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Review

Does maternal diet during pregnancy and lactation affect outcomes in offspring? A systematic review of food-based approaches

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ABSTRACT

Objectives: The aim of this study was to investigate the relationship between maternal diet during pregnancy and lactation and development of atopic disorders in childhood.
Methods: We included studies published up to August 2011 that assessed food-based maternal dietary interventions or that examined associations between maternal dietary intake during pregnancy and/or lactation and allergic outcomes (eczema, asthma, hay fever, and sensitization) in their children.
Results: We included 42 studies (>40 000 children): 11 intervention studies (including 7 randomized control trials), 26 prospective cohort studies, 4 retrospective cohort studies, and 1 case–control study. In the randomized control trials, no significant difference was noted overall in the prevalence of eczema and asthma in the offspring of women on diets free from common food allergens during pregnancy. The prospective cohorts investigated a large number of potential associations, but reported few significant associations between maternal dietary intake and development of allergy. Maternal diets rich in fruits and vegetables, fish, and foods containing vitamin D and Mediterranean dietary patterns were among the few consistent associations with lower risk for allergic disease in their children. Foods associated with higher risk included vegetable oils and margarine, nuts, and fast food.
Conclusion: This review did not find widespread or consistent links between mothers' dietary intake and atopic outcomes in their children. However, maternal consumption of Mediterranean dietary patterns, diets rich in fruits and vegetables, fish, and vitamin D-containing foods were suggestive of benefit, requiring further evaluation.

Introduction

The prevalence of allergic diseases in most industrialized countries has increased over the past 20 y and is now estimated to affect one in five individuals [1–3]. Common manifestations of allergic disease include allergic rhinitis or hay fever, asthma, eczema or atopic dermatitis, and food allergies. The risk for allergic disease is increased to about one in three if one first-degree relative (parent or sibling) is atopic and to 70% if both parents are atopic [4]. The pattern of allergy expression differs with age; with the greatest incidence of food allergy and atopic eczema peaking by 1 y of age, whereas asthma and allergic rhinitis continue to increase until around 15 y of age [5]. Many childhood allergies persist, with about 50% of childhood asthma sufferers and 80% of hay fever sufferers continuing to have symptoms into adulthood [6,7]. The cost to the health care system and the burden for the family are high [8,9] and it is estimated that asthma alone costs in excess of $34 billion annually [10].

The increase in allergic disease has occurred too rapidly (within one to two generations) to be a result of population genetic changes, so it is likely related to environmental changes.

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Throughout the industrialized world, strong evidence exists that environmental factors accompanying higher socioeconomic conditions and hygiene standards have contributed to the increased prevalence of allergic disease. Societies with fewer respiratory infections, greater use of antibiotics early in life, fewer older siblings in the household, less contact with farm animals, and general lack of early microbial exposure are repeatedly associated with the greatest burden of allergic disease [11]. The atopic predisposition is believed to arise where the infant has an innate tendency to produce immunoglobulin (Ig)E antibodies (sensitization), which in some individuals progresses to allergic disease. The allergens causing sensitization are nearly always proteins originating from the environment including pollens, house dust mite, or food. Why children are becoming increasingly sensitized to environmental allergens is a matter of debate. Several factors may be involved, including whether the child is breastfed from birth [12], the child's antioxidant status [13], the balance of ω-6 to ω-3 polyunsaturated fatty acids in the diet [14] and environmental influences such as microbial exposure, cigarette smoke, and other pollutants [15].

Because many infants develop allergic symptoms early in infancy, and exposure to allergens may be important in the development of food allergies, there is great interest in maternal dietary strategies during pregnancy and lactation that may prevent childhood allergies, and thus reduce the burden of disease. This systematic review evaluates the effect of food-based approaches in the maternal diet for the prevention of childhood allergies.

Methods

Inclusion criteria

We included studies of any design that either compared a food-based maternal dietary intervention during pregnancy and/or lactation with another intervention or no intervention. We also included studies that examined associations between maternal dietary intake during pregnancy and/or lactation and allergic outcomes in their children from that pregnancy (cohort and case–control studies). Studies with cointerventions, such as timing of introduction of solid foods into the child's diet, use of hydrolyzed formula and non-food–based interventions such as dust mite control were eligible for inclusion.

We prespecified primary outcomes as child eczema, asthma, hay fever, and food allergy and secondary outcomes as allergy symptoms, atopy or atopic disorder, dyspnea, hay fever (allergic rhinitis or allergic rhinoconjunctivitis), wheeze (and recurrent wheeze), cough, food hypersensitivity (IgE-mediated food allergy or food intolerance), and sensitization (e.g., milk, egg, nut, food, inhalant).

We excluded studies assessing infantile colic, as this was not considered an allergic outcome.

As maternal intake needed to be food based, intervention studies designed to assess the effect of dietary supplements were not eligible for inclusion, nor were those where dietary intakes were expressed only in terms of nutrients.

We excluded studies that only investigated maternal nut (including peanut) consumption during pregnancy and/or lactation because nut allergy (particularly peanut) warrants a separate examination and assessment.

Searching

We searched MEDLINE, EMBASE, and the Cochrane Library (last searched end of August 2011) and scanned reference lists of systematic reviews and other relevant retrieved papers for additional studies.

Search terms included prenatal, antenatal, maternal, mother, pregnan*, lactat*, breastf*, intake, consumption, food, diet, wheez*, dermatitis, eczema, atop*, asthma, allerg*, food allergy hypersensitivity. The full search strategy is documented in the appendix.

Study selection and data extraction

Two authors independently assessed search results against study eligibility criteria. Two authors also independently conducted data extraction for each included study.

Data synthesis

Where possible, the results of randomized controlled trials (RCTs) were pooled, using the meta-analysis program RevMan [16]. We used risk ratios with 95% confidence intervals to express dichotomous outcomes. Where statistical heterogeneity was substantial ($I^2 > 40\%$), we used a random-effects model. Differences between subgroups were assessed using interaction tests [17].

The results of non-randomized intervention studies were narratively reported.

The results from cohort and case–control studies were tabulated and narratively summarized. Where available, we reported the adjusted outcomes for these study designs. Results were presented by age of children, with the youngest age first.

Risk for bias

For RCTs, we assessed the risk for bias using the methods outlined in the Cochrane Handbook for Reviews of Interventions [17]. For other study designs we took account of the risk for selection bias, attrition bias, and reporting bias.

Results

This systematic review included 42 studies. Table 1 lists the characteristics of the included studies. Eleven studies were intervention (7 RCTs, 1 participant preference trial, and 3 non-randomized comparisons). These studies examined the effect of eliminating or restricting common allergens from the maternal diet during pregnancy and lactation. Some included cointerventions such as manipulation of the infant's diet, or environmental measures.

The remaining 31 studies were 26 prospective cohorts, 4 retrospective cohorts, and 1 case–control study. These studies looked at the association of different dietary patterns or the frequency of consumption of different foods with atopic outcomes.

One intervention trial and seven other studies were excluded [18–25]. The intervention study reported the outcomes of an intervention based on a supplement rather than food; seven cohort studies reported dietary intakes in terms of nutrients rather than foods.

Results are reported by the major clinical outcomes (eczema, asthma and wheeze, hay fever, sensitization, and food allergy) and study design (intervention or observational).

Risk for bias assessment summary

The risk for bias assessments are described in Table 1 (association studies [26–76]) and in Table 2 (intervention studies [26,29,32,34,36,39,41,42,44,45,78]). Overall, the 11 intervention studies had at least moderate risk for bias. None of the RCTs fully described the methods used to conceal allocations at the time of randomization. Only one study was able to devise a way to blind the intervention and most studies had moderate losses of participants.

The 31 association studies were of reasonable quality: 12 had low risk for bias; 2 had low to moderate risk, and 17 had moderate risk for bias.

Maternal diet and eczema in children

Intervention studies. Five RCTs and two non-randomized comparisons assessed the effects of maternal dietary restriction on eczema in their children (Table 1). The five RCTs examined the effect of avoiding one of more common allergens in the maternal diet during pregnancy and lactation. In a study that randomized 212 women to a diet free of cow's milk and egg during pregnancy and lactation or a normal diet, the development of eczema,