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The Association and Treatment of Metabolic Dysfunction-Associated Steatotic Liver Disease in Obstructive Sleep Apnoea: A Scoping Review

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The Association and Treatment of Metabolic Dysfunction-Associated Steatotic Liver Disease in Obstructive Sleep Apnoea: A Scoping Review

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Abstract

Background

Metabolic dysfunction-associated steatotic liver disease (MASLD) has become the most common cause of chronic liver disease, associated with the increasing prevalence of metabolic syndrome. Inflammatory and metabolic processes in MASLD may be linked to consequences of obstructive sleep apnoea (OSA), including intermittent hypoxia and fragmented sleep. However, the strength of this association remains unclear, and uncertainty surrounds the benefits of continuous positive airway pressure (CPAP) therapy for hepatic outcomes in OSA patients. This scoping review aims to map the current literature and identify gaps in the existing evidence to explore the relationship between MASLD and OSA in molecular level and if treatment of one condition affects the other.

Methods

A systematic search of open-access databases was conducted in accordance with the Arksey and O'Malley framework and PRISMA-ScR guidelines. Eligible studies included randomised controlled trials (RCTs) and observational studies involving individuals with OSA and/or MASLD. A structured extraction framework was used to chart data, followed by descriptive synthesis.

Results

74 studies were included (60 observational, 9 RCTs and 5 other study designs). Observational studies generally supported an independent association between OSA and MASLD, with several identifying nocturnal hypoxia as a potential mechanistic contributor. RCTs

demonstrated limited hepatic benefit from CPAP monotherapy, with improvements more closely related to weight change than correction of OSA alone.

Conclusions

The findings suggest that OSA may contribute to MASLD through both hypoxia-related and metabolic mechanisms; however, adiposity remains a major confounding factor. The largely neutral findings reported in CPAP trials reflect the multifactorial nature of MASLD, indicating that correction of sleep-disordered breathing alone may be insufficient without concurrent strategies targeting metabolic dysfunction.

Key words: MASLD, OSA, CPAP and RCT

Introduction

Obstructive sleep apnoea (OSA) is a common sleep-related breathing disorder caused by recurrent upper airway obstruction during sleep, resulting in episodes of hypopnea (reduced airflow) or apnoea (complete airflow cessation). Accompanying symptoms include loud snoring, oxygen desaturation, fragmented sleep and excessive daytime sleepiness. Using a limited sleep study with polygraphy or full polysomnography, the number of apnoea and hypopnea events per hour is quantified with the apnoea-hypopnea index (AHI) as mild (≥ 5), moderate (≥ 15) and severe (≥ 30).[1] OSA is increasingly recognised as a global health burden; approximately 936 million adults aged 30-69 years worldwide have mild-to-severe OSA, including around 425 million individuals with moderate-to-severe disease requiring

46 treatment.[2] A growing body of evidence has shown that sleep-disordered breathing
47 contributes to rising rates of obesity, insulin resistance, hypertension and dyslipidaemia.[3]
48
49 Metabolic dysfunction-associated steatotic liver disease (MASLD) describes a
50 clinicopathological spectrum ranging from simple steatosis to metabolic steatohepatitis
51 (MASH), fibrosis and cirrhosis in the absence of aetiologies including significant alcohol
52 consumption, viral hepatitis or inherited disorders. Closely associated with the rising
53 prevalence of metabolic syndrome, MASLD is now the most common form of chronic liver
54 disease in developed countries.[4] Previously known as non-alcoholic fatty liver disease
55 (NAFLD), the updated nomenclature emphasises the role of metabolic dysfunction in the
56 underlying pathophysiology of the disease. Evidence suggests that chronic oxidative stress
57 contributes to inflammation, MASH progression and fibrosis as a result of a “two hit”
58 mechanism, or more recent models depicting a parallel “multiple hit” theory driven by
59 genetic factors, gut microbiota alterations and insulin resistance.[5,6]
60
61 Considering the shared metabolic risk factors of both conditions, increasing attention has
62 been directed at the potential overlap between OSA and MASLD. Intermittent hypoxia and
63 sleep fragmentation in OSA are drivers of oxidative stress, dyslipidaemia and impaired
64 glucose tolerance, processes which are catalysts for the development of hepatic steatosis. As
65 a result, interest has increased in whether OSA is a possible contributor to liver injury. The
66 relationship between these two diseases raises another crucial area of investigation: whether
67 continuous positive airway pressure (CPAP) therapy, the gold standard treatment for OSA,
68 provides benefit in decreasing liver biomarkers and reversing steatosis and fibrosis. Presently,
69 studies have yielded conflicting results; therefore, this scoping review aims to systematically

map the current literature and identify the gaps regarding the independent association between OSA and MASLD, as well as the effects of CPAP therapy on liver outcomes.

Methods

The objectives were identified using the PCC (Population, Concept, and Context) framework. *Population* includes adults (≥18 years) diagnosed with OSA and/or MASLD, including steatosis or fibrosis. Paediatric populations were evaluated separately. *Concept* comprises the prevalence and severity of MASLD in OSA cohorts and the effects of CPAP therapy on liver outcomes. *Context* refers to clinical and population-based settings, including primary research studies not limited by geographical location. Systematic reviews, meta-analyses and narrative reviews were analysed separately.

Study Selection and Eligibility Criteria

The scoping review was conducted with a systematic literature search of randomised controlled trials (RCTs) and observational studies in accordance with Arksey and O'Malley's framework and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses for Scoping Reviews (PRISMA-ScR) guidelines.[7,8] The inclusion and exclusion criteria were predetermined and are outlined in Table 1. All paper titles and abstracts, and full-texts were screened by a single reviewer (AC) using the web-based review tool "Covidence". Decisions regarding inclusion were discussed with two academic supervisors to resolve uncertainties and ensure consistency.

Table 1. Eligibility criteria

Inclusion Criteria	Exclusion Criteria
Adults (18 years and older) diagnosed with obstructive sleep apnoea (OSA) and/or metabolic dysfunction-associated steatotic liver disease (MASLD)	Non-human/in vitro, biomechanical-only studies
Studies reporting association, prevalence, continuous positive airway pressure (CPAP), alternative interventions and liver-related outcomes	Studies not reporting OSA–MASLD association or effects of CPAP/interventions on hepatic parameters
Randomised controlled trials, cohort studies, case-control studies and case series	Case reports, editorials and conference abstracts without full articles
Published between 2006 - 2026	Not available in English

Search Strategy, Data Charting, and Synthesis

A structured literature search was carried out in the PubMed (MEDLINE), Embase and Cochrane Library databases up to February 2026. Keywords, key phrases and MESH terms included: *"sleep apnea syndromes"*, *"obstructive sleep apnea"* and *"non-alcoholic fatty liver disease"*. Searches were limited to studies published in English, involving humans and adults. (Appendix A.) The core search string was rerun in PubMed to include systematic reviews in order to identify primary studies not included in the initial search. Additional studies found were manually imported, while full-text articles unavailable through institutional access were retrieved with assistance from a medical librarian.

Table 2. Search strategy. The OSA and MASLD strings were linked using an AND operator.

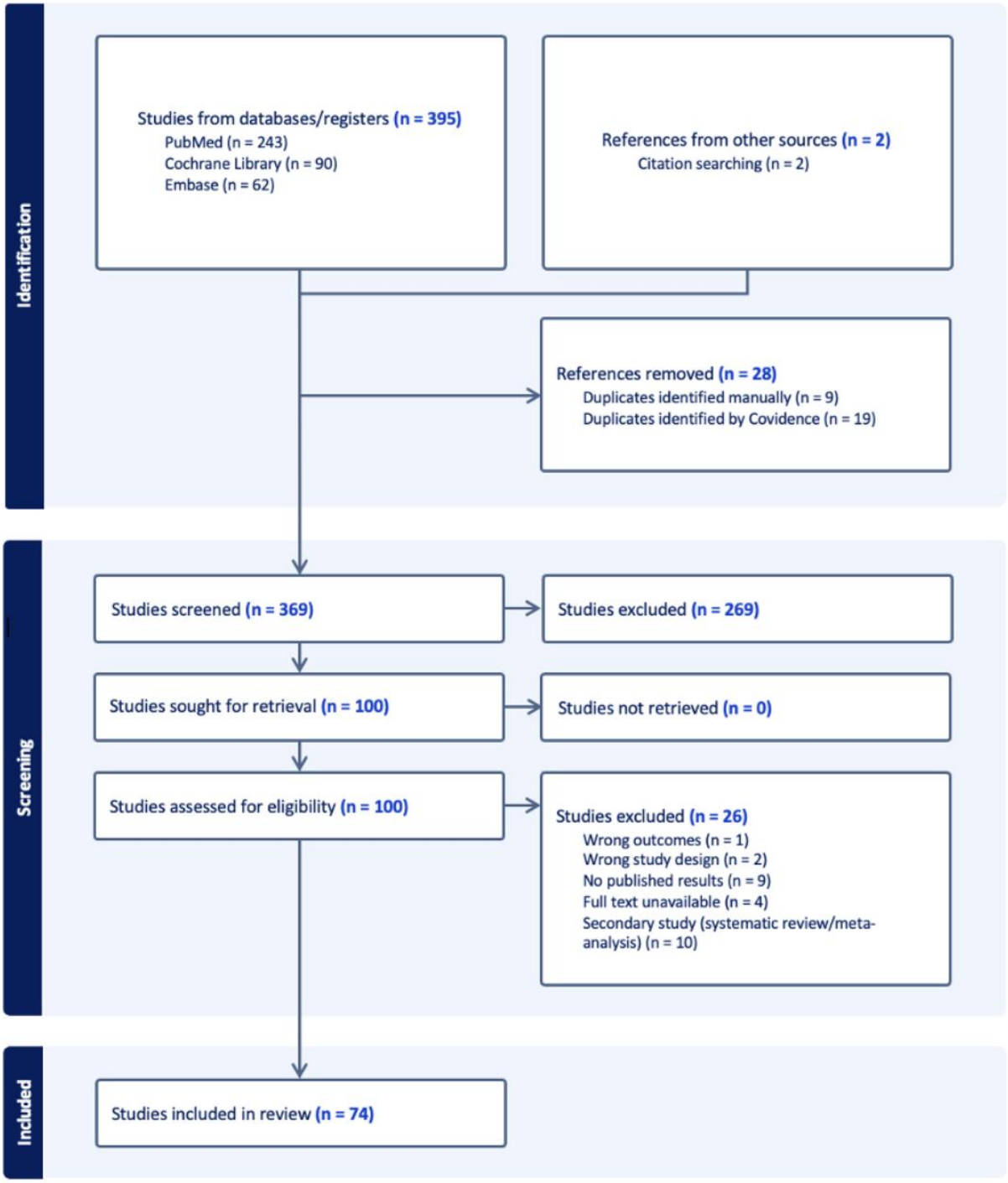
Data were extracted using a structured charting form including study characteristics, population demographics, OSA and MASLD diagnostic criteria, liver-related outcomes and interventional findings. Consistent with scoping review methodology, a formal risk of bias assessment was not performed. The intention of this review was to map and summarise the existing literature rather than to critically appraise individual studies. Data were synthesised descriptively and thematically to identify patterns relating to association, mechanistic pathways and treatment effects.

Results

Overview of Included Studies

A total of 397 studies were identified, and after screening for eligibility, 74 studies were included in this scoping review. (Figure 1.) The majority of the studies were observational in design (n = 60), comprising cross-sectional (n = 46), prospective longitudinal cohort (n = 7) and retrospective longitudinal cohort studies (n = 7). RCTs represented a smaller subset of included studies (n = 9), other designs included Mendelian randomisation (n = 1), cluster phenotyping analysis (n = 1) and diagnostic or risk modelling studies (n = 3). Details of all papers can be found in Additional file 1.

Figure 1. PRISMA-ScR Flow Diagram of Study Selection



Observational Evidence

Association of OSA and MASLD

Across the included observational studies, epidemiological and population-based data demonstrated an independent association between OSA and MASLD, including advanced liver disease phenotypes (e.g. MASH), even after adjusting for metabolic comorbidities. However, considerable heterogeneity was observed in longitudinal cohort studies, showing attenuated associations between OSA and MASLD following multivariable regression for adiposity measures, while some reported persistence in non-obese individuals. Evidence for bidirectionality varied across study designs. Three studies reported an independent reverse association, investigating the increasing stepwise incidence of OSA across liver disease indices.[9-11] Conversely, a cross-sectional study reported that MASLD did not independently predict OSA beyond male sex and obesity.[12]

CPAP findings

Nine observational studies investigated CPAP therapy. Several reported improvements in liver enzymes, steatosis and cytolysis with some associations independent of weight loss. However, this was not universal, and fibrosis parameters generally remained unchanged. Treatment duration and patient compliance were also monitored post-CPAP therapy in two studies, both observing hepatic benefits to be adherence-dependent.[13,14] Additionally, longitudinal population-based data reported reduced cumulative incidence and delayed liver disease progression with CPAP usage.[15]

Mechanistic pathways

Several studies graded the risk and association of liver disease outcomes across OSA severity categories. Increasing apnoea-hypopnea index (AHI), oxygen desaturation index (ODI) and T90 (percentage of total sleep time spent with oxygen saturation below 90%) were correlated with increasing hepatic biomarkers, steatosis and fibrosis risk. Following multivariate

adjustment, AHI was variably attenuated while nocturnal hypoxia indices such as T90 were more consistently retained as independent predictors. Recurrent hypoxia and oxygen burden demonstrated stronger associations with hepatic outcomes than apnoea events. Other cohorts reported links between OSA and impaired glucose tolerance, with higher triglyceride–glucose (TyG) index values independently associated with MASLD prevalence and severity.[16,17]

Randomised Controlled Trials

Population Characteristics

RCTs primarily showed homogeneity across patient demographics. A minority of lean OSA patients were included, as most individuals were likely overweight or obese, mean BMI ranged from approximately 28 to 35 kg/m², with most at a mean BMI of ~30-31 kg/m². RCT populations largely consisted of male subjects, with male-to-female ratios exceeding 2:1. One study investigating cardiometabolic changes assessed CPAP intervention in males only.[18] Participants generally had moderate to severe OSA at baseline, defined by AHI, ODI and Respiratory Event Index (REI). Diagnosis of MASLD was typically based on imaging modalities and serum liver enzymes, with the majority of patients exhibiting steatosis than advanced fibrosis or cirrhosis. Metabolic comorbidities such as hypertension, type 2 diabetes (T2D) and hypercholesterolaemia were reported variably across studies. Ethnic representation differed by geographic location but was often not the primary focus of subgroup analysis.

Duration of CPAP Therapy

Across the included RCTs, CPAP intervention ranged from 4 weeks to 6 months, with most trials lasting between 6 and 12 weeks. Only two studies delivered CPAP therapy for 6 months, including one focusing on the treatment of metabolic syndrome with hepatic steatosis and liver enzymes assessed as a secondary endpoint.[19,20] No randomised trials extended beyond 12 months. Changes in primary hepatic endpoints were assessed at the end of the intervention; however, long-term histological progression and fibrosis regression were not evaluated during the follow-up period of the RCTs.

CPAP Adherence

Objective CPAP adherence was monitored as part of most RCT protocols, with mean nightly usage ranging from 3 to 5 hours. Reporting of adherence data was derived from internal machine clocks and memory card downloads, with phone calls and extra visits if necessary.[18,20] In comparison with subtherapeutic or sham CPAP controls, therapeutic CPAP arms across all RCTs significantly reduced AHI, REI, % total recording time with SaO_2 <90%, minimum SaO_2, excessive daytime sleepiness using the Epworth Sleepiness Scale (ESS) and the Oxford Sleep Resistance Test (OSLER). No consistent association was observed between higher compliance and significant improvements in hepatic serum biomarkers, steatosis or fibrosis when compared with lower adherence groups in subgroup analyses examining adherence thresholds (≥3 vs <3 or ≥4 vs <4 h/night).[21]

Primary and Secondary Endpoints

Included RCTs relied on non-invasive surrogate markers of liver injury. Hepatic steatosis was primarily assessed by proton magnetic resonance derived-IHTG, controlled attenuated parameter (CAP), or CT-derived liver fat indices. Fibrosis was assessed using biomarker algorithms such as FibroMax and elastography-based liver stiffness. Secondary endpoints

included serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), alkaline phosphatase (ALP), bilirubin and cytokeratin-18 fragments. None of the included trials used liver biopsy as a primary outcome to evaluate changes in steatohepatitis, hepatocyte ballooning or histological regression.

Treatment Effect Direction

While CPAP therapy showed to be beneficial in reducing the number of respiratory events and improving nocturnal oxygen saturation in targeting OSA physiology, the majority of RCTs reported a modest and small long-term benefit in addressing metabolic syndrome and insulin sensitivity. Primary and secondary endpoints discussed above did not differ significantly between treatment groups. In the largest 6-month RCT included, between-group differences were not significant as reductions in serum ALT decreased in both therapeutic and subtherapeutic CPAP arms.[19]

Mechanistic Pathways

Mechanistic assessments used included indices of insulin resistance (e.g. HOMA-IR), metabolic and inflammatory markers (e.g. hs-CRP, cortisol, adiponectin and leptin), and lipid parameters. AHI and measures of nocturnal hypoxia, such as ODI and T90, were analysed for independent association with MASLD biomarkers. Select studies assessed changes in fibrosis and extracellular matrix-related biomarkers, including lysyl oxidase, in association with CPAP therapy.[22] Proposed mechanistic pathways in studies reporting reductions in hepatic steatosis markers were chiefly correlated with changes in weight, metabolic dysfunction and adiposity.

Adjunct Therapies

CPAP intervention was predominantly appraised as a monotherapy. The majority of trials compared therapeutic auto-adjusting CPAP to subtherapeutic (~4 cm H_2O) or sham CPAP (~0.5 cm H_2O) without integration of metabolic interventions or lifestyle programmes. In RCTs utilising adjustment models and multivariate linear regression, weight change independently correlated with intrahepatic triglyceride content and CAP rather than CPAP therapy alone.[19] Limited evidence was available regarding adjunctive metabolic therapies and the incorporation of such treatments. In one open-label RCT of oral semaglutide for obesity management, significant weight loss and reductions in liver enzymes, CAP-measured steatosis and liver stiffness were reported, although fibrosis indices, including FIB-4 and NFS, did not significantly change.[23] Only one recent trial assessing the effect of free walking alongside CPAP was included.[24]

Discussion

This scoping review synthesised current evidence investigating the relationship between OSA and MASLD and hepatic outcomes following CPAP therapy, revealing limitations and homogeneity in methodological design. Most data were cross-sectional, demonstrating a paucity in prospective longitudinal studies. Observational data generally support an association between OSA severity and liver injury. However, despite CPAP therapy providing correction of sleep-disordered breathing, RCTs showed limited improvements in MASLD without weight loss. These findings highlight the gap between associative evidence of epidemiological studies, temporality in longitudinal studies and interventional evidence assessing causality.

Across observational cohorts, hypoxia-related metrics demonstrated stronger and more consistent associations with hepatic outcomes than event frequency measures such as AHI. Indicators such as ODI, nadir oxygen saturation and T90 were repeatedly identified as independent predictors of liver injury after adjustment for metabolic factors. In contrast, AHI was frequently attenuated and lost significance following multivariate regression. These findings suggest that cumulative hypoxia and oxygen desaturation may be more relevant than the number of respiratory events alone. The “two-hit hypothesis” proposes chronic intermittent hypoxia as the second “hit”, following the initial insult of hepatic fat accumulation and steatosis secondary to insulin resistance. Hypoxia promotes oxidative stress through the generation of reactive oxygen species, leading to lipid peroxidation and pro-inflammatory cytokine activation, with subsequent stellate cell induction and acceleration of steatosis and fibrosis.[25]

Obesity and adiposity emerged as key confounders in the OSA-NAFLD relationship. While crude analyses from observational studies reported a link between OSA severity and liver injury, these associations often attenuated after adjusting for BMI, visceral fat, and metabolic parameters. Findings suggest that obesity partly mediates the relationship between OSA and MASLD. A mediation analysis implied both metabolic and hypoxia-related pathways may contribute to hepatic injury, with BMI accounting for 22% of the association.[26] It is likely that OSA amplifies metabolic injury but may not independently drive advanced liver fibrosis without adiposity.[27,28] Insulin resistance is also shown to be a co-driver of hepatic steatosis, with studies investigating surrogate markers TyG index and METS-IR, demonstrating dose-dependent relationships with MASLD in PSG-assessed patients.[16,17]

The limited hepatic improvements observed in CPAP trials may reflect the multifactorial nature of MASLD. Correcting nocturnal hypoxia alone in OSA appears insufficient to reverse hepatic steatosis if underlying metabolic dysfunction persists. One trial showed neutral metabolic effects and non-significant imaging differences in hepatosteatosis following CPAP after controlling for diet and physical activity.[20] Weight change showed a stronger association with liver improvements, aligning with the newer "multiple-hit" model proposing metabolic, inflammatory and environmental aspects interact to promote liver injury. This model is reinforced by evidence concluding additive lifestyle intervention, such as free walking exercise, enhances hepatic recovery when combined with CPAP.[24] Collectively, these findings suggest that management of MASLD in OSA cohorts may require concurrent therapeutic strategies targeting metabolic risk factors to meaningfully modify liver outcomes.

Emerging evidence points towards a phenotype-dependent relationship between OSA and MASLD, including age, body composition and comorbidities, possibly explaining the heterogeneity across studies. Sleep fragmentation measured by AHI and ageing may be stronger risk factors for liver injury in older adults (over 60 years), distinct from hypoxia and obesity narratives proposed in younger patients.[29] High-risk phenotypes, particularly T2D, may be implicated in MASLD development, emphasising the importance of screening, especially in younger men with pre-existing moderate-to-severe OSA.[30] While obesity is a dominant risk factor linking OSA and MASLD, several population studies reported persistence of association after adjustment for adiposity, suggesting OSA-related hypoxia mechanisms in non-obese populations may visibly contribute more towards liver injury.[31,32] Lean individuals remain underrepresented in RCTs as the role of hypoxia in MASLD and the effects of CPAP in this subgroup remain insufficiently investigated.

Implications for Further Research

Future studies should address the inconsistency between observational associations and the neutral CPAP findings reported in RCTs. Cross-sectional studies suggested that OSA may contribute to the risk of developing or accelerating MASLD over longer timeframes, whereas RCTs evaluated whether CPAP therapy provided short-term benefits in improving liver markers or reversing hepatic steatosis. This distinction between regression and prevention may partly explain the discordance. Future studies should prioritise longer trial durations as most lasted from 4 weeks to 6 months, whereas data show it may take up to 14 years for fibrosis progression.[33] Interventional studies should address the absence of histological endpoints across the current RCT evidence. Surrogate biomarkers such as ALT and AST lack sensitivity for detecting hepatosteatosis or fibrosis and may remain normal despite histologically confirmed disease. While liver biopsy remains the gold standard for assessing liver disease, its invasive nature and risk of complications are practical challenges in RCTs. Non-invasive radiological studies, including ultrasound-based elastography and MRI-proton density fat fraction, remain the most suitable options for assessments. Further research should also investigate the effects of lifestyle intervention, GLP-1 agonists or bariatric treatment alongside CPAP therapy. Additionally, these studies should recruit more diverse populations, as the current RCT cohorts mainly include overweight or obese male participants. Historically, OSA demonstrated an approximate male-to-female ratio of 2:1; however, recent epidemiological data reports rising prevalence among women globally.[34,35] There is also a paucity of paediatric and adolescent literature, despite the growing obesity epidemic and studies which suggest early-life hypoxia may correlate with MASLD severity.[36,37] Overall, mechanistic and longitudinal trials are needed to investigate metabolic dysregulation pathways preceding liver disease, support earlier screening and treatment strategies to modify disease trajectory before adulthood.

Limitations

There are several limitations to consider. First, only articles published in English were included; grey literature and unpublished or supplementary data were excluded. Second, variability in metrics and imaging modalities for OSA exposure and MASLD outcome measurement may limit accurate comparison across studies. Another limitation is the homogeneity of the literature, dominated by observational studies, making it difficult to determine temporality and causality between independent associations reported by cross-sectional designs. Many findings attenuated when adjusted for metabolic comorbidities, emphasising the need for prospective longitudinal and interventional data.

Conclusion

This scoping review mapped the current literature examining the relationship between OSA and MASLD. Observational studies frequently support an association between OSA severity and markers of hepatic steatosis and fibrosis; however, the significance of this correlation varied substantially following adjustment for obesity and metabolic dysfunction. The multifactorial nature of MASLD development beyond chronic intermittent hypoxia is further highlighted in largely neutral RCT results investigating CPAP therapy on liver outcomes, suggesting correction of reduced oxygenation alone may be insufficient in reversing hepatic injury. Overall, findings propose that OSA may contribute to liver disease progression through multiple parallel metabolic and inflammatory “hits” rather than a causal primary disease pathway. The existing evidence underlines important gaps in published literature, including the need for further controlled interventional trials, long-term integrated lifestyle therapies, and inclusion of underrepresented population groups.

Declarations

Authors' contributions

AC, YA and NCRL conceived the study, determined the search strategy, screened studies, extracted data, and drafted the manuscript. YA and NCRL provided methodological guidance and approval of the final manuscript.

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Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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To be added to search strategy, data charting, and synthesis of methods section:

Table 2. Search strategy. The OSA and MASLD strings were linked using an AND operator.

Database	OSA String	MASLD String	Filters	Results (23/02/26)
PubMed	("Sleep Apnea Syndromes"[Mesh] OR "Sleep Apnea, Central"[Mesh] OR "Sleep Apnea, Obstructive"[Mesh] OR obstructive sleep apn*ea[tiab] OR OSA[tiab] OR OSAS[tiab] OR "sleep disordered breathing"[tiab] OR SDB[tiab] OR "apnea hypopnea index"[tiab] OR AHI[tiab] OR "oxygen desaturation index"[tiab] OR	("End Stage Liver Disease"[Mesh] OR "Liver Failure"[Mesh] OR "Non-alcoholic Fatty Liver Disease"[Mesh] OR "Liver Diseases"[Mesh] OR "Liver Cirrhosis"[Mesh] OR "Fatty Liver"[Mesh] OR NAFLD[tiab] OR MASLD[tiab] OR MAFLD[tiab] OR MASH[tiab] OR NASH[tiab] OR "fatty liver"[tiab] OR "hepatic	Search 1: ((humans[Filter]) AND (english[Filter]) AND (alladult[Filter])) Search 2: ((systematicreview[Filter]) AND (humans[Filter]) AND (english[Filter]))	Search 1: 225 Search 2: 18

	ODI[tiab] OR "intermittent hypoxia"[tiab])	steatosis"[tiab] OR steatohepatitis[tiab] OR "steatotic liver disease"[tiab] OR "liver fibrosis"[tiab] OR "liver stiffness"[tiab] OR cirrhosis[tiab])		
Embase	('sleep apnea syndromes'/exp OR 'sleep apnea syndromes' OR 'obstructive sleep apnea'/exp OR 'obstructive sleep apnea' OR 'obstructive sleep apn*ea':ti,ab OR osa:ti,ab OR osas:ti,ab OR 'sleep disordered breathing':ti,ab OR sdb:ti,ab OR 'apnea hypopnea	('nonalcoholic fatty liver'/exp OR 'nonalcoholic fatty liver' OR 'nonalcoholic steatohepatitis'/exp OR 'nonalcoholic steatohepatitis' OR 'metabolic fatty liver'/exp OR 'metabolic fatty liver' OR nafld:ti,ab OR masld:ti,ab OR mafld:ti,ab OR mash:ti,ab OR nash:ti,ab OR 'fatty	([adult]/lim OR [aged]/lim) AND [humans]/lim AND [english]/lim AND ‘clinical trial’/it	60

	index':ti,ab OR ahi:ti,ab OR 'oxygen desaturation index':ti,ab OR odi:ti,ab OR 'intermittent hypoxia':ti,ab)	liver':ti,ab OR 'hepatic steatosis':ti,ab OR steatohepatitis:ti,ab OR 'steatotic liver disease':ti,ab OR 'liver fibrosis':ti,ab OR 'liver stiffness':ti,ab OR cirrhosis:ti,ab)		
Cochrane Library	([mh "Sleep Apnea Syndromes"] OR obstructive sleep apn*ea:ti,ab,kw OR OSA:ti,ab,kw OR OSAS:ti,ab,kw OR "sleep disordered breathing":ti,ab,kw OR SDB:ti,ab,kw OR "apnea hypopnea index":ti,ab,kw OR AHI:ti,ab,kw OR "oxygen	([mh "Non-alcoholic Fatty Liver Disease"] OR NAFLD:ti,ab,kw OR MASLD:ti,ab,kw OR MAFLD:ti,ab,kw OR MASH:ti,ab,kw OR NASH:ti,ab,kw OR "fatty liver":ti,ab,kw OR "hepatic steatosis":ti,ab,kw OR	N/A	90

	desaturation index":ti,ab,kw OR ODI:ti,ab,kw OR "intermittent hypoxia":ti,ab,kw)	steatohepatitis:ti,ab,kw OR "steatotic liver disease":ti,ab,kw OR "liver fibrosis":ti,ab,kw OR "liver stiffness":ti,ab,kw OR cirrhosis:ti,ab,kw)		

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