

Airborne house dust mite antigen after vacuum cleaning

SIR,—The major role of the house dust mite (*Dermatophagoides pteromyssinus*), and its main allergen Der p1, in the production of symptoms in patients with asthma is well recognised.¹ To reduce domestic dust levels, doctors recommend frequent vacuum cleaning,² which often results in an unexpected increase in patients' symptoms. This deterioration can probably be attributed to the small Der p1 particles (10–40 µm in diameter) passing through the vacuum cleaner and becoming suspended in the air, from where they are easily inhaled. This suspension of particles would not arise if vacuum cleaners could trap small particles.

We have compared the airborne Der p1 levels after vacuum cleaning of carpets with a conventional cleaner ("Hoover Junior") and a high filtration machine ("Medivac"; filtration of 99.99% of particles at 0.3 µm). Air was sampled in sixteen domestic rooms by the Rothero-Mitchell sampler with Whatman GFF filter (60 l/min for 5 h) before and after cleaning in a standardised manner with each of the two vacuum cleaners. Dust samples were weighed and analysed blind for Der p1 with an ELISA technique ("Biorad 3550 Absorptiometer", ALK Laboratories). Although total airborne dust levels did not differ before and after cleaning by either method (table), airborne Der p1 increased significantly after conventional vacuuming but not after medivac use.

DER P1 AND DUST LEVELS* IN AIR BEFORE AND AFTER VACUUM CLEANING

	Airborne Der p1 (ng)	Airborne dust (mg)
<i>Hoover</i>		
Before	1.76 (1.14)	58.51 (1.35)
After	5.35 (2.21)†	46.66 (1.26)
<i>Medivac</i>		
Before	1.74 (1.04)	65.0 (1.29)
After	1.78 (1.03)	39.35 (1.35)

*Geometric means (SEM).

†Before vs after, $p = 0.016$.

These results suggest that conventional vacuum cleaning is not only unlikely to eliminate Der p1 but also actually increases the airborne levels. Therefore such cleaning cannot be recommended for house dust mite control in the homes of asthmatics.

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Calcitonin nasal spray for painful diabetic neuropathy

SIR,—Calcitonin exerts an analgesic effect in painful diabetic neuropathy, via enhanced β -endorphin concentrations in cerebrospinal fluid.¹

A 21-year-old woman who had been diabetic for six years presented with hyperaesthesia and pain in the legs and difficulty in walking, despite good glucose control with monocomponent insulins twice daily. Her symptoms did not improve during six weeks' treatment with vitamin B₁₂ and phenytoin. She was therefore given salmon calcitonin nasal spray ('Prontocalcin', Dompe Farmaceutici, Italy) 100 IU daily. She tolerated this treatment well. After two weeks her symptoms diminished, and the treatment was continued for another two weeks. During calcitonin treatment fasting glucose concentration ranged from 75 to 105 mg/dl, whereas blood calcium and phosphorus concentrations remained unaltered at 9.4–9.1 and 3.7–3.5 mg per dl, respectively. I think that calcitonin nasal spray may become a useful drug for the treatment of painful

neuropathy, especially since it is well tolerated and easy to use.

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Dehydroepiandrosterone sulphate in Alzheimer's disease

SIR,—Sunderland et al¹ have described a strong correlation between age and the serum concentration of dehydroepiandrosterone sulphate (DHEA-S) in 19 healthy controls ($r = 0.91$). They reported 48% lower DHEA-S concentrations in 10 patients with Alzheimer's disease (AD) compared with that in 10 age and sex matched controls. Since there are also sex differences in serum DHEA-S,² Sunderland et al were uncertain about the clinical relevance of their findings.

We have studied 34 healthy women aged 18–81 years and found a correlation between age and serum DHEA-S of 0.79. Blood samples were taken after an overnight fast and all samples were stored at -20°C and tested in the same assay (solid phase ¹²⁵I, Diagnostic Products Corp).

Unlike Sunderland et al we found no difference in serum DHEA-S between 10 drug-free women with AD^{3,4} (mini mental state score 14.9 [SD 7.4]) and age-matched healthy women:

Group	Mean (SD) of Age (yr)	DHEA-S (ng/ml)
AD (n = 10)	75.4 (4.6)	475.4 (496.0)
Controls (n = 10)	72.0 (4.4)	566.3 (366.7)

Our series, unlike Sunderland's, was all-female and our patients were on average 14 years older.

Dr Späth-Schwalbe and co-workers (June 9, p 1412) report DHEA-S studies in AD patients of very similar age to ours; their results and those of Sunderland et al also differed.

DHEA-S is an excellent indicator of adrenal androgen output but its specific role in AD has yet to be established.

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SIR,—Dr Späth-Schwalbe and colleagues report that plasma DHEA-S concentrations in 7 patients with Alzheimer's disease (AD) were similar to those in 7 age and sex matched controls. Sunderland and colleagues had reported that DHEA-S levels were, on average, 48% lower in 10 AD patients than in 10 matched controls.¹ We have also investigated the matter using plasma samples that had been stored at -30°C for about one year. The samples had been taken from patients attending the memory clinic at the Institute of Psychiatry, London, and meeting diagnostic criteria for AD² and from their spouses. Sufficient plasma was available from 17 patients with AD (median age 63; 8 men, 9 women) and 15 controls (median age 67; 5 men, 10 women). None had been taking psychotropic drugs. DHEA-S was measured, "blind", by radioimmunoassay.³

Two methods were used to allow for differences in age and sex between patients and controls—namely, multiple log-linear