concern about misdiagnosis. First, GI infections frequently result in diarrhea, whereas individuals with eating disorders typically report delayed gastric emptying or constipation. Second, there are very few GI infections for which the recommended treatment is anti-infective agents, particularly in children and adolescents; rather, the most common treatment is symptomatic, eg, rehydration. Thus, while some clinical symptoms of an eating disorder can mimic GI disorders, we believe that it is unlikely that an individual would be misdiagnosed as having a GI infection mimicking an eating disorder if antibiotics were prescribed.

Giel et al also discuss the reported incidence rate of anorexia nervosa and bulimia nervosa noting that the incidence of anorexia nervosa was unexpectedly higher than that of bulimia nervosa. Epidemiological studies in Norway, Germany, Finland, Portugal, Holland, and the United Kingdom show a similar pattern, with the prevalence (point and lifetime prevalence) of anorexia nervosa higher than bulimia nervosa (see a review by Galmiche et al). Thus, we believe that the reported incidence of anorexia nervosa and bulimia nervosa in our study reflect the European population rather than misclassification of anorexia nervosa as an infection.

The issues of detection and diagnosis in eating disorders remains our highest priority, and we welcome scrutiny and suggestions to improve as a field. We agree that a psychosomatic approach in medicine greatly benefits the diagnosis and treatment of eating disorders. We have taken every effort to be transparent about the lack of causal mechanisms between inflammation and eating disorders and welcome replication, validation, and experimental testing of the results presented in our article. We look forward to continuing to use epidemiological register-based studies to inform well-designed longitudinal studies in eating disorders to test cause-and-effect models.

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