Correspondence

Reply

To the Editor:

We would like to thank Dr Daschner[1] for his interesting and relevant comments related to the findings of our epidemiologic study, which demonstrated that atopy was significantly associated with *Anisakis*-related allergic symptoms in fish-processing workers. The proposal that Dr Daschner[1] makes is that workers with clinical sensitization to house dust mites (HDMs) could be more prone to demonstrate sensitization to *Anisakis* because of cross-reactivity.

Because our epidemiologic study was a cross-sectional study of all seafood processors in 2 factories (n = 579) and not a cohort study, it is not possible to determine with absolute certainty which characteristic manifested first—atopy (prevalence: 37%), sensitization to HDM only (25%), or sensitization to *Anisakis* (8%). However, a more detailed analysis of our data supports our hypothesis that exposure to *Anisakis* proteins appears to be an independent risk factor for allergic disease in atopic individuals. Our data demonstrate a poor correlation between sensitization (skin prick test > 3 mm compared with negative control) to *Anisakis* and HDM (Spearman $r = 0.13$; $P < .05$), and for that matter between sensitization to *Anisakis* and any of the other 6 individual common inhalants tested: Bermuda grass, rye grass, cockroach, cat, dog, and mold (Spearman r values < 0.20). This was confirmed by the slight agreement between sensitization to *Anisakis* and HDM ($\kappa = 0.11$; $P < .05$), between sensitization to *Anisakis* and the other individual common inhalants tested ($\kappa < 0.17$; $P < .05$), as well as between *Anisakis* sensitization and overall atopy ($\kappa < .12$; $P < .05$). The relatively low correlation and agreement between sensitization to *Anisakis* and any other tested inhalant allergens suggest that cross-reactivity between these allergens may not be critical in our study population. However, confirming this would require inhibition studies using the sera of workers sensitized to these different allergens.[2]

Studies in patients and animals have indicated that parasites can alternately decrease or increase atopy and/or allergies, depending on variables such as parasite load, species, and the time kinetics of the infection.[3,4] In the study by Daschner et al,[5] a negative association was found between acute parasitism by *Anisakis simplex* and allergic sensitization to aeroallergens. This is in line with the hygiene hypothesis, which suggests that infections by pathogens such as helminths may reduce the incidence of allergic disease. In contrast, we did not find a negative association between *Anisakis* sensitization and sensitization to aeroallergens. As Daschner[1] suggests, this may illustrate the importance of the route of sensitization, because the former study was conducted among patients with acute parasitism after ingestion of parasitized fish, whereas our study focuses on subjects who may have been exposed to *Anisakis* proteins in various ways: through ingestion, skin contact, or inhalation as well as previous infection with live larvae. Such variables may influence the subsequent response to other allergens in various ways, which are currently being investigated in our laboratory.

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