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Reply. We appreciated the letter by Bhat and colleagues¹ and fully support the role of the clinical pharmacist in the management of patients with inflammatory bowel diseases (IBD). Pharmacists have several tasks ranging from patient education/counseling to medication management, from monitoring/follow-up to prevention of adverse events. Counseling with a clinical pharmacist has been associated with improvements in acceptability and adherence to treatment of patients with IBD, thus reducing the risk of relapse.² Interestingly, in patients with other chronic diseases, such as asthma and type 2 diabetes, pharmacist interventions led to better disease control and improvements in patient satisfaction and quality of life.³ In addition, during the current health emergency setting, pharmacists could assess compatibility and possible interactions between IBD drugs (eg, steroids, immunosuppressants, biologics, and small molecules) and Coronavirus Disease 2019 (COVID-19) therapy in subjects with suspected severe acute respiratory syndrome coronavirus 2 infection and in those with a confirmed COVID-19 diagnosis. Moreover, they could also monitor the pharmacovigilance data, excluding any late adverse events. A multidisciplinary approach that includes the pharmacist activity is essential to ensure optimal patient care and not to overlook important aspects related to safety, efficacy, and costs of drugs.⁴ The European Crohn's and Colitis Organization also emphasizes the importance of the clinical pharmacist by recognizing collaboration with at least 1 pharmacist/pharmacologist/health care professional educated in pharmacology as an indicator of excellence in the quality of care of patients with IBD.⁵ However, although there are undoubted advantages of multidisciplinary management, there is still a gap in clinical practice regarding integrated care to patients with IBD, and the clinical pharmacist is not a frequent component of the IBD unit.⁶ Further efforts are needed to implement the active role of pharmacists within the IBD units to improve quality of care for patients with IBD. As pharmacists are present in all the hospitals, specific IBD educational training should be promoted by scientific societies.

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Conflicts of interest

Laurent Peyrin-Biroulet has served as a speaker, consultant, and advisory board member for Merck, AbbVie, Janssen, Genentech, Mitsubishi, Ferring, Norgine, Tillots, Vifor, Hospira/Pfizer, Celltrion, Takeda, Biogaran, Boehringer-Ingelheim, Lilly, HAC-Pharma, Index Pharmaceuticals, Amgen, Sandoz, Forward Pharma GmbH, Celgene, Biogen, Lycera, Samsung Bioepis, and Theravance. Silvio Danese has served as a speaker, consultant, and advisory board member for Schering-Plough, AbbVie, MSD, UCB Pharma, Ferring, Cellerix, Millenium Takeda, Nycomed, Pharmacosmos, Actelion, Alphawasserman, Genentech, Grunenthal, Pfizer, Astra Zeneca, Novo Nordisk, Cosmo Pharmaceuticals, Vifor, Johnson & Johnson, Nikkiso Europe GMBH, and Theravance. The remaining author discloses no conflicts.



Most current article

<https://doi.org/10.1053/j.gastro.2020.12.072>

The Noticeable Crosslink between miR-122 and Metabolic Dysfunction



Dear Editors:

I read the recent article by Chai et al¹ with great interest. The authors have shown that the beneficial role of increased hepatic miR-122 induced by an RAR-related orphan receptor A (RORA) agonist in the improvement of hepatic lipotoxicity, liver fibrosis, and body weight in nonalcoholic steatohepatitis (NASH) was mediated through the activation of RORA to reveal not only local but also remote action for alleviating NASH progression.¹ However, the linkage between miR-122 and metabolic modulation remains incompletely clear.

The activation of RORA was implicated in metabolic circadian rhythm, and the dietary composition of free fatty acids connected with RORA–hepatic miR-122–triglyceride circuitry seemed crucial. According to my previous commentary on miR-122 in hepatic lipid metabolism, the effect of hepatic miR-122 on metabolic shift can be altered under different nutritional conditions.² In early stage NASH, the compensated mechanism is consistent with the findings by Chia et al because hepatic miR-122 levels in the high-fat diet group were lower than those in the normal diet group, whereas circulating and white adipose tissues (WAT) miR-122 levels were higher in high-fat diet group than those in normal diet group. Furthermore, silencing endogenous hepatic miR-122 by antagomiR-122 was accompanied by