

ORIGINAL ARTICLE

Maternal antenatal peanut consumption and peanut and rye sensitization in the offspring at adolescence

A. S. Kemp^{1,2,3}, A-L Ponsonby^{3,4}, T. Dwyer^{3,4}, J. A. Cochrane⁴, A. Pezic³ and G. Jones⁴

¹Department of Allergy and Immunology, The Children's Hospital, Westmead, NSW, Australia, ²Discipline of Paediatrics and Child Health, The Children's Hospital at Westmead, University of Sydney, NSW, Australia, ³Murdoch Children's Research Institute, Melbourne, Australia and ⁴Menzies Research Institute, University of Tasmania, Hobart, Australia

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Summary

Background There is considerable controversy whether maternal peanut ingestion during pregnancy might influence sensitization in later life.

Objective To examine whether maternal peanut ingestion during pregnancy might increase sensitization in the offspring.

Methods A population-based longitudinal cohort study with 16 years follow-up was conducted ($N = 373$). Subjects were recruited at birth as part of an infant health study. Maternal antenatal peanut consumption was documented at birth and peanut and rye sensitization were determined by measurement of serum-specific IgE at age 16.

Results Peanut sensitization was common (14%). In the entire cohort ($n = 310$), there was no association between antenatal peanut ingestion and peanut sensitization ($P = 0.17$). However, there was a strong association between antenatal peanut ingestion and decreased risk of rye sensitization and peanut sensitization in those ($n = 201$) without a family history (FH) of asthma (Rye OR 0.30, 95% CI 0.14–0.63, $P = 0.001$ and Peanut OR 0.18, 95% CI 0.04–0.78, $P = 0.02$). There was an increased risk of rye sensitization in those ($n = 108$) with a FH of asthma and antenatal peanut ingestion (Rye OR 2.69, 95% CI 1.11–6.51 $P = 0.03$). It was considered that these sensitizations were likely to be related to the presence of IgE antibodies to cross-reacting carbohydrate epitopes common to rye and peanut allergens.

Conclusions and Clinical Relevance Antenatal peanut ingestion may influence the development of IgE antibody to cross-reacting carbohydrate epitopes in later life. Genetic factors may modify this association.

Keywords adolescence, allergy, antenatal, cross reacting carbohydrate determinants, peanut, rye grass, sensitization

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Correspondence:

A.Kemp, Environmental and Genetic Epidemiology Research, Murdoch Children's Research Institute, Royal Children's Hospital, Flemington Rd, Parkville, VIC 3052, Australia. E-mail: andrew.kemp1@bigpond.com.au

Introduction

A number of IgE antibodies to peanut determinants have been described. These include antibodies to non-carbohydrate-containing determinants such as Ara h1, 2 and 3 and to cross-reacting carbohydrate determinants (CRCD), which are also found in other plant allergens, in particular in rye grass pollen [1, 2]. CRCD antibodies do not necessarily provoke clinical reactions and there is controversy as to whether they have clinical relevance [2, 3]. The prevalence of CRCD antibodies is uncertain; however, they appear to be more common than antibodies to determinants such as Ara h1 [2, 4].

The prevalence of IgE-mediated sensitization to peanut has increased substantially during the 20th century. In the Isle of Wight, UK, peanut sensitization increased threefold over 6 years from 1.1% in 1988 to 3.3% in 1994 in 3- to 4-year-old children [5] and in the United States, the rate of self-reported peanut allergy in children doubled between 1997 and 2002 [6]. Increased peanut exposure in earlier childhood appears to be associated with decreased peanut allergy [7]. It is likely that environmental factors influence the development of peanut sensitization.

There has been considerable controversy whether peanut exposure during pregnancy might influence the development of IgE antibodies against peanut. A recent survey of

systematic reviews and original studies that had investigated the effect of peanut avoidance in pregnancy highlighted the uncertainty in this area and concluded 'irrespective of whether or not there is a family history (FH) of allergy, that there is no evidence of danger from eating peanuts in pregnancy [8]. Most studies addressing the question of antenatal food allergen avoidance have examined cohorts of high-risk children with a FH of atopy or atopic disease; the effect of such avoidance on pregnancies at low risk with respect to atopy is less clear. However, such trials in high-risk cohorts have failed to demonstrate a protective effect of antenatal avoidance of cow's milk, egg [9] and peanut [10] on subsequent allergy. The possibility that antenatal avoidance might even be deleterious was raised by the observation that persistent intolerance to egg was significantly more common in children of the mothers on an antenatal egg exclusion diet [11].

The Tasmanian infant health cohort [12] provides the opportunity to examine the influence of antenatal peanut ingestion on the presence of IgE peanut and rye antibodies in a population-based longitudinal cohort study with 16 years follow-up.

Methods

In 1988 and 1989, there were 13 592 live births in Tasmania. A scoring system selected newborn infants at a higher risk of sudden infant death syndrome (SIDS) for possible participation in a Tasmanian infant health study (TIHS) [12]. The scoring system weighted infants according to their birth weight, season of birth, gender, mother's age, duration of second stage of labour and intention to breastfeed. All multiple births were included. In Southern Tasmania in 1988 and 1989, 1500 infants were born who were eligible to join the study. Of these, the mothers of 1433 infants agreed to an in-hospital interview and 80% filled a food frequency questionnaire (FFQ) regarding diet in the third trimester of pregnancy. At age 8, TIHS cohort children were followed up for studies on bone density, blood pressure in 1996 and HDL cholesterol and vitamin D in 1997 [13]. Using school lists, 1156 of these subjects were identified (or 81%, which is in close agreement to the Australian Bureau of Statistics data on annual outward migration rates from Tasmania of 2.5%). A total of 888 took part. In 2004/2005, a total of 730 were traced and approached to participate in a follow-up study of bone health and allergy at age 16. Of these, 139 failed to respond to the first or subsequent contacts, 129 responded but declined, 38 failed to attend for assessment and nine were not included for other reasons.

Allergen sensitization

Of the 415 participants, we obtained sufficient sera for peanut and rye IgE testing in 373 and of these 371 mothers

had recorded their FH of asthma. Three hundred and ten had data on specific IgE and a completed food frequency questionnaire and of these 309 also had data on a FH of asthma and 305 data concerning infantile eczema. Allergen sensitization was determined by the ImmunoCAP (Pharmacia Diagnostics AB, Uppsala, Sweden). A test result ≥ 0.35 kU_A/L of peanut-specific IgE was regarded as positive peanut or rye sensitization.

Antenatal food ingestion

Soon after giving birth, 80% of mothers completed a self-administered FFQ. The questionnaire was developed by the Commonwealth Scientific and Industrial Research Organization for self administration by adults [14]. This recorded dietary intake during the third trimester of pregnancy for 179 categories of food. The answers were coded to read the frequency of ingestion of peanuts, any nuts, fish and yoghurt. The frequencies were categorized as never or very rarely, one to four times per month and five and over times per month. A dichotomous category of never or very rarely vs. any was also made. For any nuts eaten antenatally, this included peanuts and also the items, nuts-salted and cooked, and unsalted fresh nuts. For the fish intake, the coding included eating chicken/fish patty, seafood (prawns, lobster and crab), fish fried, fish steamed/grilled (without batter), canned fish (tuna, salmon, etc.), Mornay, salted fish (e.g. herrings, anchovies) and fish fingers. Two versions of the FFQ were used but the frequency of these items was recorded the same way in both. Those mothers who had frequencies of peanut ingestion of one to four or over five times a month were classified as ever eaten with regard to antenatal peanut ingestion.

At birth family asthma history and other factors

An FH of asthma in first degree relatives was determined by a positive answer to this question in the hospital interview - 'Do any of the baby's brothers or sisters, parents or grandparents have a history of asthma?' This variable was examined as a potential effect modifier. We obtained data on other potential confounders that might influence the development of allergen sensitization; maternal antenatal smoking, maternal postnatal smoking, any breastfeeding at 1 month, early introduction of solids, cat as pet at 1 month, dog as pet at 1 month, male, premature birth and low birth weight. At age 16, blood was collected for IgE testing. Information was not collected concerning symptoms of clinical peanut allergy.

Statistical analysis

Odds ratios for various exposures and allergen sensitization were calculated. The antenatal diet data were then

stratified by FH of asthma. Logistic regression was used with exposure terms for FH of asthma and either peanuts or fish or yoghurt eaten antenatally and the outcome of allergen sensitization at adolescence. The interaction between FH of asthma and antenatal food intake was examined by a product term of FH of asthma and the relevant antenatal food exposure. In this model, the significance (*P*-value) for the interaction term was assessed by the Wald test applied to the coefficient of the product term [15]. Variables for potential confounders were added as covariates to the model. Missing data were excluded from the analyses. Ethics approval was obtained from the Human Research Ethics Committee at Children's Hospital at Westmead, Sydney and the University of Tasmania at Hobart, Australia.

Results

Concurrent data on antenatal food consumption and sensitization were obtained on 310 infants (74.7% of those who agreed to participate and 42.5% of the original participants located in Tasmania). Table 1 compares demographic factors in the original cohort (*n* = 1437). As compared with the original cohort, children in the present study appeared to belong to higher socio-economic families (older maternal age, more maternal university education, less paternal unemployment) but importantly did not differ to children born in 1988/1989 (Southern region) and not followed up with regard to specific IgE antibody data in relation to FH of asthma and maternal antenatal peanut consumption.

Table 2 shows the prevalence of IgE antibodies against common allergens. There was a high rate of sensitization to rye grass 37.5% and house dust mite (HDM) 42.5%. Fourteen percent (53/373) of adolescents had evidence of peanut IgE antibodies. This may be compared with a prevalence of 0.5% (2/373) of sensitization to cow's milk and 0.8% (3/374) sensitization to egg. The distribution of specific IgE antibody scores in kU_A/L for the 53 peanut IgE-positive subjects was 0.35–0.7, *n* = 17; 0.7–3.5, *n* = 24; 3.5–17.5, *n* = 11 and > 17.5, *n* = 1. The median peanut IgE level was 1.13. Subjects with a positive FH of asthma were not less likely to have mothers who consumed any pe-

nuts during pregnancy (*P* = 0.36) and were more likely to subsequently develop asthma [OR 2.30 (1.52, 3.49)]. Interestingly we observed that only one subject of those sensitized to peanut failed to react to rye grass. We calculated the risk of sensitization to other allergens by peanut sensitization. There was an extremely high relative risk 86.5 (95% CI 12.1, 619.1) of rye sensitization (Table 3). The magnitude of the RR of other sensitizations ranged from 3 to 6 (Table 3). These observations suggested that we were detecting CRCD common to peanut and rye grass. We examined the influence of peanut ingestion in pregnancy on allergen sensitization. Overall, there was no significant effect of antenatal peanut consumption on rye, OR (95% CI); 0.67 (0.40, 1.14) peanut sensitization OR 0.71 (0.34, 1.49) or cat sensitization OR 0.81 (0.41, 1.59). We then examined the association between allergen sensitization and antenatal peanut ingestion with regard to the presence or absence of an FH of asthma (Table 4). This demonstrated an association of antenatal peanut ingestion and reduced rye and peanut sensitization in those without an FH of asthma [rye (OR 0.30, *P* = 0.001) and

Table 2. Prevalence of allergen specific sensitization and characteristics of the adolescent follow-up sample

Year of birth	1988/1989
Region of birth (Tasmania)	South (97.4%) North (2.65%)
Year of follow-up	2004/2005
Number	374
Age at follow-up	Mean = 16.30, SD = 0.45
Inhalant sensitization	
HDM Der p1	41.7 (156/374)
HDM Der f1	34.8 (130/374)
HDM	42.5 (159/374)
Rye	37.5 (140/373)
Cat	17.4 (65/373)
Alternaria	10.2 (38/374)
Dog	3.2 (12/373)
Any inhalant	53.9 (201/373)
Food sensitization	
Peanut	14.2 (53/373)
Egg	0.8 (3/374)
Milk	0.5 (2/373)
Any food	14.5 (54/373)

Table 1. Demographic features of Tasmanian infant health cohort and current study

Comparison of the 1988/1989 southern births and those with specific IgE data			
	Without specific IgE data	With specific IgE data	<i>P</i> -value
Family history of asthma	36.8% (396/1075)	35.1% (127/362)	0.55
Peanuts eaten antenatally	26.3% (195/742)	29.8% (90/302)	0.25
Mother antenatal smoking	54.2% (579/1069)	46.3% (168/363)	0.01
Maternal education university or above	5.9% (63/1074)	9.4% (34/363)	0.02
Mother's age, mean (SD)	23.9 (4.7)	25.2 (5.0)	<0.001
Father unemployed at child's birth	27.8% (292/1051)	13.8% (49/354)	<0.001

peanut sensitization (OR 0.18, $P=0.02$)). In contrast in those with an FH of asthma, there was increased rye (OR 2.69, $P=0.03$) and peanut (OR 2.80, $P=0.06$) sensitization. There was also a decrease in cat sensitization associated with antenatal peanut consumption (OR 0.31, $P=0.04$). Following adjustment for rye sensitization, this effect was not apparent for cat [OR 0.56 (95% CI 0.17, 1.83)] or peanut [OR 0.34 (95% CI 0.07, 1.70)]. Also, the interaction between FH of asthma and either peanut or cat sensitization was not evident after adjusting for rye sensitization.

In addition, there was an increased rye sensitization [OR 2.69 (95% CI 1.11, 6.51)] in those with an FH of asthma who did ingest peanut in pregnancy. The findings concerning the antenatal consumption appeared specific for antenatal peanut ingestion as there was no significant effect for those with and without an FH of asthma of antenatal ingestion of other foods (yoghurt or fish) on either rye sensitization [with FH asthma yoghurt OR 1.37, $P=0.3$ fish OR 1, $P=0.99$; without FH of asthma yoghurt OR 0.96, $P=0.93$ fish OR 1.16, $P=0.79$ or peanut sensitization (not shown)]. We examined whether the apparent difference in effect between antenatal peanut consumption and peanut or rye sensitization by FH of asthma persisted after adjustment for various confounding factors such as cohort entry score components, male sex, maternal antenatal smoking, premature birth, low birth weight, maternal postnatal smoking, any breastfeeding at 1 month, cat as pet at 1 month, dog as pet at 1 month or early introduction of solids. The difference in effect of antenatal ingestion on peanut and rye sensitization by FH of asthma persisted ($P<0.01$) after adjustment for these potential confounders.

Table 3. Peanut sensitization and risk of other sensitizations

	Risk ratio	95% CI	<i>P</i> -value
Rye	86.54	12.10 to 619.08	<0.001
House dust mite	4.14	2.29 to 7.48	<0.001
Mould	3.48	2.12 to 5.71	<0.001
Cat	3.11	1.92 to 5.03	<0.001
Dog	6.14	4.00 to 9.42	<0.001

Table 4. Difference in association between maternal antenatal peanut ingestion and allergen sensitizations in those with and without a family history of asthma

Sensitization	No family history of asthma					Family history of asthma					Difference in effect
	Never eaten %	Ever eaten %	OR	95% CI	<i>P</i> -value	Never eaten %	Ever eaten %	OR	95% CI	<i>P</i> -value	<i>P</i> -value
Rye	41.7 (58/139)	17.7 (11/62)	0.30	0.14, 0.63	0.001	30.0 (24/80)	53.6 (15/28)	2.69	1.11, 6.51	0.03	<0.001
Peanut*	15.8 (22/139)	3.2 (2/62)	0.18	0.04, 0.78	0.02	12.5 (10/80)	28.6 (8/28)	2.80	0.98, 8.03	0.06	0.003
Cat*	18.0 (25/139)	6.5 (4/62)	0.31	0.10, 0.95	0.04	16.3 (13/80)	32.1 (9/28)	2.44	0.91, 6.58	0.08	0.007
House dust mite	44.3 (62/140)	33.9 (21/62)	0.64	0.35, 1.20	0.17	41.3 (33/80)	46.4 (13/28)	1.23	0.52, 2.93	0.63	0.23
Mould	9.3 (13/140)	8.1 (5/62)	0.86	0.29, 2.52	0.78	11.3 (9/80)	7.1 (2/28)	0.61	0.12, 3.00	0.54	0.73

*In a further multivariate analysis, the difference in effect of antenatal peanut consumption on these specific sensitizations was not independent of rye grass sensitization.

Level of peanut ingestion

The maternal frequency of peanut ingestion during pregnancy was one to four times per month in 23.6% (73/309) and five and over times a month in 5.5% (17/309). Overall, 65.0% (201/309) of adolescents ingested peanuts, with 45.3% (140/309) ingesting peanuts one to four times a month and 19.7% (61/309) five and over times a month.

Maternal consumption in pregnancy was not related to peanut ingestion at 16. For those with an FH of asthma and mother ate peanuts antenatally, 64.3% of offspring ate peanuts at age 16 whereas for those with an FH of asthma and mother did not eat peanuts antenatally, 60.0% ate peanuts at age 16, $P=0.69$. For those with no FH of asthma and mother ate peanuts antenatally, 69.4% ate peanuts at age 16 whereas for those with no FH of asthma and mother did not eat peanuts antenatally, 66.2% ate peanuts at age 16, $P=0.66$.

As household peanut consumption appears to be a major factor associated with the emergence of peanut allergy [16], antenatal ingestion may be just a marker for postnatal exposure in childhood. We examined current peanut ingestion in adolescence. There was no association between any maternal antenatal peanut consumption and adolescent consumption [$\kappa=0.027$ (95% CI) 0.058–0.112]. There was no association between current (in the past 12 months) ingestion of peanut and peanut sensitization in adolescence (results not shown).

No significant association was found between current peanut ingestion and peanut sensitization in the adolescents when stratified according to the presence or absence of an FH of asthma (results not shown).

Infantile eczema

It has been suggested that the presence of eczema might facilitate peanut sensitization via the cutaneous route [17]. Nineteen percent (59/305) of subjects had eczema at ages 0–2 years: 22% (13/59) of adolescents with a history of infantile eczema were sensitized to peanut whereas 11% (28/246) of those without infantile eczema became

sensitized ($P = 0.03$). For rye grass, 49% (29/59) of adolescents with a history of infantile eczema were sensitized as compared with 31% (77/246) of those without infantile eczema ($P = 0.01$).

Discussion

The principal findings of this study are that there was an association between maternal ingestion of peanut during pregnancy and the development of rye and peanut IgE antibodies and this association differed at birth by FH of asthma. Maternal ingestion of peanut during pregnancy was associated with a significant reduction of rye IgE and peanut IgE antibodies at age 16 in subjects who did not have an FH of asthma. This effect of antenatal peanut ingestion was a substantial one, lowering the risk for the development of rye and peanut IgE antibodies by approximately three- and fivefold, respectively. This effect was specific for antenatal peanut consumption and not related to antenatal consumption or avoidance of two other foods examined (fish and yoghurt).

Nature of peanut and rye immunoglobulin E antibodies

It is likely that our findings reflect the development of antibodies to CRCD. Ubiquitous plant structures such as profilins and CRCD readily stimulate the production of IgE antibodies. These antibodies usually lack biologic activity. CRCD have carbohydrate chains attached to a peptide backbone. Plant pollens provide some of the best documented examples of specific IgE reactivity to carbohydrate-containing determinants being found in 25–50% of pollen allergic patients. CRCD are found between plant pollens and plant foods and appear to be particularly prominent between grass pollens and peanut allergens. Thus, Guilloux found 51% (21/41) of grass pollen-allergic subjects [4] and Van der Veen (29%) 50/173 contained antibodies to CRCD of peanut [2]. Other cross-reactions have also been described between plant peptide-based allergens such as pathogenesis-related proteins and profilins.

The prevalence of IgE antibodies to peanut in our cohort not selected for atopy was high (14%) as compared with the prevalence of clinical peanut allergy or sensitization on skin prick testing found in other populations. In addition, the extremely high concordance between peanut and rye sensitization compared with other allergens such as cat or HDM indicates that antibodies to CRCD are being detected. Pollen exposure is considered to be a principal stimulus for the development of the antibodies. Guilloux et al. described the frequent occurrence of peanut IgE in patients sensitized to grass pollen, which was not associated with any symptoms related to peanut ingestion [4] and antibodies to CRCD were detected in a high proportion of the patients. These cross-reactive IgE antibodies directed to carbohydrate determinants are detected with spe-

cific IgE antibody testing but not necessarily with skin testing [2, 4]. The prevalence of these antibodies in a community-based population is unclear. In line with other studies, our findings suggest that such antibodies are common in a population not selected for atopy.

Relation to other studies of antenatal exposure or avoidance

A limited number of studies have examined the influence of maternal food allergen avoidance during pregnancy and these have failed to demonstrate a protective effect. A number of trials [11, 18, 19] concluded that there was no proven benefit of antenatal food allergen avoidance of cow's milk and egg. However, in contrast to the present study all these studies focused on 'high-risk' subjects with an FH of allergy and the issue of a differing association among subjects without an FH of atopy was not examined. Furthermore, they did not address the question of peanut allergy. In a small atopic cohort, Frank et al. [20] found that mothers who consumed peanuts more than once a week during pregnancy were more likely to have a peanut-allergic child than mothers who consumed peanuts less than once a week. Frank et al. did not examine the effect in a non-atopic cohort. In a large community-based cohort, Lack et al. did not find a significant association between maternal peanut consumption and peanut allergy up to 38 months of age; however, data on peanut consumption during pregnancy were not obtained prospectively [17]. Zeiger et al. [10] found that peanut avoidance during pregnancy and early childhood did not alter peanut sensitization at 7 years of age. The possibility that exposure might suppress the development of allergy was suggested by Falth-Magnusson who found that persistent intolerance to egg at 5 years of age in a high-risk cohort was significantly more common in children of the mothers who excluded eggs during pregnancy and early lactation [11]. Falth-Magnusson speculated that the intervention of maternal dietary avoidance might possibly be harmful if it interacts with the normal induction of tolerance [11].

It has been suggested that the presence of eczema might facilitate peanut sensitization via the cutaneous route [17]. We found an increased peanut 22% vs. 11% and rye 49% vs. 31% sensitization in children with and without infantile eczema. The most likely explanation for these findings is that infantile eczema is a marker for an atopic diathesis; however, the possibility that the eczema may also have promoted sensitization by the cutaneous route cannot be fully excluded. However, our findings indicate a substantial number of children became sensitized in the absence of infantile eczema.

Although CRCD are not necessarily of clinical relevance, the observation that these IgE antibodies can be modified by antenatal allergen ingestion is of significant interest as it implicates a possible role for antenatal

allergen exposure in modifying the development of IgE antibodies in later life. The effect on rye sensitization is complex with a decreased association demonstrable in those without an FH of asthma and an increased association with rye grass sensitization in those with a positive FH. It remains to be demonstrated that such effects would operate for those IgE antibodies relevant to clinical rye grass or peanut allergy.

Implications

Atopic disease is considered to result from a combination of environmental and genetic influences and an FH of atopy may be an effect modifier of environmental factors [21]. A differential effect on those with an FH of allergy or asthma of other environmental influences has been observed in some studies concerning breast feeding [22, 23], cat exposure [24], HDM exposure [25], environmental tobacco smoke [26] but was not observed in other studies with early life infections [27] and breast feeding [28, 29]. Further work is required to separately assess potential effect modification by maternal allergy rather than an at-birth complete FH of asthma. The differential effect on those with and without an FH of asthma raises the question whether advice regarding allergy protection obtained from high-risk cohorts might in fact be adverse if applied either deliberately or inadvertently to populations that are not at high risk. In the United Kingdom, it was found that the majority of mothers who adopted a peanut avoidance strategy were those at low risk, even though the advice was not directed at them [30]. Current opinion does not recommend antenatal peanut avoidance in either high- or low-risk pregnancies.

In an experimental model of food allergy, prenatal ovalbumin exposure induced tolerance and the balance between tolerance and sensitization depended on the amount and timing of maternal allergen intake during pregnancy [31]. In the present paper, the asthma status of the family is also a factor determining the balance between tolerance and sensitization associated with prenatal exposure. Postulated mechanisms of tolerance have included an induction of suppressive regulatory T cells and an increased maternal TGF- β production with transfer to offspring during breast feeding. However, it has been questioned whether neonatal T cell responses to allergens actually reflect in utero sensitization and it has been suggested that these so-called responses reflect the activity of naive recent thymic emigrant CD4T cells [32]. Thus, the mechanism whereby antenatal exposure to peanut might reduce sensitization in later life must remain speculative.

Strengths and limitations of the study

The strengths of the present study were the documentation of maternal antenatal peanut and nut consumption at the

time of birth. Other studies that have explored the question of maternal antenatal peanut ingestion and subsequent sensitization or allergy have collected these data retrospectively when the children were 5–7 years of age. The prevalence of peanut ingestion in our study (29% of mothers) was the same as that of Lack (29%) [17] but less than that observed by Hourihane (57%) [30]. Recruitment was as part of a community-based study and not one directed to subjects at high risk of atopic disease. As the subjects were initially recruited for a study of non-atopic conditions (SIDS and bone health), the potential for selection bias for those interested in or affected by atopic disease is likely to be minimal. Limitations include the fact that loss to follow-up might introduce unrecognized biases, food frequency questionnaires may be subject to inaccuracies, relatively limited numbers of subjects are available for analysis and antenatal ingestion may be an indicator of post-natal intake in the offspring. However, our observations that (1) maternal antenatal peanut consumption was not associated with adolescent consumption and (2) adolescent consumption was not associated with peanut sensitization provide some reassurance that maternal antenatal peanut consumption is not merely a marker for postnatal child peanut consumption. It remains a possibility that the association between FH of asthma peanut consumption and peanut IgE may be influenced by other confounders (e.g. presence of eczema, having other food allergies in the family).

Further work is required to separately assess potential effect modification by maternal allergy rather than an at-birth FH of asthma. It is also important to recognize that this study primarily concerns rye and peanut IgE antibodies, which are likely to be predominantly to CRCD and the finding of an effect on such antibody development does not necessarily mean that a similar effect would be observed on those antibodies that result in clinical disease.

Conclusions

Antenatal peanut ingestion may protect against peanut sensitization to carbohydrate-containing epitopes for those without an FH of asthma but not for those with an FH of asthma. Thus for some epitopes, antenatal avoidance might inadvertently contribute to an increase in sensitization.

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