Reply to Röser and Stensvold

To the Editor—We appreciate the interest that Röser and Stensvold [1] have taken in our recent report by raising anisakiasis as a differential diagnosis in the family cluster of illness attributed to Dientamoeba fragilis [2]. However, the
clinical and epidemiological aspects of the case make anisakiasis improbable.

The hallmark of the anisakiasis is nausea and vomiting, often associated with marked abdominal pain [3]. A small subset of patients may proceed to a chronic relapsing illness associated with milder symptoms, including abdominal pain. In others the illness may present with allergic symptoms. Approximately one-third of patients develop a peripheral eosinophilia [3]. No patient in the cluster we described reported an acute illness and none presented with the typical symptoms of nausea, vomiting, or marked abdominal pain, nor urticarial rash or angioedema. The clinical response to metronidazole in the 2 symptomatic cases would also not be consistent with anisakiasis.

Epidemiological data do not support the presence of Anisakidae species implicated in human disease on the east coast of Australia. To our knowledge, there have been no cases of anisakiasis diagnosed in this population despite the widespread consumption of raw fish. In our correspondence with the New South Wales Food Authority, they reported that no Anisakidae species were identified in the food chain through their surveillance activities.

It is clear the family we described was exposed to human fecal material as evidenced by the mix of enteric parasites identified in the stool specimens. Recently, a cyst stage of *D. fragilis* has been described for the first time [4]. It was demonstrated using an animal model that transmission and symptomatic disease occurs in rats exposed to these cysts, but not trophozoites [4]. In light of this new evidence, the theory that the eggs of *Enterobius vermicularis* are central in the transmission of *D. fragilis* should be critically reassessed.

The role of polymerase chain reaction (PCR) in the diagnosis of *D. fragilis* disease needs to be further assessed. Molecular studies in our community have identified *D. fragilis* in 5.2% of patient specimens, more commonly in symptomatic patients [5]. The strikingly higher prevalence observed by Röser and Stensvold should be interpreted with caution as correlation with light microscopy was not performed [6]. Perhaps some PCR primer sets are less specific or their sensitivity is set to detect *D. fragilis* below clinically significant levels. It is notable that the only case in our cluster without symptoms or peripheral eosinophilia was negative for *D. fragilis* on multiple repeat stool microscopy examinations, but was positive on PCR.

Historically, the absence of an animal model has hindered the ability of *D. fragilis* to fulfil Koch’s postulate as a cause of gastrointestinal disease and eosinophilia. Nevertheless, there are many reports that incriminate *D. fragilis* as a legitimate cause of enteric disease and eosinophilia [7, 8]. We would encourage clinicians to consider the pathogenic role of *D. fragilis* in symptomatic patients with positive microscopy when no alternative diagnosis is identified.

**Note**

**Potential conflicts of interest.** All authors: No potential conflicts of interest.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

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**References**


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