

Daily sedentary behavior exceeding 5 hours is independently associated with MAFLD: a cross-sectional study from NHANES 2017–2020

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Abstract

Introduction: Metabolic dysfunction-associated fatty liver disease (MAFLD) has emerged as a predominant global cause of chronic liver disease, linked to various health issues. Although sedentary behavior (SB) has been associated with MAFLD, an exact upper limit for sedentary time has not been established. This study aimed to investigate the dose-response relationship between sedentary time and MAFLD and identify potential reference values for future research and recommendations.

Material and methods: The study analyzed 7,705 participants from the 2017–2020 NHANES cycle, using transient elastography to identify MAFLD and liver fibrosis or cirrhosis. Sedentary time and physical activity levels were determined through a detailed questionnaire, with physical activity classified by official guidelines. Restricted cubic spline and piecewise regression analyses examined the association between sedentary time and MAFLD.

Results: Sedentary time demonstrated a linear dose-response relationship with MAFLD (p for non-linear = 0.647, p overall < 0.001), with sedentary behavior over 5 h daily significantly associated with higher odds of MAFLD. After adjustment for confounders, each additional sedentary hour was associated with a 7.7% increase in the odds of MAFLD (p < 0.001). Males had a higher prevalence of MAFLD, liver fibrosis, and cirrhosis than females (p < 0.001). Further analysis revealed no significant difference by sex in the association between sedentary time and MAFLD (p for interaction = 0.0974).

Conclusions: Prolonged sedentary time is associated with higher odds of MAFLD in US adults. Reducing excessive sedentary behavior may represent a potential strategy for MAFLD prevention, although further longitudinal studies are needed to establish evidence-based sedentary time recommendations.

Key words: NHANES, metabolic dysfunction-associated fatty liver disease, sedentary behavior, adults, the metabolic syndrome.

Introduction

Metabolic dysfunction-associated fatty liver disease (MAFLD), formerly known as non-alcoholic fatty liver disease, now represents a growing challenge in clinical hepatology, a trend that closely parallels the rising rates of obesity, diabetes, and metabolic syndrome (MetS) [1–6]. Over the past three decades, MAFLD has been the sole liver dis-

ease with a rising prevalence in the US, currently affecting approximately 30% of adults, and projections indicate that over 50% of adults may be affected by MAFLD by 2040 if current trends persist [5–7]. Furthermore, MAFLD is recognized as a potentially progressive condition leading to non-alcoholic steatohepatitis (NASH), liver fibrosis (LF), hepatocellular carcinoma (HCC), and culminating in liver transplantation, and is also strongly associated with mortality from all causes and cardiovascular disease (CVD) among US adults [6, 8, 9]. There is increasing awareness of the need to address MAFLD as a critical global public health concern due to its substantial economic burden and detrimental effects on health [5, 8, 10]. Despite the lack of approved pharmacotherapy for first-line treatment, lifestyle interventions – such as enhancing physical activity (PA) and reducing sedentary behavior (SB) – remain the cornerstone of MAFLD prevention and management [1, 2, 7, 11–13].

SB encompasses all waking activities involving minimal energy expenditure (≤ 1.5 METs) involving maintenance of a seated, reclining, or lying posture [13, 14]. During the post-pandemic period, SB has become increasingly pervasive in modern living [15]. Notably, approximately 36% of the examined US population was identified as sedentary, with an additional 48% exhibiting low levels of PA [16]. Furthermore, an expanding body of epidemiological research suggests that sedentary lifestyles have contributed to adverse health outcomes, including obesity, diabetes, insulin resistance (IR), MetS, CVD, cancer, and mortality, independent of PA [14, 17–19]. Many of these conditions are critical factors in the pathogenesis and progression of MAFLD. Specifically, IR is a central component of MetS and serves as the strongest predictor of MAFLD development [20]. Consequently, SB has become a major public health challenge driven by its widespread occurrence and associated healthcare costs [16, 21].

The 2018 Physical Activity Guidelines for Americans first addressed the risks associated with SB in adults; similarly, the World Health Organization guidelines on PA and SB explicitly recommend that all populations and age groups reduce sedentary time, although neither provides a specific quantitative guideline for SB [12, 14, 22–24]. In recent years, the rise in sedentary behavior has been a likely contributing factor to the accelerated development of MAFLD [25]. Furthermore, evidence indicates that SB is an independent predictor of MAFLD, with a 20% increased risk associated with SB [7, 11, 17, 18, 26]. Data from recent research support a dose-response correlation of SB with MAFLD incidence; however, none have identified a definitive threshold effect

[7, 10, 21, 24]. Unfortunately, the existing body of evidence regarding a potential dose-response association between SB and MAFLD remains limited and inconsistent.

Therefore, to delineate the specific dose-response association and potential threshold linking SB to MAFLD, we conducted an analysis of the nationally representative National Health and Nutrition Examination Survey (NHANES) cohort within a cross-sectional design.

Material and methods

Study sample

This study was based on data sourced from the pre-pandemic survey cycles of NHANES (2017 to March 2020), with a specific focus on liver ultrasound transient elastography (TE) tests. Detailed characterization of NHANES has been reported elsewhere [5, 7, 26–28]. Out of 15,560 participants, 7,705 adults with complete TE, sedentary activity, and covariate data were included. Exclusions were as follows: participants under 18 ($N = 5,867$), incomplete TE data ($N = 1,925$), missing CAP or LSM data ($N = 1$), and missing sedentary activity data ($N = 62$). The participant inclusion process is detailed in Figure 1. This study used de-identified, publicly available data from NHANES. The original NHANES investigation was carried out under the approval of the NCHS Ethics Review Board, with informed consent obtained from all participants. Accordingly, this analysis did not require additional ethical approval.

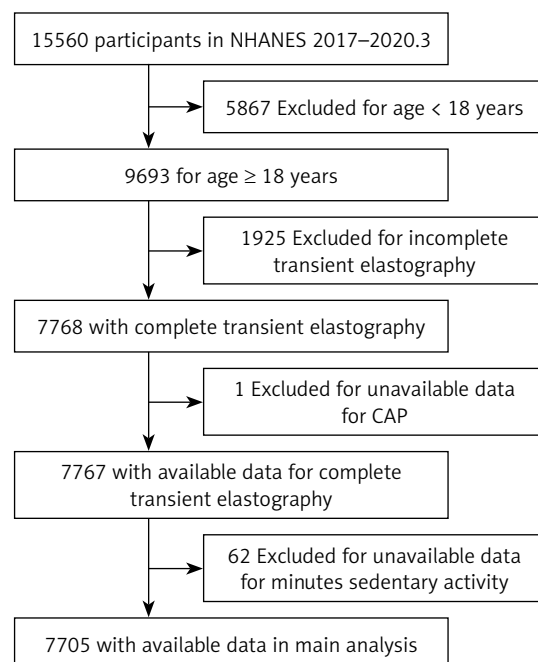


Figure 1. Flowchart showing the selection of study participants

CAP – controlled attenuation parameter.

Primary exposure

The study population of adults (≥ 18 years) was administered the NHANES 2017–2020 physical activity survey, modeled after the Global Physical Activity Questionnaire. Detailed information is available at https://wwwn.cdc.gov/Nchs/Data/Nhanes/Public/2017/DataFiles/P_PAQ.htm. The questionnaire assessed SB, such as sitting while watching TV, working, using computers, and driving, excluding sleep. It was validated for US adults, showing reliable test-retest results [15]. A total of 9,610 participants completed the questionnaire, while 83 did not. Participants were divided into SB quartiles with the following ranges: < 3 , 3–5, 5–8, and ≥ 8 h of sedentary time daily.

Outcomes

With a noninvasive technique having demonstrated reliability for steatosis and fibrosis assessment, hepatic steatosis and stiffness were measured via transient elastography (FibroScan 502 V2 Touch; M/XL probes) [1, 12, 17]. Participants aged 12 and older, excluding those who were pregnant, unable to lie down, or had electronic implants/lesions at the examination site, were eligible. The study included only those with complete examinations (fasting ≥ 3 h, ≥ 10 valid readings, and an IQR for stiffness measurements of $< 30\%$). The diagnostic criteria were as follows: hepatic steatosis corresponded to CAP ≥ 263 dB/m; clinically significant fibrosis and cirrhosis were identified by LSM cutoff values of ≥ 6.3 kPa and ≥ 12.5 kPa, respectively [29, 30]. Furthermore, MAFLD was defined by hepatic steatosis co-occurring with at least one of the following: overweight/obesity, type 2 diabetes, or ≥ 2 metabolic abnormalities, as detailed in the international expert consensus statement [1].

Covariates

Covariates were chosen in accordance with established confounders in the literature and clinical relevance. The demographic covariates were age, sex, race/ethnicity, the poverty-income ratio, and educational level. Moreover, alcohol intake was classified according to NIAAA guidelines [31], and smoking status was defined by serum cotinine concentrations as low (< 0.015 ng/ml), intermediate (0.015 – 3 ng/ml), or high (≥ 3 ng/ml) [32]. Triglyceride levels were stratified in accordance with the NCEP ATP III criteria [33]. Furthermore, physical activity levels were categorized with reference to established guidelines [23].

Statistical analysis

All analyses were conducted in accordance with the NHANES complex sampling design, with sur-

vey weights applied to ensure national representativeness. Continuous measures are expressed as mean $\pm$ SD and evaluated with linear regression, whereas categorical measures are presented as percentages (95% CI) and examined with the χ^2 test. The association of SB with MAFLD was examined using multivariable logistic regression. In line with the MAFLD framework, we did not use its diagnostic metabolic criteria as covariates in our main analysis to avoid over-adjustment, following established methods [34]. Additionally, to characterize and quantify the dose-risk pattern in the association of SB with MAFLD, analyses using restricted cubic splines (knot locations: 5th, 35th, 65th, 95th percentiles) and piecewise regression were performed.

Data were analyzed using R (v4.5.1), EmpowerStats (v4.2), and STATA (v16.0), and statistical significance was defined as a two-sided p -value < 0.05 .

Results

Participant characteristics

The cohort included 7,705 participants, comprising 49.7% men, with a mean age of 47.17 years. The distribution of baseline attributes across sedentary time quartiles is summarized in Supplementary Table SI. Given the particular interest in our findings regarding the predominance of MAFLD, data were further stratified by MAFLD status (Supplementary Table SII). Additionally, analysis stratified by sex (Supplementary Table SIII) revealed an increased prevalence of MAFLD, LF, and LC in men.

Associations between sedentary behavior and MAFLD

Individuals with a higher level of SB had higher odds of MAFLD. As shown in Table I, significant positive associations between prolonged SB and MAFLD were observed for quartiles Q2 to Q4, which persisted across all models [fully adjusted: OR = 1.508 (95% CI: 1.220–1.865) for Q2; OR = 1.511 (95% CI: 1.214–1.879) for Q3; OR = 2.042 (95% CI: 1.637–2.548) for Q4]. Compared to the reference group (Q1: < 3 h/day), the Q2–Q4 groups had 50.8%, 51.1%, and 104.2% higher odds, respectively. In sex-stratified analyses (Supplementary Table SIV), men had significantly higher odds in Q2–Q4 [fully adjusted: OR = 1.455 (95% CI: 1.080–1.959) for Q2; OR = 1.702 (95% CI: 1.237–2.340) for Q3; OR = 2.436 (95% CI: 1.753–3.384) for Q4], while women had significantly higher odds in Q2 and Q4 [fully adjusted: OR = 1.462 (95% CI: 1.072–1.992) for Q2; OR = 1.516 (95% CI: 1.102–2.084) for Q4]. However, no significant interaction was observed between sedentary time and sex ($p = 0.0974$ for interaction).

Table I. Association between sedentary time and metabolic dysfunction-associated fatty liver disease (MAFLD) among US adults ($n = 7,705$), National Health and Nutrition Examination Survey (NHANES) 2017–2020. Detailed legend: Model 1: Non-adjusted model; Model 2 adjusted for: sex; age; race; Model 3 adjusted for: sex; age; race; physical activity level; HBV infection; HCV infection; daily alcohol drinking status; serum cotinine levels; education; poverty income ratio

Sedentary time quartiles [h/day]	MAFLD (yes, $n = 3668$)	Model 1 OR (95% CI), p	Model 2 OR (95% CI), p	Model 3 OR (95% CI), p
Q1 (< 3)	607	Reference	Reference	Reference
Q2 (3–5)	1012	1.471 (1.201, 1.802) < 0.001	1.484 (1.201, 1.833) < 0.001	1.508 (1.22, 1.865) < 0.001
Q3 (5–8)	954	1.400 (1.143, 1.716) 0.001	1.441 (1.163, 1.786) 0.001	1.511 (1.214, 1.879) < 0.001
Q4 (≥ 8)	1095	1.743 (1.423, 2.134) < 0.001	1.898 (1.535, 2.348) < 0.001	2.042 (1.637, 2.548) < 0.001
P trend	–	< 0.001	< 0.001	< 0.001

OR – odds ratio, 95% CI – 95% confidence interval.

Moreover, as shown in Figure 2, the RCS analysis confirmed that SB has a dose-response association with MAFLD, revealing a linear association with a threshold at 5 h/day (p for non-linear = 0.647, p overall < 0.001). In subgroup analyses stratified by BMI, diabetes, and hypertension, participants with obesity, diabetes, or hypertension had a markedly higher odds of MAFLD (Supplementary Figure S1). Furthermore, piecewise regression analysis was performed to quantify this relationship (Tables II).

Discussion

Based on data from the nationally representative NHANES cohort, our analysis established a linear dose-response association between SB and MAFLD. Specifically, our investigation provides initial evidence for a dose-response relationship: sedentary time demonstrated a threshold effect at 5 h daily, significantly elevating MAFLD odds. Each additional hour was associated with a 7.7% increase in the odds – findings that remained consistent following comprehensive covariate adjustment and multiple sensitivity analyses.

Given its population-wide frequency and numerous adverse health effects, SB has garnered significant academic interest. A large prospective UK study of 360,047 adults demonstrated linear dose-response relationships between SB and 14 non-communicable diseases, including chronic

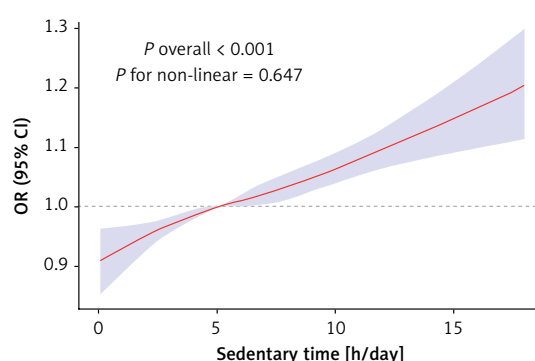


Figure 2. Dose-response relationship between sedentary time and metabolic dysfunction-associated fatty liver disease (MAFLD) risk. Detailed legend: odds ratios (ORs) and p -values were estimated with adjustment for: sex; age; race; physical activity level; HBV infection; HCV infection; daily alcohol drinking status; serum cotinine levels; education; poverty income ratio

95% CI – 95% confidence interval.

liver disease, and indicated that exceeding 6 h of daily sedentary time increases this risk – a threshold consistent with our findings [21]. Several other studies have reported a significant linear dose-response association linking total and prolonged sedentary time to MAFLD, although no specific threshold was established [7, 10, 13, 26, 27]. However, a prospective study of 338,087 UK Biobank participants revealed a nonlinear association of

Table II. Threshold effect of sedentary behavior on metabolic dysfunction-associated fatty liver disease (MAFLD) by piecewise regression, NHANES 2017–2020 ($n = 7,705$). Model 1: Non-adjusted model; Model 2 adjusted for: sex; age; race; physical activity level; HBV infection; HCV infection; daily alcohol drinking status; serum cotinine levels; education; poverty income ratio

Sedentary time (piecewise)	Model 1 OR (95% CI), p	Model 2 OR (95% CI), p
≤ 5 h/day (Reference)	1.00	1.00
> 5 h/day (per hour)	1.058 (1.031, 1.085) < 0.001	1.077 (1.043, 1.113) < 0.001

SB with MAFLD [24]. This divergence likely stems from varying definitions of sedentary time or population heterogeneity. Nevertheless, other lines of evidence consistently identify SB as an independent predictor of MAFLD.

Furthermore, evidence suggests that more than 8 h per day of sedentary time is associated with an elevated risk of MAFLD, with two studies reporting a 1.15% (95% CI: 1.14–1.50%) increase in hepatic fat content for each additional daily hour of sedentariness [7, 35, 36]. However, some findings advocate for more stringent recommendations regarding sedentary time limits [18, 24, 37]. This is particularly concerning given that US adults now average 6 h of daily sedentary time [15]. Such inactivity substantially elevates the risk of diabetes, CVD, and MAFLD, among other adverse outcomes, making the promotion of less sedentary lifestyles an urgent public health priority.

Specifically, our research indicates that for an equivalent sedentary exposure, hypertensive individuals have higher odds of MAFLD, suggesting that hypertension acts as a risk factor in sedentary populations. Given that SB is an established cardiovascular risk factor and MAFLD increases cardiovascular risk by over 60% [38], these factors may form a detrimental cycle. This pattern is pronounced among diabetic or obese individuals, where MAFLD prevalence reaches 65% and 75%, respectively [39]. The incidence of MAFLD is rising in parallel with the diabetes and obesity epidemics [6], and these conditions are key drivers of MAFLD progression [40]. Excessive and prolonged SB initiates a series of detrimental effects, including IR, vascular dysfunction, metabolic alterations in substrate utilization and muscle fiber type, increased central and ectopic fat accumulation (such as in the liver), decreased muscle mass and strength, dyslipidemia, and inflammation [41, 42]. These effects collectively drive the onset and progression of metabolic disorders, diabetes, and obesity. Consequently, reducing sedentary time is particularly crucial for individuals with common metabolic diseases, as it offers dual benefits for managing primary diseases and improving fatty liver conditions.

Interestingly, no significant sex difference was observed in the association between sedentary behavior and MAFLD (p for interaction = 0.0974). However, men exhibited a higher prevalence of MAFLD, liver fibrosis, and cirrhosis than women. Underlying this disparity are pronounced sex-based divergences in fat distribution: women typically accumulate fat in peripheral regions such as the hips and thighs, whereas men show a greater tendency toward developing visceral obesity, which has a more direct association with MetS and MAFLD [17]. Moreover, sex hormones significantly influence the development or resolution of MetS by modulating key processes such as fat

distribution, body weight, and the homeostasis of TG and fatty acids. Estradiol has been shown to confer protection against MetS, while testosterone is associated with an elevated risk [43–45]. The protective effect of estrogen against liver fat accumulation, as opposed to the risk potentially increased by testosterone, may account for the heightened susceptibility to MAFLD in women following the decline in estrogen levels during menopause [25]. Concurrently, the prevalence of MAFLD among women is projected to rise at a faster rate than in men by 2040 [6], a trend attributable to global population aging, which is increasing the proportion of postmenopausal women and consequently leading to a loss of the protective benefits of endogenous estrogen. While MAFLD was more prevalent in men, affected women exhibited comparable NASH risk and elevated risk for progressive fibrosis, particularly after age 50 [46]. Notably, NASH is now the foremost cause of liver transplantation in females [47], suggesting that they may encounter a disproportionately higher risk of adverse liver-related outcomes over time. However, subsequent studies are required to substantiate these findings.

There are some limitations in this analysis. First, the cross-sectional design precludes causal inference, a limitation inherent to the NHANES database. Second, the reliance on self-reported sedentary time rather than accelerometer data is a limitation; however, the sedentary behavior questionnaire has demonstrated acceptable reliability and validity in previous research, and the observed secular trends are unlikely to be significantly affected by measurement error. Third, the absence of a biopsy for histological validation is a limitation, although it is uncommon in large epidemiological studies due to the invasive nature of the procedure. Moreover, given its broadest accessibility and thoroughly validated bedside method, transient elastography represents the logical first choice for assessment. Fourth, despite our adjustment for multiple confounders, residual or unmeasured confounding, such as sex hormone use (including the specific route of administration for hormone replacement therapy) and dietary habits, cannot be entirely ruled out and may affect the robustness of our findings. Fifth, due to missing data, baseline characteristics may have differed between excluded and included participants, potentially introducing selection bias and limiting the generalizability of our findings.

In conclusion, our analysis revealed that sedentary time exceeding 5 h/day was an independent determinant of MAFLD, irrespective of physical activity levels. Furthermore, each additional hour of daily sedentary behavior beyond this threshold conferred a 7.7% rise in MAFLD risk. Future research should examine the relationship between

SB and MAFLD across diverse subpopulations to provide evidence for quantitative guidelines on sitting time.

Availability of data and materials

The datasets supporting the conclusions of this article are available in the NHANES repository, <https://wwwn.cdc.gov/nchs/nhanes/>.

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Ethical approval

NHANES was approved by the NCHS Ethics Review Board, with informed consent obtained from all participants.

Conflict of interest

The authors declare no conflict of interest.

References

- Eslam M, Newsome PN, Sarin SK, et al. A new definition for metabolic dysfunction-associated fatty liver disease: an international expert consensus statement. *J Hepatol* 2020; 73: 202-9.
- Shiina S, Maikhuu J, Deng Q, et al. Liver disease trends in the Asia-Pacific region for the next 50 years. *Clin Mol Hepatol* 2025; 31: 671-84.
- Wu H, Wei J, Chen W, et al. Leisure sedentary behavior, physical activities, and cardiovascular disease among individuals with metabolic dysfunction-associated fatty liver disease. *Arterioscler Thromb Vasc Biol* 2024; 44: e227-37.
- Anstee QM, Reeves HL, Kotsiliti E, Govaere O, Heikenwalder M. From NASH to HCC: current concepts and future challenges. *Nat Rev Gastroenterol Hepatol* 2019; 16: 411-28.
- Younossi ZM, Stepanova M, Younossi Y, et al. Epidemiology of chronic liver diseases in the USA in the past three decades. *Gut* 2020; 69: 564-8.
- Le MH, Yeo YH, Zou B, et al. Forecasted 2040 global prevalence of nonalcoholic fatty liver disease using hierarchical Bayesian approach. *Clin Mol Hepatol* 2022; 28: 841-50.
- Kim D, Vazquez-Montesino LM, Li A, Cholaneril G, Ahmed A. Inadequate physical activity and sedentary behavior are independent predictors of nonalcoholic fatty liver disease. *Hepatology* 2020; 72: 1556-68.
- Rong F, Mai Y, Shou L, et al. Analysis of the association between non-alcoholic fatty liver disease and mortality in United States adults. *Front Nutr* 2024; 11: 1502671.
- Li J, Hua S, Chen GC, et al. Objectively measured sedentary time, physical activity and liver enzyme elevations in US Hispanics/Latinos. *Liver Int* 2020; 40: 1883-94.
- Tsunoda K, Kitano N, Kai Y, Jindo T, Uchida K, Arai T. Dose-response relationships of accelerometer-measured sedentary behaviour and physical activity with non-alcoholic fatty liver disease. *Aliment Pharmacol Ther* 2021; 54: 1330-9.
- Wang J, Qi Y, Xin Z, et al. Isotemporal substitution of different behaviour patterns with the presence of Mafld in Chinese adults. *Liver Int* 2022; 42: 2683-95.
- Kim D, Koryn P, Cholaneril G, Ahmed A. Physical activity is associated with nonalcoholic fatty liver disease and significant fibrosis measured by fibroscan. *Clin Gastroenterol Hepatol* 2022; 20: e1438-55.
- Ma Q, Ye J, Shao C, Lin Y, Wu T, Zhong B. Metabolic benefits of changing sedentary lifestyles in nonalcoholic fatty liver disease: a meta-analysis of randomized controlled trials. *Ther Adv Endocrinol Metab* 2022; 13: 20420188221122426.
- Bull FC, Al-Ansari SS, Biddle S, et al. World Health Organization 2020 guidelines on physical activity and sedentary behaviour. *Br J Sports Med* 2020; 54: 1451-62.
- Li S, Du Y, Xu G, Bao W. Trends in sedentary behavior among US adults. *JAMA* 2025; 334: 271-3.
- Von Loeffelholz C, Roth J, Coldewey SM, Birkenfeld AL. The role of physical activity in nonalcoholic and metabolic dysfunction associated fatty liver disease. *Biomedicines* 2021; 9: 1853.
- Ren Z, Fan H, Xue Y, et al. Mediation role of metabolic syndrome between physical activity, sedentary behavior and non-alcoholic fatty liver disease: a cross-sectional study. *BMC Public Health* 2025; 25: 1661.
- Ryu S, Chang Y, Jung HS, et al. relationship of sitting time and physical activity with non-alcoholic fatty liver disease. *J Hepatol* 2015; 63: 1229-37.
- Croci I, Coombes JS, Bucher Sandbakk S, et al. Non-alcoholic fatty liver disease: prevalence and all-cause mortality according to sedentary behaviour and cardiorespiratory fitness. *The Hunt Study. Prog Cardiovasc Dis* 2019; 62: 127-34.
- Tilg H, Moschen AR, Roden M. NAFLD and diabetes mellitus. *Nat Rev Gastroenterol Hepatol* 2017; 14: 32-42.
- Cao Z, Xu C, Zhang P, Wang Y. Associations of sedentary time and physical activity with adverse health conditions: outcome-wide analyses using isotemporal substitution model. *Eclinicalmedicine* 2022; 48: 101424.
- WHO guidelines on physical activity and sedentary behaviour. Geneva: World Health Organization; 2020. Licence: cc by-nc-sa 3.0 igo.
- Piercy KL, Troiano RP, Ballard RM, et al. The physical activity guidelines for Americans. *JAMA* 2018; 320: 2020-8.
- Ge X, Wang X, Yan Y, et al. Behavioural activity pattern, genetic factors, and the risk of nonalcoholic fatty liver disease: a prospective study in the UK Biobank. *Liver Int* 2023; 43: 1287-97.
- Franco I, Bianco A, Bonfiglio C, Curci R, Campanella A, Osella AR. Leisure-time physical activity, time spent sitting and risk of non-alcoholic fatty liver disease: a cross-sectional study in puglia. *J Gen Intern Med* 2024; 39: 2788-96.
- Keating SE, Chawla Y, De A, George ES. Lifestyle intervention for metabolic dysfunction-associated fatty liver disease: a 24-h integrated behavior perspective. *Hepatol Int* 2024; 18 (suppl 2): 959-76.
- Han Q, Han X, Wang X, et al. Association of accelerometer-measured sedentary behavior patterns with nonalcoholic fatty liver disease among older adults:

- the mind-China study. *Am J Gastroenterol* 2023; 118: 569-73.
28. Wu WT, Li YJ, Feng AZ, et al. Data mining in clinical big data: the frequently used databases, steps, and methodological models. *Mil Med Res* 2021; 8: 44.
 29. Kalligeros M, Vassilopoulos A, Vassilopoulos S, Victor DW, Mylonakis E, Nouredin M. Prevalence of steatotic liver disease (MASLD, METALD, and ALD) in the United States: NHANES 2017-2020. *Clin Gastroenterol Hepatol* 2024; 22: 1330-2.e4.
 30. Cassinotto C, Boursier J, De Lédinghen V, et al. Liver stiffness in nonalcoholic fatty liver disease: a comparison of supersonic shear imaging, Fibroscan, and arfi with liver biopsy. *Hepatology* 2016; 63: 1817-27.
 31. National institute on alcohol abuse and alcoholism. drinking levels defined[cited February 11,2025; available from: <https://www.niaaa.nih.gov/alcohols-effects-health/alcohol-drinking-patterns>.
 32. Reja D, Makar M, Visaria A, Karanfilian B, Rustgi V. Blood lead level is associated with advanced liver fibrosis in patients with non-alcoholic fatty liver disease: a nationwide survey (NHANES 2011-2016). *Ann Hepatol* 2020; 19: 404-10.
 33. Berglund L, Brunzell JD, Goldberg AC, et al. Evaluation and treatment of hypertriglyceridemia: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab* 2012; 97: 2969-89.
 34. Lee H, Lee YH, Kim SU, Kim HC. Metabolic dysfunction-associated fatty liver disease and incident cardiovascular disease risk: a nationwide cohort study. *Clin Gastroenterol Hepatol* 2021; 19: 2138-47.
 35. Bowden Davies KA, Sprung VS, Norman JA, et al. Physical activity and sedentary time: association with metabolic health and liver fat. *Med Sci Sports Exerc* 2019; 51: 1169-77.
 36. Zhang X, Chen K, Yin S, Qian M, Liu C. Association of leisure sedentary behavior and physical activity with the risk of nonalcoholic fatty liver disease: a two-sample mendelian randomization study. *Front Nutr* 2023; 10: 1158810.
 37. Grøntved A, Hu FB. Television viewing and risk of type 2 diabetes, cardiovascular disease, and all-cause mortality: a meta-analysis. *JAMA* 2011; 305: 2448-55.
 38. Machado MV. Aerobic exercise in the management of metabolic dysfunction associated fatty liver disease. *Diabetes Metab Syndr Obes* 2021; 14: 3627-45.
 39. Manolis AA, Manolis TA, Vouliotis A, Manolis AS. Metabolic dysfunction-associated steatotic liver disease and the cardiovascular system. *Trends Cardiovasc Med* 2025; 35: 258-65.
 40. Newsome P, Francque S, Harrison S, et al. Effect of semaglutide on liver enzymes and markers of inflammation in subjects with type 2 diabetes and/or obesity. *Aliment Pharmacol Ther* 2019; 50: 193-203.
 41. Pinto AJ, Bergouignan A, Dempsey PC, et al. Physiology of sedentary behavior. *Physiol Rev* 2023; 103: 2561-622.
 42. Bowden Davies KA, Sprung VS, Norman JA, et al. Short-term decreased physical activity with increased sedentary behaviour causes metabolic derangements and altered body composition: effects in individuals with and without a first-degree relative with type 2 diabetes. *Diabetologia* 2018; 61: 1282-94.
 43. Hashemi L, Marijic Buljubasic A, Budoff MJ, et al. Gender-affirming hormone treatment and metabolic syndrome among transgender veterans. *Jama Netw Open* 2024; 7: e2419696.
 44. Feng B, Weng W, Man Q. Investigating the potential impact of sex hormones and adiponectin on the risk of liver fibrosis and cirrhosis: a Mendelian randomization study. *Arch Med Sci* 2025; 21: 2502-12.
 45. Ciftel S, Çiftel S, Baykan AR, Cerrah S, Çiftel E, Mercantepe F. Cardiometabolic risk in non-diabetic metabolic dysfunction-associated steatotic liver disease (MAFLD) patients: insights from the triglyceride-glucose, plasma atherogenic, and cardiometabolic index. *Arch Med Sci* 2024; 21: 401-8.
 46. Balakrishnan M, Patel P, Dunn-Valadez S, et al. Women have a lower risk of nonalcoholic fatty liver disease but a higher risk of progression vs men: a systematic review and meta-analysis. *Clin Gastroenterol Hepatol* 2021; 19: 61-71.e15.
 47. Nouredin M, Vipani A, Bresee C, et al. NASH leading cause of liver transplant in women: updated analysis of indications for liver transplant and ethnic and gender variances. *Am J Gastroenterol* 2018; 113: 1649-59.