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Executive Summary

Background

It is scientifically uncertain whether total avoidance of an allergen prevents or promotes sensitisation. Experiments with animal models suggest that high dose exposure can lead to tolerance (no adverse reaction to the food) and low dose exposure may lead to sensitisation - a potentially harmful outcome (Strobel, 2002). Clinical experience suggests that total avoidance of peanut is very difficult to achieve(Hourihane, 1997, Sicherer, 2001), so mere avoidance of obvious peanut may simply change exposure from "high" to "moderate or low" exposure. The UK prevalence of sensitisation to peanut more than doubled from 1.3% to 3.3% in 2 single centre birth cohorts born in 1989-90 and 1994-1996 (Tariq, 1996, Grundy 2002). The 1998 COT report advised peanut avoidance by infants and pregnant or lactating mothers, aimed at atopic families. This precautionary advice was issued due to uncertainty about the link between maternal peanut consumption and peanut allergy and it was considered unwise to discount possible sensitisation of offspring resulting from maternal exposure to allergen. It is not known what effect this advice may have had on the dietary habits of atopic and non-atopic mothers, or if its intended effect on the prevalence of peanut sensitisation in their offspring has been realised.

Rationale and objectives

Between 2003-2005 we studied children born between March 1999 (9 months after the COT report) and March 2000. The two basic aspects of the study - did mothers pay any attention to the COT report and did their behaviour affect the prevalence of peanut allergy in children - are key questions for the FSA trying to assess, retrospectively, the impact of the COT report and of any future strategies for affecting the dietary habits of nursing mothers.

Approach

Mothers were approached via schools and they and their child were recruited with written informed personal and parental consent. The mothers were asked in personal interviews about their own and their family's allergic conditions, their recall of the COT advice and their own peanut consumption during pregnancy and breastfeeding Children were evaluated for maternal recall of their peanut consumption in infancy, current or resolved allergic conditions and for sensitisation to common food and aeroallergens. All children with positive screening skin prick tests (SPTs) to peanut were offered a formal double-blind, placebo-controlled food challenge with peanut to confirm the diagnosis.

Outcome/ Key results obtained

The study was completed in August 2005. 1072 mother-child pairs were studied. 653 mothers (61%) recalled hearing the COT advice about peanut avoidance. Parental (mother and father) atopy rates did not affect this figure or the action taken on the advice. 376 (58%) mothers who recalled the advice changed their peanut intake while pregnant, but only 38 stopped eating all forms of peanut, which was the advice given. This figure represents 10% of those who changed their diet, 6% of those who recalled the advice and only 3.5% of the whole group. 328 (42%) mothers reduced their peanut consumption but did not eliminate peanut from their diet. The prevalence of peanut

allergy known to parents before the study, on the basis of clinical assessments, that may or may not have included formal challenges was 9/1072 = 0.8%. 29 children were found to have a positive SPT to peanut. 1 child was considered to be peanut allergic but his parent refused SPT; he had known peanut allergy and high peanut antibody levels. 30 children with positive SPT or high peanut -specific IgE gives a prevalence of peanut sensitisation of 30/1072 = 2.8% (95%CIs 1.8-3.7).The prevalence of known sesame allergy was 1/1072 = 0.1%. Four other children had positive SPT to sesame without a history of reactivity. All 5 sesame-sensitised children (0.5%) had positive SPT to peanut and 4 were considered peanut allergic. 4/30 subjects with positive SPT declined the offer of a food challenge. 6 challenges were negative. 20/1068 subjects were considered to have peanut allergy (15 by challenge and 5 by strong positive recent history and supportive skin and blood tests), giving a prevalence of peanut allergy of 1.8% (95%CIs 1.1-2.7) in this cohort of school entry children born in 1999-2000. These data represents a real increase compared to the Isle of Wight cohort of children born in 1989, before the COT report, where the prevalence of peanut allergy was 0.6%. We did not demonstrate a significant difference in the prevalence of peanut allergy in this group from the 1994-1996 cohort in the Isle of Wight. 5 children (0.5%) were found to be sensitised to sesame, by SPT but only one child (0.1%) reported sesame allergy. All 5 sesame allergic children were also sensitised to peanut

What it means and why it is important.

A majority of mothers recalled hearing the COT advice when they were pregnant. Only 3.5% recalled having adhered to the advice, for reasons that are not clear. Parental allergy status did not appear to have been an important factor in any decision taken about peanut consumption while pregnant or breast feeding.

The prevalence of peanut allergy seems to have increased despite the publication of the COT report. Sesame allergy is unusual in the UK population and all cases of sesame sensitisation were in children who were allergic to peanut and had other allergic conditions.

The prevalence of peanut allergy is continuing to increase in the 21st century. Government advice has not had a significant impact on this trend and may have been assimilated by the general population, rather than acted upon by the target group identified as at highest risk of having children who would develop allergies.

Glossary

Atopy Familial or personal tendency to produce IgE antibodies and to develop typical allergic symptoms such as asthma, eczema, or rhinitis.

DBPCFC Double-blind,placebo-controlled food challenge.

SPT Skin Prick Test

Aims and objectives of the investigation

Peanut allergy is a severe, occasionally life-threatening form of food allergy. It starts early in life and appears to persist in most cases. Management depends on awareness of potential encounters with the allergen and the appropriate provision of rescue medication, in the form of adrenaline (1). In addition to the medical consequences of peanut allergy, the consequent dietary and social restrictions can significantly impair the quality of life of those affected (2).

In June 1998 the UK Government's Chief Medical Officer's Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (known as COT) published a report on peanut allergy. This report's key finding was a recommendation that "pregnant women who are atopic [prone to develop allergies], or for whom the father or any sibling of the unborn child has an atopic disease, may wish to avoid eating peanuts and peanut products during pregnancy and breast feeding". Further, it was recommended that the infants themselves should not be exposed to these products until three years of age (3). No other government has issued such advice to families at high risk of having children that develop allergies. It has been a concern that the advice could possibly have adversely affected (increased) the prevalence of peanut allergy in the UK rather than decreasing the prevalence. Most mothers - both atopic and non-atopic - would not want their child to develop peanut allergy and it has been feared the COT advice may have been acted up on by non-high risk families, to whom the advice was not targeted.

It is scientifically uncertain whether total avoidance of an allergen prevents or promotes sensitisation. Experiments with animal models suggest that high dose exposure can lead to tolerance (no adverse reaction to the food) and low dose exposure may lead to sensitisation - a potentially harmful outcome (4). Clinical experience suggests that total avoidance of peanut is very difficult (5,6), so mere avoidance of obvious peanut may simply change exposure from "high" to "moderate" or "low" exposure. A longitudinal study of maternal egg avoidance has suggested that when atopic, pregnant women moved from high to moderate intake of egg, levels of protective antibody (IgG) to egg went down and the incidence of sensitisation (the presence of potentially harmful IgE antibodies to egg, as measured by skin prick tests [SPT]) was increased in their children at 6 months of age (7, 8). Since the COT advice about peanut was issued in

1998, medical opinion has changed and since 2004 avoidance diets in pregnancy are no longer advocated by allergists internationally as a means of primary prophylaxis of allergic disease but atopic mothers in the UK are currently still being advised to avoid peanut consumption during pregnancy as a precaution. Avoidance diets while breastfeeding are now only recommended if a breastfed child is showing symptoms of food-related diseases such as eczema (9).

The UK prevalence of sensitisation to peanut more than doubled from 1.3% to 3.3% in 2 single-centre birth cohorts born in 1989-90 and 1994-1996, born before the COT advice on peanut avoidance. The ages of the cohort at testing were slightly different, with the tests of 4-5 year olds being reported by Tariq (10) and 3-4 year olds by Grundy (11). The diagnosis was based only on history and SPT in the report by Tariq and only on history, SPT and open food challenge by Grundy.

We have measured the prevalence of sensitisation to peanut and sesame and of peanut allergy in the first school-entry cohort of children that were conceived after the date of publication of the COT report in June 1998, using the current gold standard test, the double-blind placebo controlled food challenge (12).

Experimental Procedures

Ethical approval

This study was approved by appropriate Local Research Ethics Committees (study reference numbers 062/03/t - Southampton and 03/SM/183 - Manchester).

Recruitment

Between September 2003 and August 2005 we studied children born between March 1999 (9 months after the COT report) and March 2000. Families were recruited through primary schools in Southampton and Manchester, UK. These cities were chosen due to the local availability of integrated, complete paediatric allergy services. Information sheets about the study were sent home from school with the children and research staff attended the schools to recruit interested families directly.

Questionnaire

At an arranged appointment in the school a questionnaire was administered to the child's mother (mothers exclusively were recruited) face to face, by the same research staff member, 1 only in each city. Answers were entered directly onto a laptop computer using SPSS data entry software (SPSS, Illinois, USA). Data were collected about family structure, maternal and paternal allergic conditions and smoking habits and maternal recall of the COT advice. Mothers were asked to recall their consumption, while pregnant and breastfeeding the index child, of explicit foods known to them or to the research team to contain peanut or peanut products. Children were evaluated by maternal recall of the child's peanut consumption in infancy and of current or resolved allergic conditions.

Allergy testing of children

- Skin prick testing

Children were evaluated for sensitisation to common food and aeroallergens, using skin prick testing (SPTs). Skin prick tests were performed with single-tine lancets, pricking the skin at an angle of 90 degrees. The wheal diameter, recorded at 15 minutes, was the mean of 2 perpendicular diameters. SPT for peanut was considered positive if the wheal was ≥ 3mm, in the

presence of a negative control (saline) and at least a 3mm weal to histamine (1:10w/v). Skin test reagents were from ALK-Abello, Hungerford, UK. All children with positive screening SPTs to peanut were offered a blood sample for peanut specific IgE, performed in each centre using the Immunocap© system (Phadia, Uppsala, Sweden), according to manufacturer's instructions.

- Food challenge

Formal double-blind, placebo-controlled food challenge (DBPCFC) with peanut was used to confirm the diagnosis, with written parental consent.

Exclusion criteria from DBPCFC were:

- Parental refusal
- An allergic reaction to peanut had occurred recently (within 1 year)

and skin and specific IGE measurements suggested the presence of peanut allergy (12**).**

Peanut challenges were performed using identical protocols in each centre, using peanut flour-based biscuits, prepared in Southampton by an experienced dietitian (KECG), for use in both units. Children were admitted as day cases to the Children's Day Ward in Manchester or the Wellcome Trust Clinical Research Facility in Southampton. The doses of peanut protein administered at 30 minute intervals were, 1mg, 10mg, 100mg 1g, 5g. The end point of the challenge was the identification of an objective allergic reaction with clinical signs or completion of the full challenge with no such signs up to 2 hours after the last dose (13).

Statistical analysis

Data was analysed using SPSS software (version 11.0, SPSS Corporation, Illinois, USA). The sample size of 1000 was calculated using a two sided 95% confidence interval, based on the large sample approximation for the difference between a previous study proportion of 0.616% (6/981 cases, ref 10) and a new study proportion of 1.6% (16 cases/1000) extending 0.919% from the observed difference in proportions when the sample sizes are 981 and 1000 respectively. Descriptive analyses were performed with a p value of ≤ 0.05 considered significant. Categorical data were analysed using a Chi squared or Fisher's exact test as appropriate. Univariate and multivariate logistic regression were used to explore associations between independent variables and outcome variables. Separate univariate and multivariate models were built using: 1) Maternal

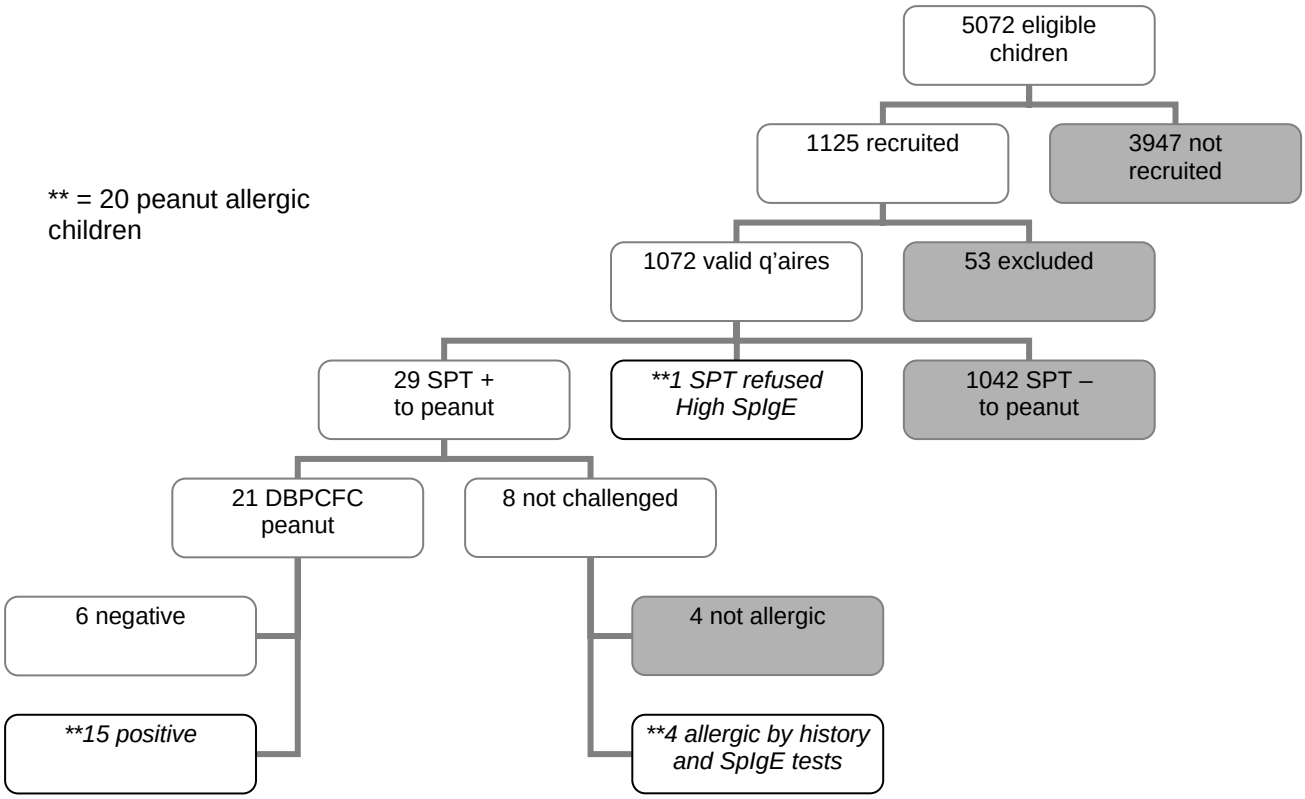
diet change (Yes/No) and 2) Peanut allergy status (Yes/No) as the 2 outcome variables. Logistic regression analysis was used to analyse associations between variables, and the relevant weight of each predictor. Variables related to maternal diet change and disease status were assessed by using a hierarchical block entry protocol. All variables significant at $p< 0.1$ were considered for inclusion in the final models. Multiple logistic regression analysis was used to simultaneously adjust for the various independent variables. Independent variables included in the analysis were selected based on selected chi-square analysis and risk factors discussed in relevant literature. The small sample size (events of interest, in this study change of maternal diet and the prevalence of peanut sensitisation and allergy) did not allow for the inclusion of a large number of independent variables in the logistic regression, due to risk of 'overfitting'.

Results

Recruitment

Families were recruited in 41 schools in Southampton and 73 schools in Manchester. In Southampton 477 of 1785 eligible families were recruited (26%). In Manchester 648 out of 3287 eligible families were recruited (19%, p = 0.001). The overall recruitment was 1125 families out of 5072 eligible families (22%). Thirty one families were excluded as SPT was refused or declined by the family, in nearly all cases (30/31) this was due to the child refusing SPT after questionnaire data had been collected. Twenty one families were excluded because the families had not been resident in the UK at the time of the COT report or the child had been born outside the UK. The final total of eligible families studied was 1072 (Figure 1).

Figure 1 Flow chart for the study



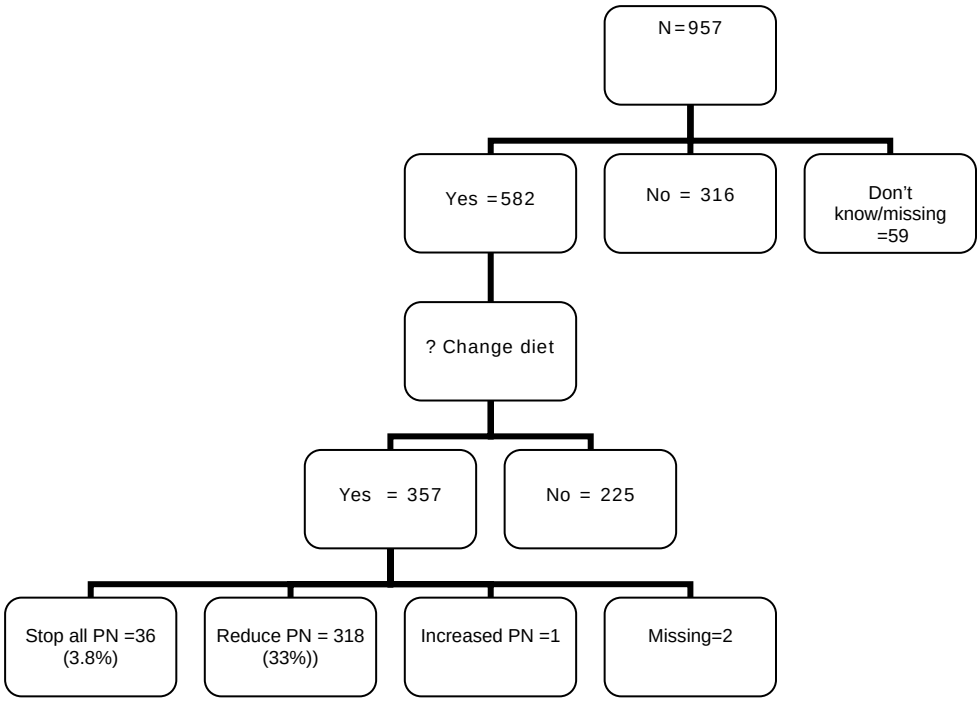
Maternal recall of peanut consumption while pregnant or breastfeeding.

Data regarding maternal and maternally-reported paternal atopic conditions are shown in Table 1. Maternal recall of the COT advice and its effect on maternal peanut consumption is shown in Figure 2. Maternal atopy had no effect on recall of the COT advice or dietary changes made on its recommendations (data not shown). Only 38 mothers (10% of those who changed their diet, 6% of those who recalled the advice and only 3.5% of the whole group) followed the COT advice by stopping consuming peanut while pregnant. Mothers who exclusively breast fed their babies reported a lower consumption of any peanut while breastfeeding (267/670, 40%) than while they had been pregnant (370/670, 55%, $p < 0.001$). Atopic and non-atopic mothers stopped eating peanut in almost equal number, 18 and 20 respectively. 328 (42%) mothers reduced their peanut consumption but did not eliminate peanut from their diet.

Table 1 Family data (n= 1072)

Mother atopic	476 (45%)
Father atopic (by maternal report)	400 (38%)
Median number of children	2 (1-11)
Birth order of screened child	1 (median)
Consumed peanut while pregnant	611 (57%)
Consumed peanut while breastfeeding	294 (27%)

Figure 2
Maternal recall of the COT advice, excl those who never ate peanut n =111 (percentages in brackets)



Logistic regression analysis

To assess the associations between putative risk factors (maternal atopy; paternal atopy; no. of children in the family; birth order of the atopic child; how often the mother ate peanuts/peanut containing products before pregnancy) and the likelihood of a mother eliminating or reducing peanut consumption during pregnancy and breastfeeding, the presence or absence of compliance with advice (Yes/No) was evaluated as the dependent variable in a logistic regression model. The estimated odds ratios (ORs) with 95% confidence intervals (C Is) for associations between independent risk factors and maternal diet change are presented in Table 2.

Birth order and paternal atopy were associated with maternal diet change. Maternal atopy did not show a significant association. Paternal atopy was significantly associated with maternal diet change even after adjusting for birth order. Mothers with more than one child were a third less likely to eliminate or reduce peanuts/peanut containing products from their diets during pregnancy and breastfeeding. In cases where father was found to be atopic, the odds of mothers

changing their diets were a third greater than when the father was not atopic. The significant association of paternal atopy was no longer evident when adjusted for the effect of siblings with a prior history of allergic disease and a mother who had never consumed peanuts. No other significant associations were found.

Table 2 Children's consumption of peanut and sesame (by maternal report)

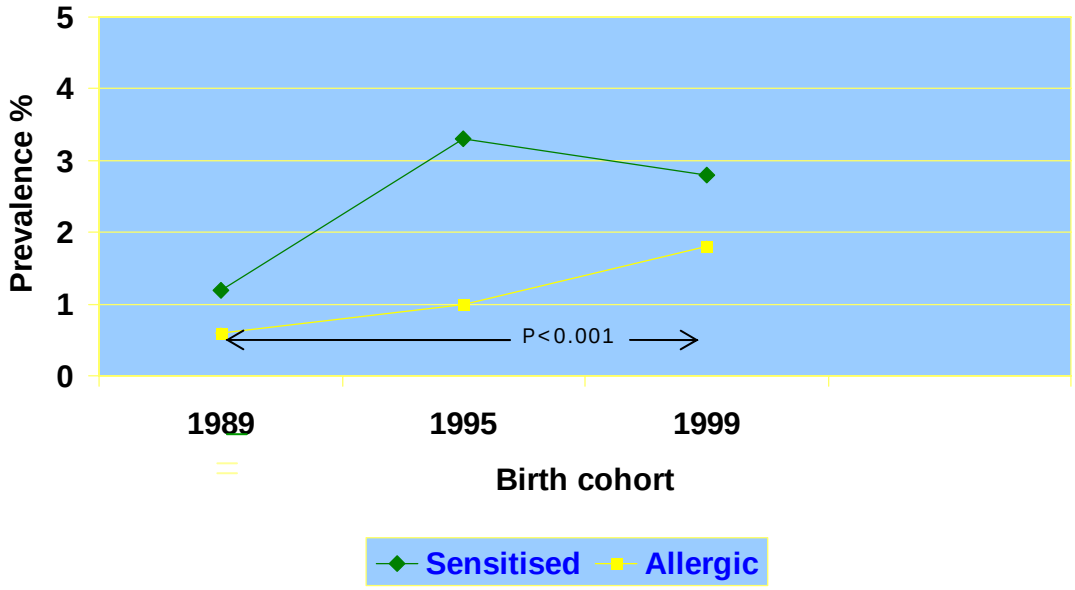
	Peanut	**Sesame**
Eaten	**695 (65%)**	**725 (67%)**
Never eaten	**345 (32%)**	**327 (30%)**
Don't know	**32 (3%)**	**19 (2%)**
Median age of introduction	**36 months (4-62)**	**Not asked**

Peanut sensitisation of children

197 children (18%) had one or more positive skin prick tests to the panel of allergens tested. 695 children (65%) were reported by their mothers to have consumed peanut (Table 2). The mean age of introduction of peanut was 36 months, which is much later than the 12.6 months reported for the first Isle of Wight cohort (Tariq, 1996). There was no difference in the age at which peanut was introduced to the diet of those who became peanut allergic and those who tolerated peanut (data not shown).

The prevalence of previously known peanut allergy was 9/1072 = 0.8%. 29 children were found to have a positive SPT to peanut. 1 child was considered to be peanut allergic but his parent refused SPT; he had known peanut allergy and high peanut antibody levels (>100 KUa/L). 30 children with positive SPT or high peanut-specific IgE gives a prevalence of peanut sensitisation of 30/1072 = 2.8% (95% CIs 1.8-3.8%), Figure 2 and figure 3. Of the 30 sensitised children, 12 mothers recalled changing their peanut consumption when pregnant.

Figure 3 Peanut allergy in 3-5 year old children in UK 1989-2005



8/29 subjects declined the offer of a food challenge. 4 of these were considered likely to have peanut allergy according to the agreed criteria outlined above and are considered allergic, 4 children who refused challenge were considered uncertain to be peanut allergic and have not been included in the estimate of prevalence (Figure 3). 6 challenges were negative, 15 were positive. 20/1072 subjects were considered to have peanut allergy (15 by challenge and 5 by strong positive recent history and supportive skin and blood tests), giving a prevalence of peanut allergy of 1.8% (95% CIs 1.1.-2.7%) in this cohort of school entry children born in 1999-2000, Figure 3). These data represent a real increase in prevalence compared to the Isle of Wight cohort of children born in 1989, before the COT report.

Of the twenty peanut allergic children 9 had mothers who were atopic and 10 had mothers who were not atopic, 1 mother's atopy status is not recorded. Mothers of 8 (40%) of the 20 peanut allergic children recalled changing their peanut consumption while pregnant, though only 1 completely stopped eating peanut. 5 of these eight were atopic mothers, and only 2 of the 10 non atopic mothers changed their diet, the remaining "changer" was of unknown atopic status, so

it appears that the avoidance (or consumption) of peanut during pregnancy had no effect on the prevalence of peanut allergy in either atopic or non atopic mothers. Children shown to have peanut allergy were more likely than non–peanut allergic children to report either "ever" or "current" asthma, eczema and rhinitis (data not shown).

A history of eczema was found to be a significant predictor of peanut allergy (table 2). Children diagnosed with peanut allergy were 7.4 times more likely to have symptoms of eczema. Type of feeding was also an important risk factor for peanut allergy. Feeding type was significantly associated (.05) with peanut allergy; children who were breast fed were 3.8 times more likely to be diagnosed as peanut allergic than those who were bottle fed. When this association was adjusted for the presence of eczema, the effect became diluted (Table 3). No other significant associations were found.

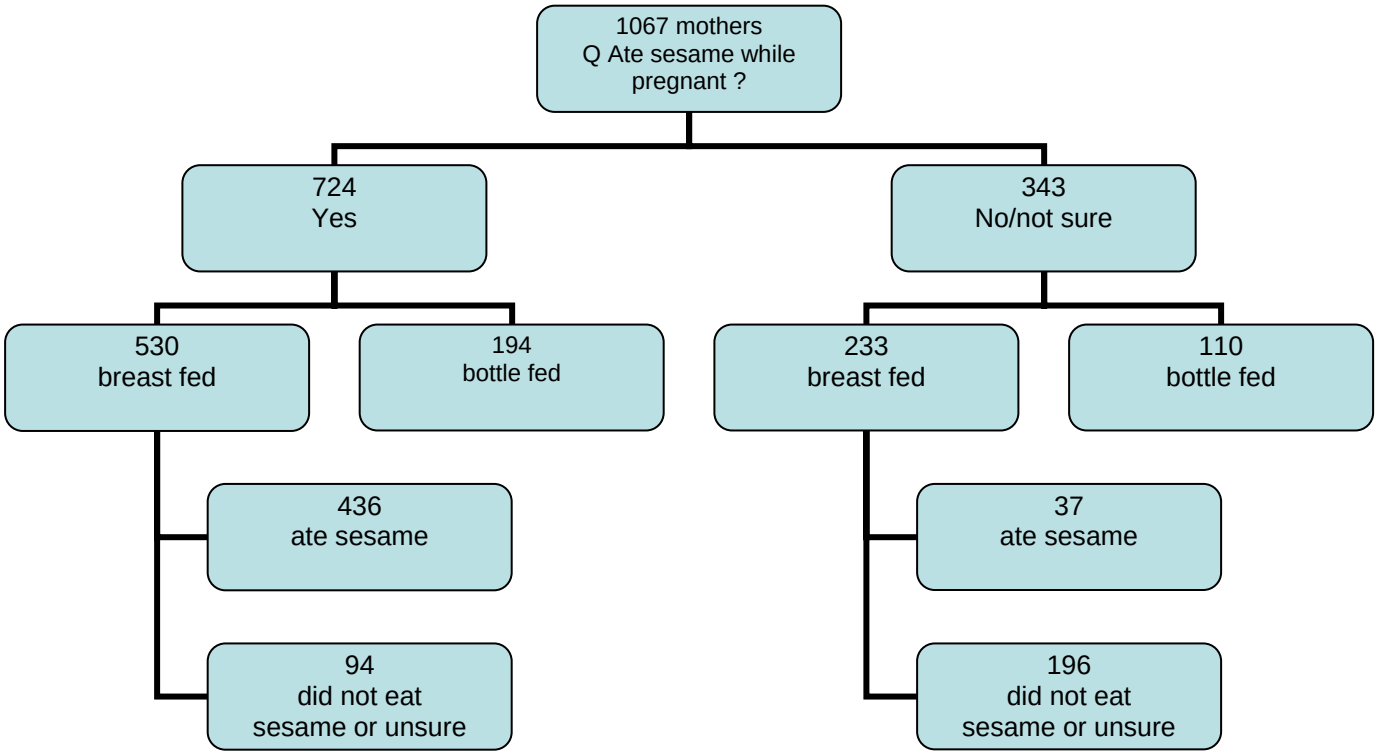
Table 3
Odd ratios for the 2 major outcomes, change of maternal diet and confirmed peanut allergy in index child

	B	Odds ratio	CI	P
1.Maternal diet change (Yes/ no)				
Birth Order	-0.454	.635	.543-.743	.00
Paternal Atopy	0.287	1.332	1.029-1.724	.02
2. Peanut allergy in child (Yes/ no)				
Breast feeding	1.15	3.16	1.00-10.8	.06
History of Eczema	1.95	7.05	2.05-24.25	.00

Sesame

Sesame was consumed frequently while pregnant but not while breastfeeding (figure 4). The prevalence of known sesame allergy was 1/1072 = 0.1%. This child had a positive SPT to sesame. 4 other children had positive SPT to sesame without a history of reactivity. All 5 sesame-sensitised children (0.5%) had positive SPT to peanut and 4 were considered peanut allergic. Therefore isolated sesame allergy was not found in this population.

Figure 4 Maternal consumption of sesame while pregnant or breast feeding



Discussion

We have demonstrated that there has been a real increase in the prevalence of peanut sensitisation and peanut allergy in the UK in the last 10 years. We have not been able to ascertain any positive or negative effect on the prevalence of the COT advice to atopic mothers about their own and their child's consumption of peanut. All cases of sesame sensitisation or sesame allergy were in children known or found to be sensitised to peanut. This reflects the findings of previous FSA funded research involving members of the UK Anaphylaxis Campaign (14).

It appears that a majority of mothers recalled hearing the COT advice when they were pregnant but only 6% appear to have adhered to the advice, for reasons that are not clear. Maternal allergy status does not appear to have been an important factor in any decision taken about peanut consumption while pregnant or breast feeding, though interestingly birth order and paternal atopy had moderate effects. It is unclear why paternal atopy would have an effect, though minor, on maternal diet and maternal atopy did not. The hoped-for effect on maternal diet in atopic mothers did not materialise and again we cannot say for sure at this stage why this might have been so. It appears that there was minimal uptake among all mothers, so any preferential uptake by the intended group (atopic mothers) may have been masked by the uptake in the "control" group of non atopic mothers. Government advice appears not to have had a significant impact on mothers' diets and was assimilated by the general population, rather than acted upon by the target group identified as at highest risk of having children who would develop allergies.

It appears that peanut has been introduced to the diet of this group of patients at a much later age than reported for the 1989 Isle of Wight birth cohort (10) but this has not affected the prevalence of peanut allergy.

This study complements a parallel study with slightly different methodology, performed on the Isle of Wight, where a whole population of slightly younger children were asked the identical questions about the COT report (Dean et al, submitted). The

prevalence of peanut sensitisation in the Isle of Wight group was 1.5% (0.8% -2.6%) but no formal challenges were performed in this slightly younger age group. With two parallel studies' findings being so similar, it is likely the findings are robust.

Our study also confirms that peanut allergy is clearly associated with other allergic diseases, with a particular association with eczema, which also persisted in logistic regression analysis. As eczema and food allergy are early steps on the allergic march, this is an expected finding.

Sesame allergy is apparently uncommon in the general population, with a single strongly suspected case and 4 sesame sensitised children. It is interesting to note that sesame consumption also changed between pregnancy and lactation (figure 4), though no advice was issued to the public about sesame avoidance or consumption.

Our study has limitations that must be acknowledged. Obviously there is potential for recall bias as mothers were asked to remember their dietary intake of peanut while pregnant between 5 and 6 years ago. The opportunity to prospectively follow up a cohort from birth was not available at the time of COT. However it is also possible that prospective follow up may have influenced dietary habits. Only 25% of eligible children were recruited in the two sites so our estimates of prevalence could conceivably be an over- or under-estimate. Checking our clinic records and prospectively seeking eligible children coming to clinic did not identify existing cases that we were not capturing, so our figures are unlikely to be an underestimate, especially considering local referral practices in each area, with identified pathways to the relevant clinic being well established. Families may have been reluctant to subject their children who might have allergies to testing, particularly in a school setting. Our clinical experience suggests this is extremely unlikely with many such families being characteristically very highly motivated to seek expert assistance for their children with allergies.

The difference in proportions of eligible families recruited in each site is a conundrum and may simply reflect differing recruitment practices of the nurses involved, despite the strategies for recruitment being intended to be identical in each city. It also reflects the fact that recruitment varied between schools from 5-70% of eligible children in both cities.

18% of children had one or more positive SPTs, which is consistent with recent data from slightly older children in the UK (15) suggesting our cohort is representative of the UK population of 4-5 year olds.

In conclusion it appears that the COT advice on peanut allergen avoidance by pregnant women and breastfeeding mothers has not affected the prevalence on peanut allergy in children at school entry. It is possible that it has affected the age of introduction of peanut to the diet of children but this has not had any discernible effect on the prevalence of peanut allergy either. This study's primary finding of a prevalence of peanut allergy of 1.8% is the highest recorded prevalence to date.

Acknowledgements

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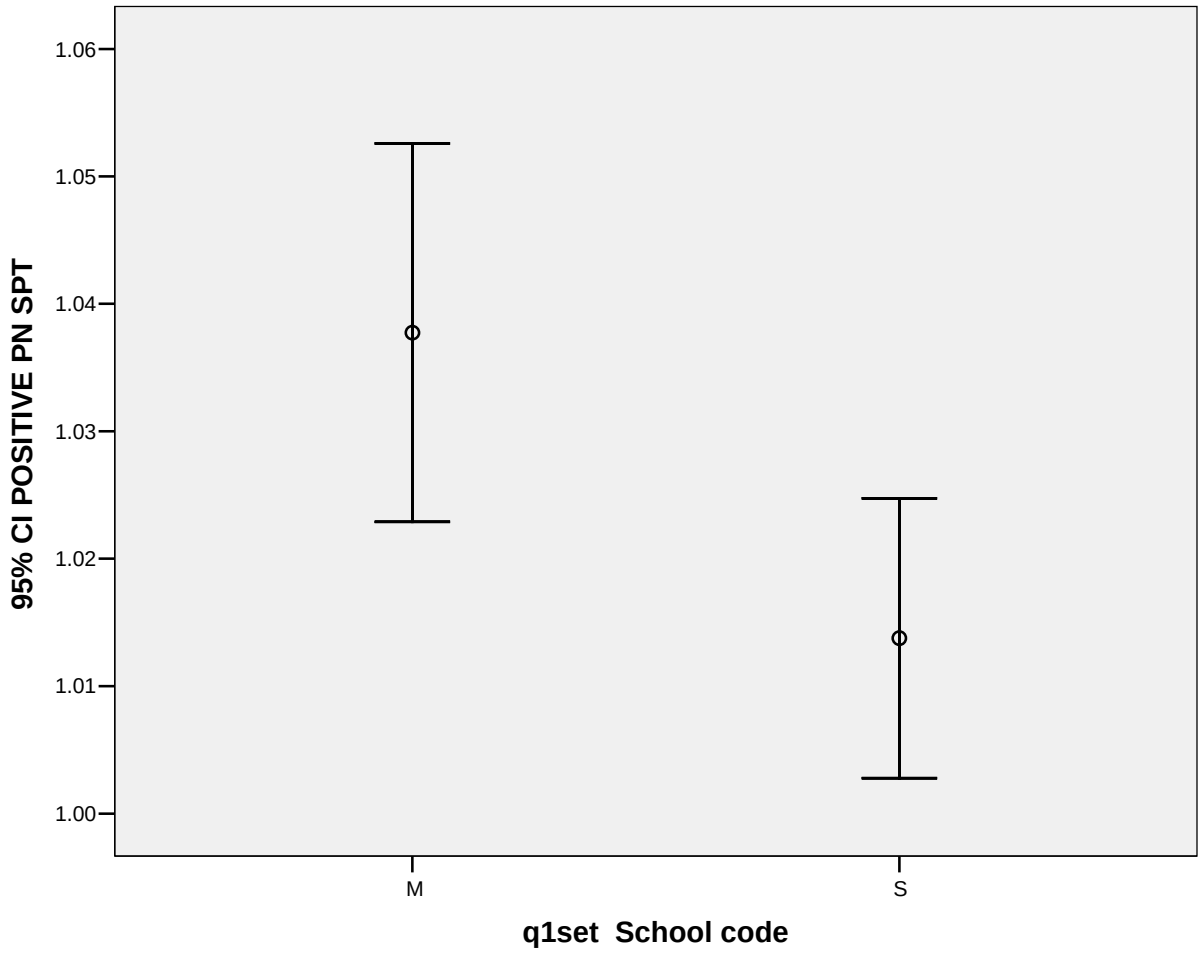
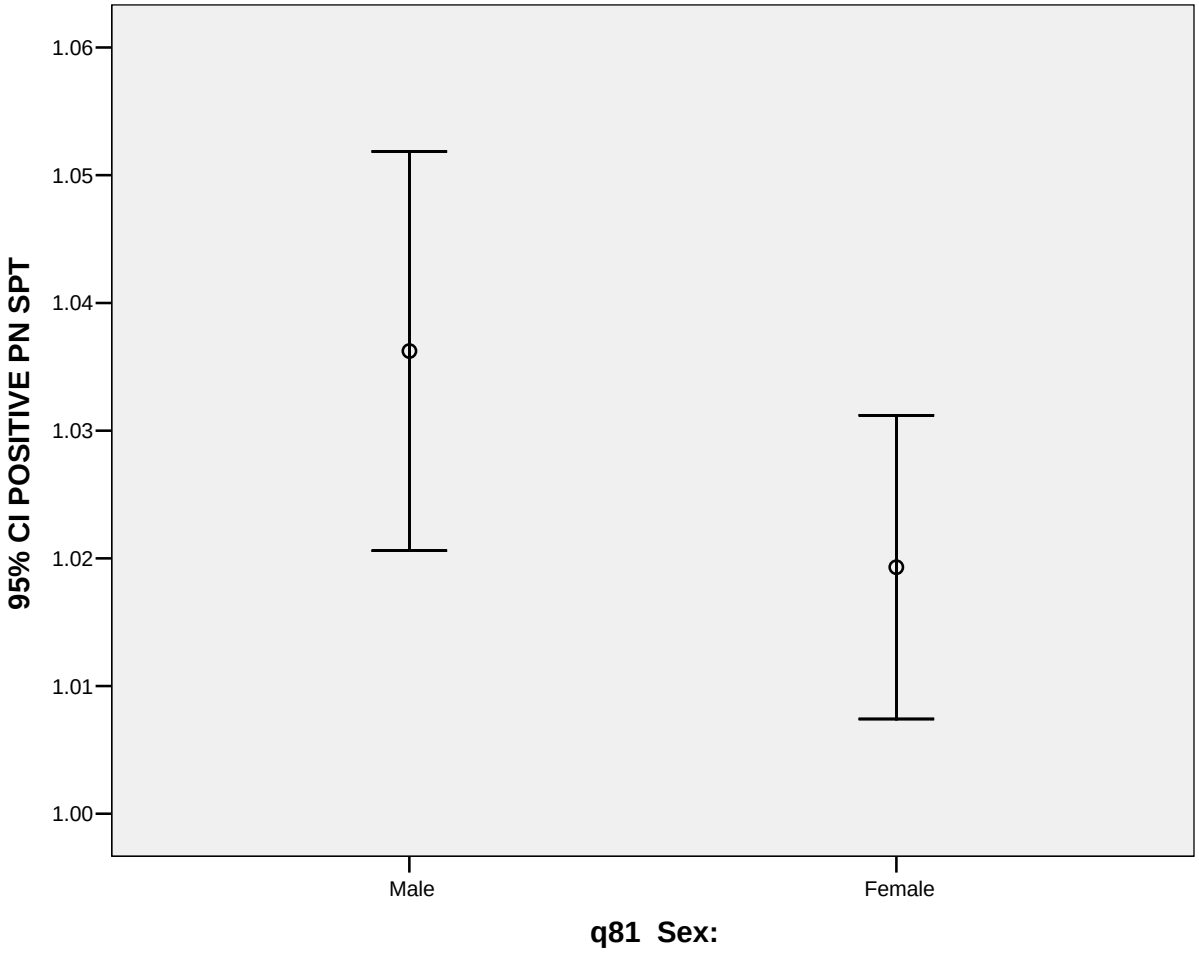
Appendix
Samples of primary SPSS figures and tables

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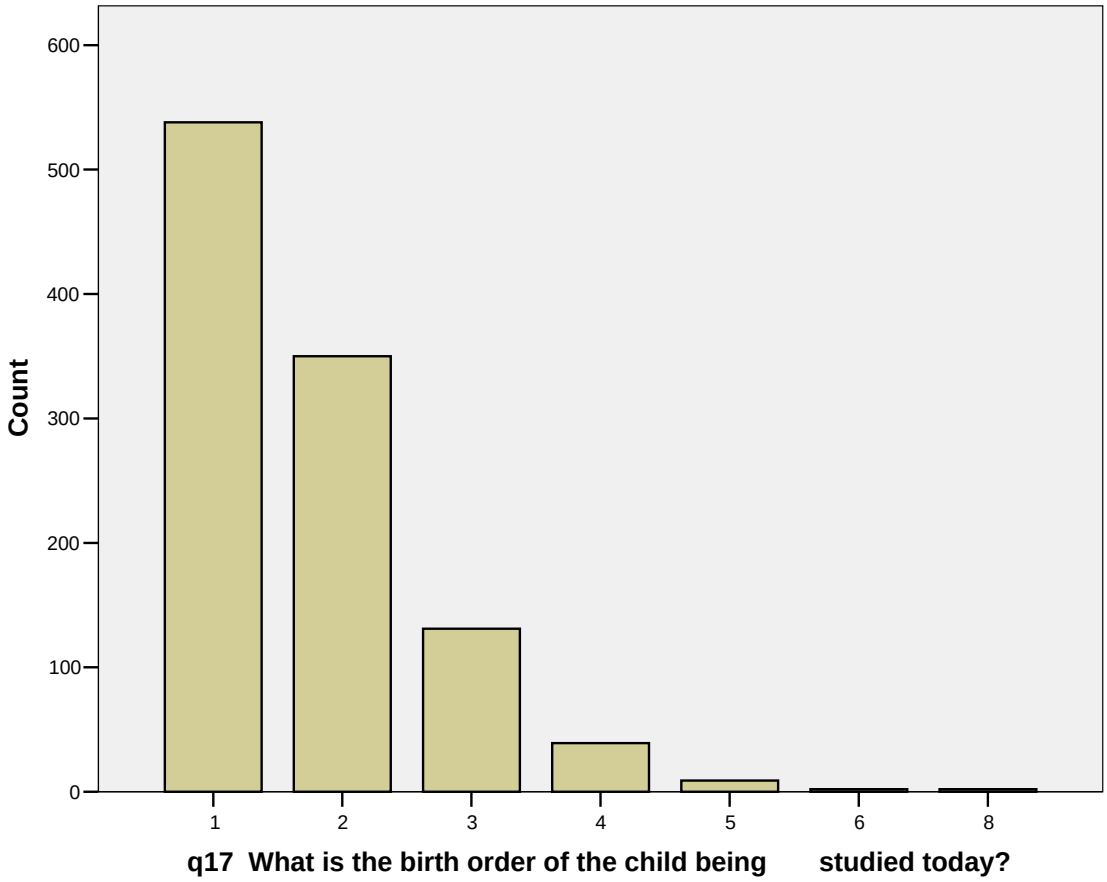
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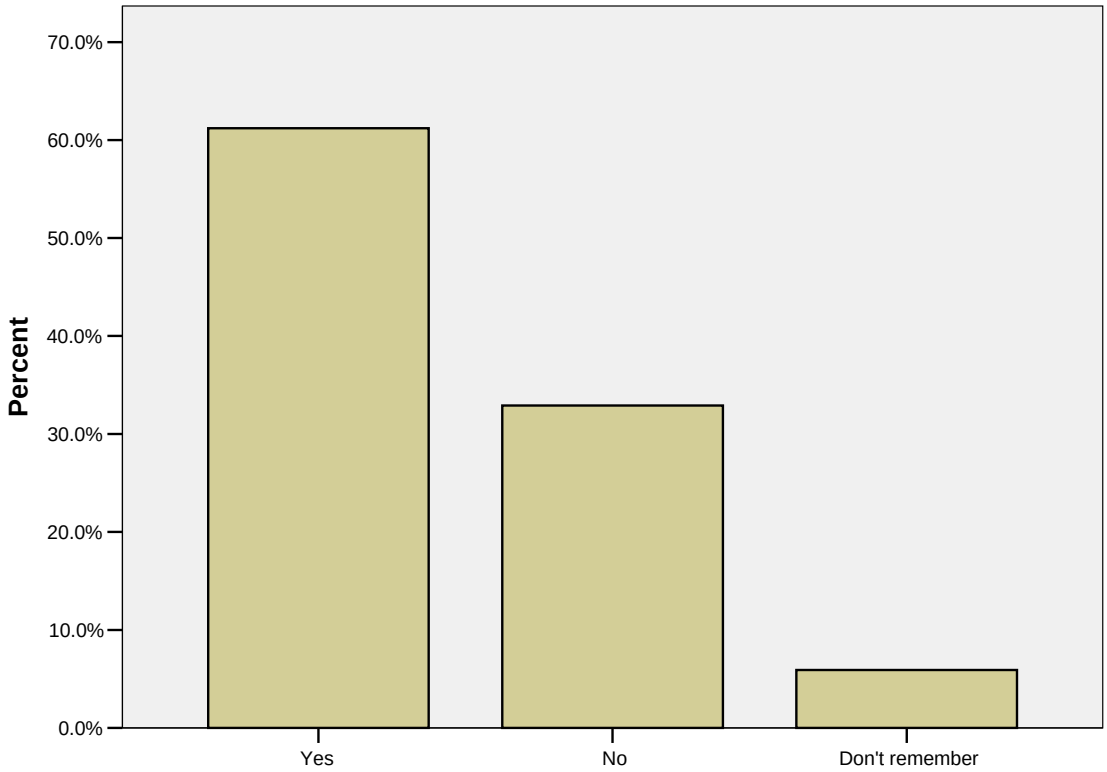
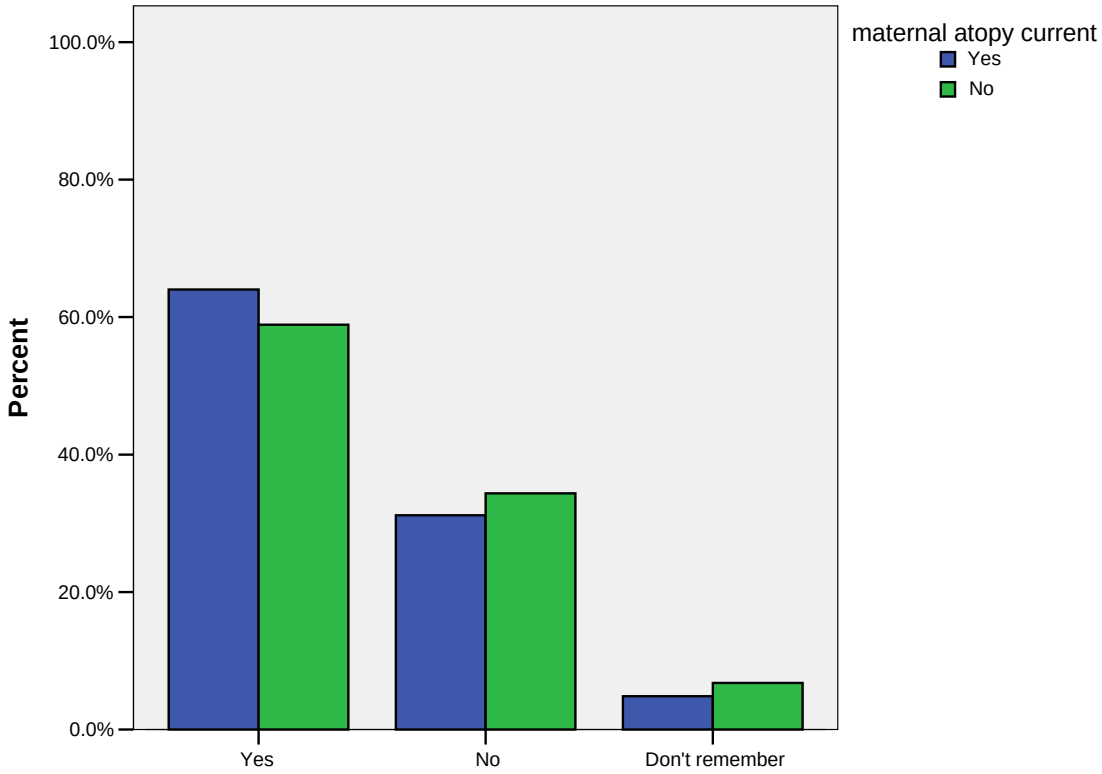
q17 What is the birth order of the child being studied today?



q31 The government issued advice in 1998 about eating peanuts whilst pregnant and breastfeeding. Do you remember hearing about that at the time?



q31 The government issued advice in 1998 about eating peanuts whilst pregnant and breastfeeding. Do you remember hearing about that at the time?

q29 What was your peanut consumption before you found out you were pregnant?

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Never ate peanuts	111	10.4	10.4	10.4
	Daily	22	2.1	2.1	12.5
	2-3 times per week	80	7.5	7.5	19.9
	Once a week	131	12.2	12.3	32.2
	Every two weeks	104	9.7	9.7	41.9
	Once a month	224	20.9	21.0	62.9
	Less than once a month	396	36.9	37.1	100.0
	Total	1068	99.6	100.0	
Missing	System	4	.4		
Total		1072	100.0		

q30 What was your peanut consumption before your antenatal booking visit?

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Never ate peanuts	496	46.3	46.4	46.4
	Daily	19	1.8	1.8	48.2
	2-3 times per week	39	3.6	3.7	51.9
	Once a week	71	6.6	6.6	58.5
	Every two weeks	61	5.7	5.7	64.2
	Once a month	120	11.2	11.2	75.5
	Less than once a month	262	24.4	24.5	100.0
	Total	1068	99.6	100.0	
Missing	System	4	.4		
Total		1072	100.0		

q32set GP

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Yes	99	9.2	9.3	9.3
	No	962	89.7	90.7	100.0
	Total	1061	99.0	100.0	
Missing	System	11	1.0		
Total		1072	100.0		

q32set Midwife

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Yes	400	37.3	37.6	37.6
	No	664	61.9	62.4	100.0
	Total	1064	99.3	100.0	
Missing	System	8	.7		
Total		1072	100.0		

q32set Health Visitor

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Yes	56	5.2	5.3	5.3
	No	1005	93.8	94.7	100.0
	Total	1061	99.0	100.0	
Missing	System	11	1.0		
Total		1072	100.0		

q32set Dietitian

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Yes	5	.5	.5	.5
	No	1056	98.5	99.5	100.0
	Total	1061	99.0	100.0	
Missing	System	11	1.0		
Total		1072	100.0		

q34 Did you change your diet on the basis of this advice?

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Yes	407	38.0	38.3	38.3
	No	657	61.3	61.7	100.0
	Total	1064	99.3	100.0	
Missing	System	8	.7		
Total		1072	100.0		

q35 If you changed your diet did you:

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	N/A	660	61.6	62.0	62.0
	Stop eating peanuts completely? Including those with a 'may	41	3.8	3.8	65.8
	Stop eating obvious peanuts but continued eating foods that	220	20.5	20.7	86.5
	Stop eating peanuts and foods containing peanut as an ingred	59	5.5	5.5	92.0
	Stop eating peanuts but not as an ingredient	83	7.7	7.8	99.8
	Increase your consumption of peanut?	2	.2	.2	100.0
	Total	1065	99.3	100.0	
Missing	System	7	.7		
Total		1072	100.0		

peanut allergy by challnge etc

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	yes	20	1.9	1.9	1.9
	no	1052	98.1	98.1	100.0
	Total	1072	100.0	100.0	

Case Processing Summary

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
peanut allergy by challnge etc * q87 Has this child ever had asthma?	1071	99.9%	1	.1%	1072	100.0%
peanut allergy by challnge etc * q91 Does he/she have eczema at the moment?	1071	99.9%	1	.1%	1072	100.0%
peanut allergy by challnge etc * q94 Does he/she have hayfever/rhinitis at the moment?	1063	99.2%	9	.8%	1072	100.0%
peanut allergy by challnge etc * q88 Does he/she have asthma at the moment?	1072	100.0%	0	.0%	1072	100.0%
peanut allergy by challnge etc * q90 Has your child ever had eczema?	1071	99.9%	1	.1%	1072	100.0%
peanut allergy by challnge etc * q93 Has your child ever had hayfever/rhinitis in the past?	1072	100.0%	0	.0%	1072	100.0%

peanut allergy by challnge etc * q87 Has this child ever had asthma?

Crosstab

Count

		q87 Has this child ever had asthma?			
		Yes	No	Don't know	Total
peanut allergy by challnge etc	yes	9	10	1	20
	no	197	822	32	1051
Total		206	832	33	1071

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	9.289[a]	2	.010
Likelihood Ratio	7.629	2	.022
Linear-by-Linear Association	5.876	1	.015
N of Valid Cases	1071		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is .62.

peanut allergy by challnge etc * q91 Does he/she have eczema at the moment?

Crosstab

Count

		q91 Does he/she have eczema at the moment?				
		N/A	Yes	No	Don't know	Total
peanut allergy by challnge etc	yes	2	10	8	0	20
	no	559	194	292	6	1051
Total		561	204	300	6	1071

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	18.399[a]	3	.000
Likelihood Ratio	18.723	3	.000
Linear-by-Linear Association	13.525	1	.000
N of Valid Cases	1071		

a. 2 cells (25.0%) have expected count less than 5. The minimum expected count is .11.

peanut allergy by challnge etc * q94 Does he/she have hayfever/rhinitis at the moment?

Crosstab

Count

		q94 Does he/she have hayfever/rhinitis at the moment?			
		Yes	No	Don't know	Total
peanut allergy by challnge etc	yes	3	14	3	20
	no	44	991	8	1043
Total		47	1005	11	1063

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	44.874[a]	2	.000
Likelihood Ratio	15.874	2	.000
Linear-by-Linear Association	.437	1	.508
N of Valid Cases	1063		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is .21.

peanut allergy by challnge etc * q88 Does he/she have asthma at the moment?

Crosstab

Count

		q88 Does he/she have asthma at the moment?				
		N/A	Yes	No	Don't know	Total
peanut allergy by challnge etc	yes	10	5	5	0	20
	no	820	83	145	4	1052
Total		830	88	150	4	1072

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	10.820[a]	3	.013
Likelihood Ratio	8.398	3	.038
Linear-by-Linear Association	8.234	1	.004
N of Valid Cases	1072		

a. 4 cells (50.0%) have expected count less than 5. The minimum expected count is .07.

peanut allergy by challnge etc * q90 Has your child ever had eczema?

Crosstab

Count

		q90 Has your child ever had eczema?			
		Yes	No	Don't know	Total
peanut allergy by challnge etc	yes	17	2	1	20
	no	457	577	17	1051
Total		474	579	18	1071

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	16.299[a]	2	.000
Likelihood Ratio	17.924	2	.000
Linear-by-Linear Association	10.254	1	.001
N of Valid Cases	1071		

a. 1 cells (16.7%) have expected count less than 5. The minimum expected count is .34.

peanut allergy by challnge etc * q93 Has your child ever had hayfever/rhinitis in the past?

Crosstab

Count

		q93 Has your child ever had hayfever/rhinitis in the past?			
		Yes	No	Don't know	Total
peanut allergy by challnge etc	yes	5	10	5	20
	no	99	905	48	1052
Total		104	915	53	1072

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	24.482[a]	2	.000
Likelihood Ratio	15.443	2	.000
Linear-by-Linear Association	.320	1	.572
N of Valid Cases	1072		

a. 2 cells (33.3%) have expected count less than 5. The minimum expected count is .99.