Paediatric steatotic liver disease has unique characteristics:

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POSITION STATEMENT

Hepatology

Paediatric steatotic liver disease has unique characteristics: A multisociety statement endorsing the new nomenclature

European Society for Pediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) | European Association for the Study of the Liver (EASL) | North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition (NASPGHAN) | Latin-American Society for Pediatric Gastroenterology, Hepatology, and Nutrition (LASPGHAN) | Asian Pan-Pacific Society for Pediatric Gastroenterology, Hepatology and Nutrition (APPSPGHAN) | Pan Arab Society for Pediatric Gastroenterology and Nutrition (PASPGHAN) | Commonwealth Association of Paediatric Gastroenterology & Nutrition (CAPGAN) | Federation of International Societies of Pediatric Hepatology, Gastroenterology and Nutrition (FISPGHAN)

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Abstract

Nonalcoholic fatty liver disease (NAFLD) has been a commonly used term and diagnosis in paediatric hepatology, gastroenterology, and endocrinology clinics for over 30 years. A multisociety Delphi process has determined a new name “Steatotic Liver Disease” (SLD) as the overarching term for disorders associated with hepatic lipid accumulation. Our Societies give our support to steatotic liver disease as the best overarching term for use in our communities. Metabolic dysfunction-associated steatotic liver disease (MASLD) overcomes many of the shortcomings of the name NAFLD. Here, we highlight several points of the new nomenclature that are of particular importance for our community and their consequences for implementation including: diagnostic criteria, considering alternate diagnoses, practical implementation, research, advocacy, and education for paediatricians. As with all nomenclature changes, it will take a concerted effort from our paediatric societies to help integrate the optimal use of this into practice.

KEYWORDS
diagnostic algorithm, fatty liver disease, metabolism

Disclaimer: ESPGHAN is not responsible for the practices of physicians and provides guidelines and position papers as indicators of best practice only. Diagnosis and treatment is at the discretion of the healthcare provider. NASPGHAN is not responsible for the practices of physicians and provides guidelines and position papers as indicators of best practice only. Diagnosis and treatment is at the discretion of the healthcare provider.

1 | INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD) has been a commonly used term and diagnosis in pediatric hepatology, gastroenterology, and endocrinology clinics for over 30 years. The term "NAFLD" has been problematic in adult medicine and possibly even more so in pediatrics: "nonalcoholic" is confusing in young children and doesn't discriminate against alcohol consumption in adolescence. The use of "fatty" is stigmatizing,[1] to an extent that may potentially impair willingness to undergo lifestyle modifications and engagement with care. To date, there has been a lack of consensus on diagnostic criteria for "NAFLD" (e.g., the degree of elevation of liver enzymes, the need for imaging for definition, and the role of liver histology). This is problematic both from a practical clinical perspective, and also in the design of research and clinical trials in the condition. Moreover, the term "NAFLD" does not describe the underlying pathophysiology, that is, the metabolic dysfunction driven by insulin resistance. In response to this last specific concern, others have previously proposed the term "metabolic dysfunction-associated fatty liver disease" (MAFLD).[2] However, although "MAFLD" as an alternative for "NAFLD" has a solid mechanistic underpinning and requires positive criteria to confer a diagnosis, it continues to include the stigmatizing word "fatty," does not specify the importance of alternative diagnoses as causes of steatosis and does not address the possibility of overlapping conditions.

We are delighted that an international, multi-society Delphi process has determined a new name; "Steatotic Liver Disease" (SLD) as the overarching term for disorders associated with hepatic lipid accumulation.[3] The process focused on establishing nomenclature and definitions for subtypes of SLD once hepatic steatosis is suspected or confirmed. In those with hepatic steatosis in the setting of cardiometabolic risk factors, this is now termed metabolic dysfunction-associated steatotic liver disease (MASLD). The overarching term "SLD" also encompasses other aetiologies, specifically alcohol-associated liver disease (ALD) and increased alcohol use overlap with MASLD (Met-ALD), other specific aetiologies (e.g., drug-induced liver injury, monogenic causes of SLD) and also provides a category for people in whom currently no cause can be identified, that is, cryptogenic SLD (Figure 1). This differential diagnosis also recognizes that MASLD may coexist with another disease or etiology. Given the high prevalence of cardiometabolic risk factors, overweight and obesity in both adults and children, combination aetiologies are not uncommon[4] and dual pathology needs to be considered (e.g., MASLD-Wilson disease). Whilst many of the conditions (e.g., autoimmune hepatitis) are individually uncommon, collectively they are a large group of disorders that may overlap with the more common MASLD.

What is Known

- "Metabolic dysfunction associated steatotic liver disease" (MASLD) is the new name for "Nonalcoholic fatty liver disease."
- Other liver diseases may masquerade as paediatric MASLD.

What is New

- Globally, paediatric societies endorse the use of MASLD.
- The diagnostic pathway for paediatric MASLD differs to that of adult MASLD.
- Ongoing education & training is need for all stakeholders to implement the use of MASLD.

As with all nomenclature changes, it will take a concerted effort from our pediatric societies to help integrate the optimal use of this into practice. We would like to highlight several points that are of particular importance for our community and their consequences for implementation.

2 | PEDIATRIC-SPECIFIC ASPECTS OF THE NEW NOMENCLATURE

First, the diagnostic pathway for pediatric SLD as described in the new nomenclature mirrors that of adults and reflects some similarities across pathophysiology at different life stages. The diagnosis of MASLD is made by the presence of both steatosis and at least one cardiometabolic risk factor. It is notable that the diagnostic criteria for MASLD do not include measurement of insulin (either fasting or dynamically as part of a glucose tolerance test). Cut-off measurements for such risk factors are not well standardized in children, particularly in those under 10 years. The consensus document suggests only one out of five cardiometabolic criteria needs to be met in children to allow the diagnosis of MASLD. We are concerned that given the current low bar for a diagnosis of MASLD (steatosis with one cardiometabolic criteria as described), that other diagnoses or overlap diagnoses may be missed. We must therefore remain vigilant for alternative or combination aetiologies of hepatic steatosis in children.

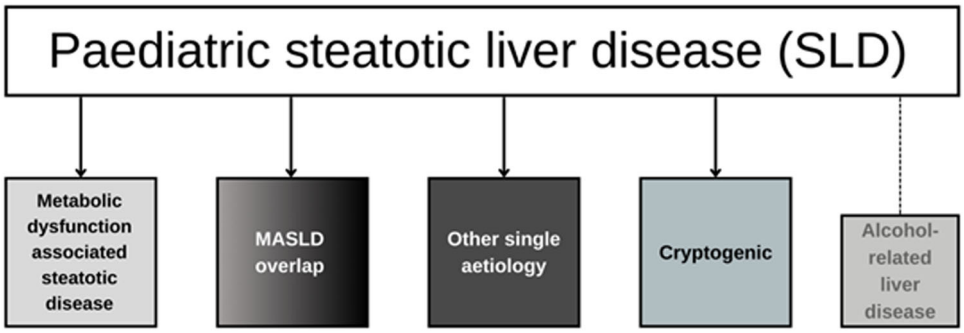

FIGURE 1 Depiction of steatotic liver disease (SLD) and subcategories in children. There are many possible aetiologies for SLD in a child. A diagnosis of MASLD may be made when the child has signs of steatosis and meets at least one cardiometabolic criterion and no evidence of other disease is found on investigation. The possibility for overlap diagnoses (MASLD plus another etiology) must always be considered and first line investigations are always indicated even in the presence of cardiometabolic risk factors. Alcohol related liver disease is a cause of SLD in children, but much less frequent than in adults. Steatosis with no identifiable cause (cryptogenic liver disease) should undergo periodic reevaluation as insulin resistance might not be evident at initial evaluation and with the rapid advances in genetic testing in particular, an affirmative diagnosis may follow on further testing. MASLD, metabolic dysfunction-associated steatotic liver disease.

The cardiometabolic criteria described in the Delphi statement are intended as proxies for insulin resistance and long-term risk of atherosclerotic disease.[3] This is analogous to the "metabolic syndrome,"[5,6] which has been used frequently throughout the literature and is a major risk factor for cardiovascular disease.[7] Here, we have continued to use "cardiometabolic criteria" for consistency with the original Delphi statement, on the basis that it reflects the same underlying pathophysiology as "metabolic syndrome."

We have recently written about the need for careful consideration of conditions such as Wilson disease, autoimmune hepatitis, and inborn errors of metabolism masquerading as SLD.[8] Whilst this is reflected in the current nomenclature change under the umbrella banner SLD, its importance in pediatrics is paramount. It should also be noted that the presence of hepatic steatosis per se induces a degree of "metabolic dysfunction," at least in terms of hepatic insulin resistance[9] and altered secretion of lipoparticles.[10] The probability of dual-diagnosis (e.g., MASLD-autoimmune hepatitis, not dissimilar to the coexistence of alcohol-related and nonalcohol-related injury in adult and adolescent patients) will rise as the prevalence of obesity increases.[4,11] So, although mentioned in the small print in the executive summary, considering alternate diagnoses is one of the key points for the assessment of children.

Pediatricians more frequently see patients with inherited metabolic disease than adult hepatologists. Inborn errors of metabolism are not uncommon indications for transplantation in children.[12] Thus "metabolic liver disease" in pediatric practice is most often used to describe a constellation of inborn errors of metabolism including urea cycle defects, organic aciduria, aminoaciduria, lysosomal disease, fatty acid oxidation defects to name but a few. In contrast, in the context of MASLD, "metabolic" refers to the syndrome of cardiometabolic abnormalities commonly associated with insulin resistance, rather than inherited single gene defects causing metabolic disease. It will require a concerted effort to make this clear to pediatricians who are less familiar with using the term "metabolic" in this way. Though children who are "lean" (have a BMI *Z* score <+1) may still have cardiometabolic risk factors, particularly in the setting of visceral adiposity or increased waist circumference, a monogenic cause of SLD must be considered with increased suspicion in children who are not overweight or obese by BMI criteria. Genetic analysis in such children may also reveal modifier variants such as p.Ile148Met in *PNPLA3*[13] which predispose them to development of steatosis and more significant liver disease at a lower range of BMI.

3 | CLINICAL IMPLEMENTATION OF MASLD

The new definition of MASLD should help assist clinicians in their assessment of children with undiagnosed liver disease (Figure 1). This holds true whether they are seen in specialist "steatotic liver clinics," obesity services, "general" or "core" pediatric services, gastroenterology clinics, or in primary care.

The diagnostic pathway starts with the suspicion of hepatic steatosis (Figure 2). The suspicion may arise completely incidentally, for example, during investigation of abdominal pain or as part of the screening in children with obesity for related comorbidity.[14] Steatosis may be suspected on the basis of elevated alanine aminotransferase (ALT), or of abnormal imaging. The thresholds for these tests were not defined in this nomenclature process and until more evidence on optimal pediatric thresholds become available the currently used thresholds still apply.[15–18]

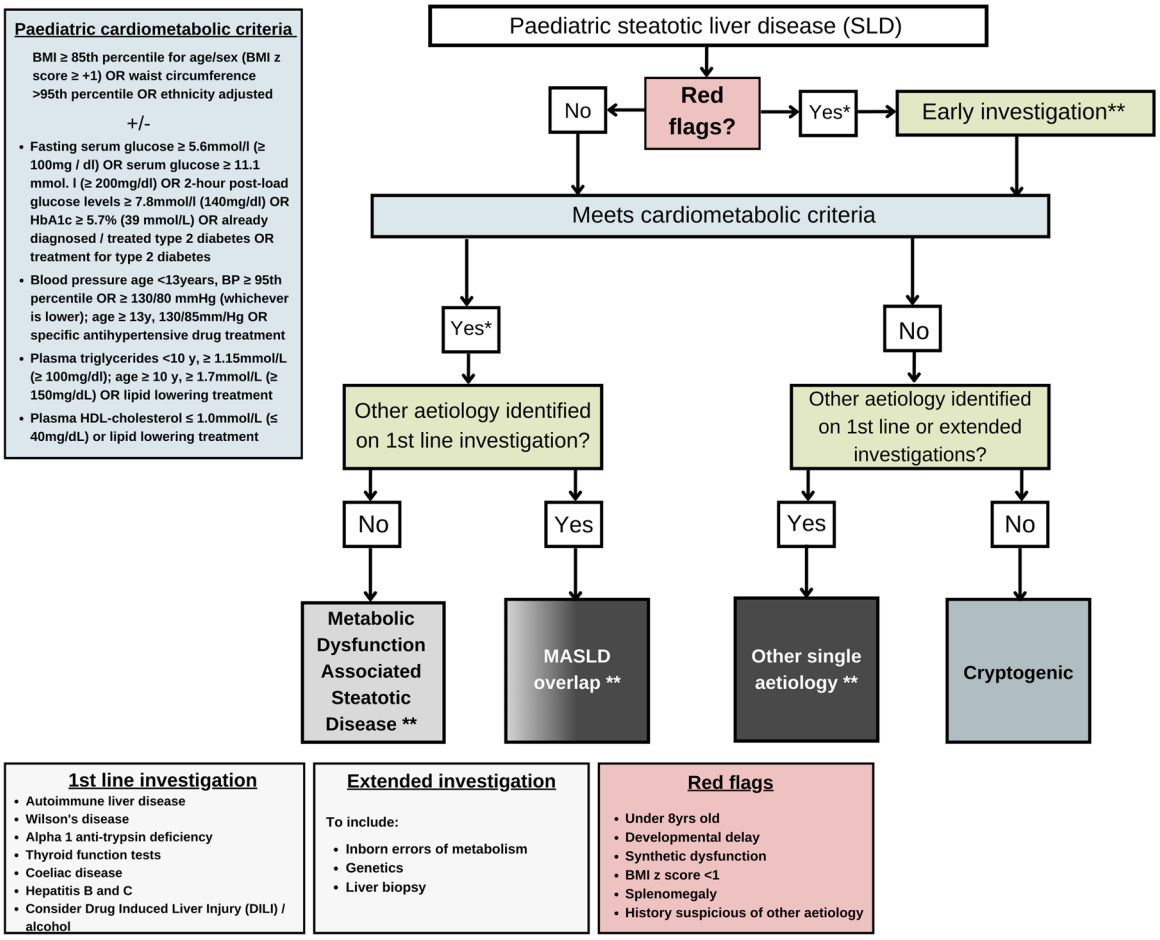

FIGURE 2 Diagnostic pathway for SLD in children. Children should all undergo first line investigation for "other etiology SLD" (i.e., non-MASLD diseases) including autoimmune hepatitis, Wilson disease, alpha-1-antitrypsin deficiency, hepatitis B and C viruses, and coeliac/thyroid disease. The child should also undergo simultaneous evaluation for the presence of cardiometabolic criteria. *If one or more cardiometabolic criteria are present, it is still possible that the child has a diagnosis of a combination of MASLD-other etiology, or even an alternative single etiology (e.g., raised triglycerides in familial hyperlipidaemia). **If the child is not overweight or obese and has no other cardiometabolic criteria, then they should undergo more extensive investigation to ascertain cause. In the case of any "red flags," even in the presence of metabolic syndrome, the child should also undergo more extensive evaluation. The precise nature of the extended investigations will depend on the clinical context and should include expert hepatology consultation. MASLD, metabolic dysfunction-associated steatotic liver disease.

Elevated ALT is obviously nonspecific and may indeed be normal in the context of even severe steatosis. In most cases, ultrasound can detect moderate to severe steatosis (>33%) but increased hepatic echogenicity may also occur in the setting of inflammation and infiltration of other substances (such as glycogen), not just fat. Magnetic resonance imaging (proton dense fat fraction or spectroscopy) is a more sensitive imaging technique in a research context to help confirm the presence of steatosis (>5% fat fraction most commonly used cut-off).[19] While vibration-controlled elastography (VCTE) is being increasingly used and studied in children and can provide estimates of liver stiffness and liver fat content, validated cut-offs are still being established for detection of increased hepatic steatosis.

All patients referred with suspicion of MASLD need investigation for a range of causes of chronic liver disease, whether they meet one or more of the cardiometabolic criteria compatible with MASLD (Figure 2). As a minimum, we expect this should include testing for autoimmune hepatitis, Wilson disease, viral hepatitis, alpha-1-antitrypsin deficiency, and coeliac disease, and screening for any alcohol use in older adolescents, as well as potential drug-induced liver injury if taking other medications. The further extent of this screen should be guided by clinical context and is reviewed elsewhere.[20] For example, there should be a low threshold of suspicion for inborn errors of metabolism, and this should be investigated accordingly.

This is particularly the case if "red flags" are present including young age (under 8 years), BMI *Z* score of <1,

neurodevelopmental delay, significant splenomegaly, synthetic dysfunction, or a history suggestive of an alternative diagnosis.[20] The failure of steatosis to improve in a child with obesity who loses weight and/or has decreasing BMI may suggest an alternative diagnosis. As mentioned, it is important that establishing an alternate diagnosis in a child does not exclude the diagnosis of MASLD as dual pathology is not uncommon. Moreover, in some circumstances, meeting the cardiometabolic criteria as they stand may indicate an alternative single etiology (e.g., low high-density lipoprotein cholesterol in abetalipoproteinaemia).

Alcohol-related liver disease is a major consideration for adult physicians investigating patients with steatosis. Chronic (excessive) alcohol consumption in adolescents leading to SLD is a much less frequent diagnosis. Whilst we acknowledge that alcohol history is an important part of assessment in young people, it is uncommon for it to be the principal diagnosis (Figure 1).

This diagnostic pathway is distinct from the staging of the severity of MASLD, and determination of risk of progression. Clear recommendations exist in adults for how to (noninvasively) assess MASLD using validated noninvasive biomarker panels and VCTE.[21,22] There is an urgent need for evidence-based guidance on the role of liver histology and for further studies of noninvasive methods of assessment (i.e., without biopsy) in children, as most biomarker panels established in adult cohorts have lower accuracy for detection of hepatic steatosis and fibrosis in children.

4 | RESEARCH IMPLICATIONS

Pediatric SLD is a rapidly moving field of research, with over 400 PubMed-listed articles published in the last year. A new name and new diagnostic criteria have the potential to improve the standardization of cohorts described as "MASLD."

Reanalyses of existing data have shown that applying MAFLD criteria to cohorts defined as "NAFLD" led to enrichment of those with nonalcoholic steatohepatitis (NASH) on biopsy,[23] and did not significantly change the proportion of young people identified with significant fibrosis or steatosis in the United States.[24] Applying the MASLD criteria to adults included in the European LITMUS consortium showed that 98% of the existing cohort fulfilled the new criteria for MASLD, thus existing cohorts are still representative despite the new diagnostic criteria.[3] Importantly, in the Delphi process it was decided that steatohepatitis and its histological definition remain unchanged. Only, the term NASH is replaced by metabolic dysfunction-associated steatohepatitis (MASH).[3] This continuity was important to maintain validity of prior biomarker development and clinical trials that have focused on steatohepatitis detection and treatment. However, pediatric steatohepatitis continues to have some unique histological features, including a higher prevalence of periportal predominant injury (inflammation and fibrosis),[25] which is not reflected in the current (MASH) or former (NASH) diagnostic criteria.

The move from NAFLD to MASLD will require a transition period. It will require care when describing existing results to clarify whether patients were defined as NAFLD, MAFLD, or MASLD. We encourage investigators to embrace MASLD (and not MASLD-/alcohol-/overlap) as a standard inclusion criterion for future studies. The requirement for exclusion of alternative diagnoses when assessing one etiology or alternatively study of potential additive or synergistic effects in case of a combined etiology, will remain as important as ever.

5 | EDUCATION AND ADVOCACY OF SLD

A new name brings the challenge of increasing complexity. Whilst the definition has been made as simple as possible, to non-experts the landscape of NAFLD, NASH, MAFLD, MASH, SLD, and MASLD is opaque.

To serve our patients effectively, we will need to provide clear educational programmes to support the nomenclature change. Endorsement of this terminology means action and will require significant resources and coordinated efforts to effect change including: online courses, in person seminars, use in electronic health records, billing, published guidelines, educational materials and websites, and outreach activities working with patient advocacy and support groups, organizations, and partner societies.

We are hopeful that the more inclusive language of SLD will act as a launchpad for increasing public awareness of the condition. Raising the profile of MASLD may facilitate earlier recognition with potential for altering the trajectory for long-term complications. SLD is a global health problem that requires concerted action from governments, health organizations, and communities to tackle.[26] We will use this new terminology as we campaign for these changes alongside affected patients and families.

6 | CONCLUSION

Determining a new name for a common condition is challenging. No set of diagnostic criteria can satisfy all permutations. Despite this, we agree that SLD is the best overarching term for use in our communities and MASLD overcomes many of the shortcomings of the name NAFLD. We will implement this nomenclature

change in clinical practice, research, advocacy, and education for pediatricians.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

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