



Investigation of trauma-induced metabolic changes in the thalamus using proton magnetic resonance spectroscopy

Trauma and thalamic metabolic changes via ¹H-MRS

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Abstract

Aim: This study aimed to noninvasively assess early neurometabolic changes in the thalamic region following trauma in adult individuals, using proton magnetic resonance spectroscopy (¹H-MRS).

Methods: Adult patients presenting with trauma but without parenchymal damage on computed tomography (CT) were prospectively enrolled. Thalamic MRS evaluations were performed on the 3rd and 15th days post-trauma to measure metabolite levels (N-acetylaspartate [NAA], choline [Cho], creatine [Cr], myo-inositol [mI], lactate [Lac], and glutamate-glutamine [Glx]) and metabolite ratios. Statistical comparisons between the two time points were made using paired t tests, with a significance threshold of $P < .05$.

Results: A significant increase in thalamic choline (Cho) levels was detected on the 3rd day after trauma ($P < .05$). In contrast, no significant changes were observed in NAA, Cr, mI, Lac, Glx levels, or in metabolite ratios ($P > .05$).

Conclusion: The findings suggest that early trauma leads to selective metabolic alterations, particularly reflected by increased Cho levels likely associated with early glial activation. Proton MRS may offer a valuable, noninvasive method for detecting early biochemical changes in the central nervous system after trauma, aiding in the early identification of patients at risk for further neurological complications.

Keywords

trauma, thalamus, proton magnetic resonance spectroscopy (¹H-MRS), choline, neurometabolic changes

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Introduction

Trauma, resulting from the transfer of kinetic, thermal, or chemical energy to tissues, is a leading cause of mortality among individuals aged 1–44 years.¹ Globally, it ranks third after cancer and cardiovascular diseases.² Beyond mortality, trauma leads to permanent disabilities and reduced quality of life. Pain emerging after trauma reflects a defensive response, transmitted from peripheral nociceptors to the thalamus and cerebral cortex.³ Alterations in this transmission may predispose individuals to chronic neuropathic pain, with the thalamus serving a central modulatory role.^{4,5}

Thalamic dysfunction following trauma has been associated with long-term neuronal damage, as indicated by reductions in N-acetylaspartate (NAA) levels observed through magnetic resonance spectroscopy (MRS).^{6–8} Conventional imaging techniques are limited in detecting early metabolic changes, whereas MRS enables noninvasive quantification of key metabolites such as NAA, choline (Cho), and creatine (Cr).⁹ These alterations may underlie the progression to chronic pain syndromes. Early identification of such metabolic disruptions could inform preventive therapeutic strategies.

Understanding the thalamus's early neurochemical responses to trauma has significant clinical relevance. Early identification

of metabolic alterations could enable clinicians to predict the risk of chronic neuropathic pain development, implement timely therapeutic strategies, and ultimately improve long-term patient outcomes. By elucidating the pathophysiological pathways involved in central pain sensitization after trauma, findings from such studies may help optimize rehabilitation programs and enhance quality of life for affected individuals.

The present study aims to investigate thalamic neurometabolite changes in adult individuals during the early post-traumatic period using proton magnetic resonance spectroscopy (¹H-MRS). By objectively characterizing early central nervous system alterations following trauma, this research seeks to contribute to a deeper understanding of trauma-related neuropathophysiology and to inform clinical approaches aimed at preventing chronic complications.

Materials and Methods

This study was designed prospectively.

Patient Selection

Adults who presented to the Emergency Department of Cumhuriyet University Research and Application Hospital between April and December 2009 and who exhibited no parenchymal damage on

Table 1. Thalamic Metabolite Concentrations Detected By MRS On The 3rd And 15th Days After Trauma

Case	Age	Sex	Trauma Cause	NAA (D3)	Cho (D3)	Cr (D3)	ml (D3)	Lactate (D3)	Glx (D3)	NAA (D15)	Cho (D15)	Cr (D15)	ml (D15)	Lactate (D15)	Glx (D15)
1	29	E	Physical assault	5.6	3.4	4.5	2.5	1.4	1.6	5.5	3.4	4.3	2.9	1.5	1.7
2	25	E	Penetrating or sharp object injury	6.1	3.7	4.7	2.4	1.3	1.6	6.0	3.9	4.8	3.2	1.5	2.1
3	36	E	Physical assault	5.5	3.6	5.4	2.8	1.1	1.5	7.2	5.1	4.4	1.9	1.4	0.9
4	29	K	Traffic accident	7.2	4.4	5.9	3.2	2.6	2.0	3.6	2.3	2.9	2.4	1.2	1.3
5	26	E	Traffic accident	6.8	4.2	4.5	1.3	2.7	1.4	3.7	2.6	3.2	2.4	1.4	1.4
6	23	K	Penetrating or sharp object injury	5.2	3.2	3.0	1.2	2.1	0.7	3.4	2.1	2.4	1.3	0.8	1.1
7	42	E	Physical assault	3.8	2.4	3.6	2.5	0.9	1.6	4.4	2.9	2.7	1.0	2.0	0.7
8	46	E	Physical assault	7.9	4.6	5.0	1.1	2.4	1.0	3.8	2.3	3.3	2.1	0.9	1.1
9	22	K	Physical assault	4.5	2.3	3.0	1.5	1.2	0.9	4.2	2.5	3.0	1.3	2.0	0.9
10	24	E	Physical assault	4.9	2.9	3.4	1.4	2.6	0.8	4.3	2.4	2.9	0.9	1.7	0.8
11	36	E	Traffic accident	3.6	2.4	2.9	1.6	1.0	0.8	4.6	3.0	3.0	0.7	2.7	0.5
12	30	E	Traffic accident	3.6	2.2	2.3	1.2	0.9	0.7	3.5	2.4	2.1	1.0	1.6	0.5
13	23	K	Traffic accident	4.7	2.9	2.7	0.7	2.0	0.4	5.9	3.2	4.3	2.5	1.7	1.6
14	49	E	Physical assault	3.6	2.4	3.0	1.9	1.3	0.9	3.4	2.1	2.8	1.3	1.4	0.9
15	38	E	Traffic accident	4.3	2.3	3.6	1.8	1.4	1.1	4.1	2.6	3.2	2.1	1.3	1.5
16	38	K	Physical assault	6.8	4.4	5.0	0.9	1.3	0.8	5.0	2.8	3.6	1.0	2.6	1.1
17	23	E	Physical assault	4.9	2.5	3.0	0.7	2.0	0.6	6.8	3.8	4.3	0.7	4.0	1.0
18	28	E	Physical assault	4.5	2.4	2.6	0.4	1.9	0.4	5.2	1.9	2.6	0.8	2.4	0.9
19	33	K	Physical assault	3.6	2.4	3.1	0.8	1.5	0.7	4.2	3.1	3.3	1.2	1.8	0.9
20	33	E	Traffic accident	3.3	2.1	2.7	2.1	2.4	0.9	1.8	1.3	1.4	1.0	1.5	1.7
21	36	E	Traffic accident	3.5	2.1	3.4	2.4	1.5	1.2	4.2	2.4	2.8	1.6	1.5	2.1
22	21	E	Traffic accident	6.1	3.5	3.8	0.9	1.7	0.8	5.9	3.7	3.5	1.1	1.4	0.9
23	48	K	Fall from height	6.3	4.6	3.9	0.6	0.9	0.9	4.8	2.9	3.1	0.9	1.2	1.3
24	44	E	Traffic accident	5.6	3.9	3.8	0.9	1.2	0.4	4.9	3.2	3.2	1.4	1.4	1.4
25	30	K	Crushing or twisting injury	6.4	4.5	5.6	0.7	1.8	0.6	4.7	2.9	4.0	1.3	0.8	1.1
26	21	E	Traffic accident	6.3	3.7	3.9	0.7	1.6	0.7	6.0	3.9	3.5	1.6	2.0	0.7

NAA, N asetilaspartin; Cho, kolin; Cr, kreatin; ml, miyoinozitol; Glx, glutamat-glutamin

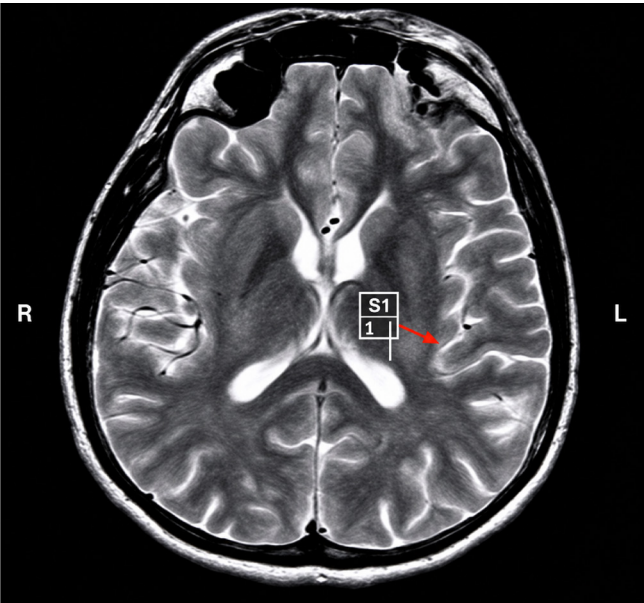


Figure 1. Spectral Pattern of MR Spectroscopy
Voxel placement in the thalamic region for proton MR spectroscopy.

brain computed tomography (CT) scans were included in the study. The emergency physician assessed the patients' overall clinical status and categorized it as moderate or good. The evaluation criteria were as follows:

- Respiratory rate: 14–20 breaths per minute
- Heart rate: 60–100 beats per minute
- Systolic blood pressure: 90–150 mmHg
- Diastolic blood pressure: 60–100 mmHg
- Glasgow Coma Scale (GCS) score: 14–15

Patients with Revised Trauma Scores between 10 and 17 and who were evaluated as neurologically normal by a neurology emergency consultant were eligible for inclusion.

Patients with chronic illnesses such as epilepsy, paraplegia, diabetes mellitus, or rheumatoid arthritis, those on long-term medication, or those with contraindications to magnetic resonance imaging (MRI) were excluded. Additionally, patients who had used analgesics or any other medications were also excluded.

All participants were thoroughly informed about the study, and written consent was obtained before participation. No sedative agents or contrast media were used during MRI examinations. Brain MRI and thalamic MRS evaluations were performed on the 3rd and 15th days following the trauma. The timing of these measurements was determined based on prior studies investigating post-traumatic changes in metabolite levels.¹⁰

Magnetic Resonance Imaging (MRI) and Spectroscopy (MRS)

MRI scans were performed using a 1.5 Tesla scanner (Exelart, Toshiba, Tokyo, Japan). Images were acquired with an axial T2-weighted fast spin-echo sequence (TR: 4000 ms, TE: 94 ms, FA: 90/160, NEX: 2, FOV: 180×220 mm, matrix: 160×320, slice thickness: 4.5 mm, interslice gap: 1 mm, duration: 1 minute 40 seconds).

The MRS examination targeted the thalamic region and was performed using a single-voxel point-resolved spectroscopy (PRESS) technique (TR: 2000 ms, TE: 136 ms, FA: 90/180, FOV: 16×16 mm, matrix: 1×1024, NEX: 128, duration: 4 minutes 16 seconds) with a voxel size of 1.5×1.5×1.5 cm. The voxel was placed over the thalamus on the slice where the thalamus was most prominently visible, avoiding the lateral ventricles, internal capsule,

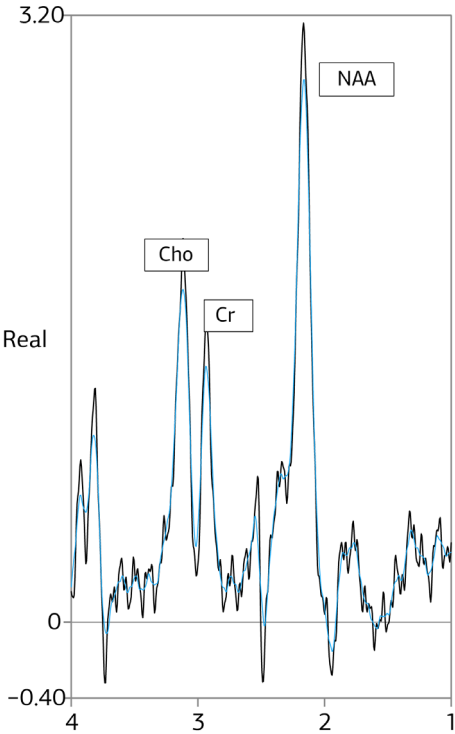


Figure 2. Voxel Localization in MR Spectroscopy
Representative MRS spectral pattern showing the evaluated metabolites.

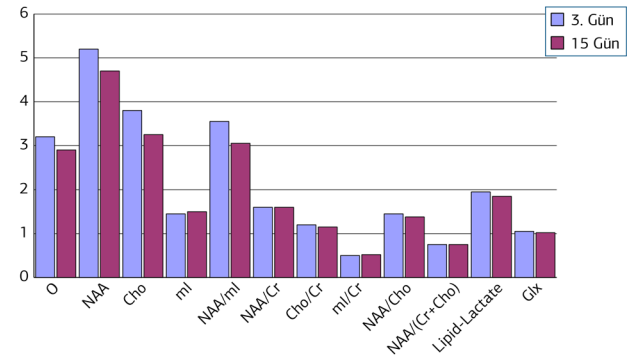


Figure 3. Comparison of Metabolite Levels and Metabolite Ratios on the 3rd and 15th Days After Trauma

Comparison of Cr, NAA, Cho, ml, lactate, and Glx levels and metabolite ratios measured on days 3 and 15 after trauma.

and lentiform nucleus. In the MRS analysis, the signal intensities of the following metabolites were evaluated: N-acetylaspartate (NAA), choline (Cho), creatine (Cr), myo-inositol (ml), glucose, lipid-lactate, and glutamate-glutamine (Glx).

Data Analysis

In our study, to more effectively demonstrate the measured metabolite changes and better highlight statistical differences, we preferred to use metabolite ratios relative to each other, utilizing an internal reference such as Cr (creatine), rather than relying solely on the raw values obtained from thalamic MRS examinations on the 3rd and 15th days after trauma.

MR spectroscopy data were acquired using the scanner's MRS unit. The parts per million (ppm) values of the acquired metabolites were recorded, and the metabolite ratios were calculated.

Ethical Approval

This study was approved by the Ethics Committee of Cumhuriyet University Faculty of Medicine (Date: 02.06.2009, Decision No: 09/152).

Statistical Analysis

Statistical analyses were performed using SPSS version 14.0 (Statistical Package for the Social Sciences). Paired t tests were used. *P* < .05 was considered statistically significant.

Reporting Guidelines

This study was reported in accordance with the STROBE guideline.

Results

The mean age of the 26 patients included in the study was 32.03 (8.67) years (range: 21–49 years); 18 patients (69.2%) were male, and 8 patients (30.8%) were female. The distribution of trauma causes revealed that traffic accidents and physical assaults were the most common, each affecting 11 patients (42.3%). Less frequent causes included penetrating or sharp object injuries in 2 patients (7.6%), followed by a fall from height and a crushing or twisting injury, each reported in 1 patient (3.8%).

The voxel placement for the MRS examination was standardized across all patients and targeted the thalamic region while avoiding adjacent anatomical structures. Figure 1 illustrates the voxel localization used in the scans. Additionally, Figure 2 presents a representative MRS spectral pattern from a patient, showing the evaluated metabolites, including NAA, Cho, Cr, ml, Lac, and Glx.

In the MRS evaluations conducted on both the 3rd and 15th days following trauma, we measured NAA, Cho, Cr, ml, lipid–lactate, and Glx metabolite levels in the thalamic region. Table 1 presents the distribution of these metabolite concentrations, along with patient demographic characteristics and trauma causes.

The comparison of the metabolites Cr, NAA, Cho, ml, lactate, and Glx, as well as the ratios NAA/Cr, Cho/Cr, ml/Cr, NAA/Cho, NAA/(Cr+Cho), and NAA/ml, obtained through thalamic MRS examinations on the 3rd and 15th days following trauma, is presented in Table 2, Table 3, and Figure 3.

When comparing the Cr, NAA, ml, lactate, and Glx levels, as well as the NAA/Cr, Cho/Cr, ml/Cr, NAA/Cho, NAA/(Cho+Cr), and NAA/ml ratios measured on the 3rd and 15th days, no significant differences were found (*P* > .05). However, when comparing the Cho values between the 3rd and 15th days, a statistically significant increase in choline on the 3rd day was observed (*P* < .05).

Table 2. Comparison Of Metabolite Levels Detected On The 3rd And 15th Days After Trauma

Measurements	Day 3	Day 15	t	P value
	(Mean ± SD)			
Cr	3.19 ± 0.88	2.87 ± 0.78	1.59	.123
NAA	5.17 ± 1.31	4.65 ± 1.19	1.73	.095
Cho	3.78 ± 1.00	3.25 ± 0.77	2.91	.007
ml	1.46 ± 0.78	1.52 ± 0.70	0.35	.728
Laktat	1.93 ± 0.89	1.85 ± 0.70	0.38	.701
Glx	1.05 ± 0.46	1.04 ± 0.37	0.07	.939

NAA, *N* asetilaspartat; Cho, kolin; Cr, kreatin; ml, miyoinozitol; Glx, glutamat-glutamin; *P* > .05.

Discussion

This study aimed to noninvasively evaluate neurometabolic changes in the thalamic region during the early/subacute phase following trauma in adult individuals, using proton magnetic resonance spectroscopy (¹H-MRS). Our findings demonstrate a significant increase in choline (Cho) levels in the thalamic region on the 3rd day after trauma. In contrast, we observed no statistically significant changes in NAA, Cr, ml, Lac, or Glx levels. These results suggest that the early effects of trauma on the central nervous system may be selective and region-specific.

The observed post-traumatic increase in Cho levels is directly associated with alterations in phospholipid metabolism of neuronal cell membranes. The levels of choline-containing compounds increase in the brain after injury because these compounds are essential for both membrane synthesis and degradation processes.^{11,12} The post-traumatic inflammatory response has been previously reported to occur early in sensory integration structures such as the thalamus.¹³

Proton MRS studies of traumatic brain injury (TBI) have shown that increased Cho is associated with injury severity. Even in mild cases of trauma, raised thalamic Cho is observed, which shows the sensitivity of this region to trauma and the contribution of early glial response.^{14,15} We provide additional insight concerning the timing of this process by demonstrating an increase in Cho levels on the third day post-trauma.

The preservation of NAA levels after injury indicates no significant neuronal integrity loss in the acute phase, as NAA is a neuron-specific metabolite and reflects neuronal loss or dysfunction.¹⁶ The absence of initial changes in NAA levels after trauma indicates that trauma may occur with limited glial activation and that there has not yet been significant neuronal degeneration.^{8,17} Past studies have reported significant reductions in NAA levels post-trauma at later intervals (weeks or greater).¹⁷

The absence of significant changes in Cr and ml levels suggests that energy metabolism and osmotic balance in the thalamus are stable in the acute phase of injury. Cr is typically thought of as a measure of energy metabolism, while ml is typically thought of as a measure of astrocytic activity. The stability of these metabolites in our study supports that early glial proliferation is likely limited to a relative increase in Cho levels.^{18,19}

According to the literature, metabolite ratios (NAA/Cr or Cho/Cr) are often regarded as more sensitive to trauma-related effects. However, the lack of any appreciable change in the observed ratios here suggests that the increase in Cho was only modest in absolute

Table 3. Comparison Of Metabolite Ratios Detected On The 3rd And 15th Days After Trauma

Measurements	Day 3	Day 15	t	P value
	(Mean ± SD)			
NAA/Cr	1.63 ± 0.15	1.63 ± 0.25	0.01	.987
Cho/Cr	1.20 ± 0.19	1.15 ± 0.16	1.03	.311
ml/Cr	0.50 ± 0.30	0.55 ± 0.24	0.72	.478
NAA/Cho	1.43 ± 0.20	1.38 ± 0.23	0.96	.346
NAA/(Cr+Cho)	0.74 ± 0.08	0.76 ± 0.09	0.79	.432
NAA/ml	3.54 ± 1.24	3.06 ± 1.02	0.86	.256

NAA/Cr, *N* asetilaspartat/kreatin; Cho/Cr, kolin/kreatin; ml/Cr, miyoinozitol/kreatin; NAA/Cho, *N* asetilaspartat/kolin; NAA/(Cho+Cr), *N* asetilaspartat/kolin+kreatin; NAA/ml, *N* asetilaspartat/miyoinozitol; *P* > .05.

value and that the stable Cr affected the ratios. This evidence suggests that, after trauma, the thalamus may display metabolic adaptation mechanisms in the first days.²⁰

There may also be different viewpoints regarding deep brain structures such as the thalamus and trauma. Some researchers argue that the thalamus is more susceptible to the secondary effects of trauma because of its dense connections with white matter;¹¹ however, others suggest that inflammation is a more significant contributor to trauma injuries than mechanical force.¹² We contribute to this area of exploration by associating the initial Cho increases with early inflammatory responses.

Future studies should utilize a larger sample size and conduct multi-regional metabolite measurements (hippocampus, cingulate gyrus) to provide more insight into metabolic change in the brain. Investigators should ideally use longitudinal designs (testing at 1 week, 1 month, and 3-month intervals) to assist in understanding the timeline of metabolic changes to TBI. Also, correlating metabolic changes with clinical measures (levels of consciousness, cognitive testing, and motor functions) would greatly improve understanding of trauma physiology.^{8,17}

In conclusion, our research indicates restricted but selective metabolic changes in the thalamic area via proton MRS in the lower-injury cohort during the early post-injury period. Proton magnetic resonance spectroscopy is a useful, noninvasive biomarker for assessing metabolic change and can serve as an early biomarker for pre-intervention assessment in acute neurological treatment development. Further research is needed on changes in trauma-related metabolite levels to determine trauma severity and long-term prognosis better.

Limitations

The current study had limitations, including a small sample size, assessment of only the thalamic region, and a lack of clinical correlations such as cognitive or neurological outcome scores. Additionally, confounding factors such as stress, pain, and medication history were not fully controlled. These limitations may reduce the generalizability of our findings.

Conclusion

In adult individuals exposed to trauma, a significant increase in Cho levels was observed in the thalamic region during the early phase following injury. In contrast, no significant changes were detected in the levels of NAA, Cr, ml, Lac, and Glx, or in metabolite ratios. Proton MRS has the potential to noninvasively assess the early metabolic effects of trauma on the central nervous system. It may contribute to the early detection of trauma-related neurological changes.

Our study demonstrates that the acute effects of trauma induce limited and selective metabolic alterations in the thalamus. These findings provide valuable information that may help plan early intervention strategies.

Declarations

Animal and Human Rights Statement

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed Consent

Written informed consent was obtained from all participants.

Data Availability

The datasets used and/or analyzed during the current study are not publicly available due to patient privacy reasons but are available from the corresponding author on reasonable request.

Conflict of Interest

The authors have no conflicts of interest to declare.

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Author Contributions (CRediT Taxonomy)

Conceptualization: K.K., İ.Ö.
Methodology: K.K., İ.Ö.
Formal Analysis: K.K.
Investigation: K.K.
Data Curation: K.K.
Writing – Original Draft Preparation: K.K.
Writing – Review & Editing: K.K., İ.Ö.
Supervision: İ.Ö.

Abbreviations

CT: Computed tomography
GCS: Glasgow coma scale
MRI: Magnetic resonance imaging
NAA: N-acetylaspartate
PRESS: Point-resolved spectroscopy

References

1. Almidgad A, Mustafa A, Alazaydeh S, Alshawish MM, Bani Mustafa M, Alfukaha H. Bone fracture patterns and distributions according to trauma energy. *Adv Orthop*. 2022;2022(1):8695916. doi:10.1155/2022/8695916

2. Fedeli U, Pierobon S, Grande E, Nordio M. Trends in chronic kidney disease-related mortality before and during the pandemic in Northeastern Italy. *J Nephrol*. 2024;37(2):269-278. doi:10.1007/s40620-024-02020-8

3. Rosner J, de Andrade DC, Davis KD. Central neuropathic pain. *Nat Rev Dis Primers*. 2023;9(1):24.

4. Posa L, De Gregorio D, Boccella S, Smart K. Interplay between chronic pain and affective-cognitive alterations: shared neural mechanisms, circuits, and treatment. *Front Pharmacol*. 2025;10(1):1605670. doi:10.3389/fphar.2025.1605670

5. Li D, Mai JW, Deng J, et al. A neural circuit from thalamic paraventricular nucleus via zona incerta to periaqueductal gray for the facilitation of neuropathic pain. *Neurobiol Dis*. 2024;202(1):106382. doi:10.1016/j.nbd.2024.106699

6. Gustin SM, Wrigley PJ, Youssef AM, et al. Thalamic activity and biochemical changes in individuals with neuropathic pain after spinal cord injury. *Pain*. 2011;152(5):1026-1035.

7. Baslow MH. Functions of N-acetyl-L-aspartate and N-acetyl-L-aspartylglutamate in the vertebrate brain: role in glial cell-specific signaling. *J Neurochem*. 2000;75(2):453-459. doi:10.1046/j.1471-4159.2000.0750453.x

8. Wang AM, Pradhan S, Coughlin JM, et al. Assessing brain metabolism with 7-T proton magnetic resonance spectroscopy in patients with first-episode psychosis. *JAMA Psychiatry*. 2019;76(3):314-323. doi:10.1001/jamapsychiatry.2018.3637

9. Ostojic J, Kozic D, Ostojic S, et al. Decreased cerebral creatine and N-acetyl aspartate concentrations after severe COVID-19 infection: a magnetic resonance spectroscopy study. *J Clin Med*. 2024;13(14):4128. doi:10.3390/jcm13144128

10. Vagnozzi R, Signoretti S, Cristofori L, et al. Assessment of metabolic brain damage and recovery following mild traumatic brain injury: a multicentre proton magnetic resonance spectroscopic study in concussed patients. *Brain*. 2010;133(11):3232-3242. doi:10.1093/brain/awq200

11. Bartnik-Olson BL, Alger JR, Babikian T, et al. The clinical utility of proton magnetic resonance spectroscopy in traumatic brain injury: recommendations from the ENIGMA MRS working group. *Brain Imaging Behav*. 2021;15(1):504-525. doi:10.1007/s11682-020-00330-6

12. Swanberg KM, Campos L, Abdallah CG. Proton magnetic resonance spectroscopy in post-traumatic stress disorder—updated systematic review and meta-analysis. *Chronic Stress*. 2022;6(1):1-20. doi:10.1177/24705470221128004

13. Veeramuthu V, Seow P, Narayanan V, et al. Neurometabolites alteration in the acute phase of mild traumatic brain injury (mTBI): an in vivo proton magnetic resonance spectroscopy (1H-MRS) study. *Acad Radiol*. 2018;25(11):1446-1455.

14. Javaid S, Farooq T, Rehman Z, et al. Dynamics of choline-containing phospholipids in traumatic brain injury and associated comorbidities. *Int J Mol Sci*. 2021;22(21):11313. doi:10.3390/ijms222111313

15. Hillary FG, Liu WC, Genova HM, Maniker AH, DeLuca J. Examining lactate in severe TBI using proton magnetic resonance spectroscopy. *Brain Inj*. 2007;21(6):627-635. doi:10.1080/02699050701426964

16. Visser K, de Koning ME, Ciubotariu D, Kok MGJ, van der Naalt J, Haitsma IK. An exploratory study on the association between blood-based biomarkers and subacute neurometabolic changes following mild traumatic brain injury. *J Neurol*. 2024;271(4):1985-1998. doi:10.1007/

s00415-023-12146-7

- 17.** Amorini AM, Lazzarino G, Di Pietro V, et al. Severity of experimental traumatic brain injury modulates changes in concentrations of cerebral free amino acids. *J Cell Mol Med.* 2017;21(3):530-542. doi:10.1111/jcmm.12998
- 18.** Lin JC, Mueller C, Campbell KA, et al. Investigating whole-brain metabolite abnormalities in the chronic stages of moderate or severe traumatic brain injury. *PM R.* 2022;14(6):661-671.
- 19.** Lefebvre G, Chamard E, Proulx S, et al. Increased myo-inositol in primary motor cortex of contact sports athletes without a history of concussion. *J Neurotrauma.* 2018;35(7):1035-1041. doi:10.1089/neu.2017.5254
- 20.** Berger L, Holshouser B, Nichols JG, Pivonka-Jones J, Ashwal S, Bartnik-Olson B. White matter metabolite ratios predict cognitive outcome in pediatric traumatic brain injury. *Metabolites.* 2023;13(7):778. doi:10.3390/metabo13070778

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