

Capacity to Measure Reduced Neurotransmission in Artillery Soldiers After Live Fire Training Offers New Avenues for Risk Management

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INTRODUCTION

Return to health and resilience are core goals of the armed forces. Yet there is currently no literature reporting on what is deemed to be a safe level of low level Military Occupational Blast (MOB) exposure, leading to an increasing interest in neurological damage following repetitive low-level MOB [1]. The recent paper "reduced neurotransmission in blast exposed artillery soldiers after live fire training" describes the effect of acute blast exposure to artillerymen [2]. This objective evaluation of a person's neurochemistry before and after firing training has implications for future management of frontline defenders. It offers the opportunity to investigate injury thresholds (e.g. extent of level and blast exposure) based on the level neurochemical dysregulation. It can also be used to test the presumption that blast affects the brain in the same way as physical impact.

The study demonstrates that a non-invasive brain chemistry (neurochemical) assessment, and capacity to monitor an individual over time, is feasible using a clinical 3 tesla siemens prisma MRI scanner with 64 channel head and neck coil. The protocol used was a two dimensional Correlated Spectroscopy (COSY) previously applied to evaluate glioma, PTSD, and breast deregulation [3,4]. The protocol provides an unambiguous assignment of those neurochemicals mobile on the MR timescale [5].

DESCRIPTION

An important aspect of this study is the multidisciplinary team that oversaw the psychological and clinical evaluation of the participants. The neuroradiologists reported no adverse findings in any of the participants in this study. The psychologists and psychiatrist evaluated all participants to ensure there were no confounding factors in addition to the acute exposure to artillery blast.

The 2D COSY technology provides a new modality to evaluate frontline defenders after exposure to blast and provide

information on the presence and extent of central nervous system dysregulation based on their neurochemistry. It provides a new avenue for risk prediction, stratification. And development of tailored screening strategies. The technology offers the opportunity to evaluate the effectiveness of MOB risk mitigation strategies.

New capacity to manage the health of front line defenders individually

The 2D COSY technology can record and monitor the extent of effect of blast on an individual's brain in response to varying levels of exposure to blast over the lifetime of their military service. Importantly it can identify neuro dysregulation in the acute phase and enables appraisal of interventions that facilitate recovery rather than transition to a chronic phase. It can be a reliable, non-invasive technique to distinguish extent, return to normal, partial response or failure of treatment response for each person's condition. Each person can be their own control.

The neurochemical pathways affected may, given time, lead to prophylactic treatment

Of the nine neurochemicals affected by firing training, seven are part of three neurochemical pathways considered to be at the terminus of the neurons and suggestive of early markers of disruption. These are glutathione, glutathione cysteine moiety and glutamine/glutamate levels indicative of a glutathione redox imbalance [6,7]. Such an imbalance was suggested in animal models to be an early marker of neurodegeneration [8]. The myoinositol/creatine and choline levels affected are consistent synthesis of inositol and precursors of inositol lipids and inositol phosphates that are pivotal for cell signaling [9].

The third mechanistic pathway are the two fucose- α (1-2)-glycans "3" and "6" and the newly assigned fucose- α (1-6) linkage [10]. These fucosylated glycans are located at the terminus of the neurons and are implicated in the molecular mechanisms that

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underlie neuronal development, learning and memory [11-13]. The intrinsic variability of glycan structures enables sugars to encode specific information, which being recognised by receptors, can be translated into a specific biological process.

Acute to chronic phase transition prevention

If the effect of blast on the individual can be documented in the acute phase, prior to the transition to the hard-to-treat chronic phase, the person may recover and return to the range recorded for the healthy unexposed cohort. The treatment could be a simple rest and withdrawal from blast exposure and reallocation of duties until neuro dysregulation is no longer evident on 2D COSY.

For return to health and resilience the soldiers would benefit by longitudinal evaluation by this technology: Annually pre and post deployment at onset, completion, and 4 weeks after firearms and explosives training. It is noteworthy that the frontline defender was examined 7 days after the MOB exposure as it can take that long for the neurochemistry to be affected.

Translation into the clinic

In order to make this technology available the data analysis and datamining is being automated and classifiers developed to evaluate each person's deviation from normal as well as comparison with other conditions such as pain [14,15].

CONCLUSION

The study demonstrated that a non-invasive brain chemistry (neurochemical) assessment, and capacity to monitor an individual over time, is feasible. It provides a new avenue for risk prediction, stratification and development of tailored screening strategies. The technology offers the opportunity to evaluate the effectiveness of MOB risk mitigation strategies.

CONFLICT OF INTEREST

Professor Mountford is a founder and shareholder of the company DatChem Pty Ltd. This is the vehicle by which the technology will be made available in collaboration with Griffith university.

REFERENCES

1. Tosh N, Watson J, Lukas D, Tremewan R, Beard J, Galloway G, et al. Two-dimensional correlated spectroscopy records reduced neurotransmission in blast-exposed artillery soldiers after live fire training. NMR Biomed. 2023;e4934.

- 2. Ramadan S, Andronesi OC, Stanwell P, Lin AP, Sorensen AG, Mountford CE. Use of *in vivo* two-dimensional MR spectroscopy to compare the biochemistry of the human brain to that of glioblastoma. Radiology. 2011;259(2):540-549.
- Quadrelli S, Tosh N, Urquhart A, Trickey K, Tremewan R, Galloway G, et al. Post-traumatic stress disorder affects fucose-α (1-2)-glycans in the human brain: Preliminary findings of neuro deregulation using *in vivo* two-dimensional neuro MR spectroscopy. Transl Psychiatry. 2019;9(1):27.
- 4. Santamaria G, Naude N, Watson J, Irvine J, Lloyd T, Bennett I, et al. Breast tissue chemistry measured *in vivo* in healthy women correlate with breast density and breast cancer risk. J Magn Reson Imaging. 2022;56(5):1355-1369.
- 5. Rae CD, Williams SR. Glutathione in the human brain: Review of its roles and measurement by magnetic resonance spectroscopy. Anal Biochem. 2017;529:127-143.
- 6. Moustapha A. Neurodegenerative diseases: Potential effect of glutathione. 2020.
- 7. Aoyama K, Nakaki T. Impaired glutathione synthesis in neurodegeneration. Int J Mol Scis. 2013;14(10):21021-21044.
- 8. Ye C, Greenberg ML. Inositol synthesis regulates the activation of GSK-3 α in neuronal cells. J Neurochem. 2015;133(2):273-283.
- Tosh N, Quadrelli S, Galloway G, Mountford C. Two new fucose-α (1-2)-glycans assigned in the healthy human brain taking the number to seven. Sci Rep. 2019;9(1):18806.
- 10. Kleene R, Schachner M. Glycans and neural cell interactions. Nat Rev Neurosci. 2004;5(3):195-208.
- Murrey HE, Ficarro SB, Krishnamurthy C, Domino SE, Peters EC, Hsieh-Wilson LC. Identification of the plasticity-relevant fucosealpha (1-2)-galactose proteome from the mouse olfactory bulb. Biochemistry. 2009;48(30):7261-7270.
- 12. Murrey HE, Hsieh-Wilson LC. The chemical neurobiology of carbohydrates. Chem Rev. 2008;108(5):1708-17031.
- 13. Siddall PJ, Stanwell P, Woodhouse A, Somorjai RL, Dolenko B, Nikulin A, et al. Magnetic resonance spectroscopy detects biochemical changes in the brain associated with chronic low back pain: A preliminary report. Anesth Analg. 2006;102(4):1164-1168.
- Stanwell P, Siddall P, Keshava N, Cocuzzo D, Ramadan S, Lin A, et al. Neuro magnetic resonance spectroscopy using wavelet decomposition and statistical testing identifies biochemical changes in people with spinal cord injury and pain. Neuroimage. 2010;53(2): 544-552.