

Letter: diarrhoea in obese patients—a new nosographic entity?

EDITORS,

With great interest, we read the paper by Ballou et al showing that chronic diarrhoea correlates with obesity and that the risk of diarrhoea increases with the severity of obesity.¹ Through an accurate analysis of the United States National Health and Nutrition Examination Survey (2009-2010), the authors found that 72.3% were overweight or up to severely obese. Obesity and related metabolic syndrome represent major challenges in Western countries for both patients who believe themselves healthy, and clinicians. Cardiologists, diabetologists and hepatologists are commonly involved in the management of obese patients, whereas gastroenterologists only rarely. However, a high number of patients are referred to gastroenterology outpatient clinics for chronic diarrhoea.² The causes are unknown, and a combination of mechanisms may come into play. Here we provide a tentative explanation of plausible mechanisms underlying obesity-related chronic diarrhoea. First, obese patients usually eat and chew for a longer period of time over 24 hours than non-obese people leading to persistent vagal stimulation that promotes gastric emptying. Continuous eating (often even during the night) causes the absence of inter-digestive fasting motor pattern with an increased fed motility pattern exacerbating small intestine mixing and propulsive activity. This enhances colonic filling and, therefore, the evacuation stimulus. Second, the overload of ingesta reaching the gut lumen may overcome the digestive/absorptive capacities, while exerting an osmotic effect. Further to the total amount, the composition of the diet (eg, high calorie, “junk” food, low fibre) may play a crucial role by inducing dysbiosis that, in turn, produces colorrhetic metabolites.³ Such dysbiosis, mainly characterised by a decrease of *Bacteroidetes* to *Firmicutes* ratio and a relative abundance of *Faecalibacterium prausnitzii* and *Akkermansia muciniphila*, alter bile acid composition, impair the epithelial barrier (“leaky gut”) and activate mucosal inflammation and secreto-motor reflexes.⁴ This is why we propose the term “metabolic diarrhoea” to address such pathophysiological complexity underlying obesity-related chronic diarrhoea. We believe that a prompt recognition of this new entity and a multidisciplinary approach may help physicians to offer appropriate management including lifestyle changes for patients. A combination of agents affecting bile salt composition, microbiota, mucosal inflammation and gut motility, in addition to dietary education, may be useful to treat this condition.

ACKNOWLEDGMENTS

Declaration of personal interests: None.

FUNDING INFORMATION

This work was partially supported by the Fondo Incentivazione Ricerca (FIR) and Fondi Ateneo per la Ricerca (FAR) to R De G (from University of Ferrara).

LINKED CONTENT

This article is linked to Ballou et al paper. To view this article, visit <https://doi.org/10.1111/apt.15500>.

Rachele Ciccocioppo¹
Roberto De Giorgio² 

¹Gastroenterology Unit, Department of Medicine, A.O.U.I. Policlinico G.B. Rossi, University of Verona, Verona, Italy
²Department of Medical Sciences, St. Anna University Hospital, Ferrara, Italy
Emails: dgrrrt@unife.it;
roberto.degiorgio@unife.it

ORCID

Roberto De Giorgio  <https://orcid.org/0000-0003-0867-5873>

REFERENCES

1. Ballou S, Singh P, Rangan V, Iturrino J, Nee J, Lembo A. Obesity is associated with significantly increased risk for diarrhoea after controlling for demographic, dietary and medical factors: a cross-sectional analysis of the 2009–2010 National Health and Nutrition Examination Survey. *Aliment Pharmacol Ther.* 2019;50:1019-1024.
2. Camilleri M, Malhi H, Acosta A. Gastrointestinal complications of obesity. *Gastroenterology.* 2017;152:1656-1670.
3. Araújo JR, Tomas J, Brenner C, Sansonetti PJ. Impact of high-fat diet on the intestinal microbiota a small intestinal physiology before and after the onset of obesity. *Biochimie.* 2017;141:97-106.
4. Maruvada P, Leone V, Kaplan LM, Chang EB. The human microbiome and obesity: moving beyond associations. *Cell Host Microbe.* 2017;22:589-599.

AP&T correspondence columns are restricted to letters discussing papers that have been published in the journal. A letter must have a maximum of 500 words, may contain one table or figure, and should have no more than 10 references. It should be submitted electronically to the Editors via <http://mc.manuscriptcentral.com/apt>.