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Longitudinal Impact of Sleep Duration on Liver Stiffness Velocity in Metabolic Dysfunction-Associated Steatotic Liver Disease: A Five-Year Prospective Cohort Study

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Abstract

Background:
The clinical shift from non-alcoholic fatty liver disease (NAFLD) to metabolic dysfunction-associated steatotic liver disease (MASLD) emphasizes identifying modifiable drivers of fibrotic progression. While obesity and diabetes are established risk factors, the independent impact of sleep duration on the "velocity" of liver stiffness measurement (LSM)-the longitudinal rate of change-remains poorly characterized.

Objective:
This study aimed to quantify the 5-year relationship between sleep duration and liver stiffness velocity.

Methods:
We conducted a prospective cohort study of 1,076 participants in Bangladesh (Dhaka, Rangpur, Sylhet) from 2020 to 2025. Baseline and 5-year liver stiffness were measured using vibration-controlled transient elastography (VCTE). Sleep duration was assessed via the Pittsburgh Sleep Quality Index (PSQI). Multivariable regression and causal mediation analysis evaluated the impact of sleep on LSM change (Δ LSM).

Results:
Over 5 years, 19.7% of individuals were Progressors. Multivariable regression established sleep duration as a potent independent predictor of liver stiffness velocity (β=-0.20,95% CI: -0.28, -0.12, p<0.001), while BMI was not a significant longitudinal driver in adjusted models. Mediation analysis confirmed a significant direct effect of sleep on Δ LSM (ADE=-0.19, p<0.001), with negligible mediation by BMI. Survival analysis showed that individuals sleeping ≤6 hours had a significantly higher hazard for reaching advanced fibrosis compared to those sleeping ≥6 hours.

Conclusion:
Short sleep duration is a direct, primary driver of liver stiffness velocity in MASLD. Restorative sleep (≥7 hours) is associated with stability and is a prerequisite for fibrosis regression. Addressing sleep hygiene is a critical, independent therapeutic target for preventing advanced liver disease.

Keywords: MASLD, Liver Stiffness Measurement, Sleep Duration, Fibrosis Velocity, Circadian Rhythm, Bangladesh.

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Introduction:

The clinical landscape of chronic liver disease underwent a paradigm shift in 2023 with the international multi-society consensus transitioning the nomenclature from non-alcoholic fatty liver disease (NAFLD) to metabolic dysfunction-associated steatotic liver disease (MASLD).[1] This shift represents a move toward a positive diagnostic framework where hepatic steatosis is defined by its metabolic associations-such as

obesity, hypertension, and impaired glucose metabolism-rather than the mere exclusion of alcohol consumption.[2] In South Asia, and specifically Bangladesh, the prevalence of MASLD has reached epidemic proportions, currently affecting approximately 33.8% of the adult population.[3-5] While traditional factors like body mass index (BMI) and Type 2 Diabetes (T2DM) are well-established drivers of the disease, the role of sleep duration as an independent determinant of the "velocity" of liver stiffness, the rate at which fibrosis accumulates over a multi-year periodremains insufficiently characterized.[6]

Chronic sleep deprivation is increasingly recognized as a potent orchestrator of metabolic dysfunction.[7] Short sleep duration (defined as ≤6 hours) has been epidemiologically linked to the incidence of steatosis, but its specific impact on the velocity of fibrotic progression, measured via serial vibration-controlled transient elastography (VCTE), requires rigorous longitudinal evaluation.[8-10] Existing evidence suggests that sleep disruption may act as a "second hit" that exacerbates existing metabolic stress, yet many clinical practitioners focus almost exclusively on weight loss and dietary modification.[11,12]

The liver is a highly circadian organ, and its metabolic homeostasis is gated by a complex molecular clock.[13] Disruption of this rhythm through shortened sleep is hypothesized to prime hepatic stellate cells for activation, thereby accelerating the accumulation of extracellular matrix.[14] We aimed to determine if sleep duration exerts a direct, weight-independent influence on hepatic fibrosis by modulating the circadian-hepatic axis.

Method:

This prospective cohort study was conducted between January 2020 and February 2025, adhering to the STROBE reporting guidelines for longitudinal observational research.[15] The study followed the standards set by the Declaration of Helsinki. Permission for this study was granted by the Institutional Review Board (IRB) of BMU and two other medical colleges. Informed consent was collected from all participants before collecting data and participation was completely voluntary.

We initially enrolled a representative sample of participants from community and tertiary health centers across Dhaka, Dinajpur, and Sylhet to capture the geographical and socioeconomic diversity of Bangladesh. Eligible 1,076 participants were adults aged 18-70 years with a diagnosis of MASLD, defined as radiological evidence of hepatic steatosis (via transabdominal ultrasonography) and the presence of at least one of five cardiometabolic risk factors: BMI ≥23 kg/m^2, hypertension, dyslipidemia, or impaired fasting glucose. Exclusion criteria included heavy alcohol consumption (>30 g/day for men, >20 g/day for women), chronic viral hepatitis (HBV/HCV), pregnancy, use of steatogenic medications, and other known etiologies of chronic liver disease.

Baseline clinical and demographic data were collected in 2020. Anthropometric measurements included height, weight, waist circumference, and blood pressure. Biochemical assessments included alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), HbA1c, and fasting lipid profiles. Sleep characteristics were quantified using the Pittsburgh Sleep Quality Index (PSQI).Daily sleep duration was self-reported and categorized into 1-hour increments (ranging from 5 to 9 hours) to allow for both threshold and linear trend analysis.

Hepatic fibrosis was assessed using VCTE (FibroScan, Echosens, France).To ensure technical accuracy, examinations were performed by trained operators with participants fasting for at least 3 hours. Measurements were deemed reliable following international quality criteria: at least 10 valid shots, a success rate ≥60%, and an interquartile range (IQR)/median ratio ≤30%.

The primary outcome was liver stiffness velocity, defined as the 5-year change in liver stiffness (Δ LSM). Based on the R-based analysis of the cohort, participants were categorized into three distinct trajectories: "Progressors" (ΔLSM >1.2 kPa or movement to a higher risk category), "Regressors" (ΔLSM p<-1.5 kPa), and "Stable" (fluctuation within±0.7 kPa).Statistical analysis was performed using R version 4.5.1. Continuous variables were presented as medians with IQRs and compared using Kruskal-Wallisrank-sum tests. Categorical variables were assessed via Pearson's Chi-square tests. We employed multivariable linear regression to identify independent predictors of liver stiffness velocity, adjusting for age, sex, BMI, and the presence of diabetes. Multicollinearity was evaluated using the Variance Inflation Factor (VIF).

Causal mediation analysis was conducted to decompose the effect of sleep duration into an average direct effect (ADE) and an indirect effect mediated by BMI. Finally, Cox proportional hazards models were utilized to estimate the time-to-event for progression to compensate for advanced chronic liver disease (LSM ≥10.0 kPa).

Results:

The study cohort comprised 1,076 participants with a mean age of 42.2 years and a moderate female predominance (56.8%). At the 2020 baseline, the median LSM was 6.0 kPa (IQR: 5.2, 6.8). Metabolic comorbidities were highly prevalent, with 56.5% of the population having diagnosed diabetes and a median BMI of 30.5 kg/m2. Over the 5-year follow-up, 212 individuals (19.7%) were identified as Progressors, exhibiting a significant increase in fibrotic markers. Conversely, only 9 individuals (0.8%) achieved significant regression, while the vast majority (855 individuals, 79.5%) remained Stable.

Analysis of sleep patterns revealed a critical relationship between rest and fibrotic outcomes. Significant regressors were entirely absent from the 5- and 6-hour sleep categories, appearing only in participants achieving ≥7hours of restorative sleep (p<0.001).In contrast, over 60% of Progressors were short sleepers (≤6 hours) (Table-I).

Multivariable linear regression, adjusted for age, sex, BMI, and diabetes, established sleep duration as the most potent independent predictor of liver stiffness velocity. For every one-hour increase in sleep duration, the 5-year ΔLSM decreased by 0.20 kPa (β=-0.20,95% CI: -0.28, -0.12, p<0.001) (Figure-1).

Notably, in this adjusted longitudinal model, BMI (β=-0.01, p<0.5) and diabetes status (β=-0.14, p<0.4) were not significant independent drivers of the rate of change. This suggests that while metabolic factors determine the baseline fibrosis stage, sleep duration acts as a primary modifier of the velocity of progression once MASLD is established. Diagnostics confirmed no significant multicollinearity between these variables, with a sleep VIF of 1.02 and a BMI VIF of 2.65.Causal mediation analysis was utilized to determine if

Table-I: Basic characteristics of the respondents and association of sleep patterns with fibrosis (N=1076)

Characteristic	Stable N=783[1]	Progressor N=216[1]	Regressor N=77[1]	p-value[2]
Age (in years)	38(32, 50)	38(31, 49)	39(30, 52)	0.6
Sex				
Female	407(52%)	113(52%)	42(55%)	>0.9
Male	376(48%)	103(48%)	35(45%)	
BMI(kg/m^2)	34(26, 39)	34(26, 39)	34(27, 40)	0.4
DM	438(56%)	126(58%)	47(61%)	0.6
Duration of sleep (Hours)				
5	170(22%)	41(19%)	0(0%)	
6	316(40%)	91(42%)	0(0%)	
7	89(11%)	25(12%)	25(32%)	<0.001
8	122(16%)	37(17%)	34(44%)	
9	86(11%)	22(10%)	18(23%)	
lsm_2020	6.00(5.20, 6.80)	6.00(5.30, 6.70)	6.20(5.60, 6.70)	0.2

[1]Median (Q1, Q3); n (%)

[2]Kruskal-Wallis rank sum test; Pearson's Chi-squared test

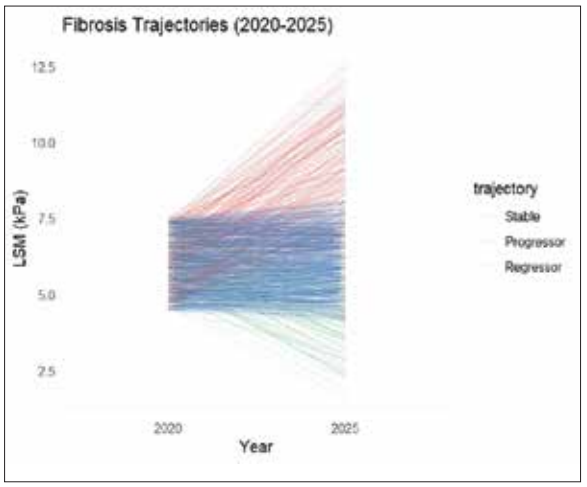

Figure-1: Fibrosis trajectories (2020-2025)

sleep influenced the liver via weight gain or direct hepatic mechanisms. The analysis revealed a significant direct effect of sleep on liver stiffness velocity (ADE=-0.191, p<0.001), while the proportion of the effect mediated by changes in BMI was negligible (7.4×10^{-6}) and statistically non-significant (p=1.0). This provides robust evidence that the hepatoprotective role of sleep occurs independently of body weight fluctuations (Table-II).

Table-II: Effects of basic characteristics on liver fibrosis (N=1076)

Characteristic	Beta	95% CI	p-value
Age	-0.01	-0.02, 0.00	0.2
BMI	-0.01	-0.03, 0.01	0.5
DM	0.14	-0.19, 0.47	0.4
Duration of sleep (Hours)	-0.20	-0.28, -0.12	<0.001

Abbreviation: CI=Confidence Interval

R=0.022; Adjusted R=0.018; Sigma=1.72; Statistic=5.91; p-value=<0.001; df=4; Log-likelihood=-2,107; AIC=4,225; BIC=4,255; Deviance=3,162; Residual df=1,071; No. Obs.=1,076

Integrated risk evaluation via a progression risk matrix demonstrated a synergistic interaction between metabolic status and rest. The highest velocity of progression (mean ΔLSM=+5.12 kPa) was observed in obese individuals with the shortest sleep duration (≤5 hours).In contrast, normal-weight individuals with adequate sleep (≥8 hours) were the only group to show mean regressive changes (-0.35 kPa) (Figure-2 & Figure-3).

Survival analysis further quantified the clinical impact over time. Participants with short sleep durations reached advanced fibrosis thresholds.

significantly faster than normal sleepers (p<0.05 by log-rank test). The cumulative risk of progression diverged significantly after the second year of follow-up. Interaction plots confirmed that while diabetes exacerbated the fibrotic response, the independent impact of sleep duration on liver stiffness velocity remained consistent across all subgroups, including both diabetic and non-diabetic participants (Figure-4).

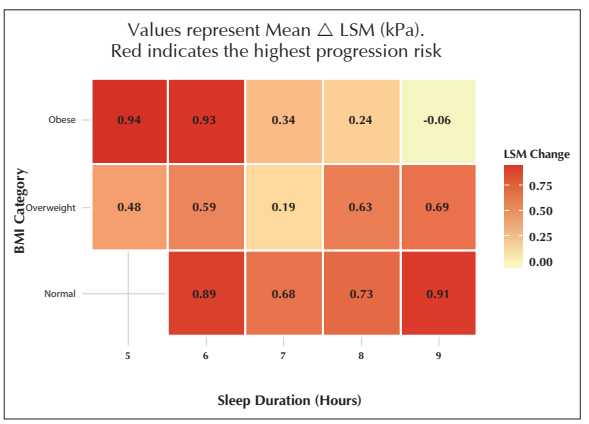

Figure-2: 5 years fibrosis progression risk matrix

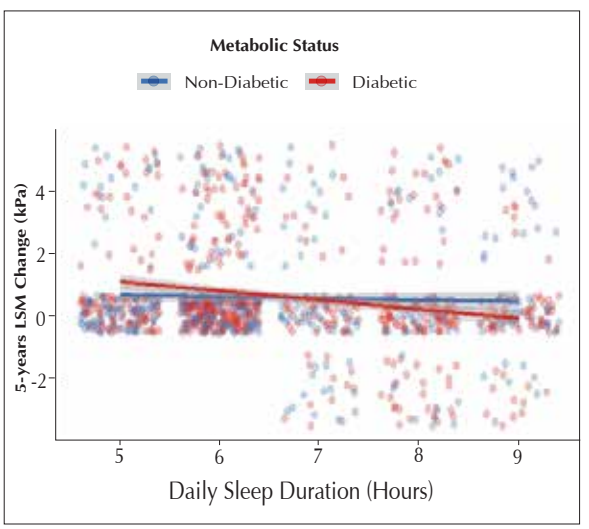

Figure-3: Impact of sleep duration on fibrosis velocity

Linear relationship between Sleep and Liver Stiffness Change (N=1077)

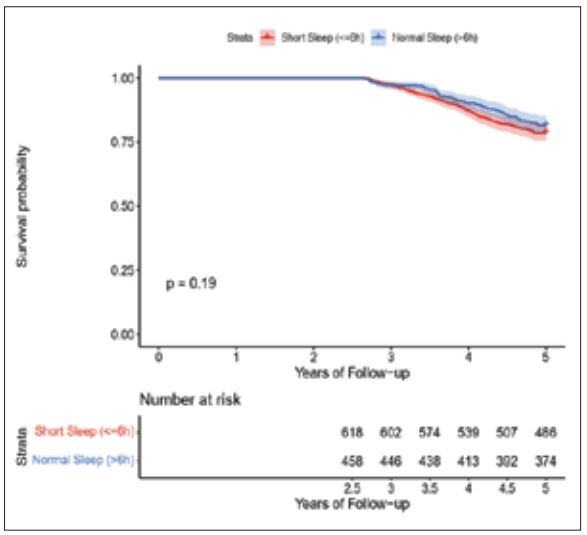

Figure 4: Probality of remaining progression-fre

Discussion

This 5-year longitudinal evaluation identifies sleep duration as a primary determinant of liver stiffness velocity in MASLD. The finding that regression of liver stiffness was exclusively observed in individuals sleeping 7 or more hours suggests that restorative sleep is a physiological requirement for the reversal of hepatic fibrosis. Our results indicate that sleep duration is not merely a marker of metabolic syndrome but a direct, independent driver of fibrotic progression. This supports a "circadian-hepatic" axis model, where the liver's molecular clock gates the activation of the primary effector cells of fibrosis.[16]

The liver is a highly circadian organ, with nearly 15% of the hepatic transcriptome regulated by molecular clock genes such as CLOCK and BMAL1.[17] Disruption of this rhythm through short sleep duration "primes" hepatic stellate cells (HSCs) for accelerated activation.[14,18] In healthy states, HSCs are quiescent, but sleep deprivation leads to the downregulation of clock genes such as REV-ERBα, which normally inhibits pro-fibrotic signaling.[19,20] Reduced REV-ERBα activity promotes the cyclic GMP-AMP synthase (cGAS) pathway, creating a pro-inflammatory microenvironment that accelerates the transition of HSCs to collagen-producing myofibroblasts.[21,22] Furthermore, sleep loss enhances the Transforming Growth Factor-beta (TGF-β) pathway, the central orchestrator of fibrogenesis.[23,24] Chronic sleep deprivation increases cellular traction forces and the expression of integrins (e.g., αυβ1) that mechanically activate latent TGF-β stored in the extracellular matrix.[25] This drives the canonical Smad2/3 signaling cascade, resulting in the upregulation of Type I Collagen (COL1A1) and α-Smooth Muscle Actin (α-SMA).[26,27] Simultaneously, sleep loss truncates melatonin production, removing a natural antioxidant "brake" on the NLRP3 inflammasome and reactive oxygen species (ROS) production, both of which are critical for sustaining the fibrotic niche.[28,29]

Clinically, the divergence of progression-free survival curves beginning at Year 2.5 indicates that the cumulative stress of sleep deprivation manifests as detectable fibrotic accumulation over a relatively short clinical horizon.This highlights the utility of serial non-invasive monitoring. While a single LSM value provides a snapshot of current damage, tracking liver stiffness velocity identifies patients who are at high risk for future liver-related events even if their current stage is low.Our study suggests that sleep hygiene should be integrated as a core therapeutic pillar in MASLD management, particularly for "high-velocity" progressors.

Future therapeutic strategies may leverage these circadian pathways. For example, REV-ERB agonists and melatonin supplementation have shown potential in preclinical models to stabilize HSC quiescence and reduce oxidative stress.In clinical practice, addressing nocturnal hypoxemia via CPAP in patients with sleep apnea and advocating for consistent sleep-wake cycles could provide a synergistic benefit to existing weight-loss and pharmacological interventions.By recognizing the liver as a circadian organ, hepatologists can move beyond traditional risk factors toward a more holistic management of metabolic dysfunction-associated liver disease.

Limitations:

This study has limitations, including potential recall bias from self-reported sleep data and the use of vibration-controlled transient elastography instead of liver biopsy. It did not consider specific genetic variants like PNPLA3 or detailed nutrient compositions. Future research should focus on objective actigraphy and multi-omics integration to confirm these direct metabolic pathways and further refine our understanding of liver stiffness measurement velocity.

Conclusion:
Short sleep duration (≤6 hours) is an independent and direct driver of liver stiffness velocity in MASLD. Restorative sleep (≥7 hours) is a physiological prerequisite for the regression of fibrosis, while the combination of obesity and sleep deprivation significantly accelerates hepatic stiffening. These findings underscore the clinical importance of sleep hygiene in slowing the progression of metabolic liver disease. Clinicians should incorporate sleep duration as a modifiable risk factor in the standard MASLD workup. By identifying high-velocity progressors through serial VCTE monitoring and addressing sleep disruption, practitioners can improve long-term outcomes and mitigate the risk of cirrhosis.

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