Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

2. Taxonera C, et al. Aliment Pharmacol Ther 2020;52:276–283.
3. Mao R, et al. Lancet Gastroenterol Hepatol 2020;5:425–427.
4. Norsa L, et al. Gastroenterology 2020;159:371–372.
5. Scaldaferri F, et al. United European Gastroenterol J 2020;8:775–781.
6. Lopetuso LR, et al. Endosc Int Open 2020;8:E980–E984.
7. Papa A, et al. Am J Gastroenterol 2020. https://doi.org/10.14309/ajg.0000000000000830. Online ahead of print.
8. Tursi A, et al. Gut 2020;69:1364–1365.

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Lessons From COVID-19, ACE2, and Intestinal Inflammation: Could a Virus Trigger Chronic Intestinal Inflammation?

Dear Editor:

We read with interest the comprehensive review by D'Amico et al[1] on diarrhea during coronavirus disease-19 (COVID-19) infection, caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2).[1]

It is now established that SARS-CoV-2 infectivity is mediated by an interaction between viral spike proteins and ACE2 expressed on target mucosal membranes, with subsequent shedding of the ACE2 ectodomain following cellular entry.[2] Multiple downstream effects of this interaction may perpetuate inflammatory response, including reduced Ang 1-7 levels (the effector peptide of the alternative renin-angiotensin system [RAS] pathway), elevated angiotensin II (the effector peptide of the classical RAS pathway), increased tumor necrosis factor-α, and tryptophan deficiency.[3] Given it has previously been shown that all components of the RAS can be identified in enteric mucosa biopsies, suggesting locally active intestinal RAS, it is perhaps unsurprising that the SARS-CoV-2 enteric interaction is sufficient enough to initiate a symptomatic inflammatory response.[4]

Interestingly, ACE2 activity is lower in inflamed colonic biopsies of patients with inflammatory bowel disease compared with those with normal bowel mucosa.[4] Given SARS-CoV-2 results in ACE2 alteration in mucosal membranes, it is feasible that these 2 pathologies may ultimately share a proinflammatory pathway.[4]

Inflammatory bowel disease is considered a consequence of a dysregulated and inappropriate immune interaction to intestinal microorganisms, with most literature to date focused on bacterial dysbiosis. Recent recognition that both eukaryotic viruses and bacteriophages contribute significantly to the gut microbiome, and the fact that phages are closely associated with bacterial virulence, raises the possibility that alterations may perturb symbiosis and generate a dysregulated immune response.[5]

When considering multisystem consequences of COVID-19, perpetuation of inflammation and fibrosis in the lungs has been described, placing affected individuals at risk of long-term respiratory morbidity.[6] The persistence of intestinal inflammation and development of fibrosis, and implications for long-term gastrointestinal morbidity, remain to be seen. The study of this potential phenomenon may hold vital clues toward understanding any postulated role that enteric viruses may play in the pathogenesis of inflammatory bowel disease. This warrants careful consideration.

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References

1. D'Amico F, et al. Clin Gastroenterol Hepatol 2020;18:1663–1672.
2. Hoffmann M, et al. Cell 2020;181:271–280.
3. Garg M, et al. Gastroenterol 2020 Apr 27.
4. Garg M, et al. Gut 2019;69:gutjnl-2019-318512.
5. Ungaro F, et al. United European Gastroenterol J 2019;7:999–1007.
6. George PM, et al. Lancet Respir Med 2020;8:457–481.

Conflicts of interest
The authors disclose no conflicts.

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Cost-effectiveness of Telemedicine-directed Specialized vs Standard Care for Patients With Inflammatory Bowel Diseases in a Randomized Trial

Dear Editor:

We read with attention the recently published article by de Jong et al[1] about the cost-effectiveness analysis of telemedicine-directed specialized versus standard care for patients with inflammatory bowel diseases (IBD). The authors published in 2017 the largest multicenter clinical trial evaluating telemedicine in IBD, enrolling a broad spectrum of patients representative of daily clinical practice. In this new article, they recently found that telemedicine was cost-effective compared with standard care, using the economic data collected alongside their pragmatic clinical trial.