Correspondence

Archives of Disease in Childhood, 1972, **47**, 321.

Coeliac Disease and IgA Deficiency

Sir,

In our paper 'IgA deficiency in children. A clinical study with special reference to intestinal findings', which appeared in the October 1971 issue (p. 665), the last sentence of the summary ('Results show that the increased incidence of autoimmune disease reported in IgA deficiency in adults also holds true in children; i.e. that there is a raised incidence of coeliac disease with or without symptoms in IgA deficiency') wrongly implied that we consider coeliac disease to be an autoimmune disease. Though there is some evidence (e.g. Wall *et al.*, 1970) to support this idea, and the increased incidence of both coeliac disease and autoimmune disease in IgA deficiency could be interpreted as such, strong evidence against this also exists. Coeliac disease does not meet the commonly accepted criteria for an autoimmune disease (Witebsky *et al.*, 1947), and the role that gluten plays in the aetiology of the disease cannot be explained in terms of autoimmunity. We feel that immune mechanisms are involved in the pathogenesis of coeliac disease, but that it is not an autoimmune disease. Our original intention was to point out that both autoimmune diseases and coeliac disease occur with raised frequency in IgA deficiency, independently of each other. The connexion between IgA deficiency and coeliac disease is probably based on impaired local immune defence of the gut, which allows more antigenically active material to penetrate the intestinal mucosa in IgA-deficient persons. This phenomenon is reflected in the high incidence of precipitating antibodies to dietary proteins (found by us among others) in this deficiency state.

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RS Virus and Bronchiolitis

Sir,

The article by Ross and her colleagues in the October number of the *Archives* (**46**, 702) prompts us to restate our views on the possible pathogenesis of 'bronchiolitis' caused by RS virus infection in infancy. Our observations are based on the analysis of nearly 300 infants with RS virus bronchiolitis studied in Newcastle over the past 3 years, the evidence and the deductions are as follows:

RS virus, being ubiquitous, infects most children by the time they are 2 years old (Neligan *et al.*, 1970). This infection can occur in the first few days of life when it produces mild respiratory tract symptoms. The majority of children with bronchiolitis are between the ages of 2 and 6 months; it is uncommon below 6 weeks and never occurs under 3 weeks of age. Infants with bronchiolitis often have specific antibody in nasal secretion at the onset of their acute illness (Scott and Gardner, 1970; Kim *et al.*, 1969), and rapidly rising neutralizing antibody titres can be detected in the nasal secretions of even the youngest infant with bronchiolitis (Scott and Gardner, 1970; Kim *et al.*, 1969). There is only scanty virus in the lungs of infants with bronchiolitis compared with the large amount present in pneumonia (Gardner, McQuillin, and Court, 1970). These facts suggest that maternal antibody plays no effective role in prevention, and that in affected children hypersensitivity is a possible mechanism in the production of bronchiolitis.

We would welcome serious evidence which would help to confirm or disprove our hypothesis, because the eventual prevention of RS virus infection depends on detailed knowledge of its pathogenesis. We were therefore disappointed that our friends in Glasgow appear to dismiss an allergic hypothesis on the basis of their findings in 16 children with bronchiolitis, only 6 of whom fell in the critical first 3 months of life. And the essence of their case is based on the serological examination of these 6 children. This is an extremely small number in a 5-year period and we wonder how representative these children were. We know that the primary infection with RS virus can occur in the first week and therefore sensitization could follow in the first few weeks of life. We are not told whether any of these 6 children in the Glasgow study were less than 6 weeks old. We cannot accept their view that the complement-fixation test is as sensitive as the neutralization test, especially if the delicate plaque reduction technique is employed, nor