[4] Nelson JD, Kusmiesz H, Jackson LH. Treatment of Salmonella gastroenteritis with ampicillin, amoxicillin or placebo. *Pediatrics* 1980;**65:**1125–30.

Dr Chin and Dr Tarlow comment:

We thank Dr Yagupsky for his interest in our article. We did not imply that the finding of *S typhi* in a stool culture should be ignored. This is a potential health hazard as was pointed out in our paper. Typhoid fever can indeed be a life threatening condition as exemplified by our first case. We agree that the introduction of antibiotic treatment has reduced the mortality of typhoid fever. We do not, however, feel that all infants (including those with minimal gastrointestinal symptoms) require oral antibiotics. Although a septicaemic infection may occur later in some patients with mild symptoms, antibiotic treatment does not completely prevent a major clinical relapse after treatment has been stopped. It is not our policy to treat all healthy contacts, even if their stool cultures are positive.

Although the paper by Nelson *et al*[4] did not refer specifically to the use of antibiotics in *S typhi* infection, the randomised prospective double blind study did show that oral antibiotics were of no benefit to infants and children with acute salmonella gastroenteritis.[1] Furthermore, the treated patients had an increased risk of symptomatic relapse. We are not aware of any recent or similar study relating to *S typhi* gastroenteritis. Lastly, one must not forget that non-typhoid salmonella infections can also cause serious complications in infants.

**Reference**

[1] Nelson JD, Kusmiesz H, Jackson LH. Treatment of Salmonella gastroenteritis with ampicillin, amoxicillin or placebo. *Pediatrics* 1980;**65:**1125–30.

## Incidences of childhood coeliac disease and transient gluten intolerance move discrepantly in UK and Sweden

Sir,

Current criteria for the diagnosis of childhood coeliac disease were formulated by the European Society for Paediatric Gastroenterology and Nutrition. Coeliac disease was defined as a lifelong disorder in which the small intestinal mucosa is abnormal as a result of exposure to gluten in the diet. The mucosal damage improves on treatment with a gluten free diet but recurs within two years of reintroduction of gluten. In some cases, however, this gluten challenge does not bring about a mucosal relapse within the two year period. These children have so called transient gluten intolerance. In an earlier study we reported an incidence of transient gluten intolerance of 19% among children with an initial flat small intestinal mucosa that resembled that seen in coeliac disease.[1]

Since 1975 the incidence of coeliac disease seems to have decreased sharply in the UK[2] while the high incidence in Sweden has remained unchanged. On the other hand, we have observed a steep decline in the number of children with transient gluten intolerance diagnosed during the same period. This is in accordance with the findings of Walker-Smith who reported no new cases of transient gluten intolerance in children born after 1975 at the Queen Elizabeth Hospital for Children.[3]

The fact that the incidences of childhood coeliac disease and transient gluten intolerance are moving discrepantly in the United Kingdom and Sweden is interesting but puzzling, and suggests that factors other than gluten ingestion are involved in the pathogenesis of coeliac disease.

**References**

[1] Stenhammar L, Johansson C-G. The incidence of coeliac disease in children in south-east Sweden. *Acta Paediatr Scand* 1981;**70:**379–81.
[2] Logan RFA, Rifkind EA, Busuttil A, Gilmour HM, Ferguson A. Prevalence and "incidence" of coeliac disease in Edinburgh and the Lothian region of Scotland. *Gastroenterology* 1986;**90:**334–42.
[3] Walker-Smith JA. Food sensitive enteropathies. *Clin Gastroenterol* 1986;**15:**55–69.

L Stenhammar, P Ansved, G Jansson, *and* V Jansson
*Departments of Paediatrics, Central Hospital, Norrköping, Central Hospital, Västervik, University Hospital, Linköping and Central Hospital, Jönköping, Sweden*