Food and nutrient intakes and asthma risk in young adults[1–3]

Rosalie K Woods, E Haydn Walters, Joan M Raven, Rory Wolfe, Paul D Ireland, Frank CK Thien, and Michael J Abramson

ABSTRACT

Background: Some aspects of diet are relatively newly recognized potential risk factors for asthma, but the evidence to date is conflicting.

Objective: The goal was to determine whether the food and nutrient intakes of adults with asthma differ from those of adults without asthma.

Design: This was a community-based, cross-sectional study of 1601 young adults ($\bar{x}$ ± SD age: 34.6 ± 7.1 y) who were initially recruited by random selection from the federal electoral rolls in Melbourne in 1999. Subjects completed a detailed respiratory questionnaire, a validated semiquantitative food-frequency questionnaire, skin-prick testing, and lung function tests, including a methacholine challenge test for bronchial hyperreactivity (BHR). A total of 25 nutrients and 47 food groups were analyzed by using multiple logistic regression with alternate definitions of asthma and atopy as the outcomes.

Results: Whole milk appeared to protect against current asthma (odds ratio: 0.66; 95% CI: 0.46, 0.97), doctor-diagnosed asthma (0.73; 0.54, 0.99), BHR (0.68; 0.48, 0.92), and atopy (0.71; 0.54, 0.94). Conversely, soy beverage was associated with an increased risk of current asthma (2.05; 1.19, 3.53), doctor-diagnosed asthma (1.69; 1.04, 2.77), and BHR (1.65; 1.00, 2.71). Apples and pears appeared to protect against current asthma (0.83; 0.71, 0.98), asthma (0.88; 0.78, 1.00), and BHR (0.88; 0.77, 1.00).

Conclusions: The consumption of dairy products, soy beverages, and apples and pears, but not of nutrients per se, was associated with a range of asthma definitions. Dietary modification after diagnosis is one possible explanation for this finding. Intervention studies using whole foods are required to ascertain whether such modifications of food intake could be beneficial in the prevention or amelioration of asthma. *Am J Clin Nutr* 2003;78:414–21.

KEY WORDS Diet, asthma, atopy, food, nutrients, community-based study, epidemiology

INTRODUCTION

Asthma is now recognized as a national health priority in Australia. It is a common source of morbidity and a significant cause of preventable mortality in many countries, contributing to the deaths of 454 Australians in 2000 (1, 2). Diet is a relatively recently recognized potential risk factor for asthma, although the evidence to date is conflicting (3, 4).

Current research interest has focused on the possible protective effect of long-chain (n−3) fatty acids in fish (5–7); dietary antioxidants such as vitamin C, vitamin E, and selenium (8, 9); and the possible deleterious effects of polyunsaturated fatty acids (10–12) and sodium (13, 14). At present, the evidence is insufficient to implicate any of these as causal risk factors for asthma. This is partly because of inconsistent findings, differences in asthma definitions, and methodologic deficiencies in many of the studies conducted to date. To determine whether diet may be a risk factor for asthma in Australian adults, we assessed the usual dietary intake of young adults by using a validated semiquantitative food-frequency questionnaire. Specifically, the aims of the present study were to determine whether the intakes of fish, fruit and vegetables, vitamin C, vitamin E, and sodium differ between those with and without asthma. A range of definitions of asthma was used, from self-reported asthma to epidemiologic and physiologic definitions of asthma, because no consensus definition of asthma exists.

SUBJECTS AND METHODS

Study population

Adults aged 20–44 y, originally recruited from the electoral rolls of southeastern Melbourne (15), were invited to attend our laboratory for further investigation. Briefly, a computer-generated random sample of 4455 eligible adults aged between 20 and 44 y was selected by using the federal electorates of Higgins, Hotham, and Goldstein (the inner southeastern suburbs Melbourne, the capital city of Victoria). A cross-sectional postal survey was conducted by using the European Community Respiratory Health Survey screening questionnaire (16), which was based on the validated International Union of Tuberculosis and Lung Disease bronchial symptoms question-

[1] From the Departments of Epidemiology & Preventive Medicine (RKW, RW, and MJA), Respiratory Medicine (JMR), and Allergy, Asthma & Clinical Immunology (FCKT), Central and Eastern Clinical School, Monash University, and The Alfred Hospital, Melbourne; the Discipline of Medicine, School of Medicine, University of Tasmania, Hobart, Australia (EHW); and the National Institute of Clinical Studies, Melbourne (PDI).

[2] Supported by the National Health and Medical Research Council of Australia (NHMRC), the Victorian Health Promotion Foundation, and the Dairy Research and Development Corporation. RKW held an NHMRC postdoctoral public health fellowship (#987087).

[3] Address reprint requests to M Abramson, Department of Epidemiology & Preventive Medicine, Central and Eastern Clinical School, The Alfred Hospital, Commercial Road, Melbourne, Victoria 3004, Australia. E-mail: michael.abramson@med.monash.edu.au.

Received August 19, 2002.

Accepted for publication April 16, 2003.

naire (17). A response rate of 72% ($n = 3194$) was achieved for the postal survey.

All subjects were then invited to our laboratory for further investigation (overall response rate = 36%, or 1601/4455). There was no significant difference in mean age (33.3 compared with 33.7 y; $P = 0.16$), sex (45.5% compared with 46.2% male; $P = 0.68$), and smoking status (those who had ever smoked; 42.7% compared with 42.3%; $P = 0.80$) between those who only completed the postal survey and those who attended the laboratory phase of the study. Those who attended the laboratory phase of the study were more likely to have doctor-diagnosed asthma (20.2% compared with 16.1%; $P = 0.003$) and were less likely to be current smokers (21.0% compared with 25.6%, $P = 0.002$) than were those who only completed the postal survey. The participants in this study completed an interviewer-administered detailed respiratory questionnaire, which was previously used in the European Community Respiratory Health Survey (18). The Standing Committee on Ethics in Research on Humans at Monash University and The Alfred Hospital Ethics Committee approved this project.

Food-frequency questionnaire

Participants completed a validated semiquantitative food-frequency questionnaire, which assessed their habitual food intake over the preceding 12 mo. The questionnaire was originally designed to derive estimates of nutrient intake for a large prospective study of Australian adults from ethnically diverse backgrounds (19). The validity of the food-frequency questionnaire was previously determined with the use of biomarkers for energy (basal metabolic rate multiplied by an activity level factor), protein, and potassium (daily urinary excretion averaged across 4 separate 24-h periods over 1 y). The Pearson correlation coefficients for energy, protein, and potassium were 0.27, 0.35, and 0.32, respectively (19). These correlations are typical of dietary questionnaires. A total of 25 nutrients (energy, total fat, saturated fat, polyunsaturated fat, monounsaturated fat, cholesterol, protein, carbohydrate, alcohol, fiber, β-carotene, calcium, folate, iron, magnesium, niacin, phosphorus, potassium, retinol, riboflavin, sodium, thiamine, vitamin C, vitamin E, and zinc) and 47 food groupings (**Appendix A**) were examined.

Total fish intake was defined as the sum of steamed, grilled or baked, fried, and canned fish as reported in the food-frequency questionnaire. Total nonfried fish intake was determined as the sum of steamed, grilled or baked, and canned fish.

The categories for fruit (citrus, stone fruit, bananas, berries, melons, apples and pears, tropical, canned and frozen, and fruit juice) and vegetables (root, cruciferous, green leafy, marrow, stalks and stems, bulbs, pods, capsicum and peppers, shoots, tomatoes, and mushrooms) were constructed according to the food variety checklist used previously by Savige et al (20). Total fruit intake was defined as the sum of all fruit, and total vegetable intake was defined as the sum of all vegetable categories.

Alcohol was converted to a categorical variable on the basis of the known risk profile of alcohol (21). The categories were as follows: no risk, low risk (1–19.9 g alcohol/d for men, 1–9.9 g/d for women), moderate risk (20–39.9 g/d for men, 10–19.9 g/d for women), high risk (40–60 g/d for men, 20–40 g/d for women), and highest risk (> 60 g/d for men, > 40 g/d for women).

Daily intakes of energy and nutrients were derived through multiplication of the food item frequency response by the standard portion size for each of the food items and were adjusted at the individual level through application of the computed portion size factor, as required. The food database used nutrient data from McCance and Widdowson (22) for vitamin E and folate, and all other nutrients were derived from the Australian NUTTAB 95 database (23).

Lung function and skin-prick tests

Lung function was measured with a rolling seal spirometer (SensorMedics, Yorba Linda, CA). Initial forced expiratory volume in 1 s was recorded as the best of 5 blows that met the American Thoracic Society criteria. Methacholine (Provocholine; Methapharm Inc, Brantford, Canada) was delivered by a Mefar 3B dosimeter (Mefar SRL, Bovezzi, Italy) up to a cumulative dose of 2 mg (10 μmol).

Skin-prick testing for 7 aeroallergens (*Dermatophagoides pteronyssinus*, cat dander, ragweed, rye grass, *Cladosporium, Alternaria,* and *Aspergillus*), 5 food allergens (cow milk, peanut mix, shrimp, egg white, and wheat), and positive (histamine) and negative controls was performed on the flexor surface of the forearm with standardized allergen extracts (Bayer Corporation, Sydney, Australia).

Definitions

Atopy was defined as a ≥ 3-mm wheal diameter in response to any allergen (24, 25). *Bronchial hyperreactivity* (BHR) was defined as a provocative dose of ≤ 2 mg inhaled methacholine causing a 20% drop in forced expiratory volume in 1 s. *Current asthma* was defined as self-reported wheeze in the past 12 mo and BHR (26). *Asthma* was defined as an answer of yes to the questions "Have you had an attack of asthma in the last 12 mo?", "Have you been woken by an attack of shortness of breath at any time in the last 12 mo?", or "Are you currently taking any medicine for asthma?" (18). *Doctor-diagnosed asthma* was defined as an answer of yes to the questions "Have you ever had asthma?" and "Was this confirmed by a doctor?" *Not diagnosed asthma* was defined as an answer of no to the questions "Have you ever had asthma?" or "Was this confirmed by a doctor?" Consequently, a subject could appear in more than one category.

Statistical analysis

Statistical analyses were performed by using the SPSS statistical package (version 10.0; SPSS Inc, Chicago). Because distributions were right-skewed, all nutrient data were log transformed.

For a sample size of 1000 subjects, 17% with current asthma, the power of a statistical comparison of exposure between affected and unaffected subjects depended on the anticipated distribution of the exposure in the 2 groups and the level of statistical significance. Using vitamin E intake as an example of dietary exposure, we assumed a log-normal distribution of vitamin E intake in both groups. For a two-tailed test with significance level of 1% (ie, $P = 0.01$), we had 90% power to detect a difference of 0.36 SDs in the logarithm of vitamin E intake between subjects affected and unaffected by current asthma.

For all univariate analyses, Student's *t* test and chi-square tests were used for continuous and categorical data, respectively. *P* values of < 0.01 were considered statistically significant because of multiple comparisons.

Logistic regression models were used for each of the 5 outcome variables to adjust the estimates of food and nutrient associations for the effects of age, sex, body mass index, smoking status, region of birth, and family history of asthma. Because almost all of the nutrients were highly intercorrelated (Pearson's $r > 0.80$), regres-

TABLE 1
Subject characteristics[1]

Item	Current asthma	Asthma	Doctor-diagnosed asthma	BHR	Atopy
n					
Cases	180	345	361	261	715
Noncases	893	1121	1103	812	423
Age (y)[2]					
Cases	33.3 ± 7.5[3]	33.8 ± 7.2[3]	33.5 ± 7.1[3]	34.2 ± 7.5[4]	34.8 ± 7.3
Noncases	35.4 ± 7.3	34.9 ± 7.0	34.9 ± 7.1	35.3 ± 7.3	35.4 ± 7.3
Sex (% male)					
Cases	48.3	45.5	44.7	43.1[4]	54.3[3]
Noncases	49.4	46.3	46.5	51.2	36.8
BMI (kg/m^2)[2]					
Male cases	26.8 ± 3.9	26.8 ± 3.9	26.3 ± 3.8	26.8 ± 4.1	26.5 ± 3.9
Male noncases	26.5 ± 3.9	26.4 ± 4.0	26.6 ± 4.1	26.5 ± 3.9	26.5 ± 4.2
Female cases	26.2 ± 5.1[4]	25.7 ± 4.9	25.5 ± 5.0	25.4 ± 4.6	25.4 ± 4.8
Female noncases	25.1 ± 4.7	25.2 ± 4.7	25.3 ± 4.7	25.3 ± 4.9	25.4 ± 4.7
Atopy (%)					
Cases	90.0[3]	79.9[3]	85.3[3]	84.0[3]	100
Noncases	57.4	57.2	54.7	56.0	0.0
Ever smoked (%)					
Cases	47.8	45.0	45.7	46.2	41.6[4]
Noncases	43.8	45.2	45.0	43.9	48.3
Current smokers (%)					
Cases	25.6	25.1	21.6	22.9	20.1
Noncases	20.6	21.3	22.4	20.9	22.9
FEV_1 (% of predicted)[2]					
Cases	100.6 ± 15.1[3]	103.9 ± 15.6[3]	104.5 ± 16.1[3]	101.4 ± 14.4[3]	108.5 ± 14.7[3]
Noncases	111.3 ± 13.2	111.4 ± 13.1	111.4 ± 12.9	112.2 ± 13.0	111.4 ± 13.0
Family history of asthma (%)					
Cases	51.7[3]	53.1[3]	55.8[3]	51.5[3]	43.8[4]
Noncases	39.1	35.6	34.7	37.9	36.8
Born in Australia (%)					
Cases	90.0[4]	88.0[3]	87.8[3]	87.0	84.7
Noncases	82.6	81.6	81.6	82.8	83.7

[1] Case definitions are not mutually exclusive. The total numbers of cases and noncases may not be exactly the same as in Subjects and Methods because of missing data. BHR, bronchial hyperreactivity; FEV_1, forced expiratory volume in 1 s.
[2] $\bar{x}$ ± SD.
[3,4] Significantly different from noncases: [3] $P \leq 0.01$, [4] $P < 0.05$.

sion models were fitted separately for each nutrient, after the inclusion of total energy.

We expected that any dietary exposure that had a strong biological association with asthma would be detected across several outcomes in our analysis, but that the size of effect and significance might vary because of limitations such as measurement error, misdiagnosis, and person-to-person biological variability. Therefore, we were interested in consistent patterns of findings across several outcomes. Because of this, we chose not to perform further adjustments for multiple comparisons (27).

RESULTS

The respiratory questionnaire was completed by 1601 young adults ($\bar{x}$ ± SD age: 34.6 ± 7.1 y), the food-frequency questionnaire by 1469, the methacholine challenge for BHR by 1075, and skin-prick testing by 1141. Overall, 16.7% (n = 180) of the subjects met our definition of current asthma, 23.5% (n = 345) were defined as having asthma, 24.7% (n = 361) were defined as having doctor-diagnosed asthma, 24.3% (n = 261) had BHR, and 62.8% (n = 715) were atopic. The subjects' characteristics, comparing those with and without current asthma, asthma, doctor-diagnosed asthma, and atopy, are summarized in **Table 1**. Unsurprisingly, those meeting any of the case definitions were more likely to be atopic, to have worse lung function, and to have a family history of asthma than were the corresponding control subjects. Women with current asthma were slightly more obese than were those without [mean body mass index (in kg/m^2) of 26.2 compared with 25.1; P = 0.04].

Relation between foods and asthma

There were no significant univariate associations between current asthma or asthma and any of the food variables (**Table 2**). However, those with doctor-diagnosed asthma were significantly less likely to consume butter (Table 2) than were those without doctor-diagnosed asthma. Atopic subjects consumed fewer tomatoes than did nonatopic subjects (mean = 0.57 daily equivalents compared with 0.64; P = 0.01). Subjects with BHR were less likely to consume whole milk (26% compared with 36%; P = 0.002) and were more likely to consume low-fat cheese (19.1% compared with 11.2%; P = 0.002) than were those without BHR. However, the overall quantity of dairy products consumed (data not shown), or of alternatives such as soy beverage, was not significantly different for any outcome.

TABLE 2
Usual (or daily) consumption of selected foods, by subgroups[1]

Food item	Current asthma (*n* = 180 cases, 893 noncases)	Asthma (*n* = 345 cases, 1121 noncases)	Doctor-diagnosed asthma (*n* = 361 cases, 1103 noncases)	Not diagnosed asthma (*n* = 1103)	BHR (*n* = 261 cases, 812 noncases)	Atopy (*n* = 715 cases, 423 noncases)
	%					
Rice (daily)	5.5	7.2	6.6	8.9	3.1	7.9
White bread	33.3	37.6	34.0	40.5	36.6	37.8
Red meat (daily)	54.4	54.3	50.8	56.5	55.7	55.6
Dairy products						
<250 mL milk/d	43.3	44.8	41.0	44.2	45.0	45.6
Other dairy (daily)[2]	60.6	62.9	62.7	64.2	63.0	62.8
Whole milk	25.6	33.0	28.7	35.4	26.0	32.2
Soy beverage	11.7	9.2	9.4	6.7	10.3	7.9
Ricotta cheese	15.0	8.6	9.9	8.0	13.4	9.6
Low-fat cheese	16.7	16.1	17.1	12.2	19.1	13.4
Butter	8.9	10.9	8.6	13.8[3]	9.2	11.9
Fish						
Total (daily)	55.0	54.3	56.1	52.8	55.3	54.4
Nonfried (daily)	32.2	31.0	30.7	33.6	32.4	31.2
Fried (daily)	35.0	36.2	39.2	33.5	35.9	36.3
Fruit						
1–2 pieces/d	40.6	36.2	38.7	38.6	40.8	40.3
Apples and pears (daily)	41.7	44.0	49.7	47.2	44.7	47.3
Berries (daily)	30.6	30.2	30.7	29.2	26.3	28.0
Vegetables						
2–4 servings/d	56.1	58.6	59.4	58.5	56.9	58.0
Green leafy (daily)	31.7	33.9	35.6	34.5	33.2	34.2
Tomatoes (daily)	32.2	33.0	31.8	31.6	29.8	27.9

[1] BHR, bronchial hyperreactivity.
[2] Other dairy includes cheese, ice cream, yogurt.
[3] Significantly different from doctor-diagnosed asthma, $P = 0.008$ (*see* text).

When the food-asthma associations were adjusted for the effects of other known potential risk factors (age, sex, smoking, body mass index, region of birth, and family history of asthma), significant associations with current asthma and the alternate definitions of asthma and atopy emerged (**Table 3**). The major consistent findings across outcomes were the associations for the types of dairy products consumed. The consumption of whole milk was negatively associated with current asthma ($P = 0.03$), doctor-diagnosed asthma ($P = 0.05$), BHR ($P = 0.014$), and atopy ($P = 0.02$). Butter consumption was also negatively associated with doctor-diagnosed asthma ($P = 0.006$) and BHR ($P = 0.04$).

Conversely, the consumption of soy beverage was positively associated with an increased risk of current asthma ($P = 0.01$), doctor-diagnosed asthma ($P = 0.04$), and BHR ($P = 0.05$). Similarly, current asthma and BHR were positively associated with ricotta cheese consumption ($P = 0.009$ and 0.04, respectively). The consumption of low-fat cheese was positively associated with doctor-diagnosed asthma ($P = 0.04$) and BHR ($P = 0.007$). However, type of margarine was not significantly associated with any of the asthma outcomes or atopy (data not shown).

Among fruit and vegetable intakes, the consumption of apples and pears was negatively associated with current asthma ($P = 0.02$), asthma ($P = 0.05$), and BHR ($P = 0.05$). The consumption of tomatoes ($P = 0.04$) was negatively associated with atopy despite the fact that atopic subjects consumed fewer tomatoes than did those without atopy (Table 3). Other isolated findings for fruit, vegetables, fish, red meat, and rice were not consistent across different outcomes (Table 3).

Relation between nutrients and asthma

There were no significant univariate associations between any of the nutrients examined and current asthma, asthma, doctor-diagnosed asthma, BHR, or atopy (**Table 4**). When the nutrient-asthma associations were adjusted for the effects of other known and potential risk factors (age, sex, smoking, body mass index, region of birth, family history of asthma, and total energy), no consistent associations with any of the asthma or atopy definitions emerged (**Table 5**). No consistent associations were found between intakes of β-carotene, retinol, vitamin C, vitamin E, or sodium and current asthma, asthma, BHR, or atopy (Table 5). Isolated findings were that retinol and sodium appeared to be negatively associated with asthma ($P = 0.03$) and atopy ($P = 0.05$), respectively. Other isolated findings for cholesterol, carbohydrate, fiber, folate, and alcohol were not consistent across the asthma outcomes.

DISCUSSION

This is the first study in which associations of both food and nutrients with asthma have been reported. Our results indicate that in young adults some whole foods, but not nutrients, are associated with current asthma, alternate definitions of asthma, and atopy.

One of the strengths of this study was our ability to examine nutrient intake in relation to several asthma outcomes. Previous

TABLE 3
Adjusted odds ratios (and 95% CIs) for selected associations between foods and asthma and atopy[1]

Food item	Current asthma (n = 180 cases, 893 noncases)	Asthma (n = 345 cases, 1121 noncases)	Doctor-diagnosed asthma (n = 361 cases, 1103 noncases)	BHR (n = 261 cases, 812 noncases)	Atopy (n = 715 cases, 423 noncases)
Rice	0.57 (0.31, 1.01)	0.97 (0.68, 1.40)	0.77 (0.52, 1.15)	0.51 (0.31, 0.84)[2]	1.00 (0.72, 1.40)
White bread	0.72 (0.51, 1.02)	0.82 (0.62, 1.10)	0.70 (0.52, 0.93)[2]	0.91 (0.68, 1.23)	0.81 (0.63, 1.05)
Red meat	0.93 (0.85, 1.03)	0.93 (0.86, 1.01)	0.91 (0.84, 0.99)[2]	0.96 (0.88, 1.04)	1.00 (0.93, 1.07)
Dairy products					
Milk	1.01 (0.59, 1.71)	0.81 (0.51, 1.28)	1.22 (0.79, 1.86)	1.41 (0.90, 2.21)	1.25 (0.81, 1.92)
Other dairy[3]	1.06 (0.94, 1.19)	1.11 (1.00, 1.23)[2]	1.08 (0.97, 1.19)	1.01 (0.91, 1.12)	0.97 (0.88, 1.07)
Whole milk	0.66 (0.46, 0.97)[2]	1.04 (0.77, 1.40)	0.73 (0.54, 0.99)[2]	0.68 (0.48, 0.92)[2]	0.71 (0.54, 0.94)[2]
Soy beverage	2.05 (1.19, 3.53)[2]	1.53 (0.92, 2.53)	1.69 (1.04, 2.77)[2]	1.65 (1.00, 2.71)[2]	1.60 (0.96, 2.67)
Ricotta cheese	1.94 (1.18, 3.19)[2]	1.07 (0.67, 1.71)	1.45 (0.93, 2.26)	1.62 (1.04, 2.55)[2]	1.20 (0.78, 1.84)
Low-fat cheese	1.29 (0.82, 2.03)	1.30 (0.88, 1.92)	1.50 (1.03, 2.19)[2]	1.72 (1.16, 2.53)[2]	1.02 (0.70, 1.48)
Butter	0.63 (0.36, 1.10)	0.45 (0.18, 1.14)	0.52 (0.32, 0.82)[2]	0.61 (0.38, 0.98)[2]	0.77 (0.54, 1.11)
Fish					
Total	0.98 (0.88, 1.10)	1.02 (0.93, 1.12)	1.06 (0.96, 1.16)	0.99 (0.90, 1.09)	0.98 (0.90, 1.07)
Nonfried	0.98 (0.84, 1.14)	1.00 (0.87, 1.13)	0.98 (0.86, 1.11)	0.99 (0.86, 1.18)	0.92 (0.82, 1.03)
Fried	1.00 (0.83, 1.19)	1.06 (0.91, 1.2 3)	1.18 (1.02, 1.36)[2]	0.99 (0.85, 1.16)	1.07 (0.94, 1.23)
Fruit					
Total	0.99 (0.95, 1.03)	0.99 (0.96, 1.02)	1.01 (0.98, 1.05)	0.98 (0.95, 1.01)	0.98 (0.95, 1.01)
Apples and pears	0.83 (0.71, 0.98)[2]	0.88 (0.78, 1.00)[2]	0.99 (0.88, 1.12)	0.88 (0.77, 1.00)[2]	0.99 (0.89, 1.11)
Berries	1.09 (0.90, 1.33)	1.03 (0.87, 1.22)	1.11 (0.94, 1.31)	0.95 (0.79, 1.13)	0.86 (0.74, 1.00)[2]
Vegetables					
Total	1.00 (0.97, 1.03)	0.99 (0.97, 1.02)	1.00 (0.97, 1.02)	0.99 (0.97, 1.02)	0.99 (0.97, 1.02)
Green leafy	0.82 (0.67, 1.00)[2]	0.90 (0.76, 1.06)	0.99 (0.85, 1.16)	0.86 (0.72, 1.02)	0.91 (0.78, 1.05)
Tomatoes	1.14 (0.93, 1.39)	1.10 (0.92, 1.31)	1.16 (0.97, 1.38)	1.07 (0.89, 1.29)	0.84 (0.71, 0.99)[2]

[1] Food intake was calculated as the response category obtained from the questionnaire, which was adjusted for portion size at an individual level and multiplied by the frequency in daily equivalents. Multivariate models included age, sex, smoking, BMI, region of birth, family history of asthma, and the specified food variable. The reference categories were those without current asthma, asthma, doctor-diagnosed asthma, bronchial hyperreactivity (BHR), or atopy, respectively.

[2] $P < 0.05$.

[3] Other dairy includes cheese, ice cream, and yogurt.

studies generally relied on only self-reported data or laboratory data. We used self-reported, epidemiologic, and physiologic definitions of asthma. However, different definitions of asthma do lead to different results, highlighting the importance of careful consideration of the outcome to be used when looking for diet-asthma associations.

The associations between dairy products and asthma definitions were unexpected. We did not expect to find that whole milk and butter would be negatively associated with asthma, nor did we expect that the consumption of soy beverage, ricotta cheese, and low-fat cheese would be positively associated with asthma. Because these associations were found across the range of asthma definitions used in this study, we do not believe that these are chance findings. However, the results of previous intervention trials of dairy product consumption and asthma outcomes were negative (28–31).

One possible explanation for our findings is that persons with asthma may change the types of dairy products they consume after their asthma is diagnosed (eg, change from drinking whole milk to soy beverage and to the use of lower fat cheeses where possible). This explanation is also consistent with our findings in that the overall intake of dairy products was not significantly different between those with and without asthma. Furthermore, we showed previously that > 50% of persons with asthma modify their food intake and that dairy products are the food item most commonly avoided (32). We think it unlikely that more of the asthmatic subjects had milk allergy before their asthma was diagnosed. We found previously that the prevalence of positive results on a skin test to cow milk was only 0.7% among young adults, and there was little association with food-related symptoms (33).

The results of some studies have suggested that antioxidants, particularly vitamins C and E, may protect against the development of asthma (3, 8, 34–37). The Nurses' Health Study (8) found that a high dietary intake of vitamin E is associated with a significantly reduced risk of late-onset asthma. Although this was a large, well-designed cohort study, asthma was defined by self-report only and no information was presented on atopy. Our results do not support the hypothesis that these nutrients affect asthma prevalence in adults.

Many experts believe that persons with high fruit and vegetable intakes (and therefore a high intake of antioxidants) have a lower risk of cancer and cardiovascular diseases. However, it is difficult to wholly attribute the protective effect to antioxidants alone, because fruit and vegetables contain many other constituents, some of which may have not been studied (38). Indeed, although the epidemiologic evidence for a beneficial effect of eating fruit and vegetables on asthma is increasing, the effectiveness of dietary supplementation has often not been shown (39). Our results are consistent with this conclusion: we found that the consumption of whole fruit, in the form of apples and pears, did protect against asthma, whereas antioxidant nutrients were not associated with asthma. However, we did not find a significant association between

TABLE 4
Median nutrient intakes, by subgroup[1]

Nutrient	Current asthma (n = 180 cases, 893 noncases)	Asthma (n = 345 cases, 1121 noncases)	Doctor-diagnosed asthma (n = 361 cases, 1103 noncases)	Not diagnosed asthma (n = 1103)	BHR (n = 261 cases, 812 noncases)	Atopy (n = 715 cases, 423 noncases)
Energy (kJ)	8544 (4000)	8416 (4115)	8326 (4339)	8164 (4006)	8367 (4115)	8589 (4141)
Total fat (g)	75.9 (47.2)	73.5 (42.1)	73.1 (47.5)	74.2 (43.9)	75.4 (45.5)	77.4 (47.8)
Saturated fat (g)	30.0 (18.3)	29.4 (17.8)	29.2 (18.9)	29.5 (18.5)	29.5 (18.2)	30.6 (20.0)
Polyunsaturated fat (g)	11.5 (8.6)	10.7 (8.7)	11.3 (8.9)	11.2 (8.1)	11.5 (8.4)	11.5 (8.5)
Monounsaturated fat (g)	27.2 (18.0)	25.5 (15.3)	25.1 (16.9)	26.2 (16.2)	26.6 (16.5)	27.2 (17.5)
Protein (g)	91.3 (47.2)	88.0 (48.9)	88.3 (49.1)	87.5 (43.4)	88.8 (44.4)	91.9 (45.8)
Carbohydrate (g)	219 (104)	208 (97.4)	208 (117)	210 (107)	215 (104)	220 (113)
Fiber (g)	21.9 (11.1)	21.3 (11.0)	22.0 (11.6)	21.1 (11.0)	21.7 (11.5)	22.0 (11.4)
Alcohol (g)	13.8 (23.6)	11.5 (23.8)	10.3 (21.5)	10.6 (21.4)	12.9 (21.3)	11.9 (23.3)
β-Carotene (μg)	2234 (1817)	2238 (1748)	2231 (1774)	2146 (1466)	2216 (1691)	2236 (1545)
Calcium (mg)	892 (365)	868 (400)	890 (439)	877 (390)	898 (378)	894 (366)
Cholesterol (mg)	250 (156)	243 (155)	246 (169)	253 (148)	246 (147)	263 (161)
Folate (μg)	278 (163)	272 (158)	283 (164)	270 (142)	278 (164)	281 (149)
Iron (mg)	13.4 (7.1)	12.8 (7.4)	13.1 (7.3)	12.7 (6.8)	13.3 (7.4)	13.4 (6.8)
Magnesium (mg)	308 (133)	298 (143)	301 (139)	297 (138)	307 (127)	305 (139)
Niacin (mg)	23.3 (15.0)	22.7 (14.2)	22.9 (13.5)	21.9 (12.3)	22.4 (14.3)	23.2 (13.0)
Phosphorus (mg)	1583 (740)	1506 (737)	1523 (754)	1515 (681)	1561 (704)	1577 (699)
Potassium (mg)	2882 (1125)	2794 (1410)	2833 (1330)	2768 (1244)	2845 (1145)	2872 (1243)
Retinol (mg)	344 (276)	348 (260)	346 (263)	360 (249)	350 (260)	361 (249)
Riboflavin (mg)	2.42 (1.28)	2.31 (1.28)	2.36 (1.32)	2.27 (1.28)	2.42 (1.34)	2.39 (1.28)
Sodium (mg)	2894 (1477)	2685 (1371)	2667 (1472)	2608 (1367)	2792 (1466)	2750 (1410)
Thiamine (mg)	1.60 (0.94)	1.57 (0.92)	1.58 (0.92)	1.52 (0.86)	1.58 (0.94)	1.59 (0.89)
Vitamin C (mg)	116 (82.8)	119 (87.2)	121 (94.6)	116 (90.9)	116 (90.2)	122 (91.9)
Vitamin E (mg)	6.35 (3.45)	6.14 (3.46)	6.33 (3.67)	6.24 (3.32)	6.36 (3.44)	6.50 (3.55)
Zinc (mg)	11.6 (6.3)	11.1 (6.1)	11.2 (6.2)	11.0 (5.8)	11.3 (6.1)	11.6 (6.0)

[1] Interquartile range (75th percentile − 25th percentile) in parentheses. BHR, bronchial hyperreactivity. There were no significant univariate associations between any of the nutrients and the case definitions.

total fruit and vegetable intake and asthma. Alternatively, fruit and vegetable intake may be a marker of other lifestyle factors (eg, exercise) that were not assessed.

We did not find an association between fish intake and asthma. A large epidemiologic study conducted in the United States (the second National Health and Nutrition Examination Survey) also failed to find an independent significant association between fish intake and respiratory symptoms in adults (40). However, only self-reported outcome measures of wheezing and chronic bronchitis, and not asthma, were used in that analysis. Conversely, a protective association between oily fish intake and asthma was found in Australian schoolchildren (5), but a subsequent intervention study by the same group failed to find any change in asthma severity in children after dietary fat manipulation (41). Furthermore, we were also unable to confirm previously reported associations between the intake of polyunsaturated fatty acid margarine and asthma (11).

Similarly, the lack of association between sodium intake and asthma is consistent with some previous studies (42–44) but not others (13, 45, 46). However, although we did not find a statistically significant association, there did appear to be a trend toward sodium intake being positively associated with the asthma definitions, with odds ratios of ≈3. Interestingly, the trend was for a negative association with atopy. Devereux et al (47) found a relation between urinary sodium excretion and BHR only in an urban sample of Newcastle men and not in rural West Cumbria or shipyard workers. A well-controlled clinical trial found that an increase in dietary sodium from 80 to 200 mmol/d was associated with a 0.73 doubling dose increase in bronchial reactivity to methacholine (45). Another clinical study concluded that patients with BHR have increased sodium influx into cells stimulated by a serum factor (46). A systematic review of controlled clinical trials of salt intake and asthma found no significant differences in asthma outcomes between persons consuming low-salt or normal- and high-salt diets and concluded that the evidence is currently insufficient to advise on whether dietary salt reduction has any place in the management of asthma (48).

Given the large number of comparisons made in the present study, some of our statistically significant findings may have been due to chance alone. Unfortunately, it is not really possible to determine which findings are due to chance. However, given that the associations between dairy products, soy beverage, and apples and pears were reasonably consistent across all the outcome measures, we are confident that these represent real underlying patterns of association. We also acknowledge that this was a cross-sectional study and therefore our results do not indicate cause and effect. It is important that the results are interpreted within this context and that the appropriate longitudinal and intervention studies are conducted to verify our findings. In particular, if possible, it would be of interest to assess usual dietary intake before any asthma diagnosis and again after diagnosis to ascertain whether the interpretation of our findings can be verified.

In conclusion, we found evidence that food but not nutrient intake is associated with current asthma and alternate definitions of asthma and atopy in young adults. Given that this was a cross-sectional

TABLE 5
Adjusted odds ratios (and 95% CIs) for nutrients and alternate definitions of asthma and atopy[1]

Nutrient	Current asthma (n = 180 cases, 893 noncases)	Asthma (n = 345 cases, 1121 noncases)	Doctor-diagnosed asthma (n = 361 cases, 1103 noncases)	BHR (n = 261 cases, 812 noncases)	Atopy (n = 715 cases, 423 noncases)
Energy (kJ)	1.03 (0.62, 1.73)	0.97 (0.63, 1.50)	1.06 (0.69, 1.62)	0.97 (0.62, 1.52)	0.71 (0.48, 1.05)
Total fat (g)	0.61 (0.25, 1.46)	0.56 (0.26, 1.20)	0.55 (0.26, 1.15)	0.88 (0.41, 1.92)	0.76 (0.37, 1.53)
Saturated fat (g)	0.66 (0.35, 1.25)	0.71 (0.41, 1.24)	0.58 (0.34, 1.00)	0.86 (0.49, 1.50)	0.98 (0.60, 1.61)
Polyunsaturated fat (g)	1.02 (0.63, 1.64)	0.75 (0.50, 1.12)	1.00 (0.67, 1.49)	1.11 (0.73, 1.68)	0.90 (0.62, 1.29)
Monounsaturated fat (g)	0.73 (0.33, 1.59)	0.68 (0.35, 1.34)	0.60 (0.31, 1.17)	0.89 (0.45, 1.77)	0.59 (0.31, 1.11)
Protein (g)	1.25 (0.41, 3.85)	1.14 (0.44, 2.96)	1.45 (0.57, 3.71)	1.08 (0.41, 2.87)	1.14 (0.48, 2.67)
Carbohydrate (g)	1.63 (0.49, 5.38)	1.00 (0.37, 2.76)	3.70 (1.34, 10.19)[2]	1.22 (0.43, 3.45)	1.44 (0.58, 3.60)
Fiber (g)	1.18 (0.62, 2.22)	0.96 (0.56, 1.64)	1.72 (1.01, 2.94)[2]	1.02 (0.58, 1.77)	1.05 (0.64, 1.70)
Alcohol (g)	1.11 (0.96, 1.28)	1.07 (0.95, 1.21)	0.94 (0.83, 1.05)	1.04 (0.92, 1.18)	1.01 (0.91, 1.12)
β-Carotene (μg)	1.04 (0.72, 1.50)	1.24 (0.91, 1.69)	1.32 (0.97, 1.80)	1.04 (0.75, 1.43)	1.06 (0.80, 1.40)
Calcium (mg)	1.08 (0.58, 2.02)	0.73 (0.43, 1.23)	1.07 (0.64, 1.78)	1.58 (0.92, 2.73)	1.24 (0.77, 1.99)
Cholesterol (mg)	0.58 (0.34, 1.00)	0.84 (0.52, 1.34)	0.67 (0.43, 1.07)	0.61 (0.38, 0.99)[2]	1.04 (0.68, 1.60)
Folate (μg)	1.07 (0.54, 2.12)	1.41 (0.79, 2.51)	2.21 (1.24, 3.94)[2]	1.11 (0.61, 2.00)	0.96 (0.57, 1.61)
Iron (mg)	1.26 (0.57, 2.80)	0.83 (0.42, 1.63)	1.82 (0.95, 3.49)	1.34 (0.67, 2.67)	0.95 (0.52, 1.75)
Magnesium (mg)	1.46 (0.54, 3.96)	0.98 (0.42, 2.25)	1.94 (0.86, 4.38)	1.39 (0.58, 3.30)	1.33 (0.62, 2.84)
Niacin (mg)	1.12 (0.47, 2.64)	1.63 (0.79, 3.35)	1.01 (0.99, 1.03)	0.84 (0.40, 1.78)	0.90 (0.47, 1.71)
Phosphorus (mg)	1.63 (0.53, 5.04)	0.72 (0.28, 1.86)	1.39 (0.55, 3.51)	1.57 (0.59, 4.17)	1.27 (0.54, 2.99)
Potassium (mg)	0.85 (0.32, 2.27)	1.01 (0.45, 2.28)	1.92 (0.86, 4.29)	1.04 (0.44, 2.44)	1.31 (0.62, 2.75)
Retinol (mg)	0.77 (0.54, 1.10)	0.71 (0.53, 0.96)[2]	0.81 (0.60, 1.09)	0.96 (0.70, 1.31)	1.01 (0.76, 1.33)
Riboflavin (mg)	1.16 (0.64, 2.11)	1.00 (0.60, 1.65)	1.34 (0.81, 2.20)	1.37 (0.81, 2.31)	1.06 (0.68, 1.68)
Sodium (mg)	3.27 (0.89, 11.93)	2.12 (0.71, 6.27)	2.82 (0.97, 8.19)	2.36 (0.78, 7.17)	0.37 (0.14, 0.99)[2]
Thiamine (mg)	1.20 (0.63, 2.29)	1.17 (0.68, 2.03)	1.71 (1.00, 2.94)	1.15 (0.66, 2.02)	0.83 (0.51, 1.37)
Vitamin C (mg)	0.93 (0.65, 1.32)	1.19 (0.88, 1.60)	1.33 (0.99, 1.78)	0.96 (0.71, 1.31)	0.97 (0.74, 1.27)
Vitamin E (mg)	0.89 (0.43, 1.85)	0.62 (0.33, 1.15)	1.20 (0.65, 2.22)	1.26 (0.67, 2.38)	1.01 (0.58, 1.77)
Zinc (mg)	1.28 (0.47, 3.45)	1.02 (0.44, 2.36)	1.64 (0.71, 3.76)	1.43 (0.60, 3.41)	1.20 (0.56, 2.57)
Alcohol risk categories[3]					
Low	0.56 (0.26, 1.23)	0.70 (0.36, 1.36)	0.49 (0.26, 0.91)[2]	0.59 (0.30, 1.19)	1.15 (0.61, 2.16)
Moderate	0.66 (0.29, 1.47)	0.70 (0.35, 1.40)	0.46 (0.24, 0.88)[2]	0.64 (0.31, 1.31)	0.94 (0.48, 1.81)
High	0.49 (0.20, 1.89)	0.70 (0.34, 1.47)	0.29 (0.14, 0.60)[2]	0.50 (0.23, 1.08)	0.89 (0.45, 1.79)
Highest	0.98 (0.36, 2.63)	1.47 (0.62, 3.47)	0.65 (0.28, 1.52)	0.87 (0.35, 2.17)	1.53 (0.63, 3.68)

[1] BHR, bronchial hyperreactivity. Multivariate models included age, sex, smoking, BMI, region of birth, family history of asthma, energy, and the specified nutrient.

[2] $P < 0.05$.

[3] For alcohol categories, "never have alcohol" was the reference group (*see* text for definitions of alcohol categories).

study, we were unable to establish a cause-and-effect relation for the food-asthma associations that were found. Dietary modification after diagnosis may be a possible explanation of our findings, or these dietary factors might be a marker of other lifestyle factors (eg, exercise) that were not assessed. Future research is required to ascertain whether modification of food intake may be important in the prevention of asthma.

We acknowledge Cathryn Wharton, who assisted with data collection and data cleaning; Judi Wicking, who assisted with the data collection phase of the study; and the Cancer Council of Victoria, for optical scanning of the food-frequency questionnaires.

RKW was involved in study design, obtaining ethics approval and funding, data analysis, and writing of the manuscript. EHW, PDI, FCKT, and MJA were chief investigators of the study and were involved in the design of the experiment, obtaining funding, and reviewing the manuscript. JMR was involved in study design, data collection, and reviewing the manuscript. RW and MJA assisted with the statistical analysis of the data and revision of the manuscript. No author had any financial or personal relation with any of the sponsoring organizations.

REFERENCES

1. Bauman A, Mitchell CA, Henry RL, et al. Asthma morbidity in Australia: an epidemiological study. Med J Aust 1992;156:827–31.
2. Abramson M, Kutin J, Bowes G. The prevalence of asthma in Victorian adults. Aust N Z J Med 1992;22:358–63.
3. Seaton A, Godden DJ, Brown K. Increase in asthma: a more toxic environment or a more susceptible population? Thorax 1994;49:171–4.
4. Weiss ST. Diet as a risk factor for asthma. In: Chadwick DJ, Cardew G, eds. The rising trends in asthma. Ciba Foundation Symposium 206. Chichester, United Kingdom: John Wiley & Sons, 1997:244–57.
5. Hodge L, Salome CM, Peat JK, Haby MM, Xuan W, Woolcock AJ. Consumption of oily fish and childhood asthma risk. Med J Aust 1996;164:137–40.
6. Thien FC, Woods RK, Walters EH. Oily fish and asthma—a fishy story? Further studies are required before claims can be made of a beneficial effect of oily fish consumption on asthma. Med J Aust 1996;164:135–6.
7. Schwartz J. Role of polyunsaturated fatty acids in lung disease. Am J Clin Nutr 2000;71(suppl):393S–6S.
8. Troisi RJ, Willett WC, Weiss ST, Trichopoulos D, Rosner B, Speizer FE. A prospective study of diet and adult onset asthma. Am J Respir Crit Care Med 1995;151:1401–8.
9. Monteleone CA, Sherman AR. Nutrition and asthma. Arch Intern Med 1997;157:23–34.
10. Hodge L, Peat JK, Salome C. Increased consumption of polyunsaturated oils may be a cause of increased prevalence of childhood asthma. Aust N Z J Med 1994;24:727 (letter).

11. Haby MM, Peat JK, Marks GB, Woolcock AJ, Leeder SR. Asthma in preschool children: prevalence and risk factors. Thorax 2001;56:589–95.
12. Woods RK, Thien FCK. Polyunsaturated fats and childhood asthma. Thorax 2002;57:94 (letter).
13. Burney PGJ, Neild JE, Twort CHC, et al. Effect of changing dietary sodium on the airway response to histamine. Thorax 1989;44:36–41.
14. Antonios TFK, McGregor GA. Deleterious effects of salt intake other than effects on blood pressure. Clin Exp Pharmacol Physiol 1995;22:180–4.
15. Woods RK, Walters EH, Wharton C, Watson N, Abramson M. The rising prevalence of asthma in young Melbourne adults is associated with improvement in treatment. Ann Allergy Asthma Immunol 2001;87:117–23.
16. DeMarco R, Zanolin ME, Accordini S, et al on behalf of the ECRHS. A new questionnaire for the repeat of the first stage of the European Respiratory Health Survey (ECRHS). A pilot study in Italy on the influence of the length of the questionnaire on the response rate. Eur Respir J 1999;14:1044–8.
17. Abramson MJ, Hensley MJ, Saunders NA, Wlodarczyk JH . Evaluation of a new asthma symptoms questionnaire. J Asthma 1991;28:129–30.
18. Burney PGJ, Luczynska C, Chinn S, Jarvis D for the European Community Respiratory Health Survey. The European Community Respiratory Health Survey. Eur Respir J 1994;7:954–60.
19. Ireland P, Jolley D, Giles G, et al. Development of the Melbourne FFQ: a food frequency questionnaire for use in an Australian prospective study involving an ethnically diverse cohort. Asia Pac J Clin Nutr 1994;3:19–31.
20. Savige G, Wahlqvist M, Lee D, Snelson B. Agefit: fitness and nutrition for an independent future. Sydney, Australia: Macmillan, 2001:145–7.
21. National Health and Medical Research Council. Draft dietary guidelines for adults. Internet: http://www.health.gov.au/nhmrc/advice/diet.htm (accessed 24 January 2002).
22. Paul AA, Southgate DAT. McCance and Widdowson's the composition of foods. 4th ed. London: Her Majesty's Stationery Office, 1987.
23. Department of Community Services and Health. Composition of foods, Australia volume 1. Canberra, Australia: Australian Government Publishing Service 1989.
24. Haahtela T. Skin tests used for epidemiologic studies. Allergy 1993;48(suppl):76–80.
25. Backman A. Skin tests for epidemiologic studies. Allergy 1994;49:493–4.
26. Toelle BG, Peat JK, Salome CM, Mellis CM, Woolcock AJ. Towards a definition of asthma for epidemiology. Am Rev Respir Dis 1992;146:633–7.
27. Savitz DA, Olshan AF. Multiple comparisons and related issues in the interpretation of epidemiological data. Am J Epidemiol 1995;142:904–8.
28. Haas F, Bishop MC, Salazar-Schicchi J, Axen KV, Lieberman D, Axen K. Effect of milk ingestion on pulmonary function in healthy asthmatic subjects. J Asthma 1991;28:349–55.
29. Onorato J, Merland N, Terral C, Michel FB, Bousquet J. Placebo-controlled double-blind food challenge in asthma. J Allergy Clin Immunol 1986;78:1139–46.
30. Woods RK, Weiner J, Thien F, Abramson M, Walters EH. Do dairy products induce bronchoconstriction in adults with asthma? J Allergy Clin Immunol 1998;101:45–50.
31. Nguyen MT. Effect of cow milk on pulmonary function in atopic asthmatic patients. Ann Allergy Asthma Immunol 1997;79:62–4.
32. Woods RK, Weiner J, Thien F, Abramson M, Walters EH. Patients' perceptions of food-induced asthma. Aust N Z J Med 1996;26:504–12.
33. Woods RK, Stoney R, Bailey M, et al. Reported adverse food reactions overestimate true food allergy in the community. Eur J Clin Nutr 2002;56:31–6.
34. Schwartz J, Weiss ST. Relationship between dietary vitamin C intake and pulmonary function in the first National Health and Nutrition Examination Survey (NHANES I). Am J Clin Nutr 1994;59:110–4.
35. Hatch GE. Asthma, inhaled oxidants and dietary antioxidants. Am J Clin Nutr 1995;61(suppl):625S–30S.
36. Bodner C. Dietary antioxidants and adult onset wheeze. Eur Respir J 1996;9(suppl):419S–20S.
37. Soutar A, Seaton A, Brown K. Bronchial reactivity and dietary antioxidants. Thorax 1997;52:166–70.
38. Mackerras D. Antioxidants and health—fruits and vegetables or supplements? Food Australia 1995;47(suppl):S1–21.
39. Smit HA, Grievink L, Tabak C. Dietary influences on chronic obstructive lung disease and asthma: a review of the epidemiological evidence. Proc Nutr Soc 1999;58:309–19.
40. Schwartz J, Weiss ST. Dietary factors and their relation to respiratory symptoms. Am J Epidemiol 2000;132:67–76.
41. Hodge L, Salome CM, Hughes JM, et al. Effect of dietary intake of omega-3 and omega-6 fatty acids on severity of asthma. Eur Respir J 1998;11:361–5.
42. Britton JR, Pavord I, Richards K, et al. Dietary sodium intake and the risk of airway hyperreactivity in a random adult population. Thorax 1994;49:875–80.
43. Zoia MC, Fanfulla F, Bruschi C, et al. Chronic respiratory symptoms, bronchial responsiveness and dietary sodium and potassium: a population based study. Monaldi Arch Chest Dis 1995;50:104–8.
44. Lieberman D, Heimer D. Effect of dietary sodium on the severity of bronchial asthma. Thorax 1992;47:360–2.
45. Carey OJ, Locke C, Cookson JB. Effects of alterations of dietary sodium on the severity of asthma in men. Thorax 1993;48:714–8.
46. Tribe RM, Barton JR, Poston L, Burney PG. Dietary sodium intake, airway responsiveness and cellular sodium transport. Am J Respir Crit Care Med 1994;149:1426–33.
47. Devereux G, Beach JR, Bromly C, et al. Effect of dietary sodium on airways responsiveness and its importance in the epidemiology of asthma: an evaluation in three areas of northern England. Thorax 1995;50:941–7.
48. Ardern KD, Ram FSF. Dietary salt reduction or exclusion for allergic asthma (Cochrane Review). In: The Cochrane Library, Issue 4. Oxford, United Kingdom: Update Software, 2001.

APPENDIX A

Food categories

Category	Foods included
Cereals	Breakfast cereals, rice, pasta, bread, crackers, cookies and cake, and savory pastries, pizza, and hamburger
Dairy	Milk and cheese, ice cream, and yogurt
Meats and alternatives	Red meat, chicken, other meats and deli items, eggs, nuts (including peanut butter), legumes, fish (total, fried, grilled, canned, and total nonfried)
Fruit	All, citrus, stone (apricot, peach, and avocado), bananas, berries, melons, apples and pears, tropical (pineapple and mango), other (canned and frozen), and fruit juice
Vegetables	All, root (potato, beetroot, and carrot), cruciferous (broccoli, Brussels sprouts, cauliflower, and cabbage), green leafy (lettuce, endive, and silverbeet), marrow (cucumber, pumpkin, and zucchini), stalks and stems (celery), bulbs (garlic and onion), pods (peas and beans), capsicum, and peppers, shoots (bean sprouts), tomatoes (including canned, dried, sauce, and paste), and mushrooms
Alcohol	All, beer, wine, and other
Other	Chocolate, corn chips, flavored milk, sugar, jam and honey, and Vegemite[1]

[1] Kraft Foods, Port Melbourne, Australia.