, M., Troyanskaya, M., Reece, C., Wilde, E., Fischer, B. L., Jones, S. E., Agarwal, R., Levin, H. S., & Rao, S. M. (2015). Disruption of caudate working memory activation in chronic blast-related traumatic brain injury. *NeuroImage: Clinical*, 8, 543–553. <https://doi.org/10.1016/j.nicl.2015.04.024>
- Olsen, C. M., & Corrigan, J. D. (2022). Does traumatic brain injury cause risky substance use or substance use disorder? *Biological Psychiatry*, 91(5), 421–437. <https://doi.org/10.1016/j.biopsych.2021.07.013>
- O'Neill, T. J., Davenport, E. M., Murugesan, G., Montilolo, A., & Maldjian, J. A. (2017). Applications of rs-fMRI to traumatic brain injury. *Neuroimaging Clinics of North America*, 27(4), 685–696. <https://doi.org/10.1016/j.nic.2017.06.006>
- Owen, A. M., McMillan, K. M., Laird, A. R., & Bullmore, E. (2005). N-back working memory paradigm: A meta-analysis of normative functional neuroimaging studies. *Human Brain Mapping*, 25(1), 46–59. <https://doi.org/10.1002/hbm.20131>
- Palacios, E. M., Sala-Llloch, R., Junque, C., Roig, T., Tormos, J. M., Bargallo, N., & Vendrell, P. (2012). White matter integrity related to functional working memory networks in traumatic brain injury. *Neurology*, 78(12), 852–860. <https://doi.org/10.1212/WNL.0b013e31824c465a>
- Piantino, J. A., Iliff, J. J., & Lim, M. M. (2022). The bidirectional link between sleep disturbances and traumatic brain injury symptoms: A role for glymphatic dysfunction? *Biological Psychiatry*, 91(5), 478–487. <https://doi.org/10.1016/j.biopsych.2021.06.025>
- Runyan, A. C., Philippi, C. L., Pessin, S., Velez, C. S., Wade, B. S. C., Drennon, A. M., Cooper, D. B., Kennedy, J. E., Bowles, A. O., Lewis, J. D., Reid, M. W., York, G. E., Newsome, M. R., Wilde, E. A., & Tate, D. F. (2022). Comparing resting-state connectivity of working memory networks in U.S. service members with mild traumatic brain injury and posttraumatic stress disorder. *SSRN Electronic Journal*. <https://doi.org/10.2139/ssrn.4033310>
- Salehi, N., Nahrgang, S., Petershagen, W., Dembek, T. A., Pedrosa, D., Timmermann, L., Weber, I., & Oehr, C. R. (2024). Theta frequency deep brain stimulation in the subthalamic nucleus improves working memory in Parkinson's disease. *Brain: A Journal of Neurology*, 147(4), 1190–1196. <https://doi.org/10.1093/brain/awad433>
- Segal, O., & Elkana, O. (2023). The ventrolateral prefrontal cortex is part of the modular working memory system: A functional neuroanatomical perspective. *Frontiers in Neuroanatomy*, 17, 1076095. <https://doi.org/10.3389/fnana.2023.1076095>
- Smith, K. (2012). CT scan does not predict outcome of mild traumatic brain injury. *Nature Reviews Neurology*, 8(9), 474–474. <https://doi.org/10.1038/nrneurol.2012.164>
- Smith, L. G. F., Milliron, E., Ho, M.-L., Hu, H. H., Rusin, J., Leonard, J., & Sribnick, E. A. (2019). Advanced neuroimaging in traumatic brain injury: An overview. *Neurosurgical Focus*, 47(6), E17. <https://doi.org/10.3171/2019.9.FOCUS19652>
- Sours, C., George, E. O., Zhuo, J., Roys, S., & Gullapalli, R. P. (2015). Hyper-connectivity of the thalamus during early stages following mild traumatic brain injury. *Brain Imaging and Behavior*, 9(3), 550–563. <https://doi.org/10.1007/s11682-015-9424-2>
- Tan, S. Z. K., Fung, M.-L., Koh, J., Chan, Y.-S., & Lim,

- L. W. (2020). The paradoxical effect of deep brain stimulation on memory. *Aging and Disease*, 11(1), 179–190. <https://doi.org/10.14336/AD.2019.0511>
- Voytek, B., & Knight, R. T. (2010). Prefrontal cortex and basal ganglia contributions to visual working memory. *Proceedings of the National Academy of Sciences*, 107(42), 18167–18172. <https://doi.org/10.1073/pnas.1007277107>
- Wallis, G., Stokes, M., Cousijn, H., Woolrich, M., & Nobre, A. C. (2015). Frontoparietal and cingulo-opercular networks play dissociable roles in control of working memory. *Journal of Cognitive Neuroscience*, 27(10), 2019–2034. https://doi.org/10.1162/jocn_a_00838
- Witt, K. (2021). The impact of the basal ganglia on working memory: Evidence from parkinson's disease. *Movement Disorders*, 36(1), 13–15. <https://doi.org/10.1002/mds.28358>
- Yin, S., Bi, T., Chen, A., & Egner, T. (2021). Ventromedial prefrontal cortex drives the prioritization of self-associated stimuli in working memory. *The Journal of Neuroscience*, 41(9), 2012–2023. <https://doi.org/10.1523/JNEUROSCI.1783-20.2020>