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EDITORIAL

Diet is a key factor in the pathogenesis of metabolic dysfunction-associated steatotic liver disease in the pediatric population☆

La alimentación es pieza clave en la patogénesis de la enfermedad hepática esteatósica asociada a disfunción metabólica en la población pediátrica

In recent years, the scientific community has advanced significantly in understanding steatotic liver disease, promoting a consequential change in nomenclature. Traditionally known as nonalcoholic fatty liver disease (NAFLD), that term has been replaced by the more accurate and stigma-free designation, metabolic dysfunction-associated steatotic liver disease (MASLD).[1] In a joint collaboration, the American Association for the Study of Liver Diseases, The European Association for the Study of the Liver, and the Latin American Association for the Study of the Liver, supported by numerous international scientific societies, including the Latin American Society of Pediatric Gastroenterology, Hepatology and Nutrition (LASPGHAN), developed the term MASLD in an effort to focus attention on the main cause of the disease: metabolic dysfunction.[2] The term NAFLD was historically defined as the accumulation of fat in the liver, in the absence of significant alcohol use or other secondary causes. However, that implicitly negative definition created ambiguity, especially in the pediatric population, in which alcohol is not a relevant factor. In response to those limitations, the new term, MASLD, has a positive and clinically more useful focus, based on diagnostic criteria that include evidence of liver steatosis, together with at least one metabolic risk factor, such as obesity, type 2 diabetes, or dyslipidemia.[3]

The worldwide prevalence of MASLD is on the rise, reflecting parallel trends in childhood obesity. Between 7 and 14% of the pediatric population is estimated to present with obesity, and in adolescents, prevalence of the condition may reach 38%.[4] In Latin American countries, such as Mexico and Brazil, in which dietary patterns have rapidly been westernized, studies suggest a growing burden of MASLD in children and adolescents.

The transition to MASLD is not merely a semantic adjustment. It involves the explicit recognition that this disease is part of a broader spectrum of metabolic diseases, positioning it as a hepatic component of metabolic syndrome and facilitating more comprehensive diagnostic and therapeutic strategies.[1] Even though obesity is one of the main risk factors for MASLD, not all obese patients develop the disease, while at the same time, there are non-obese patients who do develop it. This suggests a complex interaction between genetic, epigenetic, hormonal, and primordially, nutritional factors.[3,5] In fact, standing out among the different factors that contribute to its development, are hereditary metabolic diseases, viral infections, adverse effects of medications, and in particular, a proinflammatory and hypercaloric diet, as important triggers of the disease.[6,7]

Nutrition and MASLD: what we eat is important

A study conducted on Brazilian pediatric patients is one of the first to systematically analyze the dietary intake of children and adolescents with MASLD.[8] In that cross-sectional analysis, the patients with MASLD were found to have a lower intake of proteins, vitamin E, zinc, and iron, compared with those without the disease, highlighting not only an energy imbalance but also deficiency in key antioxidant micronutrients. Despite the fact that after the multivariate adjustment, only the waist-height index remained the independent predictor, the study suggests that dietary quality may influence the appearance of the disease. Those findings are in line with the results of previous research. A recent

☆ See related content at DOI: 10.1016/j.rgmxen.2024.12.005, Santos P.Q. Metabolic dysfunction-associated steatotic liver disease and dietary intake characteristics in children and adolescents: A cross-sectional study. Rev Gastroenterol Mex. 2025.

Iranian study showed that the empirical lifestyle index for hyperinsulinemia (ELIH) was significantly associated with a higher risk of MASLD in obese children and adolescents, reinforcing the importance of the dietary and behavioral environment in the development of the disease.[9] Likewise, studies, such as those by Jain et al. in India and Tian et al. in China, confirm that elevated consumption of free sugars, saturated fats, and ultra-processed foods, together with a low intake of fiber and omega-3, are correlated with a higher incidence of liver steatosis in children.

The work by Santos et al.[8] provides relevant evidence in a Latin American context, underlining the importance of a diet, not only balanced in macronutrients, but also rich in antioxidant micronutrients. Low intake of vitamin E, zinc, and iron could indicate an important role of oxidative stress in the pathogenesis of pediatric MASLD, a phenomenon already suggested in previous studies and that is fertile ground for research and targeted dietary prevention. Contrasting with studies on Asian populations that show a more direct relation between glycemic load, or the glycemic index, and the appearance of MASLD, the need for adapting preventive strategies to specific cultural and dietary patterns in each region stands out.

Currently, there is no approved pharmacologic treatment for MASLD in children. Thus, lifestyle modification, including a balanced diet, regular physical activity, and reduced screen time, continue to be the first-line intervention.

Regarding future perspectives, noninvasive markers for early diagnosis, as well as possible pharmacologic interventions targeting the gut-liver axis, gut microbiota, and specific metabolic pathways involved in inflammation and liver fibrosis are being evaluated.

The approach to MASLD, from research to public healthcare policy, requires ongoing international collaboration. The recent re-definition of the term is a key step toward the better understanding, destigmatization, and effective management of this growing pediatric liver disease burden.

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