Atopic Dermatitis and Breastfeeding

To the Editor.—

We read with interest the article by Moore et al1 regarding atopic dermatitis (AD) in the first 6 months of life. Their conclusions that both prenatal and perinatal factors affect the incidence of AD are intriguing. We think, though, that a more detailed discussion should have been attempted regarding the influence of breastfeeding on the incidence of AD. The authors only write that “adjustment for infant feeding type at 3 and 6 months . . . did not materially alter any of the risk estimates.”

This conclusion is in contrast to the vast majority of research in the field of infant nutrition. Indeed, there is suggestion in some articles that exclusive breastfeeding may raise the incidence of AD. Miyake et al2 concluded that breastfeeding may be associated with increased incidence of AD, especially among children without a family history of allergy. Similar conclusions were reached by Bergmann et al,3 who suggested that for every month of breastfeeding there is an increased risk of AD. However, these authors (and others) have not addressed other potential causes for AD, which may have modified their statements. For example, Karmaus et al4 investigated the adverse effects of environmental factors and reached the conclusion that for children with high levels of dichlorodiphenyl dichloroethylene, breastfeeding did not have a protective effect against allergic manifestations. They concluded that contaminants may therefore reduce the protective effects of breast milk.

However, numerous studies suggest that breastfeeding does reduce the risk of AD. Gdalevich et al5 performed a meta-analysis of the association between breastfeeding during the first months of life and incidence of AD. They concluded that breastfeeding should be strongly recommended to mothers of infants when there is a family history of atopy as a means of preventing atopic eczema. Similar conclusions were reached by Schoetzau et al,6 who followed 856 exclusively breastfeeding infants and 256 partially breastfeeding or formula-fed infants for 1 year. Kull et al7 followed 4089 Swedish infants up to 2 years and also found protective effects of breastfeeding on the incidence of asthma and AD. Heine et al8 reached a similar conclusion regarding the protective effects of breastfeeding against AD but added that high birth weight and day care attendance increased the risk of AD in the first year of life. In the Moore et al article,9 birth weight was found to be similar between the 2 groups (with and without AD), but day care attendance was not studied.

The data regarding the protective effects of human milk against infections and allergic manifestations are strong if not overwhelm-

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In Reply.—

We thank Kilchevsky et al for their comments and brief review of the association between infant feeding type and risk of atopic dermatitis. We would like to make 2 points in reply.

The first point is a plea not to confuse lack of confounding with lack of prediction. The fact that adjustment for infant feeding type did not alter the risk estimates for other predictors does not preclude infant feeding itself from being associated with atopic dermatitis. Effects of infant feeding type and breastfeeding duration were beyond the scope of our analysis, but we intend to examine them in future analyses.

The second point is that perhaps the strongest evidence that breastfeeding protects against development of atopic dermatitis derives from a large, randomized, controlled trial of breastfeeding promotion, which showed approximately half of the odds of atopic eczema in the first year of life among the intervention group compared with controls.1 Nevertheless, breast milk is a complex substance; it is a source of allergens, nutrients, and antibodies. The influence of breastfeeding on the risk of allergy or wheeze may
Chickenpox Outbreak in a Highly Vaccinated School Population

To the Editor.—

We commend Tugwell et al1 for reporting on the important issue of chickenpox outbreaks in a highly vaccinated school population. However, the authors present conclusions on the time since vaccination as a risk factor with a measure of certainty not supported by the data.

Varicella is a highly contagious disease, and varicella cases can be infectious before rash onset. Exposure may occur on school buses, in lunchrooms, in hallways, and on playgrounds; therefore, it is difficult to ascertain the likelihood of exposure. The authors presume that there was zero exposure for children in the unaffected classrooms. These assumptions do not reflect the reality of exposure through population mixing.

It is important to know the degree of exposure in each class before drawing conclusions regarding duration of vaccine protection. Because the authors stratified classes based on the risk of exposure by excluding unaffected classes, that method needs to be applied consistently. We would suggest that classes be analyzed with household exposures, because the risk of breakthrough was highest 2 to 5 years postvaccination (years of higher likelihood of varicella zoster virus exposure) and declined thereafter.3 If waning immunity was a key factor, breakthrough rates would have increased over time.

The abrupt increase in attack rates is not consistent with waning of vaccine-induced immunity. Because time since vaccination is confounded by age at exposure and correlated with age at vaccination, not taking that into consideration when interpreting findings would lead to policy change that is not necessary. Given the above biases, the definitiveness of the conclusions regarding the waning of immunity is not warranted.

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To the Editor—

A recent Pediatrics article on varicella outbreak in Oregon1 reported that >5 years since vaccination was associated with a significant increase in the risk of breakthrough disease. The co-index subjects were all unvaccinated, and the secondary subjects were vaccinated. Although vaccinated subjects are infectious, they are less infectious than unvaccinated subjects in household settings (Varicella Active Surveillance Project, unpublished data, 2003). Therefore, students exposed to the co-index unvaccinated subjects have a more intense exposure than later subjects who were mainly exposed to children with breakthrough disease. If the co-index subjects were older (and it is likely that they were, because school immunization requirements have been implemented gradually, leaving older grades with more remaining susceptibles), then the classmates exposed to the unvaccinated co-index subjects were also older. These older children are more likely to have a longer time since vaccination.

Without knowing the age of those exposed to unvaccinated subjects (ie, cases occurring after the first incubation period) as compared with the ages of those exposed to vaccinated subjects (ie, cases occurring in subsequent incubation periods), it is not possible to determine if the length of time from vaccination among subjects with breakthrough disease is a reflection of greater exposure to older unvaccinated subjects. It would be worthwhile to account for this difference in exposure. It might be possible to stratify by generation of subjects, because the first generation of subjects was exposed to highly infectious unvaccinated subjects; whereas the following generations were exposed to less-infectious breakthrough subjects. We suspect that the first generation would be older than the second generation of subjects, which might partially explain the difference in time since vaccination.
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