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Postnatal environmental tobacco smoke exposure is associated with objective markers atopy in preschool aged children

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Context

Up to 2/3 of children have evidence of exposure to environmental tobacco smoke (ETS) in the developed world.^[1] mostly from parental smoking. ETS has been highly associated asthma and allergic rhinitis. The mechanism by which ETS influences allergic conditions is unclear. Limitations have been difficulties parsing prenatal versus postnatal exposure, accurate measures of exposure, and unbiased measures of health outcomes.

This systematic review and meta-analysis examines the relationship postnatal exposure to ETS with objective markers of atopy.

Methods

Observational studies of children aged 0–18 years for the outcome of atopy were selected, excluding studies of high risk groups. The primary exposure was postnatal ETS. The primary outcomes were total IgE, any positive specific IgE (sIgE), and skin prick test (SPT). Quality control included data extraction by pairs of reviewers with triple cross-reference, bias assessments based on the Strengthening the Reporting of Observational studies in Epidemiology (STROBE) checklist and the Newcastle-Ottawa Scale (NOS). Statistical analysis of heterogeneity used the I^2 index and visual inspection of L'Abbé plots.

Results are reported as mean differences in total IgE estimated by random effects meta-analysis or by pooled odds ratios (OR) for sIgE and SPT between exposed and unexposed groups. Subgroup analyses determined differential effects by age (<7 versus 7–18 years) and study type (cross-sectional versus prospective).

Findings

Four studies (262 exposed and 172 non-exposed) demonstrated an 89.9 IU/ml (95% CI 30.9 – 148.9) elevation in total IgE in the exposed group. Removing one study to improve heterogeneity, the difference fell to 27.7 IU/ml (95% CI 7.8 – 47.7).

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Competing interests

None

Four studies (6629 subjects) demonstrated an OR of 1.12 (95%CI 1.00 – 1.25) of borderline significance ($p = 0.06$) for association with sIgE. However, the subgroup of preschool children demonstrated a significantly increased odds of sIgE (OR= 1.20, 95%CI 1.05 – 1.38) as did prospective studies (OR= 1.35, 95%CI 1.10 – 1.66). Ten studies (13,113 subjects) demonstrated increased risk of a positive SPT result (OR= 1.15, 95%CI 1.04 – 1.28), which was also greater in younger children (OR= 1.30, 95% CI 1.05 – 1.61) and prospective studies (OR= 1.43, 95% CI 1.01 – 2.01).

Commentary

This systematic review and meta-analysis lends mechanistic support to the described relationship between ETS and allergic diseases^[2] by demonstrating increased measures of atopy for ETS exposed children. The consistency across different measures of atopy and similar effect sizes to previous asthma studies^[3] supports the credibility of the relationship. Included studies cross different geographic regions and include only subjects without risk factors for atopy, thereby creating fairly generalizable results.

These findings are driven by the preschool aged children, raising the question of 1) a critical exposure period to produce sensitization, 2) the persistence of sensitization following exposure, and 3) lower exposure in older children, which has been suggested^[4]. Readers should be cautioned by the relatively few studies included in the meta-analysis and the degree of heterogeneity. The lack of consistency of findings by type of study – cross-sectional vs. prospective – casts a shadow on the quality of the selected studies.

The high potential for exposure misclassification that plagues this body of literature also effects this analysis. Postnatally exposed children likely also had prenatal exposure, which may have greater influence.^[5] Furthermore, exposure defined by biologic samples of cotinine identifies a broader group of exposed subjects than focused parental questionnaires.

Implications for practice

This study supports the strong relationship of ETS to allergic diseases in childhood by demonstrating the association with objective markers of atopy. It highlights the importance of early life exposure, which raises the question of a critical window of exposure and the longevity of the ETS influence on sensitization. This study adds support to public health strategies to decrease exposure to ETS for young children.

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