

EDITORIAL

The relationship between mite allergen exposure and asthma severity

The interesting report [1] contained in the current edition of this journal agrees with the previous findings of this group [2]. Marks *et al.* [1] prospectively followed changes in house dust mite allergen concentration in household dust and simultaneously measured indices of asthma. Fourteen per cent of the variation in non-specific bronchial reactivity was statistically explainable by the changes in house dust mite concentration. As they did not experimentally remove the mite allergen, the changes in allergen exposure may reflect a concomitant rather than a directly causal relationship. However, the authors illustrate how large an effect would be produced if the changes in mite allergen did cause that change. Fivefold simultaneous reductions in the mite allergen concentration in the bed, on the bedroom floor and on the living room floor would produce a 0.7% increase in the percentage predicted flow expiratory volume per second (FEV₁), and a modest improvement in non-specific bronchial reactivity. The order of this effect is consistent with the hypothesis of the first international workshop [3,4] which suggested that a 10-fold reduction in mite allergen exposure was required to improve symptoms, but contrasts somewhat with some of the hopes and extrapolations contained in the literature. It is consistent with our failure to demonstrate a relationship between mite allergen exposure and eczema in sensitive children [5] despite ninefold natural variation in allergen exposure and the difficulty of convincingly demonstrating that natural mite allergen exposure induces exacerbation of asthma [6,7].

In 1983 Korsgaard demonstrated, in a well controlled trial, that the risk of asthma in adults was greater if there was increased exposure to house dust mite [8]. About the same time Platts-Mills [9] demonstrated in adults that removal from mite contact of mite-sensitive adults was associated with gradual improvement in non-specific bronchial reactivity. His group later showed [10] that mite exposure was a risk factor for emergency room attendance with asthma. Similarly it was subsequently demonstrated that mite exposure increased the risk not only for asthma but also for atopic dermatitis [11]. We have also demonstrated that the severity of skin-test reactivity to house dust mite is an independent predictor of subsequent asthma severity [12]. The quantity of mite allergen in the household can apparently influence the likelihood of emergency admission of mite sensitive asthmatic children [6] and also increases the risk of readmission [7]. This is consistent with the present

theory but contrasts with the disappointing results achieved both in clinical practice and in experimental trials monitoring the clinical effects of mite removal. Although Murray's clinically effective controlled trial of mite removal procedures [13] did not measure the change in mite allergen exposure, it seems likely that the extensive measures carried out were effective by removing the mite allergen. If one dismisses all trials which do not measure the change in mites and/or their products, the problem of ineffective removal of the mites is more visible. Many commercially available techniques which work *in vitro* are not effective in practice [14].

Techniques such as vacuuming, have only at best modest effectiveness [15] but Hepa filters could be more useful in reducing aero-allergen induced symptoms [16]. Burr performed three well controlled trials [17–19] before demonstrating a modest effect of mite removal on childhood asthma [19]. Sarsfield's much quoted 1974 trial [20] monitored mite allergen removal but was uncontrolled. When a controlled trial was carried out in the same district with the same techniques [21] no significant benefit was demonstrated. The suggestion that more general changes in households such as reducing the humidity [22] by increasing the air exchange rate [23,24] is gaining favour but previous failures with attempts at this manoeuvre must be remembered [25]. Perhaps we should be moving towards the acceptance of what is achievable. Even with multiple 'effective' techniques of mite removal applied simultaneously, the effects on established asthma may be modest. This rather negative conclusion contradicts all our wishes even though it may be 'realistic'.

One of the difficulties with all such work is that of actually quantifying exposure. The present belief is that exposure to mite allergen is by inhalation in asthmatic subjects, though evidence exists [26,27] that some mite allergen exposure probably occurs via the skin in eczema. If one accepts the assumption that the mite allergen stimulus to asthma is inhaled, the relationship between the surface allergen measurements and aero-allergen becomes important. This relationship is not close [28,29]. It is likely to be majorly affected by the position and activity of the 'challenged' patient and the position of the major mite allergen pool whose disturbance briefly [30] produces detectable aero-allergen. Even standardization of the method and expression of the surface allergen results [31,32] will not allow for the different

habits or localities occupied by different patients. Our studies [33] in 1-month-old babies illustrated not only the widely varied habitats occupied by different babies in one day but also the major effect of minor changes in posture. Thus nostrils up vs nostrils down led to a threefold increase in allergen intake by our artificial 'baby'. This relationship between the surface pool and the actual allergen exposure which is relevant both to attack provocation and initial sensitization needs to be understood so that we can have a clearer idea of the dose response relationship and plan sensible interventions. The results have relevance to the much discussed idea [34] that mite exposure in infancy may generate asthma. This concept is supported by the vulnerable period for sensitization found in young animals after birth [35] and by the prospective observation [36] of a relationship between the concentration of mite allergen in the households of 1-year-old children with both the occurrence of asthma and the rapidity with which it develops. However, the causal nature of this relationship between mite allergen exposure and the risk of developing asthma has not yet been convincingly demonstrated by proper scientific trials. The papers by Marks *et al.* is a welcome contribution to this large debate [1].

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