

Correspondence

Reply

To the Editor:

We thank Gusareva et al¹ for sharing data relating specific IgE antibody measurements and total IgE among their patients with asthma in Siberia. It is interesting to compare their data with our own. The Russian cities where the children live, Tomsk, Thumen, and Irkutsk, are all south of the cities in northern Sweden (ie, below 60°N latitude). Although the climate of western Siberia would be similar to northern Sweden and not New Zealand, the prevalence of dust mite sensitivity was strikingly lower in northern Sweden. Gusareva et al¹ previously reported that among children with asthma living in the areas they studied, the prevalence of sensitization to cat allergen was highest (57%), followed by dog and dust mite (30% to 40%).² In Sweden, among wheezing children (n = 69), the prevalence of sensitization to cat, dog, and dust mite was 46.4%, 47.8%, and 11.5%, respectively.³

Further analysis of specific IgE titers and their relationship to total IgE in the 2 areas with similar climates, Siberia and northern Sweden, reveals parallels. In Sweden, high titer of specific IgE antibody (>50 IU/mL) to any allergen was uncommon. In Siberia, high titers of specific IgE to cat and dog were also relatively uncommon. High titers of specific IgE to birch in Siberia appear to be as frequent as lower titers. It is possible that this observation is explained by the time of year that some serum samples were collected (March, April, and May, although not in the same year).⁴ In both regions, only a modest correlation was observed between specific IgE antibody titers to cat, dog, and relevant pollens (birch in Siberia and birch and grass in northern Sweden). The relationship between dust mite and total IgE was not significant in northern Sweden (Table I).

The current analyses of Gusareva et al¹ reinforce our previous suggestion that the relationship between specific and total IgE is directly dependent on the relevant environmental allergen. Allergens that induce high levels of specific IgE antibody can be related to increased total IgE. This concept may seem simple, but it implies that a ranking of antibody responses to specific allergens is possible that predicts total IgE and prevalence of wheezing. We have previously suggested that titers of IgE antibody produced in response to dust mite are higher than titers of IgE to cat in New Zealand.⁵ In turn, higher titers of IgE to dust mite are related to higher total IgE and an increased prevalence of wheezing in New Zealand.³ Combining the results of Gusareva et al¹ with our own, northern Sweden, Siberia, and New Zealand

TABLE I. Associations between specific IgE antibody titers and total IgE among children living in Sweden

Allergen	Pearson correlation (r)
Cat	0.31 ($P < .001$)
Dust mite	0.22 ($P = .2$)
Grass	0.42 ($P < .001$)
Dog	0.34 ($P < .001$)
Birch	0.21 ($P = .03$)

can be ranked according to prevalence of wheezing and increased total IgE on the basis of dust mite exposure.

We agree with Gusareva et al¹ that it would be interesting to look at the relationship between specific IgE antibody and total IgE in other areas. Previously, the overlap in total IgE between allergic and nonallergic persons has complicated large epidemiologic studies. We would argue that examining a single area has been an underlying problem because allergen exposure would be the same throughout the region. By simultaneously studying areas with different allergen exposures, the allergen-specific effects can be observed.

Elizabeth A. Erwin, MD
Thomas A. E. Platts-Mills, MD, PhD

From the Asthma and Allergic Diseases Center, University of Virginia, Charlottesville.
E-mail: tap22@virginia.edu.

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