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LETTER TO THE EDITOR

Effects of rifaximin in fructose-induced steatohepatitis in rats

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To the Editor,

I read with interest the recently published article titled "Effects of rifaximin in fructose-induced steatohepatitis in rats." The article discusses how rifaximin may protect against early liver damage caused by fructose. The researchers showed that rifaximin helps reduce certain biochemical markers and promote healing in inflamed tissue, providing insight into the interaction between the gut and liver in fatty liver disease, which plays a role in metabolic disorders. These results support the idea that rifaximin may help alleviate liver inflammation and oxidative stress caused by excess fructose in the early stages of fatty liver disease, also known as metabolic dysfunction-associated steatotic liver disease (MASLD).[1]

Although this study provides important information, I believe that further interpretation is required in a few areas. While fructose consumption over 8 weeks triggered early liver inflammation, no fibrosis was observed in the studied groups. Therefore, these research findings may be most applicable to the early stages of MASLD. Longer-term treatment with rifaximin, beyond 8 weeks, may be useful in determining its effects in more advanced stages of MASLD.[2]

Second, the dosage regimen of rifaximin requires clarification. The rationale for administering rifaximin once or three times per week was not fully explained. Any pharmacological or evidence-based justification for this dosage regimen should be clarified. This research supports the idea that rifaximin may help reduce inflammation and oxidative stress that can contribute to fatty liver disease in animals exposed to high fructose levels. Therefore, the authors' study provides a valuable contribution to the expanding knowledge of gut-liver axis therapies.

I congratulate the authors on their valuable contribution and hope that these comments will support ongoing scientific discussion.

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