

#### Research Article

Predictive Value of Asprosin in Combination with Lipid Accumulation Product for Non-Alcoholic Fatty Liver Disease in Population for Medical Check-Ups

Dan  $Lv^1$ , Zepu Wang<sup>2</sup>, Yan  $Li^1$ , Shuchun Chen<sup>3\*</sup>, and Letian  $Li^4$ 





#### Research Article

# Predictive Value of Asprosin in Combination with Lipid Accumulation Product for Non-Alcoholic Fatty Liver Disease in Population for Medical Check-Ups

Dan Lv<sup>1</sup>, Zepu Wang<sup>2</sup>, Yan Li<sup>1</sup>, Shuchun Chen<sup>3\*</sup>, and Letian Li<sup>4</sup>

- <sup>1</sup>Department of Ultrasound, Hebei General Hospital, China.
- <sup>2</sup>Department of Hepatobiliary Surgery, Hebei General Hospital, China.
- <sup>3</sup>Department of Endocrinology, Hebei General Hospital, China.
- <sup>4</sup>Department of Emergency, The Second Affiliated Hospital of Xingtai Medical College, China.

#### **ABSTRACT**

Objective: To combine two indicators, asprosin and lipid accumulation product (LAP), to assess their predictive value for non-alcoholic fatty liver disease (NAFLD) and to provide a more convenient and accurate option for mass screening of the NAFLD population.

Methods: Data were collected from 1249 adult subjects who underwent physical examination, LAP was calculated and serum asprosin was measured. Statistical analyses were performed and ROC curves were constructed to assess the predictive value of asprosin, LAP and their combination for NAFLD.

Results: Asprosin and LAP levels were significantly higher in the NAFLD group than in the non-NAFLD group (P < 0.001), and asprosin and LAP were independent risk factors for the development of NAFLD (P < 0.05). The combination of asprosin and LAP had the highest predictive efficacy for NAFLD, and the AUCs were 0.841, 0.821 and 0.871 in the total, male and female populations, respectively.

Conclusion: Asprosin and LAP are both good predictors of NAFLD, and their combined performance is better than either indicator alone, and better in women than in men. The combination of asprosin and LAP may be an ideal marker for NAFLD screening and individualised monitoring and management.

**Keywords**: Asprosin; Lipid Accumulation Product; Non-Alcoholic Fatty Liver Disease; Screening; Prediction Effectiveness

#### INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is a clinical condition characterised by the abnormal accumulation of lipids in hepatocytes [1]. If left untreated, it can progress to end-stage liver disease such as non-alcoholic steatohepatitis (NASH), cirrhosis and even hepatocellular carcinoma and liver failure [2]. The global prevalence of NAFLD is estimated to be 25%, affecting approximately 173-310 million people in China, and the disease burden of NAFLD has surpassed that of other chronic liver diseases and is a major public health challenge worldwide [3]. Early and timely diagnosis and intervention of NAFLD is crucial, as early NAFLD is reversible [1]. There is an urgent need to find simple, effective and inexpensive indicators for early mass screening.

Copyright: ©2025 Chen S. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Received: April 28, 2025 Accepted: June 24, 2025 Published: June 25, 2025

Citation: Lv D, Wang Z, Li Y, Chen S, Li L, Predictive Value of Asprosin in Combination with Lipid Accumulation Product for Non-Alcoholic Fatty Liver Disease in Population for Medical Check-UpsClin Cardiol. 2025; 5(2): 1033.

#### Corresponding Author:

Shuchun Chen, Department of Endocrinology, Hebei General Hospital, 348 Heping West Road, Shijiazhuang City, Hebei Province, China



Asprosin, a novel adipokine discovered in 2016, is secreted by white adipose tissue and plays an important role in maintaining energy homeostasis, inflammation and immune response [4]. Several recent studies have confirmed the integral role of asprosin in the pathophysiology of various metabolic diseases such as NAFLD, obesity, metabolic syndrome, diabetes mellitus, polycystic ovary syndrome and cardiovascular disease [5-10]. A previous study by our team also found a strong association between asprosin and NAFLD [11]. It has been suggested that asprosin expression is increased in the serum of NAFLD patients and that asprosin may be a valuable biomarker for the diagnosis of NAFLD [8].

The lipid accumulation product (LAP) was originally proposed by Kahn as an indicator of lipid excess [12]. Recent studies have shown that LAP is a powerful tool for the identification of NAFLD. A large cross-sectional study of 40,459 Chinese individuals confirmed that LAP was highly associated with the incidence and severity of NAFLD and was a reliable predictor of NAFLD risk in Chinese adults [13]. A meta-analysis also showed that LAP is a reliable marker for screening for NAFLD [14].

However, there are currently few studies using the combined indicators to screen for NAFLD. Based on the above theory, this study combined two indicators, Asprosin and LAP, to evaluate their predictive value for NAFLD, providing a more convenient and accurate option for early mass screening of the NAFLD population, which may aid in the early screening and subsequent management of the potential NAFLD population.

#### MATERIALS AND METHODS Study Population

This study retrospectively analysed data from 1,249 adult subjects who underwent health examinations at the Physical Examination Centre of Hebei Provincial People's Hospital during 2022.01-2023.01. Inclusion criteria: (1) age ≥18 years; (2) complete data. Exclusion criteria: (1) pregnant or lactating women; (2) men drinking ≥140 g/week or women drinking ≥70 g/week; (3) people with viral hepatitis, autoimmune liver disease and other chronic liver diseases; (4) people with chronic kidney disease, severe cardiovascular disease, haematological disease and malignant neoplasm; and (5) people who had used drugs affecting hepatic lipid metabolism within six months. The study was approved by the Ethics Committee of Hebei Provincial People's Hospital and conducted according to the tenets of the Declaration of Helsinki. Informed consent was not required as all data in the study were retrospective and did not include personal and identifiable information about the subjects.

#### **Data Collection and Measurement**

In this study, information on the subjects' lifestyle and medical history was collected using a standardised self-administered questionnaire. Serum asprosin was measured using an enzyme-linked immunosorbent assay kit (ELAAB Science Inc), and basic information, general physical examination, and laboratory tests of the subjects were obtained from the electronic database of the medical examination centre. Basic information included sex and age; general physical examination included height, weight, and waist circumference (WC); and laboratory tests included triglycerides (TG),

total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and fasting plasma glucose (FPG). BMI and LAP were calculated from the above indicators using the following formulae:

BMI = Weight (kg)/Height2 (m2)

LAP (male) = TG (mmol/L) × [WC (cm) - 65].

LAP (female) = TG (mmol/L) × [WC (cm) - 58].

#### **Abdominal Ultrasound**

Abdominal ultrasound was performed by an experienced sonographer using an ultrasound machine equipped with a curved array transducer probe (4-8 MHz) to assess fatty liver based on four dimensions: liver brightness, liver and kidney echo contrast, vascular blurring, and deep attenuation [15].

#### **Statistical Analysis**

The statistical analysis was conducted using SPSS 21.0 software. For normally distributed measurements, the data were expressed as the mean  $\pm$  standard deviation (SD) and a t-test was employed to compare the two groups. For non-normally distributed measurements, the data were expressed as the median (P25, P75) and a Mann-Whitney Utest was used to compare the groups. Count data were expressed as numbers or percentages and an  $\chi 2$  test was used to compare the groups. Asprosin and LAP were grouped into quartiles, and logistic regression analysis was used to calculate the OR and 95% CI of the risk of NAFLD in each quartile group of asprosin and LAP. The predictive value of the various indicators of NAFLD was analysed using the receiver operating characteristic (ROC) curve, and P < 0.05 was considered statistically significant.

#### RESULTS

## Comparison of Baseline Data Between the Two Groups

Based on the results of abdominal ultrasonography, 1249 subjects were divided into the non-NAFLD group (n = 611) and the NAFLD group (n = 638). The proportion of males in the NAFLD group was significantly higher than that in the non-NAFLD group (P < 0.001); the levels of TC and LDL-C in the NAFLD group were higher than those in the non-NAFLD group (P < 0.05); the levels of BMI, WC , TG, FPG , asprosin, and LAP levels in the NAFLD group were significantly higher than those in the non-NAFLD group (P < 0.001), and the levels of HDL-C were significantly lower than those in the non-NAFLD group (P < 0.001) (Table 1).

#### Multifactorial Logistic Regression Analysis of the Risk of Developing Nafld

Asprosin and LAP were divided into four subgroups at the quartile level as Q1, Q2, Q3 and Q4 groups, respectively. The covariates of gender, age, BMI, WC, TG, TC, LDL-C, HDL-C, FPG, asprosin, and LAP were included in the analysis with the presence or absence of NAFLD as the dependent variable, and the results showed that BMI, TG, FPG, asprosin, and LAP were the independent risk factors for the occurrence of NAFLD (P < 0.05). After correction for each risk factor, the risk of NAFLD prevalence in the Q2, Q3 and Q4 asprosin groups was 3.841, 4.203 and 9.132 times higher, respectively, than in the Q1 asprosin group (P < 0.001, P < 0.001, P < 0.001). The risk of NAFLD prevalence in the Q2, Q3



 Table 1: Comparative analysis of baseline data between non-NAFLD and NAFLD groups

	non-NAFLD (n= 611)	NAFLD (n= 638)	Statistical value	P value
Gender, n (%)				
Males	389(63.70%)	531(83.20%)	(1.5)	<0.001
Females	222(36.30%)	107(16.80%)	61.56	
Age (years)	52(40, 59)	51(40, 58)	-1.29	0.198
BMI (kg/m2)	24.56(22.79, 26.40)	27.46(25.78, 29.41)	-17.40	<0.001
WC (cm)	88(82, 94)	96(91, 103)	-16.29	<0.001
TG (mmol/L)	1.22(0.89, 1.68)	1.86(1.42, 2.76)	-14.82	<0.001
TC (mmol/L)	5.22±1.01	5.34±1.04	-2.15	0.032
LDL-C(mmol/L)	3.27±0.75	3.39±0.72	-3.06	0.002
HDL-C(mmol/L)	1.32(1.16, 1.54)	1.19(1.05, 1.35)	-9.67	<0.001
FPG (mmol/L)	5.35(5.02, 5.79)	5.79(5.31, 6.63)	-10.43	<0.001
Asprosin(ng/mL)	20.61(14.66, 27.91)	32.35(24.34, 40.34)	-17.18	<0.001
LAP	30.80(19.88, 44.80)	61.62(42.39, 97.09)	-18.31	<0.001

Table 2: Multifactorial logistic regression analysis of NAFLD

	В	SE	Wald $\chi^2$	OR	95% CI	P value
Gender	0.189	0.226	0.700	1.209	0.775~1.884	0.403
Age	-0.011	0.007	2.540	0.989	0.975~1.003	0.111
BMI	0.168	0.045	13.868	1.183	1.083~1.292	<0.001
WC	0.014	0.018	0.619	1.014	0.979~1.051	0.431
TG	0.234	0.117	3.988	1.264	1.004~1.590	0.046
TC	-0.183	0.348	0.275	0.833	0.421~1.649	0.600
LDL-C	0.269	0.434	0.382	1.308	0.558~3.065	0.536
HDL-C	-0.466	0.447	1.088	0.627	0.261~1.507	0.297
FPG	0.254	0.061	17.425	1.289	1.144~1.452	<0.001
Asprosin						
Q1	-	-	-	1	-	-
Q2	1.346	0.218	38.145	3.841	2.506~5.887	<0.001
Q3	1.436	0.218	43.549	4.203	2.744~6.439	<0.001
Q4	2.212	0.249	78.943	9.132	5.606~14.874	<0.001
LAP						
Q1	-	-	-	1	-	-
Q2	0.677	0.243	7.788	1.969	1.223~3.168	0.005
Q3	0.987	0.425	5.391	2.682	1.166~6.169	0.020
Q4	1.410	0.291	23.561	4.097	2.318~7.240	<0.001



Table 3: ROC curve of Asprosin and LAP prediction of NAFLD risk in total population

	AUC	95% CI	Sensitivity (%)	Specificity (%)	Maximum Jordon index	Cut-of value
Asprosin	0.781	0.756~0.806	70.8	70.5	0.413	26.185
LAP	0.799	0.775~0.824	71.5	75.5	0.470	44.975
Asprosin+LAP	0.841	0.819~0.862	82.8	70.7	0.535	-

Table 4: ROC curve of Asprosin and LAP prediction of NAFLD risk in males

	AUC	95% CI	Sensitivity (%)	Specificity (%)	Maximum Jordon index	Cut-of value
Asprosin	0.771	0.741~0.801	62.5	76.9	0.394	29.110
LAP	0.768	0.737~0.799	71.8	69.7	0.415	44.900
Asprosin+LAP	0.821	0.795~0.848	76.1	73.0	0.491	-

Table 5: ROC curve of Asprosin and LAP prediction of NAFLD risk in females

	AUC	95% CI	Sensitivity (%)	Specificity (%)	Maximum Jordon index	Cut-of value
Asprosin	0.791	0.741~0.841	82.2	63.1	0.453	22.420
LAP	0.845	0.801~0.889	80.4	77.9	0.583	37.665
Asprosin+LAP	0.871	0.832~0.909	68.2	89.6	0.578	-

and Q4 LAP groups was 1.969, 2.682 and 4.097 times higher, respectively, than in the Q1 LAP group (P = 0.005, P = 0.020, P < 0.001) (Table 2).

# Roc Curves of Asprosin, Lap and Their Combination for Predicting the Risk of Developing Nafld

The ROC curves of asprosin, LAP and their combination for predicting the risk of NAFLD were plotted for the total population, different sexes, respectively (Figure 1). The results showed that the AUCs of asprosin and LAP for predicting NAFLD in the total population were 0.781 and 0.799, with optimal cut-offs of 26.185 ng/ml and 44.975, respectively, and the AUC of the combination of the two for predict-

ing NAFLD was 0.841 (Table 3). In the male population, the AUCs of asprosin and LAP for the prediction of NAFLD were 0.771 and 0.768, respectively, and the optimal cut-offs were 29.110 ng/ml and 44.900, respectively, and the AUC for the combined prediction of NAFLD was 0.821 (Table 4). In the female population, the AUCs of asprosin and LAP for predicting NAFLD were 0.791 and 0.845, respectively, and the optimal cut-offs were 22.420 ng/ml and 37.665, respectively, and the AUC of the combination of both for predicting NAFLD was 0.871 (Table 5).

#### **DISCUSSION**

There is no consensus on screening methods for NAFLD. Liver biopsy remains the gold standard for diagnosing

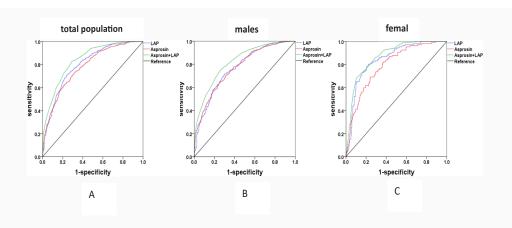


Figure 1: ROC curves for predicting the risk of developing NAFLD



NAFLD, but its clinical use is limited due to its invasive nature. Magnetic resonance imaging (MRI) and computed tomography (CT) are non-invasive diagnostic methods, but they are cumbersome, expensive and not suitable for screening [16]. Ultrasound is the most commonly used method for diagnosing NAFLD and has been endorsed by Asia-Pacific guidelines, but it is still cumbersome for remote rural areas [17,18], and to some extent ultrasound is subject to the subjective judgement of the operator [19].

LAP is an indicator of lipid excess consisting of WC and TG and may reflect visceral fat accumulation better than traditional adiposity indicators [20]. And visceral fat accumulation is considered an important risk factor for the development of NAFLD [21]. Visceral fat accumulation induces the production of several adipocytokines, such as interleukin-6, lipocalin, resistin and leptin, which increase insulin resistance and trigger macrophage infiltration and inflammatory responses, leading to increased liver inflammation and fibrosis [22]. And high levels of TG can promote the transport of free fatty acids to the liver, triggering the development of NAFLD [23].

Our study found that LAP was significantly higher in the NAFLD population than in the non-NAFLD population, and that LAP was an independent risk factor for the development of NAFLD. The AUC of LAP for predicting the development of NAFLD in the male and female populations were 0.768 and 0.845, respectively; the sensitivity was 71.8% and 80.4%, respectively; the specificity was 69.7% and 77.9%, respectively. A large cross-sectional study showed that the sensitivity of LAP in predicting the risk of NAFLD in Chinese male adults was 77% and the specificity was 75%, and the sensitivity of LAP in predicting the risk of NAFLD in Chinese female adults was 82% and the specificity was 79% [13], which is very close to the results of our study. A study by Zhang et al. compared the efficacy of anthropometric measures such as BMI, WC, waist-to-height ratio (WHtR) and LAP in predicting NAFLD in older adults and showed that LAP was the best marker for predicting NAFLD among these measures [24]. Sheng et al. also showed that LAP predicted incident NAFLD better than WHtR in both men and women, BMI, TG/HDL-C, WC, visceral adiposity index (VAI), TG, and especially in young women, LAP predicted NAFLD with an AUC as high as 0.980 [25]. Another study verified this theory pathologically by liver biopsy, and LAP predicted hepatic steatosis and NAFLD better than other common non-invasive biomarkers in the general Chinese population undergoing liver biopsy [26].

Asprosin, a fasting-induced gluconeogenic hormone, was first discovered in 2016, and many studies have shown that asprosin plays a key role in promoting glucose release from the liver and maintaining glucose homeostasis [27,28]. The potential mechanisms underlying the relationship between asprosin and NAFLD were explored in an animal study, which found that increased asprosin could exacerbate hepatocyte dysfunction and lipid aggregation, disrupt lipid metabolic homeostasis, and exacerbate inflammatory responses through ROS-mediated oxidative stress. More importantly, deletion of the asprosin gene attenuated hepatic steatosis and NASH in mice [29]. Another animal study also showed that reducing asprosin activated AMPK signalling, thereby inhibiting lipid accumulation and inflammatory infiltration in the liver [30]. The above two animal studies provide con-

clusive evidence for the relationship between asprosin and NAFLD, and it can be assumed that asprosin plays an important role in the development of NAFLD, and asprosin may become a new target for the diagnosis and treatment of NAFLD in the future. However, there are fewer studies on the relationship between human asprosin and NAFLD. High levels of asprosin have been reported to be associated with NAFLD in elderly patients with type 2 diabetes mellitus [31], and serum asprosin levels are elevated in children with obesity associated with NAFLD [32]. A previous study by our team also found that serum asprosin levels were independently and positively correlated with the hepatic steatosis indicator, the controlled attenuation parameter (CAP), in an adult population undergoing physical examination [11], suggesting that asprosin may be a predictor of NAFLD. Ke et al. have also suggested that the combination of asprosin and lipofuscin may be a novel biomarker for the diagnosis of NAFLD [8].

In this study, we found that asprosin was an independent risk factor for the development of NAFLD, the AUC of asprosin for predicting the development of NAFLD in male and female populations were 0.771 and 0.791, respectively; the sensitivity was 62.5% and 82.2%, respectively; the specificity was 76.9% and 63.1%, respectively; and the optimal cut-off values were 29.110 and 22.420, respectively. On this basis, our joint analysis of asprosin and LAP showed that the AUC of the two combined to predict incident NAFLD in the male population increased from 0.771, 0.768 to 0.821. The AUC of the two combined to predict incident NAFLD in the female population increased from 0.791, 0.845 to 0.871. And the AUC of the two combined to predict incident NAFLD in the total population increased from 0.781, 0.799 to 0.841. Therefore, the combination of asprosin and LAP may improve the prediction of NAFLD. Therefore, the combination of asprosin and LAP may improve the predictive efficacy of NAFLD and make screening for NAFLD more accurate and

The study also found that asprosin and LAP predicted NAFLD better in women than in men, whether as a single predictor or a combination of the two. This discrepancy can be attributed, at least in part, to the fact that the prevalence, quantity, and proportion of smoking and alcohol consumption are significantly higher in men than in women. These habits have been associated with the development of NAFLD [33,34], suggesting that the risk factors for developing NAFLD are more complex in men. Furthermore, the predominant distribution of body fat in women, characterised by subcutaneous storage, contrasts with the tendency of men to accumulate fat in visceral organs, a phenomenon more closely linked to central obesity [35,36]. In addition, oestrogen has been shown to be an important contributor to energy balance and metabolic homeostasis, protecting women from less damage from metabolic disease [37].

The study's merits are manifold. Firstly, the selected indicators are simple, easy to measure and low cost, which renders them suitable for large-scale screening. Secondly, the subjects were all drawn from the routine physical examination population, which is more consistent with the overall population background of screening. Furthermore, previous studies have focused on the prediction efficiency of a single indicator for NAFLD. However, this study combined the two indicators to greatly improve the prediction efficiency for NAFLD. However, the study is not without its limitations:



Firstly, the study was conducted in a single centre with relatively homogenous race. Given the recognised differences in the characteristics of NAFLD among different racial groups, further verification in diverse ethnic populations is necessary to confirm the generalisability of the findings. Secondly, although the study controlled for multiple potential confounding factors, the possibility of unmeasured confounders cannot be ruled out, such as exercise and diet structure. Finally, the necessity for further prospective studies to evaluate the findings is highlighted, given that this study is a retrospective cross-sectional study.

#### **CONCLUSION**

In conclusion, the present study has demonstrated the efficacy of both asprosin and LAP as predictors of NAFLD. The combined predictive efficacy of the two indicators is superior to that of a single indicator, and the predictive efficacy is more pronounced in females than in males. Consequently, the combination of asprosin and LAP emerges as a promising marker for large-scale NAFLD screening and can serve as an effective indicator for individualised surveillance. This study provides a simple and effective alternative for epidemiological investigations and individualised health management.

#### **FUNDING**

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit

#### AVAILABILITY OF DATA AND MATERIAL

All data used to support the findings of this study are available on request from the corresponding author.

#### **ACKNOWLEDGMENTS**

The authors would like to thank all the reviewers who participated in the review.

#### REFERENCES

- Powell EE, Wong VW, Rinella M. Non-alcoholic fatty liver disease. Lancet. 2021; 397: 2212-2224.
- Rinella ME. Nonalcoholic fatty liver disease: a systematic review. JAMA. 2015; 313: 2263-2273.
- Xiao J, Wang F, Wong NK, He J, Zhang R, Sun R, et al. Global liver disease burdens and research trends: Analysis from a Chinese perspective. J Hepatol. 2019; 71: 212-221.
- Romere C, Duerrschmid C, Bournat J, Constable P, Jain M, Xia F, et al. Asprosin, a Fasting-Induced Glucogenic Protein Hormone. Cell. 2016; 165: 566-579.
- Gozel N, Kilinc F. Investigation of plasma asprosin and saliva levels in newly diagnosed type 2 diabetes mellitus patients treated with metformin. Endokrynol Pol. 2021; 72: 37-43.
- Shabir K, Brown JE, Afzal I, Gharanei S, Weickert MO, Barber TM, et al. Asprosin, a novel pleiotropic adipokine implicated in fasting and obesity-related cardio-metabolic disease: Comprehensive review of preclinical and clinical evidence. Cytokine Growth Factor Rev. 2021; 60: 120-132.
- Hekim MG, Kelestemur MM, Bulmus FG, Bilgin B, Bulut F, Gokdere E, et al. Asprosin, a novel glucogenic adipokine: a potential therapeutic implication in diabetes mellitus. Arch Physiol Biochem. 2023; 129: 1038-1044.

- Ke F, Xue G, Jiang X, Li F, Lai X, Zhang M, et al. Combination of asprosin and adiponectin as a novel marker for diagnosing non-alcoholic fatty liver disease. Cytokine. 2020; 134: 155184.
- Summers KM, Bush SJ, Davis MR, Hume DA, Keshvari S, West JA. Fibrillin-1 and asprosin, novel players in metabolic syndrome. Mol Genet Metab. 2023; 138: 106979.
- 10. Zhang Z, Zhu L, Wang Z, Hua N, Hu S, Chen Y. Can the new adipokine asprosin be a metabolic troublemaker for cardiovascular diseases? A state-of-the-art review. Prog Lipid Res. 2023; 91: 101240.
- 11. Dan Lv, Zepu Wang, Cuiqiao Meng, Yan Li, Shuai Ji. A study of the relationship between serum asprosin levels and MAFLD in a population undergoing physical examination. Sci Rep. 2024;14: 11170.
- 12. Kahn HS. The "lipid accumulation product" performs better than the body mass index for recognizing cardiovascular risk: a population-based comparison. BMC Cardiovasc Disord. 2005;
- 13. Dai H, Wang W, Chen R, Chen Z, Lu Y, Yuan H. Lipid accumulation product is a powerful tool to predict non-alcoholic fatty liver disease in Chinese adults. Nutr Metab (Lond). 2017; 14: 49.
- 14. Ebrahimi M, Seyedi SA, Nabipoorashrafi SA, Rabizadeh S, Sarzaeim M, Yadegar A, et al. Lipid accumulation product (LAP) index for the diagnosis of nonalcoholic fatty liver disease (NAFLD): a systematic review and meta-analysis. Lipids Health Dis. 2023; 22: 41.
- 15. Hamaguchi M, Kojima T, Itoh Y, Harano Y, Fujii K, Nakajima T, et al. The severity of ultrasonographic findings in nonalcoholic fatty liver disease reflects the metabolic syndrome and visceral fat accumulation. Am J Gastroenterol. 2007; 102: 2708-2715.
- 16. Davison BA, Harrison SA, Cotter G, Alkhouri N, Sanyal A, Edwards C, et al. Suboptimal reliability of liver biopsy evaluation has implications for randomized clinical trials. J Hepatol. 2020; 73: 1322-1332.
- 17. National Guideline Centre (UK). Non-Alcoholic Fatty Liver Disease: Assessment and Management. London: National Institute for Health and Care Excellence (NICE). 2016.
- 18. Farrell GC, Chitturi S, Lau GK, Sollano JD, Asia-Pacific Working Party on NAFLD. Guidelines for the assessment and management of non-alcoholic fatty liver disease in the Asia-Pacific region: executive summary. J Gastroenterol Hepatol. 2007; 22: 775-777.
- 19. van Kleef LA, Sonneveld MJ, de Man RA, de Knegt RJ. Poor performance of FIB-4 in elderly individuals at risk for chronic liver disease - implications for the clinical utility of the EASL NIT guideline. J Hepatol. 2022; 76: 245-246.
- 20. Shu L, Zhao Y, Shen Y, Jia L, Zhang J. Interaction analysis of lipid accumulation product and family history of diabetes on impaired fasting glucose and diabetes risk in population with normotension in Eastern China: a community-based crosssectional survey. Arch Public Health. 2022; 80: 217.
- 21. Kim D, Chung GE, Kwak MS, Seo HB, Kang JH, Kim W, et al. Body Fat Distribution and Risk of Incident and Regressed Nonalcoholic Fatty Liver Disease. Clin Gastroenterol Hepatol. 2016; 14: 132-8.e4.



- 22. Tantanavipas S, Vallibhakara O, Sobhonslidsuk A, Phongkitkarun S, Vallibhakara SA, Promson K, et al. Abdominal Obesity as a Predictive Factor of Nonalcoholic Fatty Liver Disease Assessed by Ultrasonography and Transient Elastography in Polycystic Ovary Syndrome and Healthy Women. Biomed Res Int. 2019; 2019: 9047324.
- Birkenfeld AL, Shulman GI. Nonalcoholic fatty liver disease, hepatic insulin resistance, and type 2 diabetes. Hepatology. 2014; 59: 713-723.
- 24. Zhang Y, Li B, Liu N, Wang P, He J. Evaluation of Different Anthropometric Indicators for Screening for Nonalcoholic Fatty Liver Disease in Elderly Individuals. Int J Endocrinol. 2021; 2021: 6678755.
- Cai J, Lin C, Lai S, Liu Y, Liang M, Qin Y, et al. Waist-to-height ratio, an optimal anthropometric indicator for metabolic dysfunction associated fatty liver disease in the Western Chinese male population. Lipids Health Dis. 2021; 20: 145.
- Huang Q, Tan X, Wu Q, Zhao H, Chen H, Yu X, et al. Lipid accumulation product is a valid predictor of hepatic steatosis and nonalcoholic fatty liver disease. Biomark Med. 2024; 18: 123-135.
- 27. Donma MM, Donma O. Asprosin: Possible target in connection with ghrelin and cytokine network expression in the post-burn treatment. Med Hypotheses. 2018; 118: 163-168.
- 28. Duerrschmid C, He Y, Wang C, Li C, Bournat JC, Romere C, et al. Asprosin is a centrally acting orexigenic hormone. Nat Med. 2017; 23: 1444-1453.
- 29. Wang C, Zeng W, Wang L, Xiong X, Chen S, Huang Q, et al. Asprosin aggravates nonalcoholic fatty liver disease via inflammation and lipid metabolic disturbance mediated by reactive oxygen species. Drug Dev Res. 2024; 85: e22213.

- Zhang B, Lu J, Jiang Y, Feng Y. Asprosin contributes to nonalcoholic fatty liver disease through regulating lipid accumulation and inflammatory response via AMPK signaling. Immun Inflamm Dis. 2023; 11: e947.
- 31. Cui J, Liu Y, Li M, Yin J, Yang J, Xu L. Association of serum asprosin with metabolic dysfunction-associated fatty liver disease in older adult type 2 diabetic patients: a cross-sectional study. BMC Endocr Disord. 2024; 24: 27.
- 32. Liu LJ, Kang YR, Xiao YF. Increased asprosin is associated with non-alcoholic fatty liver disease in children with obesity. World J Pediatr. 2021; 17: 394-399.
- 33. Wang S, Ungvari GS, Forester BP, Chiu HFK, Wu Y, Kou C, et al. Gender differences in general mental health, smoking, drinking and chronic diseases in older adults in Jilin province, China. Psychiatry Res. 2017; 251: 58-62.
- Li Y, Jiang Y, Zhang M, Yin P, Wu F, Zhao W. Drinking behaviour among men and women in China: the 2007 China Chronic Disease and Risk Factor Surveillance. Addiction. 2011; 106: 1946-1956.
- 35. Karpe F, Pinnick KE. Biology of upper-body and lower-body adipose tissue--link to whole-body phenotypes. Nat Rev Endocrinol. 2015; 11: 90-100.
- 36. Amati F, Pennant M, Azuma K, Dubé JJ, Toledo FG, Rossi AP, et al. Lower thigh subcutaneous and higher visceral abdominal adipose tissue content both contribute to insulin resistance. Obesity (Silver Spring). 2012; 20: 1115-1117.
- 37. Tramunt B, Smati S, Grandgeorge N, Lenfant F, Arnal JF, Montagner A, et al. Sex differences in metabolic regulation and diabetes susceptibility. Diabetologia. 2020; 63: 453-461.



### **About Us**

Clinics Cardiology is an open access, double peerreviewed, pedagogical journal steadfast to publish articles in all areas of Cardiology. Clinics Cardiology is a platform which provides a useful insight of clinical studies and improves the knowledge of disease from clinical perspective. The journal mainly acts as a source of information for surgeons and researchers who are interested to acquire more knowledge in this field.



#### **Contact Info**

