

## Correspondence

## Reply

To the Editor:

We thank Des Roches and colleagues<sup>1</sup> for their kind comments about our study.<sup>2</sup> We agree that it is difficult to dissect the effect of maternal peanut ingestion during pregnancy from breast-feeding. As indicated in the article, we were able to isolate the effect of pregnancy by performing a subset analysis on 71 mothers who did not breast-feed and found a particularly strong association between frequent peanut ingestion during pregnancy and elevated peanut-specific IgE (>5 kUA/L) in that group (odds ratio, 4.99; 95% CI, 1.7-14.7;  $P < .004$ ). In addition, our analysis included a reasonably high percentage of subgroups who reported altering their peanut consumption after pregnancy (23.3% decreased and 7.8% increased consumption), adding to our multivariate analyses that consistently indicated that pregnancy consumption was the primary risk, and breast-feeding was not, even when only the latter was added to the model ( $P > .4$ ). Nonetheless, we agree that a larger trial would be needed to evaluate the relative influence more definitively, and we remain concerned about the existence of other possible causal factors including unmeasured environmental exposures.

We suggested caution in interpreting our results because our study design is observational in nature and selected children at risk of peanut allergy on the basis of personal atopic history.<sup>3</sup> We suggest caution in interpreting the case-control study by Des Roches and colleagues<sup>4</sup> as well, because, in contrast with our study, patients with peanut allergy were queried about peanut consumption after the diagnosis of peanut allergy, which could result in recall bias. In addition, their controls were nonatopic infants, whereas atopic infants without peanut allergy might have been a better matched control.

Overall, we share their frustration on the lack of definitive studies, but we must underscore, as we did in our article,<sup>2</sup> that there are several studies that have not identified peanut ingestion during pregnancy as a risk factor for peanut allergy. There is also a notion that specific allergen avoidance may prevent the occurrence of natural tolerance, which requires exposure. It is likely that many additional factors modify risk—for example, the proportion of linoleic or eicosapentenoic fatty acids in the maternal diet.<sup>5</sup> A study of a Tasmanian birth cohort<sup>6</sup> published subsequent to our work evaluated peanut sensitization at age 16 years in relation to antenatal maternal peanut ingestion and overall found no relationship ( $P = .17$ ). However, the study also evaluated sensitization to rye grass and found that among those without a family history of asthma, maternal peanut ingestion during pregnancy was related to decreased peanut and rye grass sensitization, whereas for those with a family history of asthma, it was associated with an increased risk of sensitization to rye grass (not peanut). Clearly, this is a complex area affected by a multitude of genetic and environmental factors.

Although Des Roches et al<sup>1</sup> put forth the argument that maternal avoidance may be responsible for a recent leveling off in the prevalence of peanut allergy, it could also be noted that peanut allergy appeared to have increased over the period when avoidance recommendations were promulgated.<sup>7</sup> The most recent recommendations admit a lack of evidence to suggest maternal avoidance.<sup>8</sup> We strongly agree that this is an area of investigation

in need of more research, although the practicality of appropriately powered randomized trials may be a challenge.

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