

Letter to the Editor

Prevention of asthma and allergy: Evidence-based medicine?

Editor,

Chan-Yeung et al. (1) published their interesting and surprising findings on early-life environmental determinants of asthma at school age in 380 children at risk from a Canadian birth cohort study. The randomized intervention consisted of the reduction of exposure to house dust mite, pet avoidance, avoidance of environmental tobacco smoke, encouragement of exclusive breast feeding ≥ 4 months and delayed introduction of solid foods (2, 3).

In multivariate analyses adjusted for the intervention group assignment and baseline factors, significant risk factors for asthma at age 7 yr were asthma in mothers and siblings, and atopy in children at age 1 or 2 yr (1), in line with many other studies. Significant environmental risk factors were dog ownership at age 1–2 yr (but not cat ownership) and RSV infection at age 8–12 months (but not rhinovirus infection), while maternal smoking and short breastfeeding < 4 months were protective (1).

In our recent prospective, post-bronchiolitic follow-up until 12 yr of age in 81 Finnish children, early-life exposure to dog (but not to cat) decreased the risk of asthma at age 7–12 yr (OR 0.136, $p = 0.034$), though the significance was lost in multivariate analyses (aOR 0.203, $p = 0.150$) (4). Early sensitization to pets was assessed by specific immunoglobulin E measurements and by skin prick tests, and in both univariate and multivariate analyses, early sensitization to cat (aOR 5.318, $p = 0.022$) and to dog (aOR 5.165, $p = 0.039$) increased later asthma risk (4). Chan-Yeung et al. did not report the effects of early sensitization to pets, although the data obviously are available (2). Early sensitization, merely than pet ownership, in children at high risk for atopy might explain why dog exposure was a risk factor of asthma.

In a recent prospective, post-bronchiolitis follow-up until 17 yr of age in 89 Swedish children, prenatal exposure to tobacco smoke

was an independently significant risk factor of later asthma, while postnatal exposure increased asthma risk in young adults mainly via their own active smoking (5). In addition, prenatal tobacco smoke exposure, but not postnatal exposure, impaired lung function until early adulthood (6). In the Canadian cohort study, both prenatal and postnatal tobacco smoke exposure increased asthma risk at age 2 yr (2) but decreased asthma risk at age 7 yr (1). If this result is attributed to a selection bias as the authors discussed, how much the drop-outs (30.2%) and the consequent selection bias have influenced to the other findings?

Chen-Yeung et al. (1) found that the detection of RSV at age 12 months, but not that of rhinoviruses, was predictive for asthma at age 7 yr, as was the case with recurrent wheezing in early life (7). However, many recent studies in wheezing infants (8, 9), as well as in birth cohorts (10), have stressed the role of early rhinovirus infections in the development of asthma. In our study, the risk of asthma at 7 yr of age, compared with controls, was twofold after RSV-induced wheezing but 10-fold after rhinovirus-induced wheezing in early life (8).

As Chan-Yeung et al. (1) concluded, the effects of inherited risk factors of asthma cannot be modified by any intervention. In their earlier study, however, this multifaceted intervention programme was beneficial with a 56% reduction of doctor-diagnosed asthma at age 7 (14.9% vs. 23.0% in controls, aRR 0.44, 95%CI 0.25–0.79) (3). What actually caused the beneficial effects of the intervention, as the only component of the programme which decreased asthma risk was the avoidance of dog keeping? The other components of the intervention programme either increased asthma risk (avoidance of environmental tobacco smoke and exclusive breast feeding ≥ 4 months) or had no influence (reduction of exposure to house dust mite).

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