

Government advice on peanut avoidance during pregnancy – is it followed correctly and what is the impact on sensitization?

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Abstract

Background In 1998, the UK government issued precautionary advice that pregnant or breast-feeding women with a family history of atopy, may wish to avoid eating peanuts during pregnancy and lactation. This study aimed to assess the compliance with this recommendation and investigate its impact upon peanut sensitization.

Methods A total of 858 children born immediately after the advice were followed for 2 years and assessed for peanut sensitization. A standardized questionnaire was used to ascertain history of atopy and maternal exposure to peanuts during pregnancy. Following parental consent children were skin prick tested to assess sensitization to peanuts.

Results Sixty-five per cent of mothers had avoided peanuts during pregnancy. Forty-two per cent of the mothers had heard about the government advice, and half modified their diet as a consequence. Neither maternal nor family history of atopy had any significant effect on peanut consumption. Parity did play a role, and mothers having their first child were twice as likely to change their diet ($P < 0.001$). Mothers of 77% of the children sensitized to peanuts had avoided peanuts during pregnancy. In this cohort study maternal consumption of peanut during pregnancy was not associated with peanut sensitization in the infant.

Conclusions The majority of mothers in this cohort avoided peanut consumption during pregnancy. It is likely that either the government advice is misunderstood by mothers, or that those who communicate the advice have not fully explained who it is targeted at.

Introduction

Peanut allergy is often associated with severe symptoms and can be potentially life threatening

(Sicherer *et al.*, 2001). In 1998, the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT), Department of Health issued precautionary advice that 'pregnant

or breast-feeding women who are themselves atopic, or where another first-degree relative of the child is atopic, may wish to avoid eating peanuts and peanut products during pregnancy and lactation' (Calman, 1998; Woods, 1998). This advice was mainly based on an earlier study where the researchers suggested that peanut allergy early in life may be a reflection of peanut consumption by pregnant and nursing mothers (Hourihane *et al.*, 1996).

It is unclear at present how the COT recommendation has been interpreted and implemented by health care professionals as well as pregnant and lactating women. The aim of this study was to assess the compliance of pregnant mothers with the COT recommendations and to assess whether this has subsequently influenced the rate of sensitization to peanuts in their children.

Methods

A population birth cohort was established in order to determine the prevalence of food allergy amongst children (Venter *et al.*, 2006a) on the Isle of Wight. Ethical approval for the study was obtained from the Isle of Wight, Portsmouth and South East Hampshire Local Research Ethics Committee (Ref. 09/01). The birth cohort was recruited through antenatal clinics and included all babies born on the Isle of Wight (UK) between 1 September 2001 and 31 August 2002, 3 years after the COT report was issued. Once the study information was discussed, written consent was obtained from all expectant mothers willing to participate in the study. Expectant mothers were then asked to complete a questionnaire regarding a history of allergic disease in themselves, the baby's father or any siblings of the babies.

Expectant mothers also completed a validated food frequency questionnaire (Venter *et al.*, 2006b) at 36 weeks gestation, regarding eating habits during pregnancy, which included questions regarding peanut avoidance during pregnancy. All children were prospectively followed from birth until 2 years of age when those whose parents consented were skin prick tested to peanuts with a commercial extract of standard peanut allergen (Soluprick SQ allergens-ALK Allergolog-

isk Laboratorium A/S, Horsholm, Denmark) as described previously (Venter *et al.*, 2006a). In addition a detailed medical history, with a focus on symptoms of allergic diseases, was obtained using a standardized questionnaire. This questionnaire also included the following questions (with appropriate response categories) to assess compliance with the COT advice:

Did you avoid peanuts during pregnancy?

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

Did any of the following people (a list which included GP, midwife, health visitor, dietitian, media and other) speak to you or give you information about eating peanuts and peanut containing foods during your pregnancy?

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

If you changed your diet did you

Stop eating peanuts completely?

Stop eating obvious peanuts but continued eating foods that 'may contain peanuts'?

Increase your consumption of peanut?

Do not remember.

All data were double entered by separate operators, compared and verified before analysis using SPSS (v13.0; SPSS Corporation, Chicago, IL, USA) and StatsDirect (Cheshire, UK) statistical software.

The proportion of mothers who changed their diet during pregnancy and the incidence of sensitization to peanut were calculated together with exact (Clopper Pearson) 95% confidence limits. Categorical predictors of sensitization and change in maternal diet were identified using Fisher's Exact test. Univariate and multivariate logistic regression was used to provide both crude and adjusted odds ratios, to quantify the effect of different exposures on sensitization. In view of the small number of cases of those sensitized to peanut, analysis was restricted to three potential predictors; maternal atopy, paternal atopy and birth order (classified as first or subsequent child).

Results

A total of 969 pregnant women were recruited and their babies were part of the birth cohort study

Table 1 Basic characteristics of the cohort at 2 years of age

Male : female	52% : 48%
Mother atopic	406/855 (48%)
Father atopic	326/847 (38%)
Number of children in the family, median (range)	1 (0–7)
Birth order of the index child, median (range)	2 (1–8)
Avoided peanut consumption while pregnant	545/838 (56%)

Table 2 Association between reported history of atopy and peanut consumption during pregnancy

	Did you avoid peanuts during pregnancy		
	Yes	No	Frequency (%)
Maternal history of atopy*			
Present	249	124	68
Absent	295	166	64
Family history of atopy**			
Present	404	201	67
Absent	140	91	61

*Fisher exact, two sided $P = 0.38$, odds ratio = 1.14, 95% CI = 0.85–1.54; **Fisher exact, two sided $P = 0.10$, odds ratio = 1.31, 95% CI = 0.94–1.81.

(91% of the target population, $n = 1064$). At 2 years of age 858 (88%) children were followed up of which 658 (76% of those followed up) consented by their parents to be skin prick tested. These formed the basis of the findings described here.

General characteristics of the cohort at 2 years of age are shown in Table 1. At the 2 year follow-up information regarding peanut avoidance was available from 838 mothers. Of these 545 (65%, 95% CI 62–68%) stated that they avoided peanuts during pregnancy.

As the advice on peanut avoidance was aimed at those with a family history of atopy, association of avoidance of peanuts during pregnancy with the atopic status of the families was investigated. Neither maternal atopy ($P = 0.38$) nor family history of atopy ($P = 0.10$) had an effect on peanut consumption (Table 2).

Forty-two per cent of mothers who responded, recalled knowledge of the COT report but this awareness was not linked to maternal atopy ($P = 0.63$) or family history of atopy ($P = 0.08$). Among those who were aware of them the reported

primary sources of information were midwives (12% health visitors, 9% media, 4% GPs and 9% 'others').

Logistic regression was used to assess the associations between putative risk factors (maternal atopy; paternal atopy; birth order of the atopic child) on the likelihood of peanut sensitization (Table 3) and the mother eliminating or reducing peanut consumption during pregnancy (Table 4). Although exposure to each risk factor was associated with an elevated incidence of sensitization to peanut within the study group, none of the three predictors were found to be statistically significant. Only one variable, parity (coded as first child = yes or no), was found to significantly predict change of diet. Mothers having their first child were almost twice as likely to change their diet ($P < 0.001$, OR = 1.97, 95% CI 1.37–2.84). Maternal and paternal atopy failed to emerge as independent predictors.

Of the 858 children who completed the study 658 parents consented for their child to be skin prick tested. Thirteen of these were sensitized to peanut giving an estimated incidence risk of 2.0% (95% CI 1.2–3.4%). If it is assumed those that declined testing (200) are negative, the estimated incidence risk falls to 1.5% (0.8–2.6%). Eleven of the children sensitized to peanut (11/13, 85%) had a family history of atopy with eight (62%) having a maternal history of atopy. Food challenges to peanut were not carried out as 85% of the children sensitized to peanut had a family history of atopy. The COT report recommended not introducing peanut in the diet of children with atopic family histories before 3 years of age. Food challenges with peanut would have introduced this group to peanut prior to the recommended age. Mothers of 77% (10/13) of the sensitized children stated they had avoided peanuts during pregnancy.

Discussion

Sixty-five per cent of women in this study reported avoiding peanut during pregnancy regardless of their own or their family history of atopy. Although this advice (COT report) was only issued in the UK (Calman, 1998; Woods, 1998), other countries have also reported that some pregnant

Exposure (predictor)	Unadjusted		Adjusted	
	OR (CI)	P-value	OR (CI)	P-value
First child	2.77 (0.90–8.58)	0.082	2.78 (0.89–8.67)	0.078
Maternal atopy	1.77 (0.57–5.46)	0.404	1.77 (0.57–5.48)	0.324
Paternal atopy	1.25 (0.42–3.77)	0.778	1.13 (0.37–3.45)	0.824

Table 3 Risk factors associated with sensitization to peanuts

Exposure (predictor)	Unadjusted		Adjusted	
	OR (CI)	P-value	OR (CI)	P-value
First child	2.03 (1.42–2.92)	<0.001	1.97 (1.37–2.84)	<0.001
Maternal atopy	0.92 (0.65–1.29)	0.615	0.92 (0.65–1.30)	0.639
Paternal atopy	1.10 (0.78–1.55)	0.598	1.04 (0.73–1.48)	0.820

Table 4 Factors associated with change of diet during pregnancy

women avoided peanut during pregnancy, even though this was not recommended in their country and that this avoidance was unrelated to atopic status (van Odijk *et al.*, 2004). Forty two per cent of the mothers in this cohort had recalled hearing about the COT advice whilst pregnant and on the basis of this advice, half of this group had changed their diets. Neither maternal nor paternal atopy was found to be predictive of maternal diet change with regards to peanut consumption during pregnancy. If the child was the first born, however, mothers were almost twice as likely to change their diet. This is probably not surprising as first time mothers tend to be more cautious and are likely to follow the advice very carefully (Miller & Sollie, 1989).

The COT advice had stated that 'pregnant or breast-feeding women who are themselves atopic, or where another first-degree relative of the child is atopic, may wish to avoid eating peanuts and peanut products during pregnancy and lactation' (Calman, 1998; Woods, 1998). However, the findings of this study have clearly demonstrated that the target population did not necessarily take up this advice. It is not exactly clear why so many mothers avoid peanut consumption during pregnancy. However, it is likely that the COT advice is either misunderstood by mothers or that those who communicate the advice have not fully explained who it is targeted at. Not surprisingly most mothers who had heard of the COT advice had heard of it through their midwives. At the booking appointment (which is organized in the first tri-

mester) midwives highlight certain dietary precautions to safeguard the baby's well-being. It is likely that avoidance of peanut is often communicated as blanket advice or interpreted as blanket advice regardless of the family history of atopy. It has been previously shown that this 'blanket approach' causes confusion among mothers (Turke *et al.*, 2005).

It is difficult to ascertain what impact, if any, the COT advice has had on prevalence of peanut allergy from this small cohort. It should be noted that in this cohort of children born 3 years after the COT advice the rate of sensitization at 2 years of age was 2.0%, which is not significantly different to that previously reported in another cohort of 3–4 year old children born approximately 3 years before the COT advice (Grundy *et al.*, 2002). Nevertheless, the fact that 10 out of 13 children who were sensitized to peanut (none of whom had previously knowingly consumed peanut and 85% with a family history of atopy) had mothers who had avoided peanut during pregnancy, questions the evidence base for this advice.

It is difficult to draw a comparison between these findings and the literature as few studies have attempted to address the relationship between maternal food intake/avoidance and infant sensitization and allergy. Most are observational studies which are subject to recall bias as information regarding maternal food intake was collected retrospectively, often years after pregnancy (Hourihane *et al.*, 1996; Ushiyama *et al.*, 2002). In a recent prospective study conducted in

Southampton (UK) pregnant women from high-risk families were recruited at 16 weeks of pregnancy and randomized into a group continuing with their normal diet and a group avoiding egg (Vance *et al.*, 2004). This study found that egg intake from week 20 of pregnancy was reflected in the IgG levels to egg in the pregnant women. The infants (age 6 months) of those mothers with low and high serum IgG levels i.e. low and high egg intake, were less likely to be atopic than those with mid range IgG levels, i.e. moderate egg intake. Atopy was defined by atopic dermatitis and/or positive skin prick test (>2 mm). However, this cut off value of 2 mm has major implications on the results as 2 mm is not generally accepted as a cut off point for a positive skin prick test (Haahtela, 1993). In the present study, a cut off value of 3 mm was used which is regarded as the accepted size.

In summary, in the cohort study presented here maternal consumption of peanut during pregnancy was not associated with peanut sensitization in the infant. This study also revealed the requirement for clear, consistent factual advice and information about the real risks associated with peanut consumption during pregnancy/lactation and peanut allergy in the developing child, and specifically to whom these risks apply.

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