Diet and food allergy development during infancy: Birth cohort study findings using prospective food diary data

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Background: After an era of only considering the allergenic properties of the infant diet and allergy outcomes, emerging data suggest that the overall composition of the infant diet might be a more important factor in the development of allergic disease.

Objective: We sought to assess the relationship between infant dietary patterns in the first year of life and development of food allergy by age 2 years.

Methods: We performed a nested, case-control, within-cohort study. Mothers kept prospective food diaries for the first year of life, with resultant diet data coded in a unique manner to produce new variables, which were then analyzed by using principal component analysis to identify infant feeding patterns within the study subjects.

Results: Principal component analysis of diet diary data from 41 infants given a diagnosis of food allergy based on results of double-blind, placebo-controlled food challenges in the first 2 years of life and their 82 age-matched control subjects provided an early infant diet pattern and an ongoing diet pattern. There was no difference between the study groups for the early infant diet pattern, but for the ongoing diet pattern, there was a significant difference between the groups ($P = .001$). This ongoing dietary pattern was characterized by higher intake of fruits, vegetables, and home-prepared foods, with control infants having a significantly higher healthy infant diet dietary pattern score than children who had a food allergy.

Conclusions: An infant diet consisting of high levels of fruits, vegetables, and home-prepared foods is associated with less food allergy by the age of 2 years. (J Allergy Clin Immunol 2014;133:511-9.)

Key words: *Food allergy, double-blind, placebo-controlled food challenge, principal component analysis, infant feeding, prospective food diary data*

There is "compelling evidence" for an increase in food allergy prevalence,[1] and it has been hypothesized that changes in diet might be responsible for this increase. The changes in dietary intake that have been suggested to have a causal link with allergy development are a decreased intake of fruits and vegetables,[2,3] a change in the types of fat in the diet,[3,4] or both. This has led to an interest in the role of specific nutrients, foods, or both in allergy development[5,6] and also the link between diet and existing allergic disease.[7-9]

The role of the infant diet in the development of food allergy has long been researched, with studies looking at the timing of important feeding events during infancy[10-13] or the diet's content of particular nutrients, such as long-chain polyunsaturated fatty acids,[14,15] vitamin D,[16-18] and folic acid.[19,20] However, a number of nutritional/dietary variables might be acting on the development of food allergy in infants, and therefore focusing on one nutrient or dietary characteristic (eg, timing of solid introduction) might be an oversimplification of the complex interactions taking place.

Looking at the pattern of consumption as opposed to focusing on individual nutrients can take into account nutrient interactions of known or unknown effects, a process thought to be particularly useful when looking at disease etiology. This type of analysis is popular and has been advocated as a valid method of looking at nutritional data.[21] It has been used to describe dietary patterns that might be affecting normal development[22] or disease outcome.[23] To date, no work looking at dietary pattern analysis in infants and allergy outcome have been published. The purpose of the present study was to use principal component analysis (PCA) on prospective food diary data to investigate whether infant feeding patterns, in particular a feeding pattern that could be described as meeting infant feeding guidelines (described in this article as a healthy infant diet),[24] are associated with the development of food allergy.

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Abbreviations used
DBPCFC: Double-blind, placebo-controlled food challenge
PCA: Principal component analysis
PIFA: Prevalence of Infant Food Allergy

METHODS

Study design

The Prevalence of Infant Food Allergy (PIFA) study is a prospective birth cohort of 1140 babies recruited between 2006 and 2008 and comprised the United Kingdom cohort of the EuroPrevall project.[25] Infants with food allergy from the PIFA study and their 2 age-matched control subjects were included in a case-control study that was nested within a cohort because analysis of prospective food diaries for all study infants was not possible within the study resources. Cases were diagnosed by means of double-blind, placebo-controlled food challenge (DBPCFC).[26] Control subjects were selected by approaching parents of infants in the cohort with birthdays just before or after the index participant until 2 control subjects were found. Control participants were assessed with the same symptomatic questionnaire and physical examination as the symptomatic infants to ensure they did not have food allergy and were not sensitized to any food.[26]

PIFA study

The PIFA study has a longitudinal prospective cohort design starting from birth. Pregnant women were recruited by one of the study researchers when informed consent was obtained and baseline information on socioeconomic, environmental, and family allergy history was collected. At this appointment, women were also invited to keep food diaries for their infants from birth until 1 year of age and were instructed how to do so. Mothers/caregivers returned the food diaries to the study office monthly. Symptom sheets were sent every 2 months to facilitate identification of symptomatic infants. In addition, parents were asked to contact the study team if they thought their child had signs of allergic disease. When each infant was 12 and 24 months of age, one of the parents was asked to complete the EuroPrevall telephone questionnaire.[26] The symptomatic sheet, telephone calls to the study office, and the 12- and 24-month questionnaires were used to identify any infant who might have a food allergy.

Food allergy diagnosis

Possible cases of food allergy were triaged by means of telephone, and those fulfilling the EuroPrevall-wide criteria for assessment were invited for an outpatient visit, where the EuroPrevall symptomatic questionnaires and skin prick tests were completed, a physical examination was undertaken, and a blood sample was taken.[26] Any infant with a convincing clinical history of food allergy, a positive specific IgE level to a common food allergen (≥0.35 kU/L), and/or a positive skin prick test response (≥3-mm wheal) was started on an exclusion diet for the suspected food or foods. If symptoms improved, the child attended the Southampton Wellcome Trust Clinical Research Facility for a DBPCFC.[26] The diagnostic criterion for food allergy in this study was a positive DBPCFC result or a convincing history of anaphylaxis.

Dietary intake data

Parents were asked to record daily anything their child ate or drank for the first year of life on specifically designed food diary sheets designed and instructed on their completion. Diary sets, made up of 4 weekly sheets, were sent out every 2 months, with parents returning each diary to the study office on completion. Freepost envelopes were provided. Parents were given no advice on how to feed their infant unless they were given a diagnosis of a food allergy. However, if they asked for advice, they were given the national feeding advice of the time, which was to exclusively breast-feed for 6 months, and not to introduce solids until this age.[27]

On receipt in the study center, the diaries were reviewed to ensure they were fit for purpose. Where they lacked adequate detail (eg, brand name of commercial baby food given), parents were contacted by telephone so that these data could be recorded.

For each week's diary, the foods/ingredients the child had eaten were coded and entered into Excel. For this, details of ingredients were obtained from the relevant food companies or from the recipe information provided by the parent/caregiver. If recipe/ingredient information was not provided, then ingredients of standard recipes from food composition tables[28] were used. The resultant Excel data file was run through an SAS program (SAS Institute, Cary, NC) written for the purpose, which converted the weekly yes/no data into new variables, such as the number of weeks an infant was breast-fed, when an infant first had a particular food/ingredient, and how many weeks in total an infant ate a food/ingredient. These new SAS variables were then imported into SPSS software (SPSS, Chicago, Ill), in which further analyses could be run.

Infants suspected of having a food allergy were started on an avoidance diet for the suspected foods. If the allergy was confirmed, the avoidance diet continued, but if the allergy was not confirmed, the food was reintroduced into the diet. No control child was given avoidance advice.

Statistical analysis

A descriptive analysis of the baseline characteristics obtained by means of a standard questionnaire administered at recruitment[26] was carried out for infants involved in the study by using SPSS version 17. PCAs were carried out on selected variables. The PCA is a mathematic way of explaining the pattern of correlations within a set of observed variables.[29] It is often used in data reduction to identify a small number of factors that explain most of the variance observed from a much larger number of variables but can also be used to generate hypotheses regarding causal relationships. The variables included in the model were selected by using (1) findings from previous infant feeding studies and (2) observations made during infant food diary coding. Because of the large difference between the early infant diet, which is predominantly milk based, and the infant diet after the introduction of solids, 2 separate PCAs were run on the intake data for the first year of life, with different variables selected for each. This maximized the potential of the prospective diary data to pinpoint any factors in the diet that might be initiators or promoters of food allergy development, something that could have been lost if the data were combined into one large pattern covering the first year of life.

The first PCA looked at characteristics of the early infant diet, such as duration and exclusivity of breast-feeding, infant formula use, and timing and types of solid food introduced into the infant diet. The second analysis looked at the diet from solid introduction to 1 year of life and was termed the ongoing infant diet. It incorporated such characteristics as type of foods eaten, use of commercial infant foods, and healthy versus unhealthy weaning foods, as defined by infant feeding guidelines.[30] Because each PCA included the appropriate variables for the characteristics of early or ongoing diet, there was no need to place *a priori* cutoffs for early and ongoing diet, which meant all the available data for each infant were captured in each PCA.

Reverse causation can be a problem in studies looking at the relationship between infant feeding and disease development and needs to be considered in analyses. There was no statistical difference in the age that egg, milk, wheat, and fish was introduced into the diet of the infants with food allergy compared with their control infants (data not shown), and therefore these foods were incorporated into the first PCA looking at the early infant diet. For the analysis looking at the ongoing infant diet and food allergy development, foods to which the infants were allergic (ie, milk, egg, and peanut) were not incorporated into the analysis to reduce the likelihood of reverse causality affecting the analysis. To identify what effect (if any) exclusion of these foods might have had on the results of the PCA, the analysis was also carried out including these allergenic foods. Each infant's score within each pattern was then saved as a new variable, and a Mann-Whitney *U* analysis was carried out to establish whether there was a difference in mean scores for the patterns between infants with food allergy and control infants. Finally, a multivariate analysis, which included variables associated with food allergy development

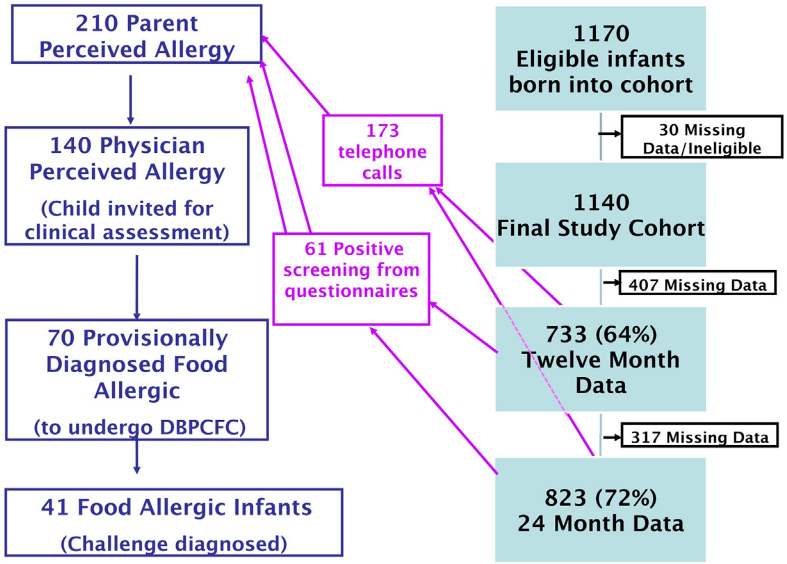

FIG 1. Flowchart of participants in the PIFA study.

by the age of 2 years (including those from the PCA, where appropriate), was carried out.

RESULTS

Participants

Infants and their mothers were from the main parent study (the PIFA study, n = 1140) and had received a diagnosis of a food allergy based on DBPCFC results (n = 41) or were their age-matched control subjects (n = 82). Fig 1 shows the number of participants involved in each stage of the main PIFA study. The study infants (n = 123) were born between January 2006 and October 2007. Median maternal age was 33 years (range, 19-43 years), and median infant weight was 3420 g (range, 2160-5060 g). Baseline characteristics for the infants given a diagnosis of food allergy and their control subjects are detailed in Table I. The demographics of the main PIFA study population (n = 1140) differed from the demographics of the community from which it was recruited because it had a large proportion of older, well-educated mothers. However, the infants in the 2 experimental groups of this study (n = 123) did not differ significantly from each other for any demographic or environmental measurement (Table I).

All infants with food allergy were given a diagnosis based on DBPCFC results (which included delayed reactions up to 48 hours after the challenge). Twenty-two reacted to hen's egg, and 20 reacted to cow's milk. Six reacted to peanut, 3 to soya, and 2 to wheat. Some infants reacted to more than 1 food. The most common symptom was vomiting (n = 17), with physician-diagnosed eczema the second most common (n = 12).

Food diary return

A total of 4489 weeks of diary data were analyzed. Median duration for food diary completion for the cases was 42 weeks, with 31 having prospective food diaries for at least 28 weeks and 17 having diaries for 52 weeks. Two infants had less than 4 weeks of data. Each control infant had at least the same number of weeks of diaries analyzed as their age-matched case. Missing data in the analysis were allocated the mean value for each variable because PCA is a measure of variance.

Patterns of early infant diet

In the PCA related to the early infant diet, 5 principal components were identified (Table II), accounting for 59% of the variance observed. The first component, which accounted for 25% of the observed variance, was characterized by infant nutrition, predominantly breast milk. The main characteristic for the second component was early solid introduction. The third component's main characteristics related to the intake of egg, fish, and wheat. The fourth component was characterized by intake of commercial baby foods, and the fifth component was characterized by formula milk intake. After the analysis was run, each infant's resultant score for each component was saved as a variable, and a Mann-Whitney *U* test was carried out to determine whether there was a difference in pattern scores between the symptomatic infants and their control subjects. There were no significant differences between the 2 experimental groups for any of the 5 early infant diet components (Table III).

Ongoing infant diet patterns

In the PCA related to the ongoing infant diet, 6 principal components were identified (Table IV), and 2 of these were significantly different between the 2 experimental groups (Mann-Whitney *U* test: component 1, $P = .015$; component 3, $P = .030$). These 2 components both described dietary patterns associated with the intake of fruits and vegetables, poultry, oily fish, ready meals, potato products, and cook-in-sauces. In the first component the high-scoring foods were those that were age appropriate (eg, commercial baby foods, toddler snacks, carrots, potatoes, and bananas), while low-scoring foods could be considered adult foods (eg, potato products, ready meals, and cook-in-sauces). For component 3, high scores were seen with adult foods, and low scores were seen with foods that could be considered healthy

TABLE I. Characteristics of the mother and infant pairs included in this prospective case-control study

	Children with food allergy (n = 41)	Control subjects (n = 82)	*P* value*
Male sex (%)	24 (58.5)	43 (52.4)	.522
Birth weight (g), median (range)	3480.0 (2160-4120)	3370.0 (2270-5060)	.913†
Birth length (cm), median (range)	53.0 (48-59)	52.0 (47-61)	.909†
Duration of pregnancy (wk)	39.5 (36-42)	40.0 (36-42)	.062†
Cesarean delivery	13 (31.7)	20 (24.4)	.255
Season of birth			.926
Spring	8 (19.5)	18 (22.0)	
Summer	17 (41.5)	37 (45.1)	
Autumn	8 (19.5)	13 (15.8)	
Winter	8 (19.5)	14 (17.1)	
Maternal age (y), median (range)	31.0 (19-43)	33.0 (22-42)	.192†
Paternal age (y), median (range)	33.5 (21-42)	34.0 (23-49)	.247†
Maternal education (%)			.448
Did not complete basic education	0	0	
Completed basic education	4 (9.7)	6 (7.3)	
Junior college/vocational training	11 (26.8)	15 (18.3)	
University/college	26 (63.4)	61 (74.4)	
Maternal antibiotic use (%)			
During pregnancy	9 (22.0)	19 (23.2)	.379
During delivery	7 (17.1)	8 (9.8)	.321
After delivery	9 (22.0)	11 (13.4)	.160
While breast-feeding	16 (39.0)	18 (22.0)	.349
Maternal multivitamin use (%)			
During pregnancy	24 (58.5)	47 (57.3)	.495
While breast-feeding	9 (22.0)	20 (24.4)	.404
Maternal folic acid supplement use (%)			
During pregnancy	36 (87.8)	69 (84.1)	.345
While breast-feeding	7 (17.1)	9 (11.0)	.320
Maternal vitamin D supplement use (%)			
During pregnancy	2 (4.9)	1 (1.2)	.241
While breast-feeding	2 (4.9)	2 (2.4)	.444
Maternal fish oil supplement use (%)			
During pregnancy	3 (9.8)	13 (17.0)	.235
While breast-feeding	2 (4.9)	5 (6.1)	.514
Maternal prepregnancy BMI (kg/m^2), median (range)	22.9 (16.6-43.0)	22.8 (16.5-49.2)	.323†
Maternal asthma (%)	11 (26.8)	11 (13.4)	.067
Maternal allergy (%)	22 (53.7)	31 (37.8)	.105
Maternal smoking (%)	1 (2.4)	3 (3.7)	1.000
Only child (%)	24 (58.5)	49 (59.8)	.570
Urban dwelling (%)	8 (19.5)	11 (13.4)	.378
Pet ownership (%)	26 (63.4)	40 (48.8)	.142

*χ^2 Test.
†Mann-Whitney *U* test.

(eg, dried fruit, lentils, and broccoli). In both cases the mean score for the control infants was associated with the healthier pattern, whereas the mean score for the symptomatic infants was associated with the less healthy pattern.

Because there were some similar elements between these 2 patterns, further analyses were run to incorporate the 2 patterns into one. This resulted in the final analysis, which produced a pattern in which the first 3 components accounted for 50% of the variance, with the first component accounting for 32% of the variance. Component 1 depicted a dietary pattern, with high positive values associated with fruit, vegetable, fish, and poultry consumption, and it was hypothesized that the component described a diet that was predominantly home cooked because at the time of data collection, broccoli, parsnips, and butternut squash (which had high scores within the component) were ingredients of few commercially prepared infant foods and would only be in the diet if they were home prepared. The component also had low/negative values associated with highly processed adult foods (eg, ready meals, cook-in-sauces, potato products, and bacon) and the use of commercial baby foods more than once a day, which showed these foods were not important in the component. Combining these characteristics indicated a diet that could be described as following infant feeding guidelines,[31] which in this article is described as a healthy infant diet. Component 2 was a pattern defined by finger foods. The highest positive values were allocated to healthy finger foods, and the low/negative values were allocated to pureed baby foods and unhealthy finger foods. The third component had high values allocated to highly processed adult foods (Table V). The PCA assigned a score to each infant in the analysis according to how his or her diet corresponded to each of the dietary components. These scores were significantly different between the symptomatic and control infants (P = .002) for component 1 but not for components 2 or 3 (Table VI).

TABLE II. Five PCA components for the early infant diets of all infants in the study (n = 123)

Variable included in the analysis	Component 1	2	3	4	5
Breast-feeding duration	0.943	0.191	0.125	0.080	0.085
Age infant first had any solids	0.102	0.885	0.051	0.149	0.029
Age infant first had hummus	−0.007	−0.031	−0.015	−0.034	0.257
Age infant first had any fish	−0.115	0.218	0.857	0.006	0.094
Age infant first had wheat	0.003	0.318	0.465	0.440	0.120
Age infant first had any baby cereal	0.095	0.883	0.023	0.091	0.039
Age infant first had commercially prepared savory baby food	0.086	0.405	−0.087	0.618	0.015
Age infant first had commercially prepared sweet baby food	−0.007	0.237	0.025	0.787	−0.015
Age infant first had yogurt/fromage frais	0.111	0.548	0.200	0.092	0.204
Age infant first had avocado	0.039	0.341	0.204	0.115	−0.112
Age infant first had carrots	0.062	0.875	0.162	0.087	0.047
Age infant first had lentils	−0.018	0.374	0.142	0.191	0.297
Age infant first had any apples	0.162	0.845	0.121	0.184	−0.076
Age infant first had banana	−0.042	0.730	0.051	0.158	0.005
Age infant first had strawberry	0.106	0.483	0.141	−0.033	−0.193
Age infant first had any cow's milk protein	0.661	0.051	0.234	0.200	−0.567
Age infant first had egg	0.152	0.043	0.644	0.014	−0.095
Age infant first had cow's milk ingredient	0.093	0.463	0.290	0.497	−0.027
Age infant first had oily fish	−0.057	0.201	0.705	0.012	0.090
Age infant first had white fish	−0.107	0.164	0.741	−0.064	0.115
Age infant first had bread	−0.003	0.161	0.566	0.223	−0.172
Age infant first had tinned baked beans	0.064	−0.223	0.147	0.470	0.368
Age infant first had infant formula	0.692	0.024	0.167	0.041	−0.498
Age infant first had blueberry	0.073	0.195	0.054	0.515	−0.208
Duration of soya and breast milk overlap	0.448	−0.079	0.234	0.262	−0.114
Duration of breast milk and any solid overlap	0.884	0.010	0.126	0.069	0.050
Duration of breast milk and infant formula overlap	0.532	0.244	0.107	0.093	0.592
Duration of fish and breast milk overlap	0.921	0.102	−0.271	0.076	0.051
Duration of wheat and breast milk overlap	0.949	0.073	−0.070	−0.097	0.044
Duration of egg and breast milk overlap	0.855	0.158	−0.110	−0.029	0.012
Duration of any milk and breast milk overlap	0.701	0.213	−0.038	−0.054	0.604

The rotation method was varimax with Kaiser normalization. Rotation converged in 6 iterations. Age and duration refer to weeks. High values demonstrate those characteristics important in the component, and very low/negative values show those characteristics that are not important in the component.

TABLE III. Mean scores for 5 components identified from PCA analysis for the early infant diets of all infants in the study (n = 123)

Mean score (SE)	Children with food allergy (n = 41)	Control subjects (n = 82)	*P* value*
Component 1	−0.099 (0.169)	0.050 (0.106)	.248
Component 2	−0.202 (0.146)	0.101 (0.113)	.109
Component 3	−0.029 (0.159)	0.014 (0.110)	.959
Component 4	−0.060 (0.127)	0.030 (0.120)	.845
Component 5	−0.130 (0.168)	0.065 (0.106)	.184

*Mann-Whitney *U* test.

Each infant's score for component 1 was included in the multivariate analysis, which showed that the association between component 1 and food allergy development remained after controlling for the effects of other potentially confounding variables (Table VII).

DISCUSSION

In this hypothesis-generating study we found that children who did not have a food allergy by the age of 2 years had a dietary pattern in later infancy characterized by higher intake of fruits, vegetables, and home-prepared foods than children who had a food allergy. Such a diet meets infant feeding recommendations of a healthy infant diet.[30] There was no difference between food allergy cases and control subjects for patterns for the early infant diet (ie, before solids were introduced).

The variables included in the early infant diet PCA were those dietary factors that have been associated with food allergy development in previous studies. These included breast-feeding,[32-34] infant formula use,[35,36] and age and nature of solid introduction.[37,38] Of these variables, only age at solid food introduction was significantly different between the groups, with control infants first receiving solids at a median age of 20 weeks compared with 18 weeks for control infants ($P = .044$). PCA on the early infant diet identified the 5 components that made up 62% of the variance. There was no difference between infants given a diagnosis of food allergy and control infants in how they scored for these 5 patterns, demonstrating that early infant feeding patterns did not have an association with the later development of food allergy. This is likely due to the overall pattern of the infant diet being similar between cases and control subjects as a result of the lack of diversity in dietary intake in early infancy because the diet mainly consists of breast milk, infant formula, or both. This lack of diversity in the overall early infant diet might be masking single dietary factors that have been associated with food allergy development in previous research, such as age of solid food introduction, which was significantly different for this dataset.

TABLE IV. Six components for the ongoing infant diet identified by means of PCA on data from all infants (n = 123)

Variable included in the analysis	Component					
	1	2	3	4	5	6
Breast-feeding duration	0.106	0.142	0.061	0.000	0.702	−0.021
Age of infant at introduction of solids	−0.102	−0.106	−0.012	−0.023	0.799	−0.089
No. of weeks apple included in infant's diet	0.904	0.225	−0.024	0.062	−0.084	0.053
No. of weeks infant had commercially prepared savory baby food	0.705	−0.108	0.158	0.460	−0.094	−0.069
No. of weeks infant had commercially prepared savory baby food more than once a day	0.280	−0.142	−0.015	0.791	−0.030	−0.137
No. of weeks infant had commercially prepared sweet baby food	0.673	0.038	0.020	0.429	−0.137	−0.049
No. of weeks infant had commercially prepared sweet baby food more than once a day	0.022	0.131	0.131	0.860	0.029	0.111
No. of weeks pizza was included in infant's diet	0.130	0.802	0.082	0.018	−0.108	−0.230
No. of weeks commercially produced potato products were included in infant's diet	0.112	−0.184	0.759	−0.074	−0.026	−0.098
No. of weeks ready meals were included in infant's diet	0.114	0.220	0.631	0.282	0.027	0.209
No. of weeks cook-in-sauces were included in infant's diet	0.084	0.177	0.767	0.069	0.068	0.029
No. of weeks carrot was included in infant's diet	0.907	0.183	0.089	0.109	−0.042	0.127
No. of weeks onion was included in infant's diet	0.861	−0.016	0.164	−0.009	0.054	0.055
No. of weeks potato was included in infant's diet	0.864	−0.016	0.143	0.007	0.003	0.032
No. of weeks peas were included in infant's diet	0.688	0.212	0.160	0.107	0.081	−0.150
No. of weeks lentil was included in infant's diet	0.506	0.307	−0.087	−0.095	0.256	0.162
No. of weeks banana was included in infant's diet	0.878	0.226	0.074	0.121	−0.058	0.115
No. of weeks peaches were included in infant's diet	0.620	0.208	0.135	0.067	0.104	0.112
No. of weeks raspberries were included in infant's diet	0.711	0.275	−0.122	0.148	0.119	0.164
No. of weeks strawberries were included in infant's diet	0.788	0.150	0.026	0.119	−0.063	0.233
No. of weeks oily fish was included in infant's diet	0.637	0.230	0.358	−0.165	0.078	0.123
No. of weeks broccoli was included in infant's diet	0.737	0.230	−0.077	0.102	0.045	−0.037
No. of weeks beef was included in infant's diet	0.878	−0.018	0.243	−0.019	−0.044	0.015
No. of weeks poultry was included in infant's diet	0.920	0.120	0.130	0.091	−0.017	−0.018
No. of weeks Marmite was included in infant's diet	0.267	0.528	0.302	0.147	−0.212	0.427
No. of weeks jam was included in infant's diet	0.277	0.050	0.133	−0.042	−0.187	0.746
No. of weeks chocolate was included in infant's diet	0.328	0.344	0.379	0.043	−0.256	−0.565
No. of weeks dried fruit was included in infant's diet	0.301	0.561	−0.056	−0.262	0.267	0.227
No. of weeks toddler packet snacks were included in infant's diet	0.582	0.481	0.042	−0.023	0.112	−0.055
No. of weeks fruit (not pureed) was included in infant's diet	0.399	0.553	0.192	0.162	0.277	0.236

The rotation method was varimax with Kaiser normalization. Rotation converged in 13 iterations. Age and duration refer to weeks. High values demonstrate those characteristics important in the component, and very low/negative values show those characteristics that are not important in the component.

The findings for the pattern analysis for the ongoing infant diet showed a difference between the 2 study groups for a dietary component which depicted a dietary pattern that was predominantly home cooked with a high intake of fruits and vegetables, little highly processed adult foods (eg, ready meals, cook-in-sauces, potato products, and bacon), and only the occasional use of commercial baby foods. (A similar pattern has been identified in previous research into infant dietary intake and described as the "infant guidelines" pattern.[31]) This association remained after adjustment for potential confounding factors, such as maternal age, education, asthma, and allergy. A possible mechanism for the observed results are the immunomodulatory effects of nutrients found in fruits and vegetables, such as vitamin C, β-carotenes, folate, and oligosaccharides, which have been suggested in previous research to influence allergic outcomes because of their effect on inflammatory processes.[39-41] That these findings are not allergen or tissue specific (ie, the effect was seen despite all the infants being included in the analysis regardless of what food to which they were reactive) further suggests that the results might be due to modification of the immune response. Additionally, a feature of the protective dietary pattern was home-prepared food, suggesting that the observed effect might be due to the overall micronutrient content of the diet because food processing is known to affect this.[42] Although commercial baby foods are often supplemented with vitamins and minerals to enhance micronutrient content, the bioavailability of these nutrients might not be the same as that of those occurring in the food naturally because the bioavailability of micronutrients is affected by a number of factors, including food preparation and food matrix.[43]

Along with introducing solids after the age of 17 weeks (the other significant factor in the logistic analysis), the importance of introducing infants to a wide variety of home-cooked foods containing plenty of fruits and vegetables has been part of infant feeding recommendations for many years. However, with the advent of more convenience foods in homes, United Kingdom infants and young children now consume large amounts of processed foods.[29] These findings suggest that this change in infant and early childhood diets might be contributing to the observed increase in allergy rates seen in the last 2 decades.[1] It might also be the reason for the perceived difference in infant allergy prevalence rates between countries[25,44] because children from different countries appear to be fed differently in the first year of life and embrace processed foods as a part of their child's diet regimen at different rates.[45,46]

The main strength of this study is in its design, with infant feeding data being collected prospectively, thus reducing the potential for experimental and recall bias. Additionally, infants were given a diagnosis of food allergy based on results on the DBPCFC, which is considered the gold standard for food allergy diagnosis. A potential limitation of the study is the generalizability of the findings to the wider population. Women who completed the study were generally older and better educated than seen in the

TABLE V. Three components for the ongoing infant diet identified by means of final PCA on data from all infants (n = 123)

Variable included in the analysis	Component 1	Component 2	Component 3
No. of weeks infant had commercially prepared savory baby food	0.727	−0.044	0.184
No. of weeks infant had commercially prepared savory baby food more than once a day	0.306	−0.084	−0.018
No. of weeks infant had commercially prepared sweet baby food	0.747	0.089	0.050
No. of weeks infant had commercially prepared sweet baby food more than once a day	0.094	0.109	0.095
No. of weeks "fast food" was included in infant's diet	0.411	−0.086	0.419
No. of weeks commercially produced potato products were included in infant's diet	0.052	0.051	0.651
No. of weeks ready meals were included in infant's diet	−0.026	0.127	0.625
No. of weeks cook-in-sauces were included in infant's diet	−0.056	0.212	0.749
No. of weeks avocado was included in infant's diet	0.265	0.270	−0.072
No. of weeks broccoli was included in infant's diet	0.728	0.201	0.058
No. of weeks carrots were included in infant's diet	0.853	0.315	0.185
No. of weeks apples were included in infant's diet	0.876	0.330	0.099
No. of weeks grapes were included in infant's diet	0.202	0.664	−0.088
No. of weeks bacon was included in infant's diet	0.048	−0.028	0.107
No. of weeks poultry was included in infant's diet	0.839	0.237	0.201
No. of weeks sausages were included in infant's diet	0.262	0.141	0.773
No. of weeks Marmite was included in infant's diet	0.209	0.530	0.343
No. of weeks jam was included in infant's diet	0.120	0.608	0.012
No. of weeks dried fruit was included in infant's diet	0.169	0.680	−0.019
No. of weeks toddler packet snacks were included in infant's diet	0.542	0.392	0.165
No. of weeks raw fruit was included in infant's diet	0.289	0.677	0.155
No. of weeks oily fish was included in infant's diet	0.500	0.418	0.387
No. of weeks "sweeties" were included in infant's diet	0.194	−0.008	0.055
No. of weeks crisps were included in infant's diet	0.259	−0.226	0.583
No. of weeks bread was included in infant's diet	0.589	0.549	0.374
No. of weeks butternut squash was included in infant's diet	0.742	0.136	−0.079
No. of weeks parsnips were included in infant's diet	0.713	0.132	0.059
No. of weeks sweet corn was included in infant's diet	0.547	0.162	0.024
No. of weeks kiwi was included in infant's diet	0.315	−0.020	0.029
No. of weeks oranges/citrus were included in infant's diet	0.287	0.577	0.154

The rotation method was varimax with Kaiser normalization. Rotation converged in 7 iterations. High values demonstrate those characteristics important in the component, and very low/negative values show those characteristics that are not important in the component.

TABLE VI. Mean scores for 3 components identified from final PCA analysis on the ongoing infant diets of all infants in the study (n = 123)

Mean score (SE)	Symptomatic (n = 41)	Control (n = 82)	*P* value*
Component 1	−0.390 (0.160)	0.185 (0.105)	.002
Component 2	−0.064 (0.110)	0.030 (0.124)	.494
Component 3	−0.143 (0.107)	0.068 (0.124)	.284

*Mann-Whitney *U* test.

TABLE VII. Multivariate logistic regression analysis for assessment of variables associated with food allergy development

Variable	Adjusted OR (95% CI)
Infant guidance score from PCA analysis	2.136 (1.233-3.700)*
Age at solid introduction (≥17 vs <17 wk)	0.252 (0.082-0.780)*
Maternal asthma	1.372 (0.410-4.593)
Maternal allergy	2.600 (0.932-7.254)
Maternal smoking	0.000 (0.000)
Maternal education (≤18 vs >18 y)	1.370 (0.233-8.052)
Maternal age (y)	0.979 (0.879-1.089)
Pet ownership	1.275 (0.489-3.327)
Female sex	0.896 (0.347-2.317)
Any siblings	0.808 (0.303-2.153)

**P* < .05.

general population and would be expected to have better diets and to be more aware of recommended best practice. However, among those women completing the study, there were no significant differences in demographic factors between cases and control subjects (Table I); thus the reported feeding differences seen between cases and control subjects are unlikely to be due to demographic differences. The observed small differences between the demographic characteristics of the infants generally occur in the same direction as previously published associations with food allergy development,[1,47] except for high maternal education, which (as a marker of other socio economic factors) has previously been shown to be associated with food allergy development. However, when considering that well-educated mothers are more likely to feed their children according to infant recommendations,[31] this observed small difference is not so surprising. For pet ownership, its relationship with food allergy development in infancy is currently under debate because most of the available data relate to pet ownership and the development of asthma/allergy in older children.[48,49]

Another potential limitation is the selection of variables for the pattern analysis. It could be argued that not including the foods to which the infants were allergic in the analysis for the ongoing infant diet (which were excluded to reduce the effect of reverse causality) limits the resultant patterns and might create false-positive associations. Consequently, the PCA was repeated with the inclusion of milk, egg, peanut, and foods that contain these ingredients. The resultant 3 components had the same foods scoring highly within each component as in the analysis that excluded the allergenic foods. Component 1 was also significantly different between the cases and control subjects ($P = .001$), but components 2 and 3 were not (analysis not shown). A final limitation of the analysis is that we did not look at diversity of the early infant diet, which has previously been shown to be associated with food allergy development.[37,48]

It is still necessary to consider the results from the perspective that some of the observed differences in mean scores might be due to food allergy rather than a cause of it. Children with food allergy are more likely to consume home-prepared foods when compared with nonallergic children because they are known to be "safe," whereas commercially prepared foods might not be so. Because commercially prepared foods, such as ready meals, cook-in-sauces, and potato products, are likely to include milk, egg, or both and have precautionary labelling concerning peanut and nut traces, these do not constitute a large part of the diet of children with food allergy.[50] Consequently, the observation that there were more commercially prepared foods in the diets of the allergic children is unlikely to be due to the food allergy.

In conclusion, the findings from this study have provided new insight into how the infant diet might modify allergy development. The possible protective nature of a healthy diet in the first year of life is a unique finding. To date, there have been observations that healthy dietary patterns can modify asthma symptoms[8,9] and even sensitization rates[7] in older children and adults, but this has not been demonstrated for the infant diet. Because advocating a healthy diet to help protect against the development of food allergy is unlikely to have any adverse health effects and fits with the core requirements of many national infant/child feeding recommendations,[51,52] embracing this as a recommendation for allergy prevention is very attractive. The problem with such advice is that compared with previous messages advocating onerous food allergen avoidance for allergy prevention,[53] it does not seem very proactive or scientific and therefore might not be readily embraced without additional promotion and justification to both health care professionals and parents alike. However, before recommendations advocating a healthy infant diet for allergy prevention are made, these results need to be replicated in larger cohorts by using the findings of this work with prospective food diaries to inform how data from both prospective and retrospective food intake questionnaires can be analyzed to investigate the relationship between the whole infant diet and allergy development.

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Clinical implications: Advocating a healthy infant diet that is predominantly home cooked and provides high levels of fruits and vegetables might be a positive way to protect against food allergy development.

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