Professor Whitehead replies as follows:

It is not easy to comment on the letter of Drs T Cooke and J Matthews as they do not give a reference to their work. They apparently used a method for a study of adenomatous colonic polyps by harvesting cells by cytological brushing, which is not comparable with our work. They showed that 18% were aneuploid and expressed "surprise" that we, in our study, did not find aneuploidy in 16 adenomatous polyps. In our discussion it is made quite plain that the conclusion that aneuploidy in polyps does not occur had only a 75–90% chance of being correct. Furthermore, Drs Cooke and Matthews do not seem to be aware that even frank carcinomas may be diploid. They also assume that the degree of dysplasia in polyps must parallel the degree of cell division activity. This is not necessarily true. The fact that adenomatous polyps and carcinomas may have a profile indicating minimal cell division activity bears this out. The reference to differences in our findings to those of Cuvelier et al is irrelevant.

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Eosinophilic gastroenteritis

I read with interest the review article by Blackshaw and Levison on eosinophilic gastroenteritis,1 but I am unhappy about the coverage of parasitic infections, which I think is misleading.

Concerning anisakiasis as a cause of eosinophilic gastroenteritis, the designation of the parasite as "Eustoma rotundatum" was abandoned long ago,2 and the invasive larvae belong to the genus Anisakis. Semantics apart, the purported British cases of 19643 were not associated with any helminths, and thus there is no evidence to suggest that either of them were, indeed, anisakiasis. The first autochthonous British cases were reported in 19794 and 1985.5 At the moment, anisakiasis is regarded as the result of actual tissue invasion by larvae rather than the passage through the gastrointestinal tract of the larvae, and therefore a diagnosis of anisakiasis requires the detection of larva or fragments thereof. In the future serodiagnosis may be of help.6

I am surprised that giardiasis is mentioned as a cause of prominent gut eosinophilia. This is not my experience when Giardia lamblia is the sole parasite present, and it is not described in standard texts or in the description of purported invasive giardiasis.7

One protozoon that is, however, associated with eosinophilic enteritis is Sarcocystis spp.8 This comes from eating meat containing Sarcocystis cysts, whereby man can become the definitive host to the sexual phase in the intestinal mucosa. This may produce an acute enteritis with eosinophilia of the lamina propria. How common the condition is remains uncertain, partly because the parasites are so small they can be readily overlooked.

Hookworm infection may reasonably be diagnosed histologically only if the worms are seen rather than from "erosions or circular channels." Notably, recorded cases of invasive ankylostomiasis are rare.9

Other helminths not mentioned in the review are well described as producing eosinophilic gastroenteritis, including oesophagostomiasis and ankylostomiasis.10 11 In the United Kingdom invasive enterobiasis happens occasionally, with eosinophilic abscesses in the large bowel wall and serosa (personal observations). More important is the omission of strongyloidiasis as infection with Strongyloides stercoralis may be life threatening. Some cases are diagnosed from intestinal biopsy, where a dense mucosal eosinophilia associated with invasive larvae is seen.10

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References

Professor Whitehead replies

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