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# Reply

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To the Editor:

In his response[1] to our recent review[2] on dust mite allergen avoidance, Tovey raises some important issues. These concern both the need to consider exposure over the full 24 hours and the relative importance of different sizes of particles carrying allergens. We do not disagree with his overall position that naturally occurring exposure is more complex than we presented. However, we suspect that the relevance of the answers to these questions may be limited by the lack of practical approaches to control exposures, especially those that occur outside the home. An important element of the primary observations about airborne allergens was the dramatic differences between natural exposure to cat and dust mite.[3] Central to those studies was the use of measurements in absolute units, that is, nanograms or micrograms of allergen proteins. In those studies, including those of Tovey et al, particles less than 5 mm accounted for only a minor fraction of the total IgE-binding fraction to dust mite.[3–5] In the hypothetical model proposed by Tovey, values for exposure are given as "% of maximal for each size." Given that small particles represent only a minor fraction of total (dust mite) allergen-containing particles, this presentation has the effect of inflating the relative contribution of small particles. Although it is possible that small particles play a greater role in promoting inflammatory responses to dust mite allergen in the lungs relevant to asthma, existing data do not support this position. For example, in the studies reported by de Blay et al in France, they found that larger particles carrying mite allergen would produce an early effect on lung function with a smaller quantity of mite allergen.[6] Although subjects who were challenged with small particles required more frequent and higher doses of oral corticosteroids, those subjects also received a much higher total dose of dust mite allergen given that they did not mount early-phase reactions. Thus, the study was not designed to study the effect of particle size on late-phase responses. The analogy with pollen allergy and thunderstorm asthma regarding particle size is interesting, but there is currently insufficient data to support the view that small particles are more clinically potent sources of dust mite allergen. We would like to highlight one aspect of Tovey et al's[7,8] recent work that strongly supports one of the messages of our article. Primary prevention to dust mite sensitization is unlikely to be fruitful in areas where dust mite prevalence is high because of the extent of exposure that occurs outside the home. In contrast, home interventions can be beneficial for those with established sensitization and allergic disease (ie, tertiary prevention) as shown recently by Murray et al.[9]

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Conflicts of interest: T. A. E. Platts-Mills has a patent on an IgE assay to a-Gal, has received assay support from Phadia/Thermo-Fisher, and has received grant support from the National Institutes of Health. J. M. Wilson declares no relevant conflicts of interest.

Overall we would maintain our conclusion that the techniques for reducing exposure in the home are well established and can be achieved without major disruption or expense. Thus, the practical approach is to recommend that mite-allergic subjects take measures to decrease exposure in the home, starting with the bedroom. In conjunction with modern forms of treatment (ie, immunotherapy, inhaled corticosteroids, leukotriene antagonists, etc), this approach can achieve asthma control in most of our patients who adopt the recommendations.

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