To the Editor:

The recent article by Wilson and Platts-Mills[1] provides an insightful review of the current rationale and practice of mite interventions. However, the clinical benefits remain debated and are often elusive.

There are a couple of ideas worth considering that may improve the practice.

The first is to better understand the sites of exposure. As the authors note[1], the bed is often chosen as the primary focus of interventions because of the time spent there and the ease of applying interventions at this site. However, the only data on the time-geography of personal mite aeroallergen exposure over 24 hour periods, from our group[2,3,4], suggests, on average, that about half the total exposure occurs within the house but not at night in bed. A further quarter occurs outside the house during transport, occupational or social situations, probably from clothing. Thus measurement challenges the established dogma. While there are limitations to our observations, if they are substantially correct then they indicate new opportunities to develop more effective interventions.

The second is to better understand the role of aeroallergen particle size in clinical outcomes. The authors[1] hypothesise about the effects of 'large particle' exposure. We additionally hypothesise that small, 'respirable' (< 5μm) mite particles may also be important.

The original description of mite aeroallergens[5] showed the ubiquitous distribution of reservoirs in houses, that aeroallergen was both difficult to detect unless reservoirs were disturbed and most was associated with large particles that settled rapidly, leaving small particles aloft. This is consistent with our recent observations[4]. As illustrated schematically in Fig 1, large-particle exposure occurs as multiple transitory spikes during the day and evening. During sleep, people seldom move and most exposure is generated on getting in and out of bed and during brief arousals between sleep cycles.

Small-particle exposure is different in several ways. Once generated, such particles can remain airborne for long periods. However, exposure may be low and transitory in well-ventilated spaces or initially on moving into an unoccupied space. In an unventilated space which is also rich in disturbed reservoirs, such as an occupied bedroom, small-particle exposure could be relatively high and prolonged. Whether small particles are also aerosolised by micro-events such as body thermals is not known.

While the total quantity of inhaled allergen associated small-particle exposure is probably a small fraction of the 24-hr total, (this has never been determined), we suggest it may be atypically clinically potent. This is indicated by relatively benign exposure to 'large-particle' pollen, which when fragmented during thunderstorms, can provoke epidemic, devastating asthma[6]. Additionally, low-level cat exposure, involving small particles, increases airway hyperresponsiveness in the absence of symptoms[7]. Perhaps the importance of size trumps quantity. Again this model, if valid, indicates new directions for improving domestic avoidance.

Ultra-low mite allergen environments such as in mountain sanatoria and allergen-free chambers provide impressive clinical benefits and lessons in the role of environmental exposures. A better understanding of the timing and nature of domestic mite exposure may help us replicate these outcomes in the home.

Euan Tovey, PhD

Woolcock Institute, University of Sydney, Australia

Legend: Figure 1

Schematic, hypothetical, representation of exposure to mite aeroallergens carried on large (>10µm) and small (<5 µm) particles over a 24-hour period. Exposure is shown as a percentage of the maxima for each size. Large-particle exposure occurs as transitory spikes associated with disturbance of reservoirs, see[4]. Small-particle exposure is transitory and low in well ventilated spaces and high and prolonged in unventilated locations. Both may contribute differently to clinical outcomes.

References

1. Wilson JM, Platts-Mills TAE. Home Environmental Interventions for House Dust Mite. Journal of Allergy and Clinical Immunology-in Practice. 2018 Jan-Feb;6(1):1-7.

2. **De Lucca SD, Tovey ER. Quantitative modelling of domestic allergen (Der p 1) using amplified ELISA assays and nasal sampling. Journal of Allergy and Clinical Immunology. 2001 Feb;107(2):S219-S.**

3. Tovey ER, Willenborg CM, Crisafulli DA, Rimmer J, Marks GB. Most Personal Exposure to House Dust Mite Aeroallergen Occurs during the Day. Plos One. 2013 Jul;8(7).

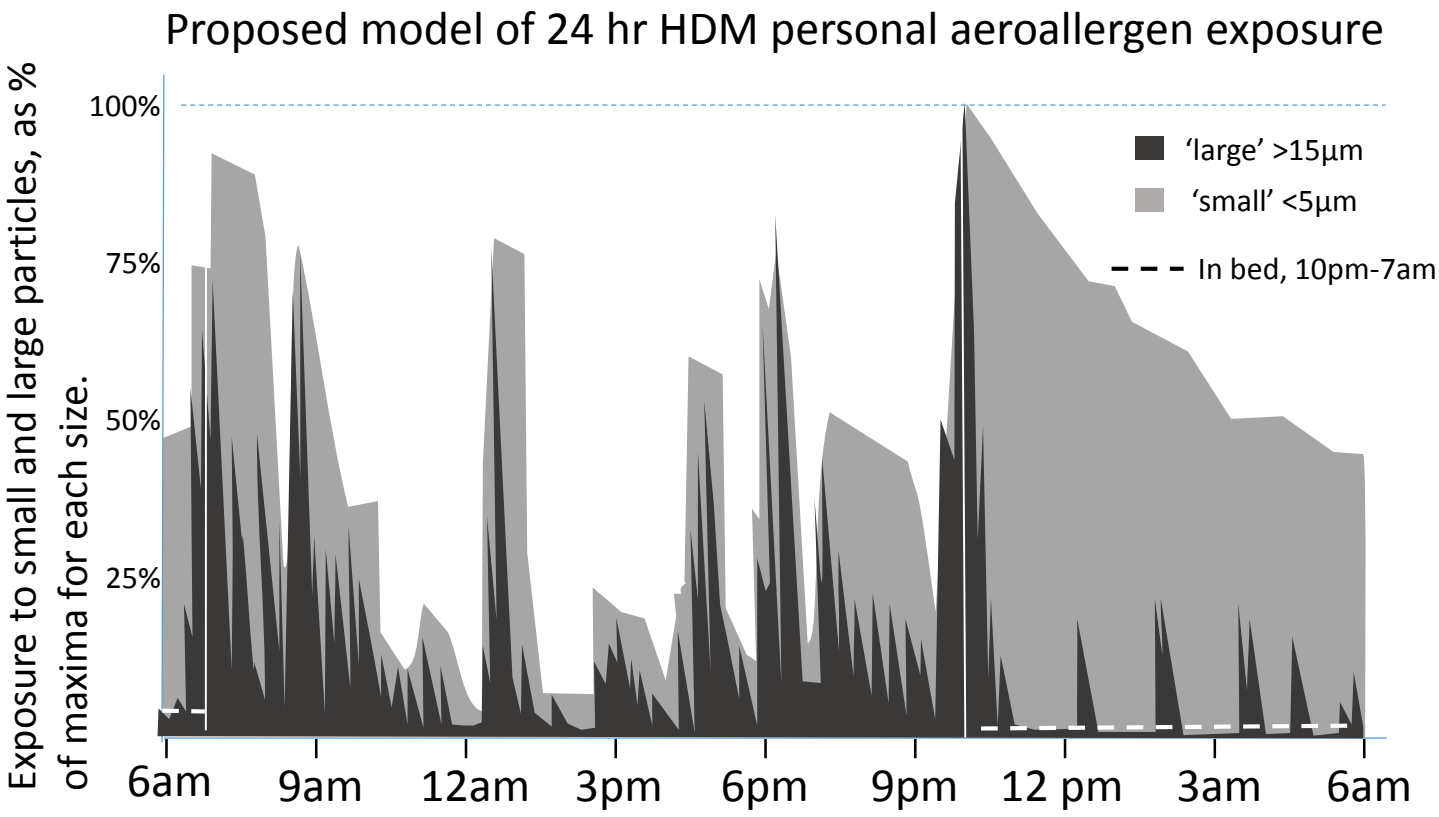
4. Tovey ER, Liu-Brennan D, Garden FL, Oliver BG, Perzanowski MS, Marks GB. Time-Based Measurement of Personal Mite Allergen Bioaerosol Exposure over 24 Hour Periods. Plos One. 2016 May;11(5).

5. Tovey ER, Chapman MD, Wells CW, Platts-Mills TA. The distribution of dust mite allergen in the houses of patients with asthma. American Review of Respiratory Disease. 1981;124(5):630-5.

6. **Woodhead M. Hospitals overwhelmed with patients after "thunderstorm asthma" hits Melbourne. BMJ-British Medical Journal. 2016 Nov;355**.

7. Zeidler MR, Goldin JG, Kleerup EC, Kim HJ, Truong DA, Gjertson DW, et al. Small airways response to naturalistic cat allergen exposure in subjects with asthma. Journal of Allergy and Clinical Immunology. 2006 Nov;118(5):1075-81.

Figure No.


Proposed model of 24 hr HDM personal aeroallergen exposure
Exposure to small and large particles, as % of maxima for each size.
100%
75%
50%
25%
'large' >15µm
'small' <5µm
In bed, 10pm-7am
6am
9am
12am
3pm
6pm
9pm
12 pm
3am
6am