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Response to Letter to the Editor “Sleep hygiene in pediatric patients with steatotic liver disease”

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Dear Editor,

We thank Josef Finsterer for his constructive comments about our manuscript entitled “Association Between Gastrointestinal Symptoms and Sleep Habits in Children with Metabolic Dysfunction-Associated Steatotic Liver Disease: a Cross-Sectional Study” (1). We appreciate the opportunity to address the points raised.

The correspondent lists numerous potential sleep disruptors, including genetic factors, personality, comorbidities, sleep habits, environmental stressors, diet, late meals, and extreme factors such as earthquakes. While we acknowledge the multifactorial nature of sleep, it is methodologically not feasible to adjust for every conceivable confounder in a cross-sectional design. Our selection of covariates was guided by established evidence, and we believe that the listed factors such as earthquakes or geopolitical events are unlikely to have systematically biased our stable, single-center cohort. We have, however, acknowledged the potential for residual confounding in our limitations section (2).

Regarding the Children's Sleep Habits Questionnaire (CSHQ), the correspondent argues that polysomnography (PSG) or actigraphy should have been used. Our study aimed to screen parent-reported sleep habits in a large sample, not to diagnose sleep disorders (2). For epidemiological screening, validated questionnaires are widely accepted. The CSHQ has been validated in Turkish children (Cronbach's $\alpha=0.78$) and has a well-established clinical cut-off (3). PSG or actigraphy are resource-intensive, impractical for 176 children, and capture only a single night. We agree that objective methods are valuable and stated in our limitations that future studies should incorporate them. The citation of Markovich et al. (4) does not invalidate the CSHQ as a screening tool; it simply highlights that questionnaire and objective measures capture different aspects of sleep.

Respectfully, we do not agree with the suggestion of routine brain imaging. None of the enrolled children had clinical signs of structural CNS disease, and performing brain MRI on asymptomatic children without neurological red flags would be neither ethical nor indicated by any current guideline.

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Finally, the correspondent raises the possibility of hepatic encephalopathy (HE) as a confounder, suggesting that serum ammonia or cerebral MRI for HE should have been performed. Hepatic encephalopathy occurs almost exclusively in patients with cirrhosis and advanced liver failure (5). Our study included children with MASLD, none of whom had clinical or biochemical evidence of cirrhosis (normal albumin, no ascites, no jaundice). Citing articles on HE in cirrhotic patients is not applicable to our non-cirrhotic pediatric population. Therefore, routine measurement of serum ammonia or cerebral imaging for HE in asymptomatic, non-cirrhotic pediatric MASLD patients is not indicated by current guidelines (6).

In summary, none of the raised criticisms invalidate our main findings: gastrointestinal symptoms are independently associated with sleep disturbances in children with MASLD, and low family income and insulin resistance are significant predictors. We maintain that the additional investigations suggested by the correspondent are not warranted in our asymptomatic, non-cirrhotic pediatric cohort, and we have clearly acknowledged the inherent limitations of a cross-sectional design in our manuscript. We thank Dr. Finsterer for his comments, which have allowed us to further clarify the scope of our study.

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