

cobalamin deficiency.² Untreated cobalamin deficiency progresses to subacute combined system disease with degenerative changes of the dorsal and lateral columns,³ and in severe cases some affected areas of the spinal cord are grossly atrophic.⁴

MRI has proved to be an important step in the evaluation of the central nervous system. A recent MRI study on demyelination of the central nervous system in children with inborn errors of the methyl-transfer pathway revealed a bright signal that resolved on correction of the metabolic deficiencies.⁵ These metabolic deficiencies cause a demyelinating process similar to that associated with cobalamin deficiency. The MRI picture in multiple sclerosis also resembles that seen in our patient. However, these changes are not seen in the spinal cord without concomitant brain abnormalities⁶ and they would not be expected to change with cobalamin administration, as they did in our patient.

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Grass pollen and asthma

SIR,—Cenk Suphioglu and colleagues (March 7, p 569) present data that support suspicions that pollen grains are too large to penetrate lower airways. What they have shown, and have shown well, is that pollen starch granules can precipitate episodes of airway narrowing. However, the conclusions drawn are not warranted since recognised criteria for causation are not satisfied. There is no evidence that particles or pollen allergy cause the disease asthma. Suphioglu et al have not shown any association with the incidence of asthma because this was not measured; this misleading use of words may encourage people to believe that avoidance of pollen starch can prevent the disease. Our data from children in New South Wales show that although many asthmatics are allergic to ryegrass, which is clearly associated with symptoms, it is a much less important risk factor for "current asthma" than allergy to housedust mites, alternaria, or cat dander. Until avoidance of all of these allergens is undertaken, it is extremely unlikely that avoidance of ryegrass allergen will have any impact on the prevalence of asthma. Nor do we yet know if allergen avoidance can prevent asthma from developing.

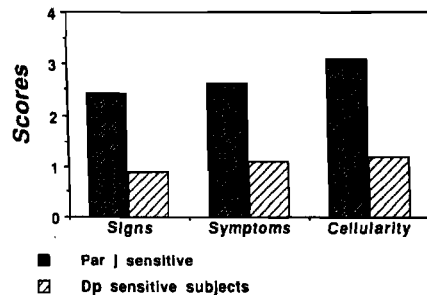
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SIR,—Suphioglu and colleagues suggest a mechanism by which starch granules released from ryegrass pollen grains after rainfall elicit allergic asthma—namely, that the granules are small enough to enter the lower airways. We report our experience in 207 patients with allergic rhinitis due to *Parietaria judaica* pollen, 193 with rhinitis due to *Dermatophagoides pteronyssinus*, and 31 healthy controls. Our aim was to establish the presence and the severity of allergic conjunctivitis. The study was done during the pollen season (spring, 1991).

Clinical evaluations were done on a four-point rating scale (0 = no symptom, 1 = mild, 2 = moderate, 3 = severe) and the cytological findings were scored (0-4, no cells or 1-5, 6-19, 11-30, or > 30 cells per microscope field). 2 weeks before the study (run-in period) allergic individuals recorded symptoms of conjunctivitis every day on a diary card (itching, photophobia, lacrimation, foreign-body sensation).² No anti-allergic drugs were allowed during the run-in.

Conjunctival symptoms were more common in the pollen-sensitive patients than in the house-dust-mite-sensitive patients



Symptom scores on patients' diary cards and sign scores and total cellular (cytology) scores reported by physicians.

Par j = *P judaica*; Dp = *D pteronyssinus*.

(89% [185/207] vs 34% [67/193]). Furthermore, their clinical and cytology scores were greater (figure). The controls showed neither clinical nor cytological reactions. All conjunctival scrapings of pollen-sensitive patients showed not only an inflammatory infiltrate (mainly neutrophils and eosinophils) but also several pollen grains (7-8 per field at 250 × focus); pollen granules frequently seemed to be ruptured along the outer envelope, thus releasing cytoplasmic contents. The greater severity of findings in pollen-sensitive subjects could be related to these granules in the conjunctiva. No pollen granules were found in conjunctival scrapings from the other patient group or from the controls.

P judaica pollen grains are subspheroidal and are rapidly ruptured by osmotic shock (ie, contact with wet surfaces), releasing allergic contents through the germinal pore.² Our findings fit very well with those of Suphioglu and colleagues. All these data suggest a combined mechanism whereby the conjunctiva of pollen-sensitive individuals selectively captures *P judaica* grains that trigger a specific-IgE-mediated response with inflammatory reaction, including the cellular infiltrate and mediator release.³ Moreover, histamine produces capillary leakage with albumin transudation, which could retain pollen grains, so prolonging allergen persistence and setting up a vicious circle with a clinically more severe pattern. A similar sequence could be hypothesised in pollen-associated asthma.

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Chlamydia pneumoniae infection and asthma

SIR,—Dr Crane and colleagues (March 28, p 814) speculate that recent worldwide increases in asthma morbidity, mortality, and prevalence could be attributable to diagnostic transfer, changes in the delivery of health care, or increased diagnosis or self-reporting. I would add another possible explanation that has not received the attention it deserves—increasing worldwide prevalence of *Chlamydia pneumoniae* infection.

Preliminary epidemiological and clinical data support the suggestion that *C pneumoniae* infection may play a part in this increase in asthma. *C pneumoniae* infection has been associated with wheezing, asthmatic bronchitis, and adult-onset asthma in a clinical population.² A patient from this study in whom asthma began in association with serologically confirmed acute *C pneumoniae* infection had a complete remission of symptoms after lengthy anti-chlamydial therapy. After a symptom-free interval of two

years, this patient had renewed chronic wheezing in association with a serologically documented reinfection with *C pneumoniae*.

In Finland, increasing prevalence of *C pneumoniae* infection since the 1970s³ correlates with striking increases in adult asthma.⁴ Since *C pneumoniae* is a recognised cause of bronchitis, we should also note an increase in acute bronchitis in adults that correlates with a rise in adult asthma between 1970 and 1981 in England and Wales.⁵ There is also a temporal relation between the appearance of *C pneumoniae* infection⁶ and adult asthma⁷ in Denmark.

I agree with Crane et al that appropriate use of bronchodilator medication is important to keep to a minimum the adverse effects on asthma. I would urge asthma researchers also to consider the possibility that *C pneumoniae* infection could be one of the underlying (and potentially treatable) causes of a disease whose management has so far been only palliative.

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Chlamydia trachomatis infection in children with wheezing simulating asthma

SIR,—*Chlamydia trachomatis*, according to WHO estimates is, after *Trichomonas vaginalis*, the most frequent sexually acquired pathogen in adults, carrying the possibility of contamination at birth of babies born to infected mothers. It is difficult to establish how older children become infected with this organism, sexual abuse apart, but serological evidence of *C trachomatis* is found in an increasing proportion of the population with time. The rate varies not just with age but also with socioeconomic and environmental factors; in prepubertal age groups, rates of 26.7% and 47.2% have been reported.^{1,2}

We have looked for *C trachomatis* infection in 20 wheezing children (13 boys, 7 girls; aged 6 months to 10 years, mean 32 months). They were negative on allergic evaluation and did not respond to bronchodilator agents. Pharyngeal and conjunctival swabs, for culture on McCoy cells and for a direct immunofluorescent test with monoclonal antibodies, were collected. In an attempt to trace the route of transmission we also examined the families of positive children.

C trachomatis was identified by culture in 7 (35%) children in the pharynx, with concordance of results by both methods in only 1 case. In these 7 children, the organism was identified in the conjunctiva in 6; 4 of these had eye symptoms, present at the time of examination in 2.

Treatment with erythromycin ethylsuccinate (50 mg/kg daily) for 2 weeks was successful in 6 of the 7 cases. In the other child, a further course of the same antibiotic eradicated the infection.

C trachomatis was demonstrated by culture of at least one site in several parents of positive children (table). In one family, where the parents were both negative, we found on further inquiry that other relatives living in the same apartment were positive. Doxycycline 200 mg twice a day for 21 days was given to the adult relatives and the cousin was treated with erythromycin for 2 weeks. Control swabs after completion of this therapy were all negative.

The asthmatic symptoms remitted in all 7 *C trachomatis* infected children, and the ocular symptoms also stopped. At 1-year follow-up these 7 children remained negative for *C trachomatis* on culture and had had no further asthma attacks.

C pneumoniae strain TWAR can be excluded as the explanation in these 7 wheezing children because this agent grows only on HeLa

C TRACHOMATIS IN FAMILIES OF SEVEN POSITIVE "ASTHMATIC" CHILDREN (A-G)

Member	Conjunctiva	Pharynx	Urethra	Cervix
A, child (M, 12 mo)	+, -*	+, -†
A, father	- , ..	- , ..	- , -	..
A, mother	- , ..	+, ..	+, -	+, +
B, child (M, 56 mo)	+, -	+, -
B, father	+, ..	- , ..	- , -	..
B, mother	+, ..	- , ..	- , -	- , -
C, child (F, 41 mo)	+, -	+, -
C, father	+, ..	- , ..	+, +	..
C, mother	- , ..	- , ..	+, -	- , -
D, child (M, 57 mo)	+, -	+, -
D, father	- , ..	+, ..	- , +	..
D, mother	- , ..	- , ..	+, -	+, -
E, child (M, 8 mo)	+, +	+, +
E, father	- , ..	- , ..	- , -	..
E, mother	- , ..	- , ..	- , -	- , -
E, cousin (26 mo)	+, +	+, +
E, uncle	- , -	- , -	+, +	..
E, aunt	- , +	- , -	+, +	- , -
F, child (F, 7 yr)	- , -	+, -
G, child (F, 8 mo)	+, +	+, -

*Results for culture, immunofluorescence (direct).

†.. = irrelevant or not done.

229 cells and not on the McCoy cells we used for our isolation procedures.

In these children, *C trachomatis* infection could have arisen through family contacts, the trigger being a primary infection in one adult.³ The findings in the family living in an overcrowded apartment suggest the possibility of *C trachomatis* spreading to children from adults directly by close daily contact with secretions (eg, saliva, tears) and/or hands or indirectly through fomites, toys, towels, and bedlinen, for example.

Our data indicate that wheezing may be another clinical expression of *C trachomatis* infection and that this organism should be sought as a routine in children who wheeze but have no demonstrable allergy and do not respond to the usual anti-asthmatic medications.

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Reversal by ceftriaxone of dilated cardiomyopathy *Borrelia burgdorferi* infection

SIR,—Dilated cardiomyopathy is a life-threatening disorder, which, when progressive, leads to chronic heart failure. It has been suggested that, in several cases, *Borrelia burgdorferi* could be associated with chronic myocarditis and dilated cardiomyopathy. Thus *B burgdorferi* could be isolated and cultured from a myocardial biopsy specimen in a patient with dilated cardiomyopathy.¹ However, data about *B burgdorferi* in dilated cardiomyopathy and its treatment are scant, especially with respect to reversibility of changes in myocardial function.²⁻⁵

We report a prospective study in which we examined specifically for *B burgdorferi* infection, serum for IgG and IgM (ELISA), and the history in 42 patients with dilated cardiomyopathy (mean left-ventricular ejection fraction [LVEF] 30% [SEM 1.2%] assessed by cardiac catheterisation). 9 (21%) patients with a mean LVEF of 34% (2.2%) were seropositive for *B burgdorferi*, and 7 of those had a typical history of tick bite and erythema chronicum