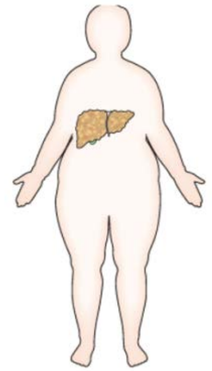

Actualización de nomenclatura “NASH a MASH” relevancia población diabética

Blanca Norero

Hepatología; CASR-RED UCHRISTUS

VIÑA DEL MAR 9 agosto 2024

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Nonalcoholic Steatohepatitis

Mayo Clinic Experiences With a Hitherto Unnamed Disease

JURGEN LUDWIG, M.D.
Department of Pathology and Anatomy

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Resident in Gastroenterology*

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Division of Gastroenterology
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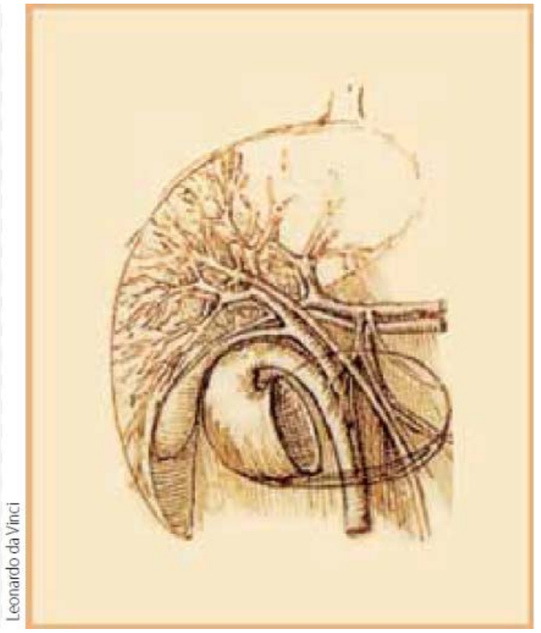
BEVERLY J. OTT
Division of Gastroenterology
and Internal Medicine

Nonalcoholic steatohepatitis is a poorly understood and hitherto unnamed liver disease that histologically mimics alcoholic hepatitis and that also may progress to cirrhosis. Described here are findings in 20 patients with nonalcoholic steatohepatitis of unknown cause. The biopsy specimens were characterized by the presence of striking fatty changes with evidence of lobular hepatitis, focal necroses with mixed inflammatory infiltrates, and, in most instances, Mallory bodies. Evidence of fibrosis was found in most specimens, and cirrhosis was diagnosed in biopsy tissue from three patients. The disease was more common in women. Most patients were moderately obese, and many had obesity-associated diseases, such as diabetes mellitus and cholelithiasis. Presence of hepatomegaly and mild abnormalities of liver function were common clinical findings. Currently, we know of no effective therapy.

Ludwig J et al , Mayo Clinic Exp. 1980.



Hepatocellular carcinoma in patients with non-alcoholic steatohepatitis

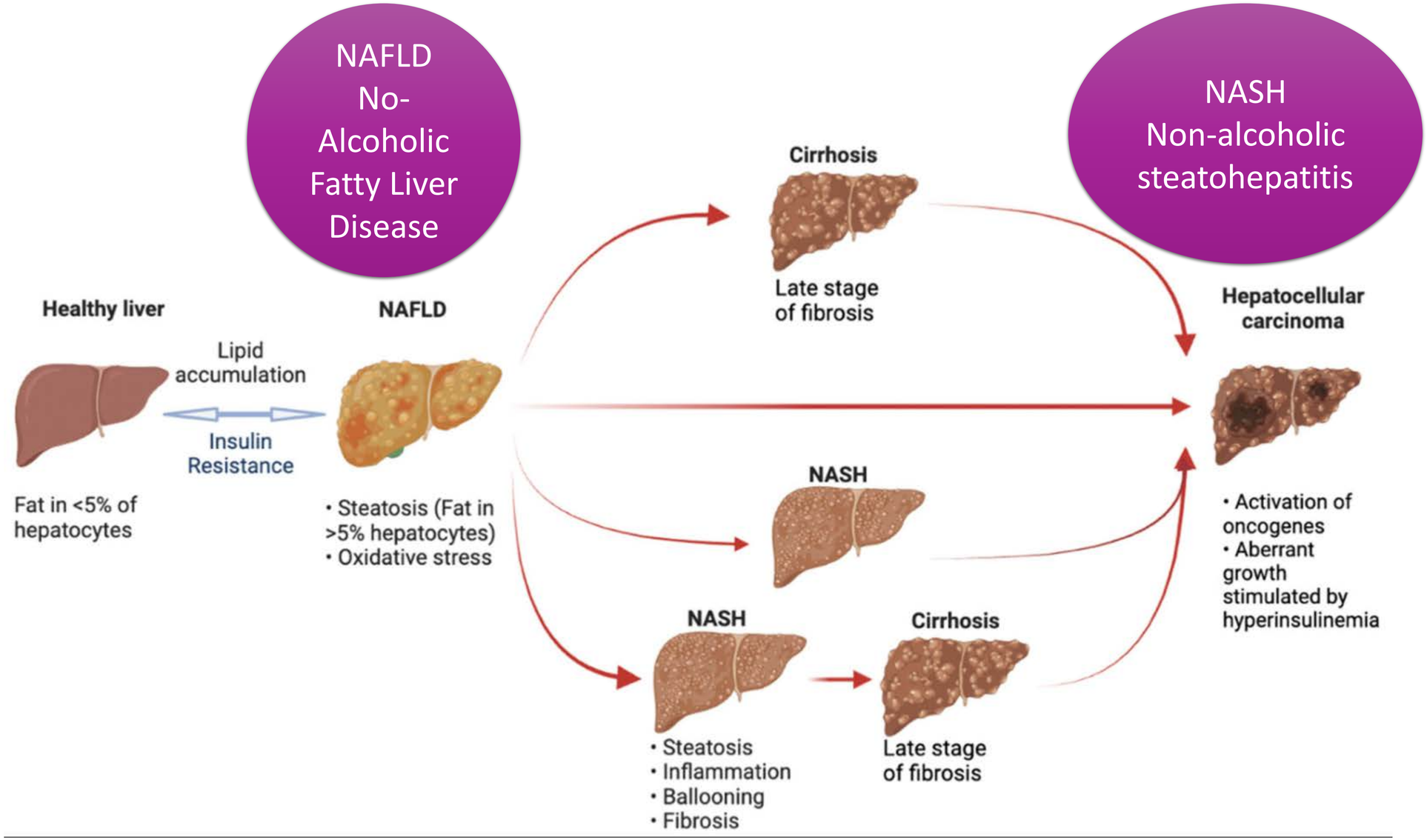
Masahiko Shimada • Etsuko Hashimoto • Makiko Taniai • ... Naoaki Hayashi • Ken Takasaki • Jurgen Ludwig • Show all authors

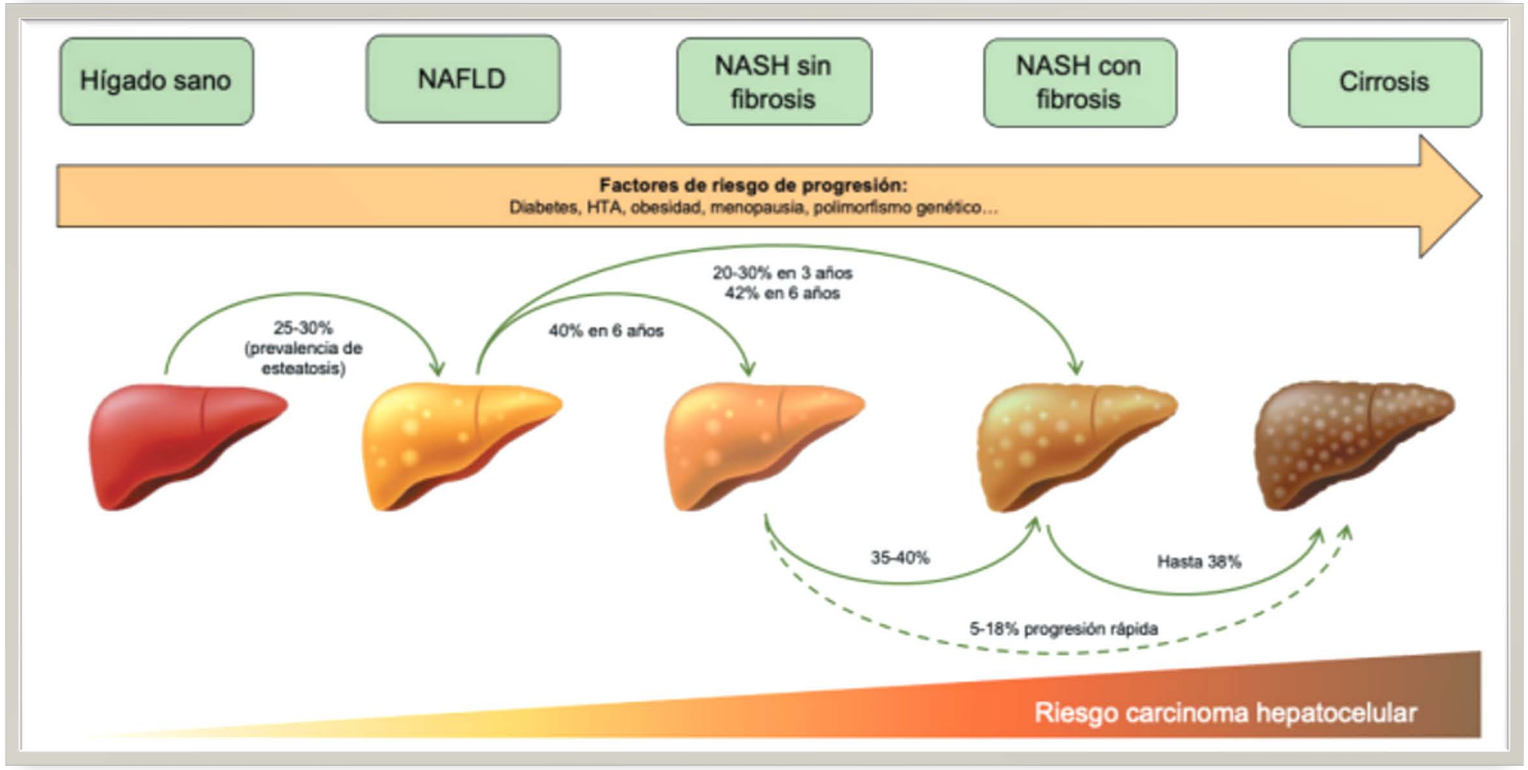
Abstract

We describe six patients with non-alcoholic steatohepatitis (NASH) and hepatocellular carcinoma (HCC). From 1990 to 2001, we treated 82 patients with NASH and observed six patients (three men and three women, aged 56–72 years) in this group who were referred with HCC or developed the complication during follow-up. In five of these six patients, NASH was associated with obesity (cases 3, 4 and 5), hyperlipidemia (case 5), or diabetes mellitus (cases 1, 3 and 6). We confirmed the presence of HCC by ultrasonography-guided tumor biopsy or surgery except in case 3 where we diagnosed the tumor by ultrasonography, computed tomography and selective hepatic arteriography. The carcinomas measured 1.5–6.0 cm in diameter and three were well differentiated. When HCC was diagnosed, cirrhosis was present in all instances. Four of the six tumor patients also had esophageal varices but only one patient had a history of variceal bleeding and ascites. Treatment of HCC consisted of surgery (cases 1 and 5), transcatheter arterial embolization or infusion and/or percutaneous ethanol injection (cases 2, 3, 4, and 6). In patients with NASH cirrhosis, the development of treatable HCC is sufficiently common to warrant regular screening for this grave complication.

Journal Hepatology 2002

Hígado Graso





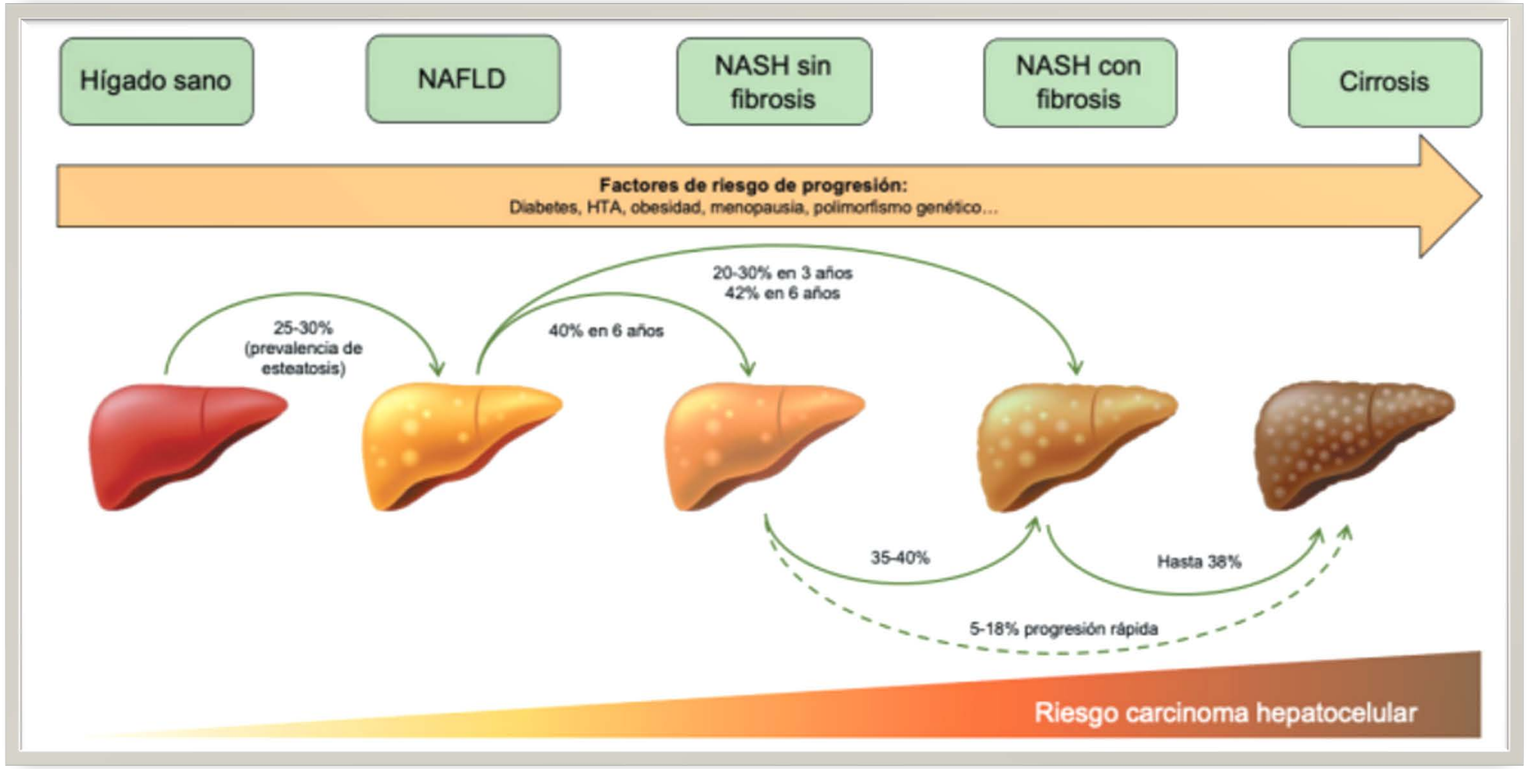
NAFLD: NOT ALCOHOLIC FATTY LIVER DISEASE

NASH: NOT ALCOHOLIC STEATOHEPATITIS

DEFINICIÓN:

IMAGEN CON ESTEATOSIS (>5%)

HISTORIA CLINICA CON MENOS DE GR (20/30) DE ALCOHOL EN MUJER/HOMBRE



DIAGNÓSTICO EXCLUSIÓN

ESTIGMA ALCOHOL

NO PERMITE COEXISTENCIA DE ETIOLOGIAS

EN INGLES “FATTY”

....

NECESDAD DE DEFINICIÓN:
- LIGADA A FISIOPATOLOGÍA
-CON CRITERIOS POSITIVOS
-PERMITA COEXISTENCIA DE FR

Expert Opinion

JOURNAL
OF HEPATOLOGY

A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement

Mohammed Eslam[1,*,†], Philip N. Newsome[2,*,†], Shiv K. Sarin[3], Quentin M. Anstee[4], Giovanni Targher[5], Manuel Romero-Gomez[6], Shira Zelber-Sagi[7], Vincent Wai-Sun Wong[8], Jean-François Dufour[9], Jörn M. Schattenberg[10], Takumi Kawaguchi[11], Marco Arrese[12], Luca Valenti[13], Gamal Shiha[14], Claudio Tiribelli[15], Hannele Yki-Järvinen[16], Jian-Gao Fan[17], Henning Grønbæk[18], Yusuf Yilmaz[19], Helena Cortez-Pinto[20], Claudia P. Oliveira[21], Pierre Bedossa[22], Leon A. Adams[23], Ming-Hua Zheng[24], Yasser Fouad[25], Wah-Kheong Chan[26], Nahum Mendez-Sanchez[27], Sang Hoon Ahn[28], Laurent Castera[29], Elisabetta Bugianesi[30], Vlad Ratziu[31,*,‡], Jacob George[1,*,‡]

Journal of Hepatology 2020

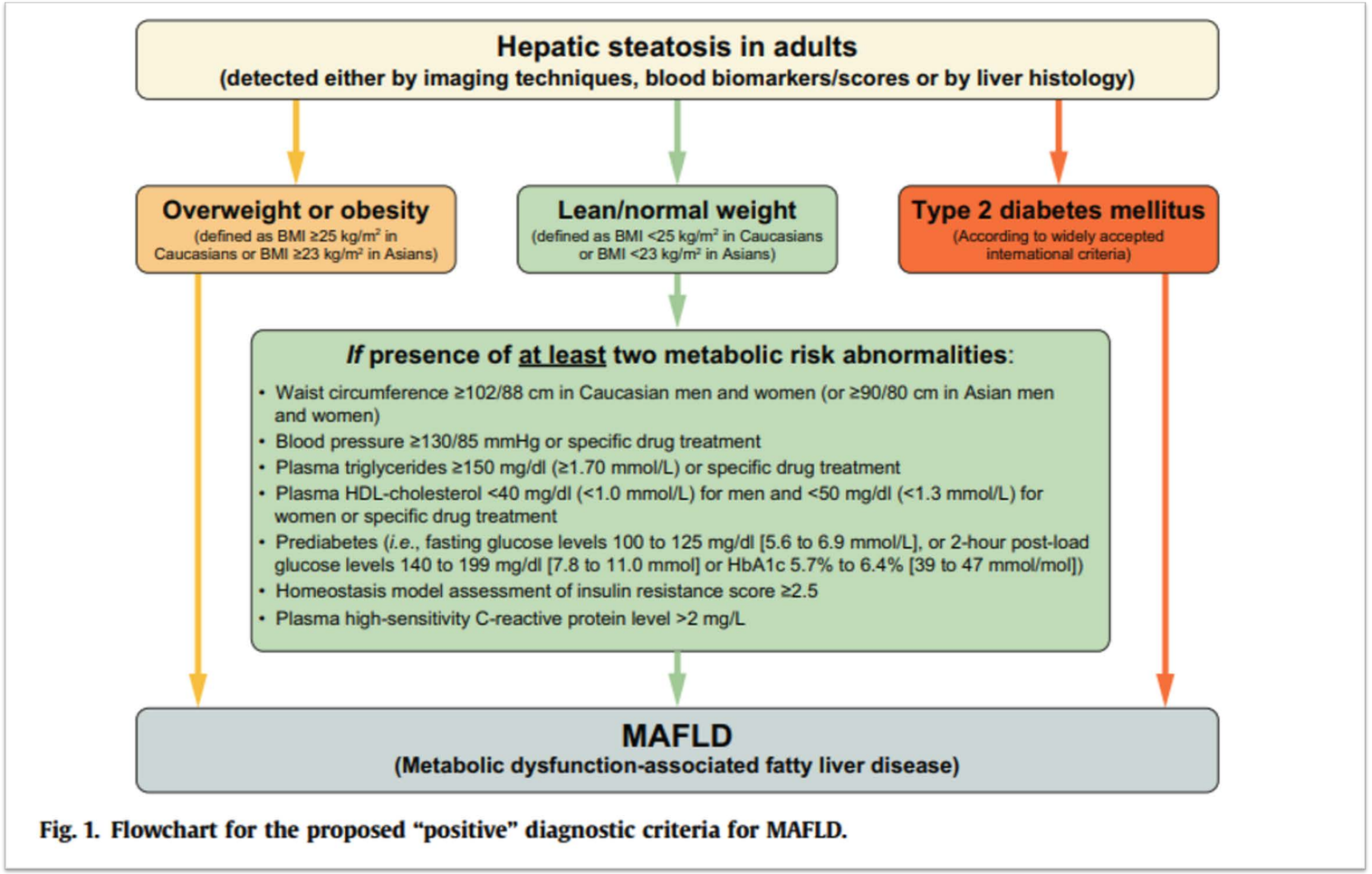
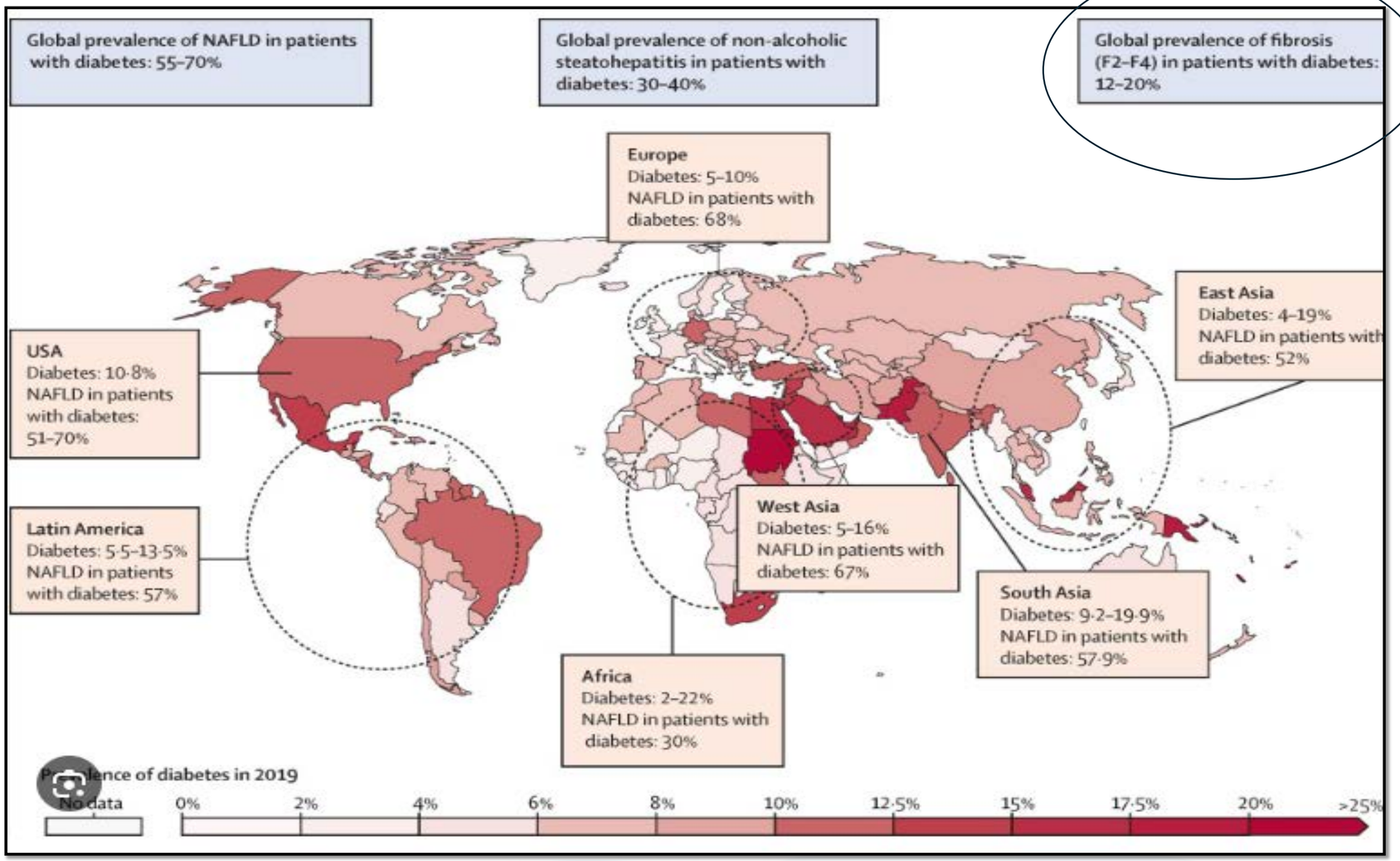


Fig. 1. Flowchart for the proposed "positive" diagnostic criteria for MAFLD.

Journal of Hepatology 2020



A global view of the interplay between non-alcoholic fatty liver disease and diabetes

Prof Norbert Stefan MD [a b], Prof Kenneth Cusi MD [c]

THE LANCET
Diabetes & Endocrinology

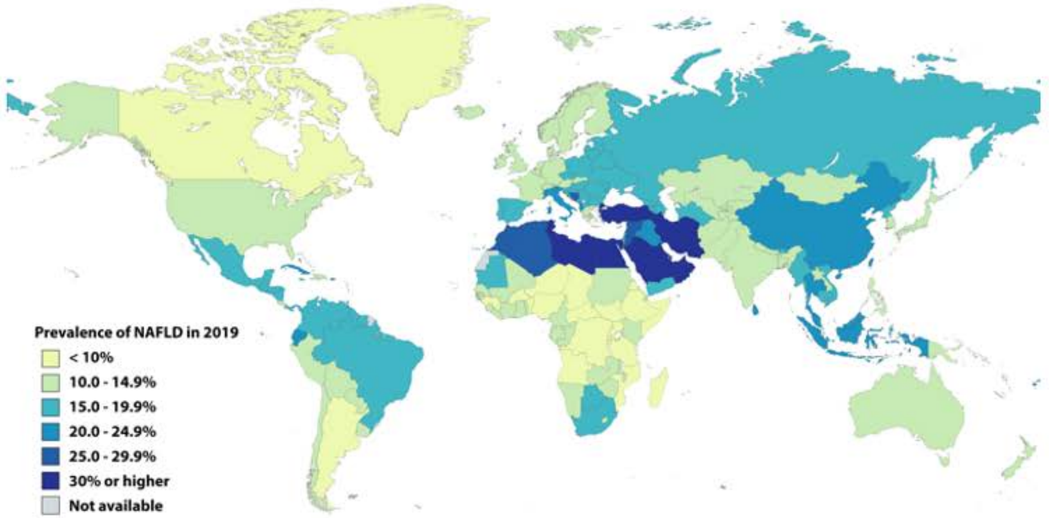
Prevalence of MASLD

Seminar

JOURNAL
OF HEPATOLOGY

Global burden of liver disease: 2023 update

Harshad Devarbhavi[1], Sumeet K. Asrani[2,*], Juan Pablo Arab[3,4], Yvonne Ayerki Nartey[5], Elisa Pose[6], Patrick S. Kamath[7]



MASLD:

- The worldwide **prevalence** of MASLD is **32.4%**.
- The percentage of **total deaths** from all causes attributable to MASLD increased from 0.10% to **0.17%**.
- MASLD represents the second-leading cause of **liver transplantation** and the leading cause among females.

ORIGINAL ARTICLE

OPEN

Diabetes and the risk of cirrhosis and HCC: An analysis of the UK Biobank

Fangzhou Ye[1,2] | Liangkai Chen[3,4] | Xin Zheng[1,2,5]

Hepatology Communications. 2023;7:e0280.

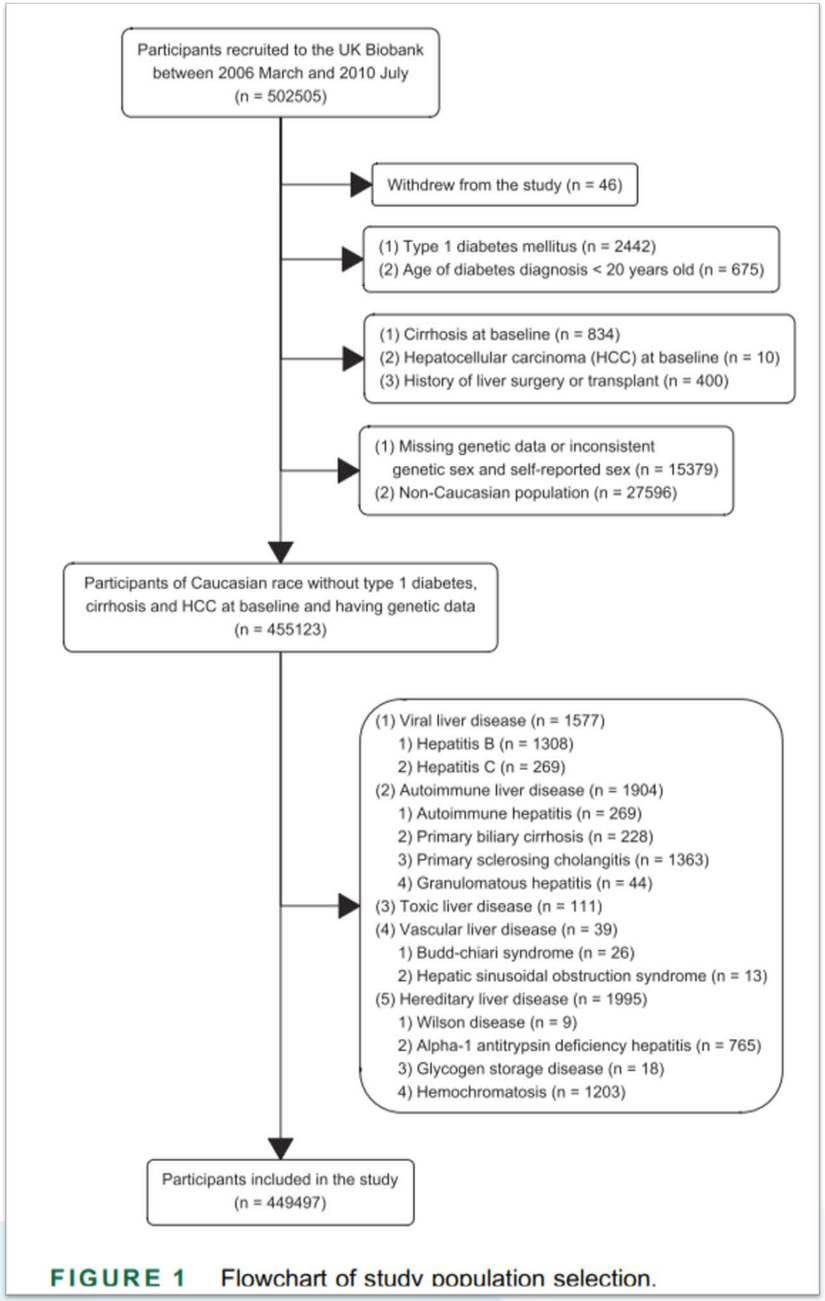


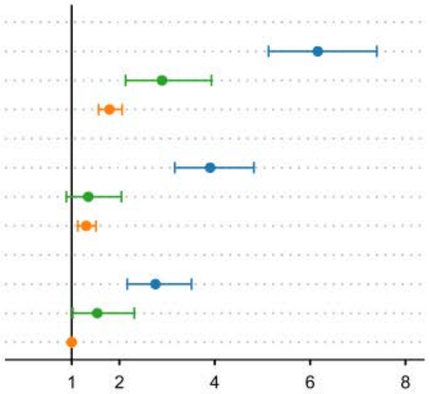
FIGURE 1 Flowchart of study population selection.

CIRROSIS INCIDENTAL

HCC INCIDENTAL

(A)

	HR (95% CI)	P value	Incident/ Total, No.	Rate/100 000 person-years
High genetic risk				
T2D	6.16 (5.13-7.40)	P < 0.001	206/7837	232.28
Prediabetes	2.89 (2.13-3.93)	P < 0.001	48/4471	93.61
No T2D and prediabetes	1.79 (1.56-2.06)	P < 0.001	561/137510	34.33
Medium genetic risk				
T2D	3.90 (3.16-4.82)	P < 0.001	130/7664	148.70
Prediabetes	1.35 (0.89-2.04)	P = 0.162	24/4504	46.17
No T2D and prediabetes	1.30 (1.13-1.51)	P < 0.001	412/137424	25.20
Low genetic risk				
T2D	2.76 (2.16-3.51)	P < 0.001	88/7433	103.96
Prediabetes	1.54 (1.02-2.31)	P = 0.040	25/4320	50.06
No T2D and prediabetes	1 [Reference]	NA	330/138334	20.04



(B)

	HR (95% CI)	P value	Incident/ Total, No.	Rate/100 000 person-years
High genetic risk				
T2D	13.81 (9.20-20.74)	P < 0.001	59/7825	66.19
Prediabetes	3.65 (1.71- 7.77)	P < 0.001	8/4407	15.78
No T2D and prediabetes	1.94 (1.35- 2.80)	P < 0.001	74/134215	4.63
Medium genetic risk				
T2D	5.83 (3.40-10.02)	P < 0.001	20/6287	27.77
Prediabetes	1.09 (0.26- 4.50)	P = 0.907	2/3742	4.62
No T2D and prediabetes	1.09 (0.71- 1.69)	P = 0.691	35/112757	2.61
Low genetic risk				
T2D	4.22 (2.45- 7.25)	P < 0.001	20/8822	19.85
Prediabetes	2.43 (1.03- 5.73)	P = 0.042	6/5146	10.09
No T2D and prediabetes	1 [Reference]	NA	48/166296	2.42

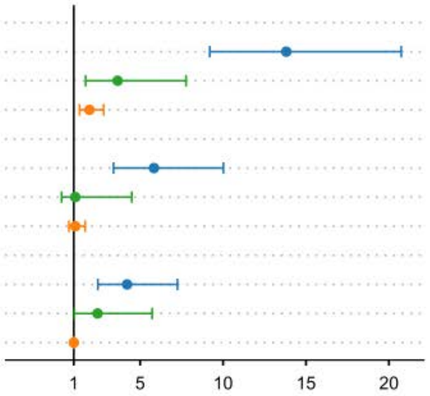


FIGURE 2 Risk of incident cirrhosis and HCC according to genetic risk and presence of diabetes or prediabetes. (A) Risk of incident cirrhosis; (B) Risk of incident HCC. Tertile of genetic risk for cirrhosis or HCC (low, medium, and high) was evaluated by polygenic risk scores, respectively. The P for interaction was 0.095 for cirrhosis and 0.081 for HCC, calculated by including a product term of diabetes status and the polygenic risk score of cirrhosis or HCC, respectively, adjusting for Model 3. Abbreviation: T2D, type 2 diabetes.

Special Article

JOURNAL
OF HEPATOLOGY

A multisociety Delphi consensus statement on new fatty liver disease nomenclature

Mary E. Rinella[1,*], Jeffrey V. Lazarus[2,3], Vlad Ratziu[4], Sven M. Francque[5,6], Arun J. Sanyal[7], Fasiha Kanwal[8,9], Diana Romero[2], Manal F. Abdelmalek[10], Quentin M. Anstee[11,12], Juan Pablo Arab[13,14,15], Marco Arrese[15,16], Ramon Bataller[17], Ulrich Beuers[18], Jerome Boursier[19], Elisabetta Bugianesi[20], Christopher D. Byrne[21,22], Graciela E. Castro Narro[16,23,24], Abhijit Chowdhury[25,26], Helena Cortez-Pinto[27], Donna R. Cryer[28], Kenneth Cusi[29], Mohamed El-Kassas[30], Samuel Klein[31], Wayne Eskridge[32], Jiangao Fan[33], Samer Gawrieh[34], Cynthia D. Guy[35], Stephen A. Harrison[36], Seung Up Kim[37], Bart G. Koot[38], Marko Korenjak[39], Kris V. Kowdley[40], Florence Lacaille[41], Rohit Loomba[42], Robert Mitchell-Thain[43], Timothy R. Morgan[44,45], Elisabeth E. Powell[46,47,48], Michael Roden[49,50,51], Manuel Romero-Gómez[52], Marcelo Silva[53], Shivaram Prasad Singh[54], Silvia C. Sookoian[15,55,56], C. Wendy Spearman[57], Dina Tiniakos[11,58], Luca Valenti[59,60], Miriam B. Vos[61], Vincent Wai-Sun Wong[62], Stavra Xanthakos[63], Yusuf Yilmaz[64], Zobair Younossi[65,66,67], Ansley Hobbs[2], Marcela Villota-Rivas[68], Philip N. Newsome [69,70,*], on behalf of the NAFLD Nomenclature consensus group

Journal of Hepatology, **December 2023**. vol. 79 | 1542–1556

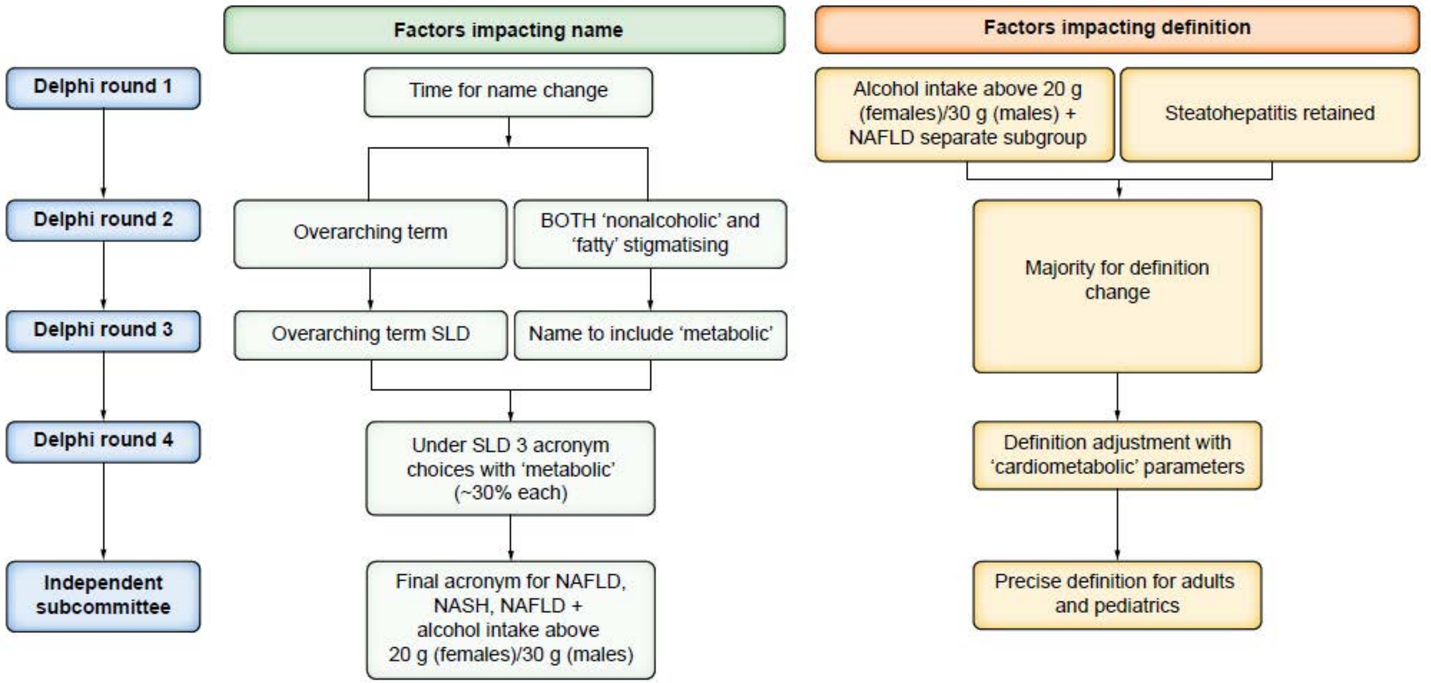
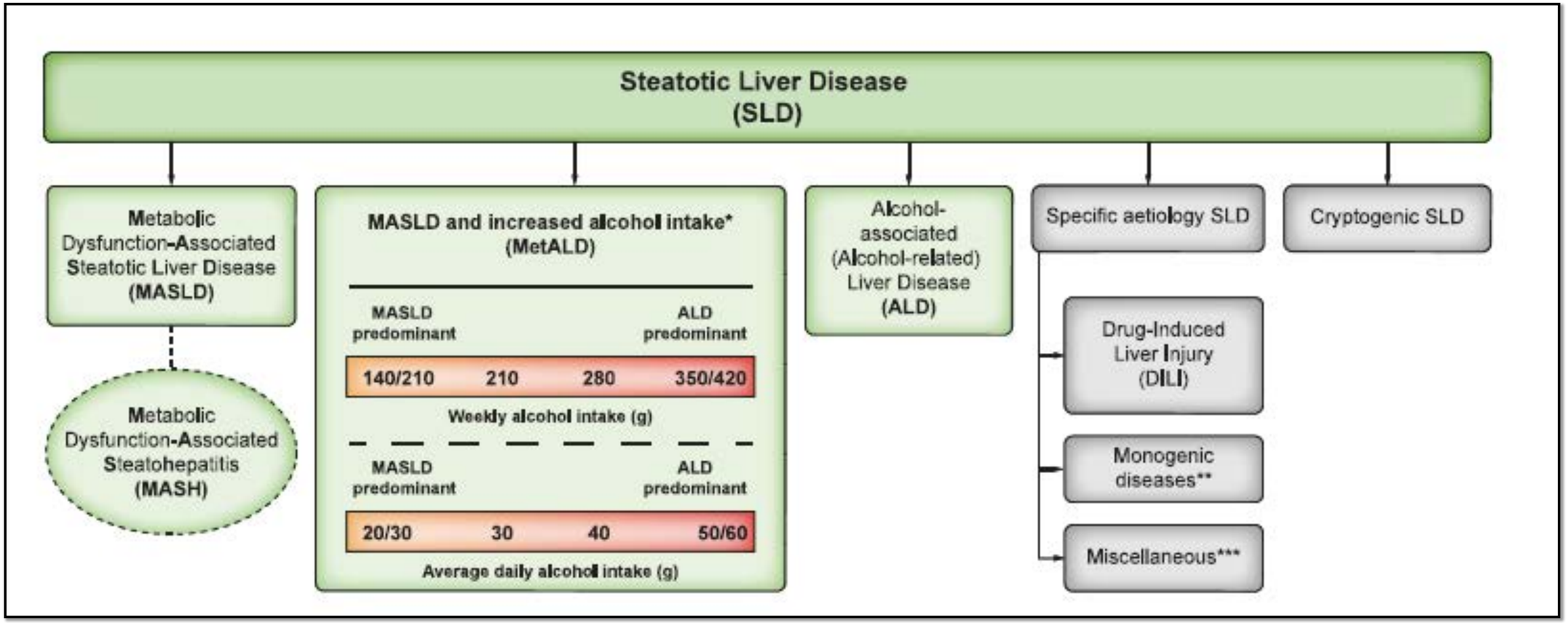
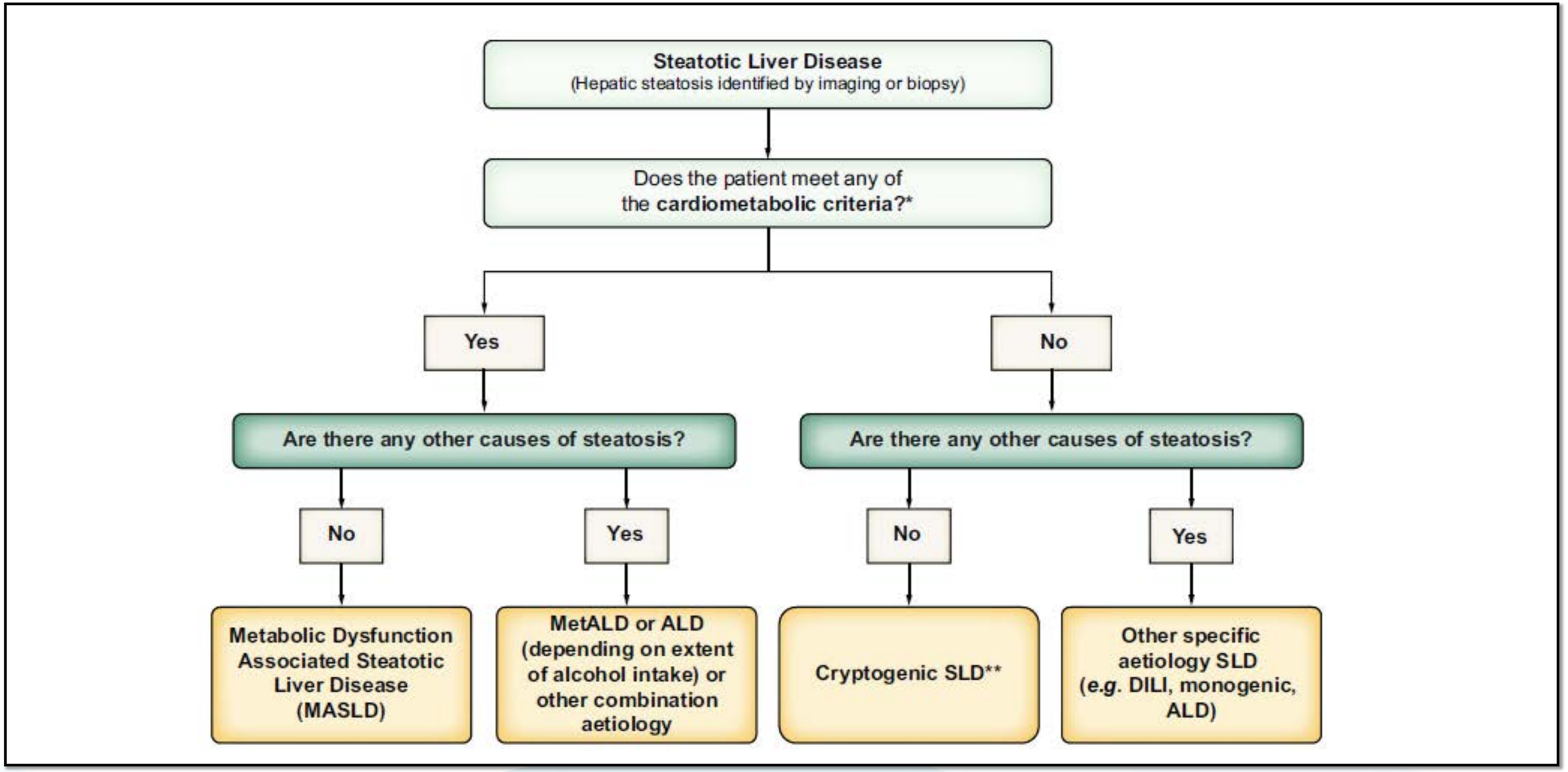


Fig. 3. Overview of main findings by Delphi round. The conclusions reached at the end of each Delphi round are depicted here. Results are shown at each corresponding Delphi round with respect to name change and definition, depicted in light green and orange, respectively. An independent subcommittee that comprised expert endocrinologists, hepatologists, paediatricians, and patients chose between the top 3 acronyms emerging from the fourth Delphi round and outlined the specifics of the definition to include cardiometabolic parameters, as dictated by the fourth Delphi round. Abbreviation: SLD, steatotic liver disease.



Journal of Hepatology, **December 2023**. vol. 79 | 1542–1556


Steatotic Liver Disease
(Hepatic steatosis identified by imaging or biopsy)
Does the patient meet any of the cardiometabolic criteria?*
Yes
No
Are there any other causes of steatosis?
Are there any other causes of steatosis?
No
Yes
No
Yes
Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD)
MetALD or ALD (depending on extent of alcohol intake) or other combination aetiology
Cryptogenic SLD**
Other specific aetiology SLD (e.g. DILI, monogenic, ALD)

*Cardiometabolic criteria

Adult criteria	Paediatric criteria
At least 1 out of 5:	**At least 1 out of 5:**
☐ BMI ≥25 kg/m² [23 Asia] **OR** WC >94 cm (M) 80 cm (F) **OR** ethnicity adjusted equivalent	☐ BMI ≥85th percentile for age/sex [BMI z score ≥+1] **OR** WC >95th percentile **OR** ethnicity adjusted equivalent
☐ Fasting serum glucose ≥5.6 mmol/L [100 mg/dl] **OR** 2-hour post-load glucose levels ≥7.8 mmol/L [≥140 mg/dl] **OR** HbA1c ≥5.7% [39 mmol/L] **OR** type 2 diabetes **OR** treatment for type 2 diabetes	☐ Fasting serum glucose ≥5.6 mmol/L [≥100 mg/dl] **OR** serum glucose ≥11.1 mmol/L [≥200 mg/dl] **OR** 2-hour post-load glucose levels ≥7.8 mmol [140 mg/dl] **OR** HbA1c ≥5.7% [39 mmol/L] **OR** already diagnosed/treated type 2 diabetes **OR** treatment for type 2 diabetes
☐ Blood pressure ≥130/85 mmHg **OR** specific antihypertensive drug treatment	☐ Blood pressure age <13 yr, BP ≥95th percentile **OR** ≥130/80 mmHg (whichever is lower); age ≥13 yr, 130/85 mmHg **OR** specific antihypertensive drug treatment
☐ Plasma triglycerides ≥1.70 mmol/L [150 mg/dl] **OR** lipid lowering treatment	☐ Plasma triglycerides age <10 yr, ≥1.15 mmol/L [≥100 mg/dl]; age ≥10 yr, ≥1.70 mmol/L [≥150 mg/dl] **OR** lipid lowering treatment
☐ Plasma HDL-cholesterol ≤1.0 mmol/L [40 mg/dl] (M) and ≤1.3 mmol/L [50 mg/dl] (F) **OR** lipid lowering treatment	☐ Plasma HDL-cholesterol ≤1.0 mmol/L [≤40 mg/dl] **OR** lipid lowering treatment

Síndrome metabólico

Efectos del cambio de nomenclatura

SLD/MASLD, sin estigma “fatty”

Criterios positivos, FRCM

FRCM; reflejo presencia de Sd. Metabólico

Falta de validación de FRCM como sustento de sd. Metabólico

Efecto cambio definición en validez de estudio clínicos previos

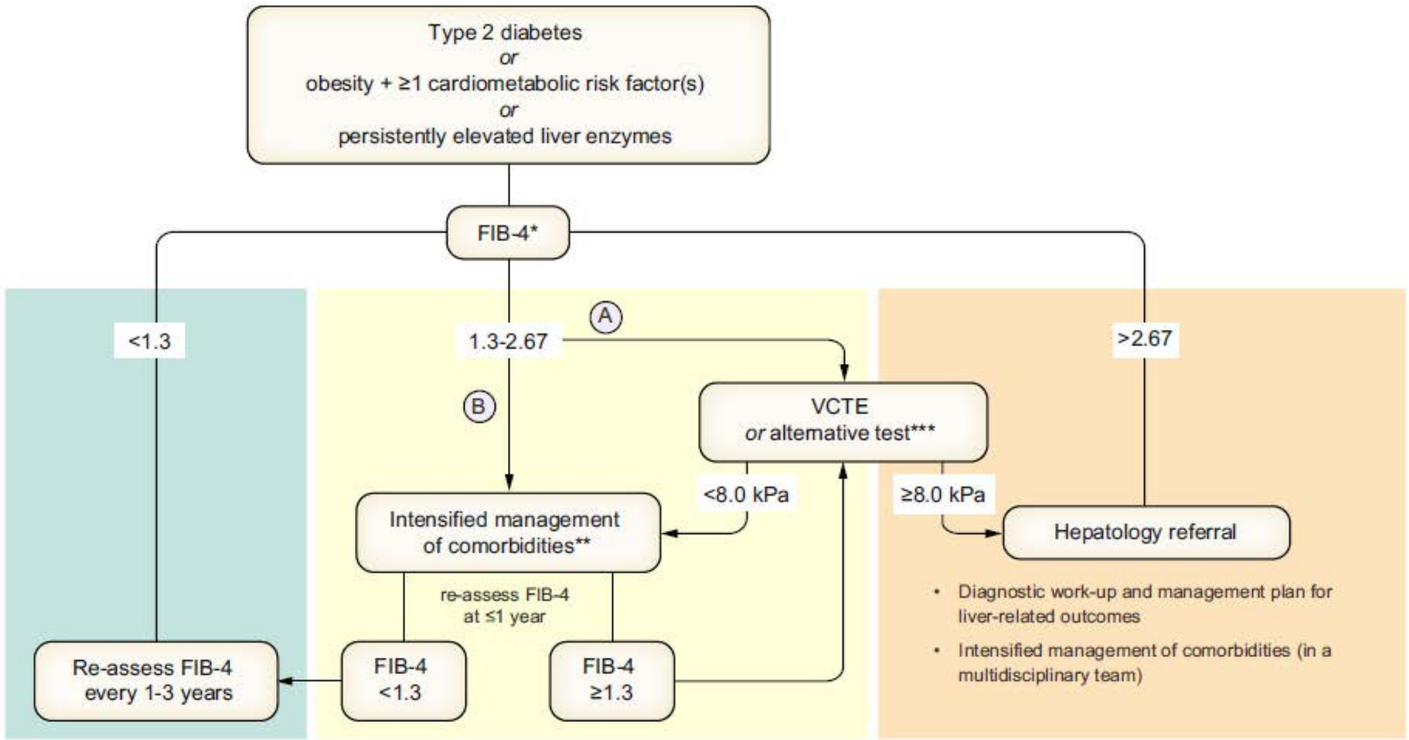


Fig. 2. Proposed strategy for non-invasive assessment of the risk for advanced fibrosis and liver-related outcomes in individuals with metabolic risk factors or signs of SLD. Individuals with (A) T2D or (B) abdominal obesity and ≥1 additional cardiometabolic risk factor(s) or (C) persistently elevated liver enzymes should undergo a multi-step diagnostic process, as indicated in the figure, to identify individuals with MASLD and advanced fibrosis. The algorithm can also be applied in case of incident finding of steatosis. This strategy is intended to identify individuals at risk of developing liver-related outcomes. ELF, enhanced liver fibrosis; FIB-4, fibrosis-4 index; GLP1RA, glucagon-like peptide-1 receptor agonist; MRE, magnetic resonance elastography; SLD, steatotic liver disease; SWE, shear wave elastography; VCTE, vibration-controlled transient elastography.

Clinical Practice Guidelines

JOURNAL OF HEPATOLOGY

EASL–EASD–EASO Clinical Practice Guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD)☆

European Association for the Study of the Liver (EASL)*, European Association for the Study of Diabetes (EASD), European Association for the Study of Obesity (EASO)

2024

Mensajes finales

-SLD; nuevo concepto global en hígado graso

-MASLD, énfasis en “M” metabolic

-Nueva definición, importancia de síndrome metabólico y FR cardiovascular en la génesis

-Enfoque Multidisciplinario

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Sociedad Chilena de Gastroenterología

—Adiós —dijo.

—Adiós —dijo el zorro—. He aquí mi secreto. Es muy simple: no se ve bien sino con el corazón. Lo esencial es invisible a los ojos.

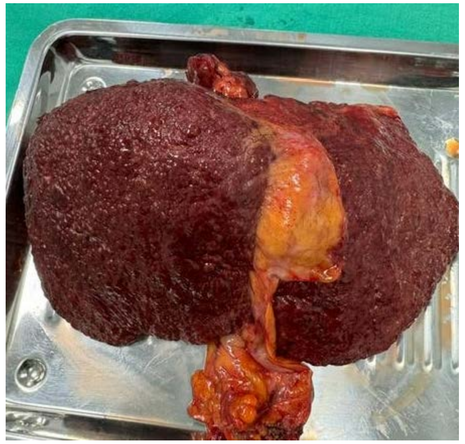
—Lo esencial es invisible a los ojos —repitió el principito, a fin de acordarse.

—El tiempo que perdiste por tu rosa hace que tu rosa sea tan importante.

—El tiempo que perdí por mi rosa... —dijo el principito, a fin de acordarse.

—Los hombres han olvidado esta verdad —dijo el zorro—. Pero tú no debes olvidarla. Eres res-

Y, tendido sobre la hierba, lloró.



Muchas gracias

Blanca Norero
blanca.norero@gmail.com

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Sociedad Chilena de Gastroenterología