

Fifty-three of these 538 illnesses were identified at scheduled visits. Therefore, 9.85% of all such illnesses were identified at scheduled visits. One hundred sixty-three mild (score 1-4) illnesses were also identified at the scheduled visits. On the basis of the proportion of moderate to severe illnesses identified at scheduled visits, we would estimate a total of 1654.6 (163/.0985) mild illnesses in the first year of life. Overall, based on the COAST data, we would estimate 7.69 illnesses (score 1+) per year, 5.81 mild (score 1-4) illnesses per year, and 1.89 moderate to severe (score 5+) illnesses per year. These numbers are in close agreement with other estimates of respiratory infection frequency in this age group.³

We agree with the authors that the manner in which respiratory tract illnesses are defined, reported, documented etiologically, and tracked longitudinally can all influence data regarding overall incidence rates. We agree that having study participants collect specimens is a promising technique for studies of illness prevalence. It should also be noted, however, that with dedicated staff and frequent communications with study participants, accurate estimates of illness prevalence can be achieved. The latter approach, which we used in our study, also allows for clinical evaluation of signs and symptoms of illness, as well as for the collection of standardized specimens suitable for both viral¹ and immunologic⁴ analyses.

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Environment-induced reactivity against autoallergens: Possible role of latex

To the Editor:

We read with interest the recent article by Schmid-Grendelmeier et al,¹ which highlighted the role of human

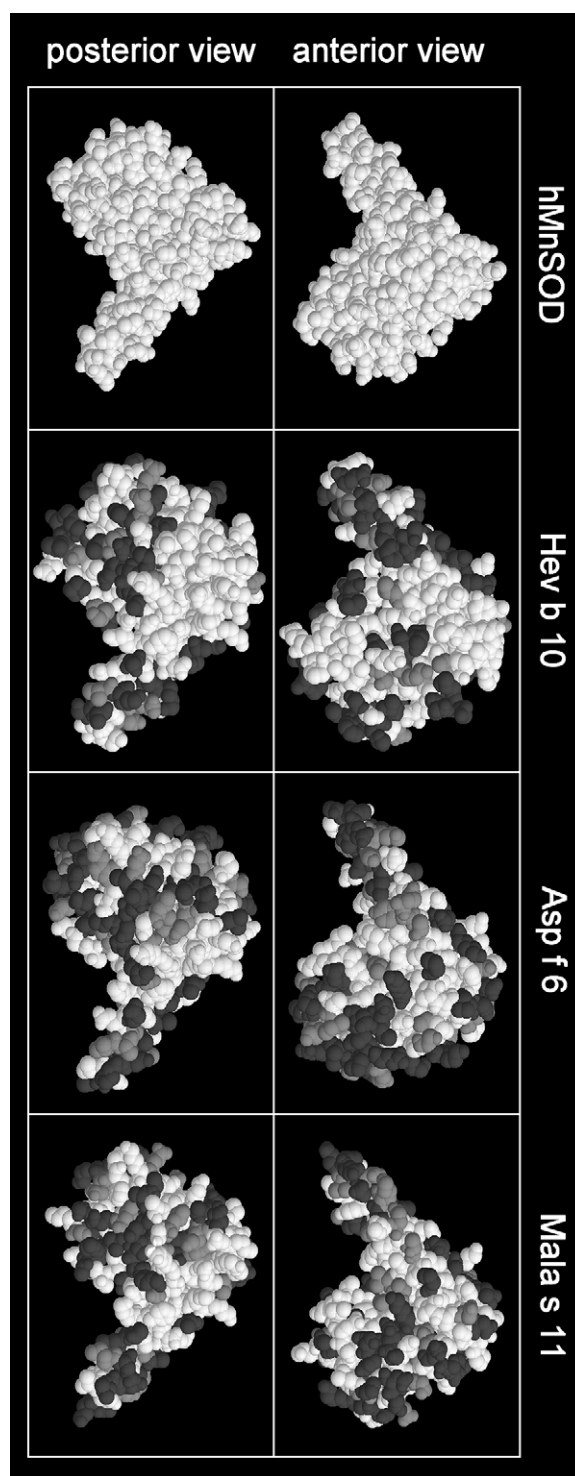


FIG 1. Molecular surface comparison among hMnSOD, *Hevea brasiliensis* (allergen Hev b 10), *Aspergillus fumigatus* (allergen Asp f 6), and *Malassezia sympodialis* (allergen Mala s 11). Identical, chemically similar, and chemically different amino acids are shown in white, light gray, and dark gray, respectively.

manganese superoxide dismutase (hMnSOD) as an auto-allergen in a relevant number of subjects affected by atopic dermatitis. The authors also demonstrated allergic cross-reactivity between hMnSOD and manganese superoxide dismutase of *Malassezia sympodialis* (allergen Mala s 11) and *Aspergillus fumigatus* (allergen Asp f 6). Based on experimental data, they suggested that Mala s 11 could trigger, through molecular mimicry, allergic reactivity against hMnSOD (and, ultimately, atopic dermatitis) in atopic subjects.

Aiming to verify whether other allergens could play a similar role, we used the National Center for Biotechnology Information BLAST (Basic Local Alignment Search Tool) to compare the amino acid sequence of hMnSOD with all the allergen sequences deposited in the Entrez Protein database ($n = 5504$ as of June 5, 2005). Only 3 allergens were significantly ($E < 0.01$) homologous: manganese superoxide dismutase of the rubber tree, *Hevea brasiliensis* (allergen Hev b 10); Asp f 6; and Mala s 11. Homologous segments spanned almost the entire length of hMnSOD in all cases. Amino acid identity was 119 of 199 (59%), 103 of 203 (50%), and 99 of 196 (50%); amino acid similarity (that is, identical plus chemically similar amino acids) was 140 of 199 (70%), 134 of 203 (66%), and 130 of 196 (66%); and E values were 4×10^{-65} , 3×10^{-57} , and 2×10^{-55} , respectively.

We also used the software SWISS-MODEL (available through Web interface at <http://swissmodel.expasy.org/SWISS-MODEL.html>) to obtain a 3-dimensional model of all these proteins and the RasMol program (version 2.6, which is freely available at <http://www.umass.edu/microbio/rasmol/getras.htm>) to visualize and compare their molecular surfaces. We found a highly significant similarity of the 3 environmental allergens with hMnSOD (Fig 1). Our *in silico* results, which suggest cross-reactivity of Hev b 10 with hMnSOD, are confirmed by experimental data by Wagner et al.²

Allergic sensitization to latex is a well known concern for public health. It can occur through skin or mucosal contact and is estimated to affect about 1% of the general

population. In atopic children, prevalence of latex sensitization is 3.9%.³ Because of the widespread diffusion of rubber manufacts, latex exposure can occur early in life (eg, through gloves used by obstetrics, latex nipples, toys) and persist at high levels even in a domestic setting. Thus, latex can be considered a frequent environmental allergen, particularly in industrialized countries.

Taken together with data from the literature,¹⁻³ our results suggest that Hev b 10, similar to Mala s 11, could act in atopic subjects as a trigger of allergic reactivity against hMnSOD, through molecular mimicry. The relevance of this mechanism in the pathogenesis of atopic dermatitis needs to be evaluated experimentally. Our data also point out the potential usefulness of *in silico* molecular analysis techniques as a valid support to the traditional, irreplaceable *in vivo* and *in vitro* research.

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