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SYSTEMATIC REVIEW OPEN ACCESS

Neuroinflammatory and Structural Brain Changes Associated with Non-alcoholic Fatty Liver Disease: A Systematic Review

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ABSTRACT

Background: From the past few years, clinicians have identified non-alcoholic fatty liver disease (NAFLD) as a common health issue that has effects that go beyond the liver dysfunction. The given systematic review is aimed at analyzing the relationship between NAFLD and compromised brain outcomes based on examination of imaging data and cognitive performance, and systemic inflammatory markers. **Methodology**: The research was carried out according to the PRISMA 2020 principles, involving the search of the literature in PubMed, Scopus, and Embase databases. Research which was associated with adult patients with NFLT was incorporated. Exclusions were made on reviews, editorials and case reports. A narrative synthesis was established from the included studies. Risk of bias was also assessed using appropriate tools. **Results:** A moderate but significant effect of NAFLD on brain alterations was found. The results suggested that brain volume was reduced, cortical brain areas were diminished, and lower scores on tests of cognitive ability were observed. Both MR and animal studies showed the cause and effect for this inflammation as the middle point. **Conclusion:** Structural brain changes are caused in research results by NAFLD, thus supporting the liver-brain connection. For future research, integrated management strategies for both liver health and brain health is much needed.

Keywords: Non-alcoholic Fatty Liver Disease, Brain, Cognitive Dysfunction, Neuroimaging, Magnetic Resonance Imaging, Inflammation, Precision Medicine

Introduction

Nonalcoholic fatty liver disease (NAFLD) had come to attention due to the damage it causes to normal liver. Brain liver connection occurs during disease progression, and according to the research findings, NAFLD played a role in neurological changes [1]. NAFLD patients suffered cognitive decline as well as brain volume and cortical changes, which might cause neuroendocrine damage [2]. As NAFLD is a global health problem, understanding the two pathways between the damage to the liver and brain dysfunction is urgently required, since both conditions accompany obesity and T2DM [3].

Recent neuroimaging and mendelian randomization studies had shown that liver fat accumulation and fibrosis had a severe effect on the cortex as well as the hippocampus and poor cognitive function [4]. These results indicated that NAFLD is not only a hepatic dysfunction but also a neuro metabolic condition that affects higher brain regions responsible for memory, executive function and emotional regulation. The reports suggested the sources for key signaling components which keep these two systems in the constant 'crosstalk' including inflammatory mediators such as C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) etc., as well as immunological disruptions like Th17/Treg imbalance (T Cells) [5]. These mediators might cause neuroinflammation, blood brain barrier disruption, and neuronal apoptosis, and in doing so, associate hepatic pathology to central nervous system decline [6]. Studies had revealed that the association between NAFLD and brain structure changes was a population specific vulnerability and the association was modulated by variables including age, sex, metabolic syndrome status and ethnicity [7,8].

This systematic research and analysis were geared to compile already existing NAFLD studies on brain structure changes and cognitive function effects with the coexistence of clinical imaging, genetic studies and functional connectivity outcomes. The effects of NAFLD on brain structures and mental processing could be explained by means of inflammation and metabolic problems. The aim of this study was to have a complete analysis for developing approaches of precision medicine and neurological risk screening protocols in metabolic disease treatment.

Methodology

In accordance to the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines published in 2020 [9], this review was performed.

Inclusion and Exclusion Criteria

Studies with defined research methods and data from adult NAFLD or patients with related liver characteristics were included. Studies including include reviews, editorials nor case reports in the evaluation were excluded.

Data Sources and Search String used

To systematically search of the literature, the databases PubMed, Embase and Scopus were used. The time frame of research studies was from 2017 till 2024. The search terms used in databases for literature search were "NAFLD", "brain" "cognition", "neuroimaging", "cortical thickness", "grey matter", and "functional connectivity." In the research study the eligibility criteria were determined using the Population, Intervention, Comparison, Outcome, and Study (PICOS) framework definitions [10].

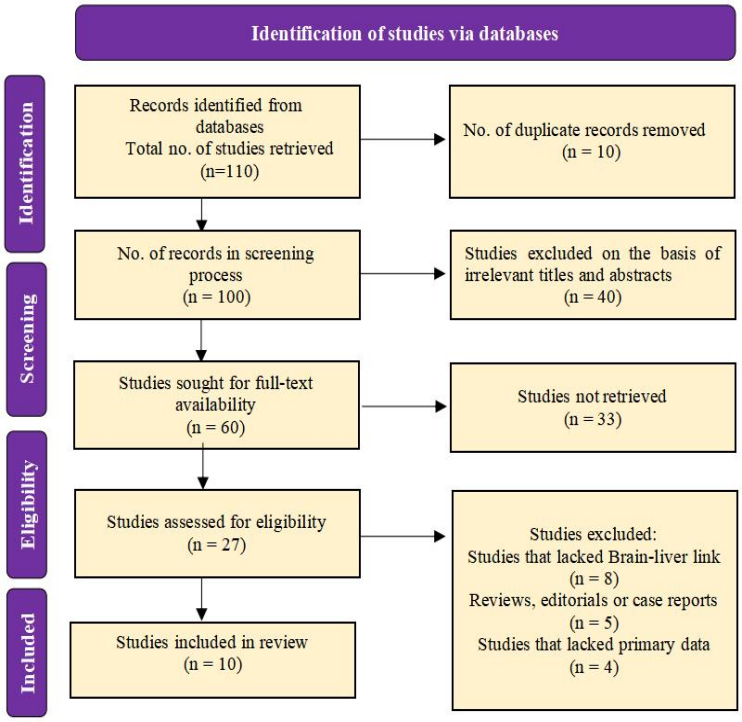

Figure 1: The PRISMA Flow Diagram was designed according to the PRISMA guidelines 2020, showing study identification, screening, assessment of eligibility, and final selection in the systematic review.

Study Selection and Data Extraction

Two independent reviewers carefully screened the data from identified sources. Imaging and liver enzyme marker, and genetic proxy testing, were primary exposures of NAFLD assessment during this phase. Studies that assessed neither of this parameter were excluded by the reviewers. In case of any disagreement, a consensus decision with a third reviewer was followed. The data were extracted by two reviewers independently. Information on study size, design type, mechanism, effects of brain-liver axis, cognitive measurement results, imaging data and statistical values were collected by the investigators. The study designs consisted of cross-sectional studies, pre-clinical examinations with Mendelian randomization (MR) work, and observational analysis as well as translational applicable studies.

Primary Outcome and Quality Assessment

A comparison was made to healthy people or those not having liver disease. Structural or functional changes in the brain (MRI, fMRI); cognitive scores; inflammatory biomarkers were considered primary outcomes. We used the Newcastle-Ottawa Scale Version 2011 for observational studies and the Cochrane Risk of Bias tool Version 2 for MR-based studies as two tools of assessment of risk of bias [11]. Because there were multiple study designs, a random effects analysis was required for the sake of measuring anticipated outcomes and methodologic variations. GRADE approach was used to determine certainty of evidence.

Results

The selection of the studies followed PRISMA rules and shown in Figure 1. Database search identified 110 records which were initially identified. After removing 10 duplicate records, ten articles were left to go through the title and abstract screening. Among them, 40 records were eliminated because they had no association to brain and liver interactions. Sixty full-text articles were identified in the retrieval process, though full-text was not available for 33 articles. As a result, a total of 27 full text articles were evaluated in terms of eligibility. At this step, 17 studies were dropped: eight articles did not even examine a definite brain liver relationship, five were reviews, editorials, or case studies and four articles did not include presuppositional data. Finally, 10 papers that satisfied the inclusion criterion were included in the qualitative synthesis.

A summary of the study characteristics included was represented in Table 1. The chosen articles were published in the period of 2022-2024 and included a variety of study and research designs cross-sectional neuroimaging studies (n = 4), Mendelian randomization probability analyses (n = 3), large population-based observational cohort studies (n = 2), and one pre-clinical longitudinal animal study. The sample size of the studies was diverse; in a study with a focused fMRI the sample consisted of 64 participants, whereas in a study with genetic and imaging consortia more than 800,000 people participated.

Table 1: Summarized view of characteristics of 10 studies selected

Author (Year)	Study Design	Sample Size	Brain–Liver Crosstalk Mechanism	Outcomes Assessed	Key Findings	Risk of Bias
Xu et al. (2023) [12]	Cross-sectional fMRI study	64	Alterations in ALFF, ReHo, FC in temporo-occipital, cerebellar, and sensorimotor areas	MoCA, DST, TMT-A, RS-fMRI markers	Non-obese NAFLD linked to abnormal brain activity; cognitive decline	Moderate
Li et al. (2023) [13]	Cross-sectional (functional connectivity analysis)	189	Disrupted intra-/inter-network topologies in DMN, visual, executive, and sensorimotor networks; fALFF and dynamic FC alterations.	MMSE, MoCA, FC metrics (fALFF, SFNC, DFNC, DR)	The T2D + NAFLD group showed greater brain dysfunction, linked to metabolic markers.	Moderate
Weinstein et al. (2023) [14]	Cross-cohort observational MRI study	5660 (NAFLD); 3022 (Fibrosis)	Liver fibrosis/NAFLD is associated with reduced brain, gray matter volumes.	Brain volumes (total, GM, hippocampus), WMH	NAFLD and fibrosis are linked to reduced brain volumes; implications	Low
Mai & Mao (2023) [15]	Mendelian Randomization	>700,000 (GWAS + MRI)	Genetic instruments for liver traits (ALT, NAFLD, PLF) linked with cortical SA, TH changes	Cortical surface area and thickness (34 brain regions)	Liver fat levels are associated with regional cortical thinning, area loss	Low
Lin et al. (2024) [16]	Mendelian Randomization	342,499 (NAFLD cases + controls), 51,665 (MRI)	Genetic variants for NAFLD, fibrosis, and activity score linked to cortical structure	Global and regional cortical SA and TH	NAFLD is linked to reduced frontal SA, increased TH in some areas	Low
Yilmaz et al. (2023) [17]	Cross-sectional (Rotterdam Study)	3,493	Subclinical liver traits are linked to changes in the volume of the brain and perfusion.	TBV, GM/WM volumes, CBF, cognitive tests	GGT and steatosis are associated with lower TBV, GM, and brain perfusion	Low
Yoshikawa et al. (2023) [18]	Literature Review	N/A	Th17/Treg imbalance via PI3K/AKT/mTOR; MAFLD-linked CNS inflammation	Neurodegeneration; psychiatric links	Immune dysregulation links MAFLD to depression and Parkinson's	High
Jiang et al. (2023) [19]	Cross-sectional (UK Biobank)	Up to 447,626	Liver fibrosis effects on the brain mediated by systemic inflammation (CRP)	Cognitive function (11 tests), GMV (MRI)	Fibrosis is linked to reduced GMV and cognition; CRP mediation supports the inflammatory axis.	Low
Mao et al. (2024) [20]	Mendelian Randomization	>800,000 (genetics + MRI)	NAFLD-linked traits causally impact cortical SA and TH	MRI-based cortical SA and TH	Liver fat is associated with reduced parahippocampal SA, cortical TH	Low
Nucera et al. (2022) [21]	Preclinical longitudinal animal study	DIAMOND mice (multiple timepoints)	MAFLD-induced systemic inflammation affects the brain	1H-MRS, cerebral volume, liver histology, neuronal morphology	MAFLD progression caused brain volume loss, neuroinflammation, and degeneration	Moderate

ALFF: Amplitude of Low-Frequency Fluctuations; ReHo: Regional Homogeneity; FC: Functional Connectivity; fALFF: fractional Amplitude of Low-Frequency Fluctuations; SFNC: Static Functional Network Connectivity; DFNC: Dynamic Functional Network Connectivity; DR: Dynamic Regional Homogeneity; MoCA: Montreal Cognitive Assessment; DST: Digit Span Test; TMT-A: Trail Making Test, Part A; MMSE: Mini-Mental State Examination; MRI: Magnetic Resonance Imaging; GWAS: Genome-Wide Association Study; SA: Surface Area; TH: (Cortical) Thickness; TBV: Total Brain Volume; GM: Gray Matter; WM: White Matter; WMH: White Matter Hyperintensities; CBF: Cerebral Blood Flow; CNS: Central Nervous System; CRP: C-Reactive Protein; ALT: Alanine Aminotransferase; NAFLD: Non-Alcoholic Fatty Liver Disease; MAFLD: Metabolic Dysfunction-Associated Fatty Liver Disease; PLF: Proton (Liver) Fat Fraction; DIAMOND: Diet-Induced Animal Model Of Non-Alcoholic Fatty Liver Disease.

Neuroimaging studies confirmed that the patients with NAFLD had decreased functional connectivity between brain networks that were involved in default mode activities, executive control, and sensorimotor systems. Resting state fMRI scans revealed consistent changes in connectivity metrics of the brain, such as ALFF, ReHo, and DFNC (ALFF: Amplitude of Low-Frequency Fluctuations; ReHo: Regional Homogeneity; and DFNC: Dynamic Functional Network Connectivity), which were more particular among NAFLD patients also diagnosed with T2DM. According to MR analyses, the genetic association between NAFLD resulted in lower surface area (SA) and thickness (TH) levels in cortical regions of the frontal and para-hippocampal cortices. DIAMOND (Diet-Induced Animal Model of Non-Alcoholic Fatty Liver Disease) mouse studies validated the findings by demonstrating how MAFLD (Metabolic dysfunction-associated fatty liver disease) caused systemic inflammation as well as thalamic degeneration. CRP joined other systemic inflammatory markers to form the link between the liver and brain tissue. The risk quality of most of the studies ranged from low to moderate. Risk assessments, excluding the papers with moderate bias strength, did not change the effect strength measurements, thus, the overall analytic stability was validated. The summary of characteristics studied was shown in Table 1. The certainty of evidence was rated as low to moderate by the GRADE assessment.

Discussion

The findings provide strong evidence that NAFLD is linked to negative brain outcomes. The results demonstrated that, even in the absence of obesity, NAFLD and its fibrotic and metabolic forms cause structural and functional alterations in the brain. A liver-brain connection that facilitates the interaction of shared metabolic and immunological systems is supported by several complementary brain assessment techniques, as well as the identification of widespread inflammation.

Numerous studies supported the consistent finding that individuals with NAFLD have smaller brains and lower grey matter tissue density. Scientific evidence confirmed that the hippocampus and frontal cortex undergo significant neuroanatomical changes because these areas regulate memory processing, executive function, along mood control [22]. FMR-based (Functional Magnetic Resonance) research indicated that default mode and executive system dysfunctions, as well as sensorimotor network dysfunctions, were likely the cause of the reported cognitive deficits in patients with NAFLD [23]. Physicians use the MMSE, MoCA, and TMT-A to evaluate the cognitive impairments seen in NAFLD patients, which included memory issues with lack of attention and decreased psychomotor speed [25]. Researchers could obtain more durable interpretations of these relationships through MR studies. Researchers used MR studies to analyze GWAS and MRI data and discovered that genetically predicted liver traits such as liver fat percentage and fibrosis, as well as elevated ALT levels, resulted in cause-specific reductions in cortical surface area and thickness [24].

Research indicated that these effects occur in particular brain regions, though their efficacy varied according to genetic factors and type 2 diabetes. The study offered key evidence that inflammation was a link between liver and brain health [25]. When present in metabolic syndrome and NAFLD conditions, the acute-phase protein CRP served as a crucial link between liver disease and cortical degeneration. Research employing DIAMOND mice supported this mechanism by demonstrating that liver inflammation causes tissue destruction in addition to affecting the health of thalamic neurons [26]. Clinical work data necessitated early neurological screening examination protocols for patients with non-alcoholic fatty liver disease 26. Cognitive evaluations were currently not incorporated into metabolic disorder care practices [27]. The findings indicated that neurocognitive deterioration begun when NAFLD developed, even before the appearance of cirrhotic liver damage or hepatic encephalopathy [28,29]. Long-term neurological complications could be reduced with the effective application of precise medical treatments, which included liver and brain health monitoring [30]. This review highlighted the current gaps in knowledge in the field of study. There had not been enough research interest in evaluating cognitive changes across NAFLD patient populations. Neurocognitive tests were required during research investigations that evaluated the overall therapeutic effects on patients to conduct a comprehensive assessment of liver disease treatments.

The review had certain limitations, among which are the high heterogeneity of the study design, populations, and results of neuroimaging which did not allow carrying out quantitative meta-analysis. Causality could not be inferred because mostly cross-sectional studies were included. Also, the possibility of publication bias, lack of full text retrievals, and use of large secondary data sets could lead to a decrease in the generalizability of the results. In future research, longitudinal and interventional research designs should be given priority to elucidate causal brain to liver pathways. Clear neuroimaging and cognitive tests should be used to facilitate meta-analyses. A combination of multi-omics, inflammatory profiling, and mechanistic animal models could be used to further understand the therapeutic targets to address liver-associated neurocognitive impairment.

Conclusion

In conclusion, NAFLD was directly involved in detrimental brain-related effects that resulted in cognitive deficits and a shrinking of the brain. Liver-brain connectivity was shown to function through inflammation and metabolic dysfunction in several research lines related to neuroimaging, MR, and animal models. Because NAFLD patients required comprehensive care, brain health assessment should be a part of screening and management strategies. To confirm causality and identify treatment points, more research must be carried out with extended and intervention-based examinations. A thorough understanding of the brain-liver correlation is necessary to develop accurate medical treatments for combined metabolic diseases of the liver and brain.

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References

41. Moretti R, Caruso P, Gazzin S. Non-alcoholic fatty liver disease and neurological defects. Ann Hepatol. 2019 Jul-Aug;18(4):563–570. https://doi.org/10.1016/j.aohep.2019.04.007
42. Miao Y, Zhang B, Sun X, et al. The presence and severity of NAFLD are associated with cognitive impairment and hippocampal damage. J Clin Endocrinol Metab. 2023 Nov 17;108(12):3239–3249. https://doi.org/10.1210/clinem/dgad352
43. Park J, Lee EY, Li J, et al. NASH/liver fibrosis prevalence and incidence of nonliver comorbidities among people with NAFLD and incidence of NAFLD by metabolic comorbidities: lessons from South Korea. Dig Dis. 2021;39(6):634–645. https://doi.org/10.1159/000514953
44. Zhang P, Zhang X, Gao B, Gao Y, Pan Y. The impact of metabolic syndrome on the cerebral cortex: a Mendelian randomization study. Cereb Cortex. 2024 Aug 1;34(8):bhae342. https://doi.org/10.1093/cercor/bhae342
45. Gangopadhyay A, Ibrahim R, Theberge K, May M, Houseknecht KL. Non-alcoholic fatty liver disease (NAFLD) and mental illness: mechanisms linking mood, metabolism and medicines. Front Neurosci. 2022 Nov 15;16:1042442. https://doi.org/10.3389/fnins.2022.1042442
46. Latif F, Saqib V, Waqar E. Dementia and non-alcoholic fatty liver disease—an unprecedented relationship. Ann Med Surg (Lond). 2022 Sep 1;81:104359. https://doi.org/10.1016/j.amsu.2022.104359
47. Giuffrè M, Merli N, Pugliatti M, Moretti R. The metabolic impact of nonalcoholic fatty liver disease on cognitive dysfunction: a comprehensive clinical and pathophysiological review. Int J Mol Sci. 2024 Mar 15;25(6):3337. https://doi.org/10.3390/ijms25063337
48. Xia T, Du M, Li H, et al. Association between liver MRI proton density fat fraction and liver disease risk. Radiology. 2023 Oct;309(1):e231007. https://doi.org/10.1148/radiol.231007
49. Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. BMJ. 2021 Mar 29;372:n71. https://doi.org/10.1136/bmj.n71
50. Schardt C, Adams MB, Owens T, Keitz S, Fontelo P. Utilization of the PICO framework to improve searching PubMed for clinical questions. BMC Med Inform Decis Mak. 2007 Jun 15;7:16. https://doi.org/10.1186/1472-6947-7-16
51. Lo CK, Mertz D, Loeb M. Newcastle–Ottawa Scale: comparing reviewers' to authors' assessments. BMC Med Res Methodol. 2014 Apr 1;14(1):45. https://doi.org/10.1186/1471-2288-14-45
52. Xu JL, Gu JP, Wang LY, et al. Aberrant spontaneous brain activity and its association with cognitive function in non-obese nonalcoholic fatty liver disease: a resting-state fMRI study. J Integr Neurosci. 2023 Jan 4;22(1):8. https://doi.org/10.31083/j.jin2201008
53. Li X, Zhang W, Bi Y, et al. Non-alcoholic fatty liver disease is associated with brain function disruption in type 2 diabetes patients without cognitive impairment. Diabetes Obes Metab. 2024 Feb;26(2):650–662. https://doi.org/10.1111/dom.15354
54. Weinstein G, O'Donnell A, Frenzel S, et al. Nonalcoholic fatty liver disease, liver fibrosis, and structural brain imaging: the Cross-Cohort Collaboration. Eur J Neurol. 2024 Jan;31(1):e16048. https://doi.org/10.1111/ene.16048
55. Mai Z, Mao H. Causal effects of nonalcoholic fatty liver disease on cerebral cortical structure: a Mendelian randomization analysis. Front Endocrinol (Lausanne). 2023 Nov 1;14:1276576. https://doi.org/10.3389/fendo.2023.1276576
56. Lin YK, Cai XR, Chen JZ, et al. Non-alcoholic fatty liver disease causally affects the brain cortical structure: a Mendelian randomization study. Front Neurosci. 2024 Jan 8;17:1305624. https://doi.org/10.3389/fnins.2023.1305624
57. Yilmaz P, Alferink LJM, Cremers LGM, et al. Subclinical liver traits are associated with structural and hemodynamic brain imaging markers. Liver Int. 2023 Jun;43(6):1256–1268. https://doi.org/10.1111/liv.15549
58. Yoshikawa S, Taniguchi K, Sawamura H, et al. Metabolic associated fatty liver disease as a risk factor for the development of central nervous system disorders. Livers. 2023 Jan 5;3(1):21–32. https://doi.org/10.3390/livers3010002
59. Jiang R, Wu J, Rosenblatt M, et al. Elevated C-reactive protein mediates the liver–brain axis: a preliminary study. EBioMedicine. 2023 Jul;93:104679. https://doi.org/10.1016/j.ebiom.2023.104679
60. Mao Z, Gao ZX, Ji T, et al. Bidirectional two-sample Mendelian randomization analysis identifies causal associations of MRI-based cortical thickness and surface area relation to NAFLD. Lipids Health Dis. 2024 Feb 23;23(1):58. https://doi.org/10.1186/s12944-024-02043-x
61. Nucera S, Ruga S, Cardamone A, et al. MAFLD progression contributes to altered thalamus metabolism and brain structure. Sci Rep. 2022 Jan 24;12(1):1207. https://doi.org/10.1038/s41598-022-05228-5
62. Filipovic B, Đuric V, Filipovic N, et al. Anatomical brain changes and cognitive abilities in patients with obstructive sleep apnea syndrome and nonalcoholic fatty liver disease. Can J Gastroenterol Hepatol. 2021 Oct 20;2021:8873652. https://doi.org/10.1155/2021/8873652
63. Shu K, Ye X, Song J, et al. Disruption of brain regional homogeneity and functional connectivity in male NAFLD: evidence from a pilot resting-state fMRI study. BMC Psychiatry. 2023 Aug 29;23(1):629. https://doi.org/10.1186/s12888-023-05071-6
64. Hashida R, Nakano D, Yamamura S, et al. Association between activity and brain-derived neurotrophic factor in patients with non-alcoholic fatty liver disease: a data-mining analysis. Life (Basel). 2021 Aug 7;11(8):799. https://doi.org/10.3390/life11080799
65. He X, Hu M, Xu Y, et al. The gut–brain axis underlying hepatic encephalopathy in liver cirrhosis. Nat Med. 2025 Feb;31(2):627–638. https://doi.org/10.1038/s41591-024-03405-9
66. Kjærgaard K, Mikkelsen AC, Wernberg CW, Grønkjær LL, Eriksen PL, Damholdt MF, Mookerjee RP, Vilstrup H, Lauridsen MM, Thomsen KL. Cognitive dysfunction in non-alcoholic fatty liver disease—current knowledge, mechanisms and perspectives. J Clin Med. 2021 Feb 9;10(4):673. https://doi.org/10.3390/jcm10040673
67. Cushman M, Callas PW, Alexander KS, et al. Nonalcoholic fatty liver disease and cognitive impairment: a prospective cohort study. PLoS One. 2023 Apr 14;18(4):e0282633. https://doi.org/10.1371/journal.pone.0282633
68. Parikh NS, Wahbeh F, Tapia C, et al. Cognitive impairment and liver fibrosis in non-alcoholic fatty liver disease. BMJ Neurol Open. 2024 Jan 23;6(1):e000543. https://doi.org/10.1136/bmjno-2023-000543
69. Bajaj JS, Gentili A, Wade JB, Godschalk M. Specific challenges in geriatric cirrhosis and hepatic encephalopathy. Clin Gastroenterol Hepatol. 2022 Aug;20(8):S20–S29. https://doi.org/10.1016/j.cgh.2022.04.035
70. De Cól JP, de Lima EP, Pompeu FM, et al. Underlying mechanisms behind the brain–gut–liver axis and metabolic-associated fatty liver disease (MAFLD): an update. Int J Mol Sci. 2024 Mar 26;25(7):3694. https://doi.org/10.3390/ijms25073694

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