ORIGINAL ARTICLE

Peanut Allergy: Is Maternal Transmission of Antigens During Pregnancy and Breastfeeding a Risk Factor?

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Abstract

Background: Peanut allergy is an important public health problem in western countries. However, the risk factors associated with this allergy remain uncertain.
Objective: To determine whether the consumption of peanuts during pregnancy and breastfeeding is a risk factor for peanut allergy in infants.
Methods: We enrolled 403 infants in a case-control study. The cases were infants aged 18 months or less with a diagnosis of peanut allergy based on a history of clinical reaction after exposure to peanuts and the presence of peanut-specific immunoglobulin E. Controls were age-matched infants with no known clinical history or signs of atopic disease. The mothers of the children filled out a detailed questionnaire about maternal diet during pregnancy and breastfeeding, the infant's diet, the presence of peanut products in the infant's environment, and family history of atopy.
Results: The mean (SD) age of cases was 1.23 (0.03) years. The groups were comparable in terms of the rate and duration of breastfeeding. However, the reported consumption of peanuts during pregnancy and breastfeeding was higher in the case group and associated with an increased risk of peanut allergy in offspring (odds ratio [OR], 4.22 [95% confidence interval [CI], 1.57-11.30 and OR, 2.28 [95% CI, 1.31-3.97] for pregnancy and breastfeeding, respectively). Overall, the infants with peanut allergy did not seem to be more exposed to peanut products in their environment than the controls.
Conclusion: Early exposure to peanut allergens, whether in utero or through human breast milk, seems to increase the risk of developing peanut allergy.

Key words: Allergy. Breastfeeding. Food. Peanut. Pregnancy.

Resumen

Antecedentes: La alergia a cacahuete es un importante problema de salud pública en los países occidentales. No obstante, los factores de riesgo asociados con esta alergia siguen siendo inciertos.
Objetivo: Determinar si el consumo de cacahuetes durante el embarazo y el período de lactancia es un factor de riesgo de alergia a los cacahuetes en lactantes.
Métodos: Se incluyeron 403 lactantes en un estudio de casos y controles. Los casos eran lactantes de hasta 18 meses de edad con un diagnóstico de alergia a los cacahuetes basado en antecedentes de reacción clínica tras la exposición a cacahuetes y presencia de inmunoglobulina E específica frente a cacahuete. Los controles eran lactantes sin antecedentes clínicos conocidos ni signos de enfermedad atópica emparejados por edad. Las madres de los niños rellenaron un cuestionario detallado sobre su dieta durante el embarazo y la lactancia, la dieta del lactante, la presencia de productos con cacahuetes en el entorno del lactante y los antecedentes familiares de atopia.
Resultados: La edad media (DE) de los casos fue de 1,23 (0,03) años. Los grupos fueron comparables en cuanto a la frecuencia y la duración de la lactancia. No obstante, el consumo de cacahuetes notificado durante el embarazo y la lactancia fue superior en el grupo de casos y se asoció con un mayor riesgo de alergia a los cacahuetes en los descendientes (oportunidad relativa [OR] = 4,22; intervalo de confianza [IC] del 95%: 1,57 11,30 y OR = 2,28; IC del 95%: 1,31 3,97 para el embarazo y la lactancia, respectivamente). En general, no parece que los lactantes con alergia a los cacahuetes estuvieran más expuestos a productos con cacahuetes en su entorno que los controles.
Conclusión: La exposición temprana a alérgenos del cacahuete, ya sea en el útero o a través de la leche materna, parece aumentar el riesgo de desarrollar alergia a los cacahuetes.

Palabras clave: Alergia. Lactancia. Alimentos. Cacahuete. Embarazo.

Introduction

Peanut allergy is an important public health problem in western countries. It affects 1 in 150 persons in the United States and has high anaphylactic potential [1-4]. Most children with peanut allergy develop a reaction on the first known exposure to peanuts [5], implying that previous, insidious exposure has occurred in early childhood. Possible sources are contaminated foods, indirect exposure through household members eating peanuts, use of cosmetic or medical products containing peanut derivatives, and peanut proteins contained in breast milk. There has been a report, for example, of an infant developing a peanut allergic reaction while being breastfed [6]. Breastfeeding has been extensively promoted in recent decades and peanut allergens are known to pass through human milk [7]. As the fetus might also be exposed to allergens in utero [8], sensitization by early exposure to peanuts during pregnancy cannot be excluded either. The purpose of this study was to determine if maternal consumption of peanuts during pregnancy and breastfeeding is a risk factor for peanut allergy in infants.

Methods

Selection of Cases and Controls

Cases and controls were recruited from our pediatric university center (Centre Hospitalier Universitaire Sainte-Justine, Montreal, Canada) between October 1998 and December 2004. Cases were selected from among infants aged 18 months or less presenting at the allergy outpatient clinic (where they were seen by A.D.R. or L.P.) with a presumptive diagnosis of allergy to peanuts in the month before the visit. Peanut allergy was defined as a history of a clinical reaction within 60 minutes of exposure to peanuts, combined with the presence of peanut-specific immunoglobulin (Ig) E (positive skin tests and/or serum-specific IgE). A clinical reaction was defined as 1 or more symptoms affecting at least 1 of the following organ systems: skin (hives, angioedema); lower or upper respiratory tract (wheezing, repetitive cough, shortness of breath, rhinitis, conjunctivitis, voice change); and gastrointestinal tract (vomiting, diarrhea). Food challenges were not performed because of ethical considerations related to the young age of the children and the risk of a life-threatening reaction. Given the estimated prevalence of the risk factors under consideration and projecting a 2-fold increase in associated risk, we determined that a case group of 200 would theoretically provide the desired 80% study power. Additional skin tests were thus performed on 248 consecutively enrolled infants with a compatible history to determine study eligibility. Of these, 36 were excluded because they had a negative skin prick test to peanut and 2 because their mothers did not speak French. Of the 210 eligible infants, 202 participated (participation rate, 97%).

The control participants were infants of 18 months of age or less who presented at the same hospital and had no known signs of atopy such as food allergy, atopic dermatitis, repeated bronchiolitis (1 episode or more), asthma, or persistent and/or repeated rhinitis. Children with a family history of atopy, however, were not excluded. The controls were recruited from among children presenting at different outpatient clinics (pediatrics, urology, ophthalmology, cardiology, dermatology, emergency, one-day surgery, and plastic surgery) and at the outpatient blood test center. A research nurse contacted the parents in the waiting room and determined eligibility based on age and the above criteria. In total, the parents of 410 potential controls were contacted; 195 infants were excluded: 77% because they had atopic manifestations (a high percentage due to the presence of eczema and/or chronic rhinitis), 19% because the infant had not been accompanied by the mother, and 4% because the mother did not speak French. Of the 215 eligible infants, 201 participated (participation rate, 93.5%).

Exposure and Confounder Data

Data were collected using a self-administrated questionnaire filled out by the infant's mother during the visit to the hospital. A food frequency questionnaire was used to evaluate patterns of food consumption. The questionnaire specifically explored the mother's diet during pregnancy and breastfeeding. The respondents were asked to specify the frequency with which they had eaten certain foods during the periods analyzed, with frequencies defined as once a day or more, 5 to 6 times a week, 2 to 4 times a week, once a week, 1 to 3 times a month, or never. In the case of peanut butter, they were also asked to report the average size of the portions consumed. The food history of the infant since birth was also investigated and the presence or absence of potential sources of peanut in the child's environment was assessed on the basis of a comprehensive list of peanut-containing products. Finally, sociodemographic information and past medical history of atopic diseases in the family were obtained.

Medical Questionnaire

The medical questionnaire included information on clinical reaction to peanuts, treatment required to control the reaction, age at the time of the first reaction, and history of exposure to peanuts. The presence of symptoms for other atopic diseases (atopic dermatitis, asthma, rhinitis, and food allergy) was also assessed in cases and their families.

Skin Prick Tests

Skin prick tests with peanuts and a panel of inhalant and food allergens (Laboratoire Omega, Montreal, Canada) were performed in cases using a sterile 25-gauge needle according to the Pepys technique [9]. Positive and negative controls were performed using histamine 2 mg/mL and saline 0.9%, respectively. The skin test site was measured after 15 minutes and a positive result was recorded if the mean wheal diameter was at least 3 mm larger than that of the negative control. Skin prick tests were also performed in controls whose parents agreed to these tests.

Total and Specific IgE

Serum peanut-specific IgE levels (IU/mL) were measured using an enzyme allergosorbent test (Allercoat 6; Bio-Rad

Laboratories, Marnes la Coquette, France) in all cases. The cutoff points were those established by the manufacturer: <0.35 IU/mL, undetectable; 0.35-0.7 IU/mL, low; 0.7-3.5 IU/mL, moderate; >3.5-50 IU/mL, high; and >50 IU/mL, very high. Total serum IgE (kU/L) was measured using an automated microparticle enzyme immunoassay (IMx, Abbott Laboratories, France). Values were considered normal if they were lower than 25 kU/L in infants aged 12 months or less and if they were lower than 60 kU/L in infants between 13 and 36 months (manufacturer's criteria). Blood tests in control children were not allowed by the ethics committee.

Statistical Analysis

Logistic regression was used to estimate odds ratios (ORs) and 95% confidence intervals (CIs) associated with maternal peanut consumption. ORs were adjusted for age, sex, family history of atopic diseases, and duration of breastfeeding (where relevant). Proportions were compared between groups using the χ^2 test. The study protocol was reviewed and approved by the ethics committee at our institution.

Results

Baseline Characteristics of Cases and Controls

The baseline characteristics of cases (202) and controls (201) were comparable (Table 1). The cases had a mean (SD) age of 1.23 (0.03) years and included 120 boys and 82 girls. The cases were slightly older than the controls and there were more boys. The mothers' age was similar in both groups but the level of education was higher in the case group. Eighty percent of infants were breastfed in both groups, although the duration was slightly longer in the case group (6.3 months vs 5.7 months). The use of nipple cream was similar in both groups.

Table 1. Baseline Characteristics of Cases and Controls

	Cases (n=202)	Controls (n=201)
Age, mean (SD), y	1.23 (0.03)	1.14 (0.04)
Boys, No (%)	120 (59)	102 (51)
Maternal age, mean (SD), y	30.5 (4.8)	30.6 (5.3)
Educational level of mothers, No. (%)		
University	101 (50)	84 (42)
Collegial Studies	46 (23)	50 (25)
Secondary school	40 (20)	43 (21)
Primary school	2 (1)	4 (2)
Others	13 (6)	20 (10)
Language spoken, No. (%)		
French	194 (96)	185 (92)
Current maternal smoking, No. (%)	29 (14)	33 (16)
Breastfeeding, No (%)	162 (80)	161 (80)
Duration of breastfeeding, mean (SD), mo	6.3 (3.8)	5.7 (3.7)
Use of nipple cream, No./ Total (%)	97/162 (60)	94/161 (58)

Clinical Characteristics of Cases

A reaction to peanuts occurred at the first known exposure in 75% of case patients. The mean (SD) age at which the first reaction to peanuts was observed was 12.1 (2.8) months. Peanut butter was reported to have caused this reaction in 87% of cases. Urticaria (81%) and angioedema (47%) were the 2 most common manifestations and vomiting and diarrhea were present in 12% of the patients. More severe reactions also occurred, such as respiratory manifestations (16%), altered consciousness (4%), and hypotension (0.5%). More than 56% of the reactions involved at least 2 organs. All the cases had a positive skin test for peanuts, with a mean (SD) diameter of 10 mm (4 mm) and a range from 3 to 24 mm. Total IgE levels were increased in 32% of infants (≥25 kU/L in those aged 12 months or less and ≥60 kU/L in those aged between 13 and 36 months). The range of total IgE was 0 to 213 kU/L (median, 19 kU/L) in the first group and 0 to 998 kU/L (median, 28.5 kU/L) in the second group. Peanut-specific IgE levels were high in 17% of cases, moderate in 53%, low in 9%, and undetectable in 21%. Sensitization to other foods and inhalants was observed in 70% and 44% of infants, respectively. Other atopic diseases were frequently reported, including atopic dermatitis (75%), asthma (16%), repeated bronchiolitis (21%), rhinitis (28%), and other food allergies (38%). All the skin tests performed in the 75 controls (37%) whose parents had agreed to such tests were negative for peanut extract.

Analysis of Data Collected by the Self-Administrated Questionnaire

The prevalence of atopic diseases such as atopic dermatitis, rhinitis, and food allergy in parents was markedly higher in the case group than in the control group (where it was not negligible) (Table 2). Peanut consumption was more common in case mothers than in control mothers during both breastfeeding (95% [154/162] vs 79% [124/157]) and pregnancy (97% [196/202] vs 88% [176/199]). Table 3 shows significantly increased ORs associated with the consumption of peanuts during pregnancy (crude OR, 4.2 [95% CI, 1.63-13.07]; adjusted OR, 4.22 [95% CI, 1.57-11.30] and breastfeeding (crude OR, 5.12 [95% CI, 2.21-13.24], adjusted OR, 2.28 [95% CI, 1.31-3.97]). The quantity of peanuts consumed during pregnancy and breastfeeding was similar in the case group (25.5 g) and the control group (24.7 g) (P=.69). As reported, peanut butter, which contains roasted peanuts, was the most common type of peanut-containing food consumed. An increased risk was also observed with the consumption of soy but not nuts or eggs during both pregnancy and breastfeeding.

We also measured the age at which other potentially allergenic foods were introduced into the infant's diet (Table 4),

Table 2. Family Atopy in Cases and Controls

	Cases (n=202)	Controls (n=201)
Mother, No. (%)		
Atopic disease	116 (57)	68 (34)
Atopic dermatitis	52 (26)	18 (9)
Rhinitis	75 (37)	37 (18)
Food allergy	26 (13)	14 (7)
Asthma	34 (17)	25 (12)
Father, No. (%)		
Atopic disease	102 (50)	58 (29)
Atopic dermatitis	25 (12)	12 (6)
Rhinitis	70 (35)	42 (21)
Food allergy	17 (8)	12 (6)
Asthma	34 (17)	9 (4)
Siblings, No./Total No. (%)		
Atopic disease	47/84 (56)	26/86 (30)
Atopic dermatitis	30/84 (36)	10/86 (12)
Rhinitis	15/84 (18)	5/86 (6)
Food allergy	11/84 (13)	8/86 (9)
Asthma	19/84 (23)	13/86 (15)

Table 3. Risk Associated With Maternal Diet During Pregnancy and Breastfeeding

	Maternal Consumption[a]		Adjusted OR[b] (95% CI)
	Cases	Controls	
Pregnancy	(n=202)	(n=201)	
Peanut	196/202 (97)	176/199 (88)	4.22 (1.57 -11.30)
Soy	94/194 (48)	62/197 (32)	1.88 (1.20 -2.94)
Nuts	169/199 (85)	157/201 (78)	1.36 (0.76 -2.41)
Eggs	196/200 (98)	199/201 (99)	0.56 (0.09-3.43)
Breastfeeding	(n=162)	(n=161)	
Peanut	154/162 (95)	124/157 (79)	2.28 (1.31 -3.97)
Soy	73/157 (46)	49/157 (31)	1.81 (1.08 - 3.03)
Nuts	132/160 (82)	110/159 (69)	1.50 (0.90 -2.52)
Eggs	159/161 (99)	155/160 (97)	1.27 (0.67-2.38)

Abbreviations: CI, confidence interval; OR, odds ratio.
[a]Data are presented as No. (%). The denominators are the total numbers for whom full information was available.
[b]Adjusted for age and sex of children, mother's level of education and smoking, presence of atopic diseases in the family, and duration of breastfeeding where relevant.

and found no significant differences between cases and controls for milk formula, cereals, peanuts, nuts, soy, or eggs.

The presence of peanuts and peanut-containing food at home was reported significantly less often in the case group. Reports of peanut exposure in daycare centers, however, was similar in both groups (with the exception of whole peanuts, which were significantly more common in the case group). Adjusting for this exposure, however, did not significantly change the risk estimate associated with the consumption of peanuts during pregnancy and breastfeeding. Daycare attendance was slightly higher in cases than in controls but the difference was not significant. (Table 5)

Table 4. Age[a] of Introduction of Various Foods in the Infant's Diet

	Cases	Controls
Milk formula	3.8 (2.7)	3.9 (3.2)
Solids	4.3 (1.8)	4.5 (1.6)
Peanuts	11.6 (2.5)	11.0 (2.6)
Nuts	10.8 (2.1)	11.8 (2.2)
Soy	7.6 (3.6)	7.3 (3.4)
Eggs	9.1 (2.4)	8.9 (2.4)

[a]Expressed as mean (SD) age in months.

Table 5. Presence of Peanut Products in the Environment

	Cases (%)	Controls (%)	*P*
Home	(n=202)	(n=201)	
Peanut butter	81	91	.002
Peanuts in chocolate	42	60	<.001
Peanuts in cereals	52	62	.027
Peanuts in cookies	43	60	<.001
Whole peanuts	27	35	.08
Daycare Center	(n=117, 58%)	(n=99, 49%)	.08
Peanut butter	47	46	.85
Peanuts in chocolate	18	14	.47
Peanuts in cereals	32	27	.56
Peanuts in cookies	38	37	.90
Whole peanuts	15	2	.006

Discussion

This study shows that early exposure to peanut proteins in human breast milk or in utero is a potential risk factor for the development of peanut allergy. Breastfeeding itself, however, does not appear to be a relevant risk factor. The results of this study support earlier recommendations that pregnant women and breastfeeding mothers with a family history of atopy should avoid eating peanut products in order to potentially reduce the number of children who develop peanut allergy [10]. However, these recommendations were withdrawn in 2007 because they were not supported by evidence-based data [11].

In this study, breastfeeding did not appear to be in itself a risk factor for the development of peanut allergy as a high rate of breastfeeding (80%) and similar breastfeeding durations were observed in both groups. Eating habits during pregnancy and breastfeeding, however, seem to be of the

highest importance. Our data show that eating peanuts during these periods was associated with the development of peanut allergy in offspring. It is well known that infants can develop an allergic reaction to food allergens passed through human milk [12] and there have been reports of peanut allergens being transmitted through human milk [7]. It thus seems plausible that early exposure to low levels of peanut allergen in human breast milk could be a significant risk factor for the development of peanut allergy in an atopy-prone population. In a murine model of oral tolerance, very low doses of potential allergens given orally have been shown to prime for subsequent systemic reactions rather than induce oral tolerance [13,14]. Lack et al [15] did not find a significant association between maternal diet and the development of peanut allergy in offspring. However, the study had low power since it included only 36 children with peanut allergy. In that study, as in ours, the maternal dietary questionnaire was administrated after the children had been diagnosed. In a more recent publication, Fox et al [16] found that maternal peanut consumption during pregnancy and breastfeeding significantly increased the risk of having a peanut-allergic child. However, when the analysis was adjusted for household environmental exposure to peanut-protein products, maternal consumption lost its significance. It is important to underscore that the study by Fox et al was limited to peanut-allergic children that presented with eczema as the predominant manifestation. A murine model has clearly shown that the loss of integrity of the skin as a barrier, as in eczema, is a risk factor for sensitization through environmental exposure [17]. Therefore, inferences from this study are probably not readily applicable to children without eczema. It is probably safe to say that the development of peanut allergy is the result of many potential factors related to genetics, the environment, and immunology that act simultaneously and possibly synergistically [18].

Maternal consumption of soy products during pregnancy and breastfeeding also seemed to be a risk factor in our group. Because soy-protein fractions are known to be homologous to major peanut proteins, cross-sensitization between soy and peanuts cannot be ruled out. Lack et al [15] reported an association between peanut allergy and intake of soy in infants (OR, 2.6 [95% CI, 1.3 to 5.2]) [15]. In our study, we found no such association but earlier exposure to soy proteins might have increased the immunologic cross-sensitization with peanuts in infants from high-risk families.

As discussed previously, the possibility that skin exposure to traces of peanut butter could be another risk factor for peanut allergy has been raised recently in the literature [15,16]. In our study, similar rates of exposure to peanuts in daycare centers were reported by both groups. In comparison, peanut exposure at home was significantly reduced in cases compared to controls but this could be because parents eliminated peanut-containing products immediately after they suspected the allergy (ie, before inclusion in the study). Moreover, maternal peanut butter consumption was similar in both groups. Overall, the cases in our population did not seem to be more exposed than the controls to peanut products in their environment. Although skin exposure is reported to be a risk factor for peanut allergy, other factors such as maternal peanut consumption during pregnancy and breastfeeding seem to play a role. Finally, the possibility of peanut sensitization upon the introduction of peanuts into the child's diet cannot be excluded as 25% of the cases had their first reaction after more than 1 exposure to peanuts in their diet.

Despite the attractiveness of the skin sensitization hypothesis, it does not explain the increased risk associated with the consumption of peanuts during pregnancy. Interestingly, Frank et al 19] observed similar results in a smaller sample of peanut-allergic children. They found that mothers who consumed peanuts more than once a week during pregnancy were more likely to have a peanut-allergic child (OR, 3.97 [98% CI, 0.73-24]). Based on this study and our own results showing that the consumption of peanuts during pregnancy and not breastfeeding itself, increases risk, it would seem that in utero sensitization could occur. Food proteins, for example, have been detected in the amniotic fluid of pregnant rats [20] and ß-lactoglobulin and ovalbumin proteins have been detected in both cord blood and placental tissue [8]. As the fetus might be in contact with food protein, early sensitization of the fetus to peanut allergens may be responsible for the development of allergy to peanuts during infancy.

We deliberately selected a group of high-risk cases based on their clinical history. As expected, the reported prevalence of atopic diseases was higher in their families than in those of the controls, although a substantial proportion of controls also had a genetic predisposition. Because of the high prevalence of family history in the controls, some might have been atopic but not yet developed clinical symptoms. Nonetheless, all of those tested were negative for peanut extract. It would, however, be interesting to repeat this study with a control group of atopic infants without peanut allergy in order to better distinguish between the impact of early environmental exposure and that of genetic predisposition on the development of peanut allergy.

The selection of a case-control design as opposed to a prospective cohort study was based on feasibility and efficiency. Differential recall between cases and controls cannot be excluded, although it would have to be quite marked to change the main conclusion of the study. The reported distribution of variables related to peanut exposure such as breastfeeding and duration of breastfeeding between cases and controls was not suggestive of such a bias. Despite the known challenges associated with properly measuring exposure in a cohort study, and the costs associated with enrollment of large enough numbers to generate a sufficient number of cases, it may be necessary to carry out a study where exposure is measured prospectively to confirm the findings of this study.

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