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Topical Review

Diet and asthma

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The role of food intolerance in asthma is well recognized, and where food avoidance measures are instituted considerable improvement in asthma symptoms and in reduction in drug therapy and hospital admissions can result. These benefits may have a greater impact in those patients with greater symptoms. However, the promise of such benefits should not result in an approach which ignores inhaled drug therapy, or in a dietary regime which is inappropriate in the face of mild symptoms. Whilst sub-optimal intake of dietary nutrients is also a recently recognized potential risk factor for asthma, available data are insufficient to implicate any as casual. A number of studies have sought to establish the role of the antioxidant vitamins, A, C and E and selenium, yet others of the elements sodium and magnesium. Sub-optimal nutrient intake may enhance asthmatic inflammation, consequently contributing to bronchial hyperreactivity. Prospective studies of supplementation therapy are needed to confirm this.

Key words: asthma; diet; food intolerance; nutrient intake.

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Introduction

The role of food intolerance in asthma is well recognized but poorly quantified. There are considerable difficulties in recognizing and diagnosing food intolerance and the logistics of doing so has dissuaded many clinicians from trying. In addition, the relatively poor understanding of the mechanisms involved in food intolerance has meant that its very existence is doubted by some. However, where food intolerance is recognized, and food avoidance measures instituted, considerable improvements in asthma symptoms and reductions in drug therapy and hospital admissions can result. It is important to clarify terminology and establish working definitions in this area. Different schemes have been suggested but the one generally accepted in the U.K., and which we use here, is that proposed by the Royal College of Physicians (1).

As well as food intolerance in asthma, other dietary factors should be considered. The potential role of certain nutrients has recently been recognized and emerging evidence suggests that a relationship may exist between suboptimal intakes and lung disease (2). The influence of dietary factors on inflammatory and antioxidant activity, and hence the development of chronic respiratory symptoms, has been examined (3–6) as have the antioxidants selenium (7,8) and vitamins A (9), C (10,11) and E (12,13) and the trace elements sodium (14–18) and magnesium (19,20).

We examine here the work done, both in food intolerance and nutrient intake in asthma, and make recommendations for current clinical practice and further research.

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Food intolerance

EPIDEMIOLOGY

Most studies of the prevalence of food intolerance are questionnaire based relying on self-reported perceived adverse responses to food. The European Community Respiratory Health Survey (ECRHS) employed a validated questionnaire (21), which has been used in epidemiological studies (22,23), but there are scant data on the reproducibility of different questionnaires used in other studies. Objective measures of food intolerance give a better idea of the true prevalence but this involves challenge of each patient with a range of foodstuffs. The double-blind placebo-controlled food challenge (DBPCFC) is considered to be the 'gold standard' (24) procedure to confirm or refute a diagnosis of food intolerance, but the logistics of this are substantial and it cannot be used as an epidemiological tool. It should also be recognized that, even in an individual with proven food intolerance, the response to ingestion of a

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particular food may vary over time. Because of this problem, estimates of the prevalence of adverse reactions to food should be regarded with caution unless they are supported by an objective measure.

Food intolerance and mild-moderate asthma

A number of studies have been conducted in a range of countries over the last 20 years in subjects with mild–moderate asthma. The populations have been a mix of general populations and selected groups and it is difficult to determine any clear pattern from these figures. In general, rates of self-reported food intolerance in asthma are found to be similar to those in the general population whether considering self-reported or objectively confirmed adverse reactions (Table 1). The discrepancy between perception and objective evidence of an adverse reaction to food is clear, the lowest figures for prevalence being in studies where food challenges have been conducted. Whilst recognizing the substantial logistical difficulties of the DBPCFC, unless it is used as a standard test, or until a simpler test off equivalent sensitivity and specificity is found, it will be difficult to determine the true prevalence of food intolerance in asthma.

Food intolerance and severe asthma

Even less information is available for patients with more severe asthma. In a clinic population of patients with brittle asthma (Birmingham Heartlands Hospital, U. K.), around two thirds reported at least one food which could exacerbate their asthma. Sixty percent of the total clinic population have subsequently undergone food challenge studies and, whether using a protocol of dietary exclusion followed by open food challenges (OFCs) or of dietary exclusion followed by DBPCFCs (34) around 50% patients showed positive responses to one or more foods, which supported the questionnaire findings (Table 2). An adverse response can be immediate (Fig. 1), late (Fig. 2) or a dual response.

These proportions cannot be extrapolated to all cases of asthma at the severe end of the spectrum but the findings would support the hypothesis that the more severe the bronchial hyperresponsiveness the more likely it is that a positive response to food may be demonstrated.

DIAGNOSIS

While some take the view that detection of food intolerance in asthmatic subjects is of limited value (35), some patients, in whom specific foods are identified as being causally related to their asthma, benefit substantially from excluding them from their diet (28,36).

History

Although a positive history of an adverse reaction to a food is helpful in deciding whether an individual patient may

Table 2. Prevalence of food intolerance in brittle asthma

	FCs	DBPCFCs 1 × daily
No. of subjects:	29	18
No. of subjects reacting to at least 1 food:	15 (52%)	12 (66%)
No. of subjects by no. of foods reacted to:		
1	0 (0%)	1 (8%)
2	6 (40%)	2 (15%)
3	4 (27%)	2 (15%)
4	5 (33%)	2 (15%)
5	0 (0%)	3 (23%)
6	0 (0%)	0 (0%)
7	0 (0%)	2 (15%)

Table 1. Prevalence of food intolerance in various populations

Country	Date	Sample size & population			% Self-reported		Confirmation by food challenge
		General population data	Asthma populations	Patients with food intolerance	Food intolerance	Asthma	
Sweden (25)	1978	—	1129	—	24·0	—	No
Switzerland (26)	1983–5	—	—	229	—	24·1	No
Wales (27)	1985	—	72	—	4·0	—	Yes
France (28)	1986	—	300	—	2·0	—	Yes
France (29)	1987	—	67	—	5·8	—	Yes
China (30)	1990	10 144	—	—	4·9	3·8	No
Italy (31)	1992	—	—	1·339	—	2·6	Yes
Australia (32)	1996	—	914	—	45·3	—	No
Turkey (33)	1996	1884	584	—	4·5	13·5	No

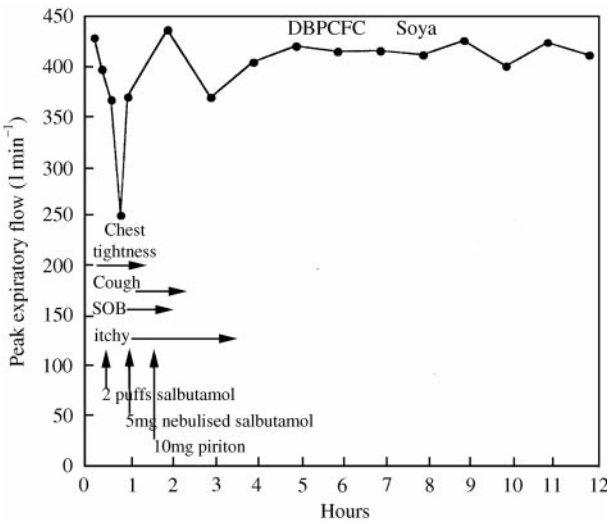

FIG 1. Immediate positive response to DBPBFC with soya in a patient with brittle asthma.

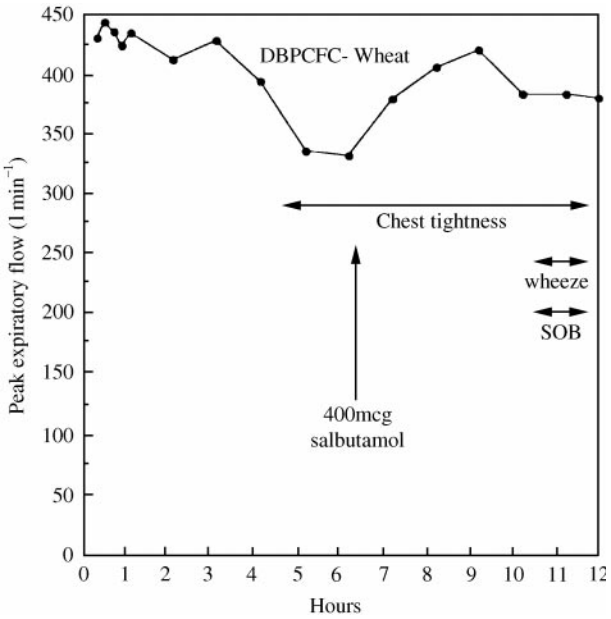

FIG. 2. Late positive response to DBPCFC with wheat in a patient with brittle asthma.

have food intolerance, some subjects with proven food intolerance on challenge give no such history. The main reason for this is that the unrecognized foods are those that are staple in the diet (e.g. wheat, milk/milk products) and presumably act as chronic potentiators of airway inflammation. Where a history is positive, the pattern of symptoms is similar to any other exacerbation of asthma, although there may be associated symptoms in other systems such as headache, abdominal pain and bloating, nasal symptoms and, occasionally, skin rash.

Skin prick tests (SPTs)

Burrows (37) stated that the use of SPTs in respiratory allergic disease should be 'used as an indicator of a subject's atopic predisposition rather than as a guide to the specific cause of his complaints'. This cautious approach was supported by later work (24), which showed them to have poor positive predictability (25–75%) for food induced asthma and they are now acknowledged as not being diagnostic for food intolerance in asthma (38).

Radioallergosorbent tests (RASTs)

These tests have also been shown to have poor positive predictability (24) (0–57%) and are considered to help in the diagnosis of food allergy but, again, not to be diagnostic in themselves (38). Equally, levels of total serum IgE have not been shown to be consistently related to the presence of a positive food challenge.

It is thus inappropriate, on the basis of skin prick tests and/or RASTs alone, to recommend restrictive diets (39).

Histamine Release Test (HRT)

The diagnostic value of the HRT in whole blood of patients with food allergy has been suggested as a diagnostic test for food allergy in asthma (40) but has yet to be tested against DBPCFC.

Top-cast/cast elisa

The cellular antigen stimulation test (CAST) is an enzyme-labelled immunosorbent assay (ELISA) which measures sulphidoleukotriene generation by leukocytes on specific allergen challenge. In one study (41), which aimed to establish the usefulness of the test in differentiating between allergic and non-allergic status, the test yielded a 100% sensitivity and did not produce any false positives. However, it was unable to distinguish between health and disease states and its role in food allergy in relation to asthma remains to be determined.

Methacholine responsiveness

While bronchial hyperresponsiveness to methacholine is well recognized as a marker of asthma in both the laboratory and occupational setting, in food challenge the picture is less clear cut. In one study, methacholine response before, and 24 h after, DBPCFCs in 11 subjects with asthma and a history of food-induced asthma and positive SPTs to the suspect food (42) revealed no difference in response after food challenge compared to placebo food challenge. However, in another study (43), methacholine hyperresponsiveness measured before and 4 h after DBPCFC in 26 food-allergic asthmatic patients, showed a greater increase in airway responsiveness following positive DBPCFC compared to negative DBPCFC ($P = 0{\cdot}03$),

without inducing acute asthma. The discrepancy between the findings of the two studies may be due to differences in study design, the first lacking environmental controls and the latter being conducted with children. In adults it still has to be shown that bronchial hyperresponsiveness is a true marker of food intolerance.

Dietary manipulation

As no single laboratory test has yet been devised to identify food intolerance or even provide a broad screening test, dietary manipulation is considered to be the cornerstone of diagnosis and treatment. The method of choice for investigators is usually that outlined by Bock *et al.* (44) which comprises three main components:

(i) *A period of dietary exclusion prior to food challenge (45)*. The length of time for such exclusion varies. Up to 14 days (38) may be necessary in order to stabilize a patient's symptoms prior to food challenge, although 5 days may be adequate (34). 'Few Foods' Diets (46) (providing the patients with a minimal number of hypoallergenic foods to provide optimal nutrition) are usually used during the period of dietary exclusion. Alternatively, a hypoallergenic formula drink (Elemental 028 Extra Liquid, Scientific Hospital Supplied Ltd., U.K.) and water can be used instead of 'normal' food and drinks.

(ii) *A DBPCFC methodology* (47). Food challenges are usually given as lyophilized foods in opaque, dye-free capsules. In preparing antigens for hiding in capsules, freeze-drying and purification have been shown to decrease or even destroy the allergenic activity of the agent (48). Whilst this is considered the most convenient method (49), it does not facilitate food being given in the 'natural form' and problems may also arise concerning the quantity of dried foods needed to elicit symptoms, the number of capsules required, and time taken for dissolution of the capsule. Less frequently, due to difficulty in devising recipes, masked foods are used for challenges (48,50). This method does, however, enable challenge foods to be given in portion-sized doses and in the 'natural form' i.e. how it was eaten when the reaction occurred. The challenge of the methodology is to devise a masking agent suitable for all the foods in the challenge panel to be masked for every subject in a study. When masked in food, the suspected agent must be undetectable by taste, smell, colour and texture.

(iii) *A subsequent diet which is nutritionally adequate*. This should be based on the evidence of a positive food challenge (47) and be acceptable to the patient.

TREATMENT

Dietary avoidance

There is conflicting evidence as to whether oral challenge with food causes a direct asthmatic response, or may lead to an alteration in bronchial hyperresponsiveness thus priming the bronchi for subsequent provocation (51). Nevertheless, the purpose of any dietary avoidance is to have a direct impact on symptoms and food intolerance and is primarily treated by dietary avoidance of the offending foods (52). The most important factor influencing the effective control of symptoms is complete avoidance, with the underlying proviso that the dietary regime is nutritionally adequate (for example if milk and milk products are to be avoided then a calcium supplement should be prescribed) and is less troublesome to the patient than any symptoms it has alleviated.

Drug therapy

In the 1970s inhaled sodium cromoglycate was developed as a therapy for patients with asthma, particularly those with an allergic component (53). Subsequently its use was assessed in asthma and urticaria due to foods. By inhalation it blocked the airway responses following food challenge (54), but in an oral dosage of 800 mg day^{-1} for 1 week, or a single dose of 1·0 g, did not block any of the asthmatic reactions. In a study of fish-induced asthma, sodium cromoglycate blocked the fall in FEV_1 either completely or significantly, in 16/20 patients (55). Ketotifen has been considered to offer help in food allergy (56) and in one study (57) of 24 patients it was found to afford protection against the bronchial response to food challenge in asthmatic subjects.

Inhaled bronchodilators offer the best treatment for an acute episode induced by food, but when the attacks are severe and rapid, and are similar in speed of onset to an anaphylactic reaction, self-injected adrenaline (EpiPen, AnaPen) is effective and essential.

Vitamin therapy

Vitamin C is considered by some to have a protective role in asthma and other allergic diseases but this is not well defined. The current literature (58) does not support a definite indication for the use of vitamin C in asthma and food intolerance

RELEVANT FOODS

Adverse responses have been documented to most foods but those found to be the most allergenic in the Western diet are egg, milk, wheat, fish, citrus fruits, peanuts and soya (51). The respiratory system is documented as being affected by adverse reactions to these foods. In patients with brittle asthma a similar pattern of food responses is seen, suggesting that it is the allergenic moieties that are of importance, rather than the end organ sensitivity, in determining which foods are important in asthma.

Although alcohol is a modest bronchodilator (59), many patients with asthma report worsening of their symptoms when it is ingested. In one study (60), 25% of a hospital outpatient population reported that at least one alcoholic drink made their asthma worse, the effects being in most patients due to congeners in the beverage although the rare

patient will respond to ethyl alcohol alone. This is particularly marked in Oriental subjects (61), mediated through acetaldehyde a metabolic product of ethyl alcohol.

Foods of minor significance nutritionally, but nevertheless cited recently as the cause of severe asthmatic responses are shellfish (62), royal jelly (63) and fenugreek (64), and are probably IgE mediated responses.

Food additives are frequently cited as a group of foodstuffs causing problems in asthma. Some patients with asthma report adverse responses to monosodium glutamate (MSG), particularly if high doses are consumed (65,66), although the mechanism is unknown. Tartrazine sensitivity is most frequently manifested as urticaria and asthma (63) although the mechanism is obscure and probably not truly allergic. There is conflicting evidence concerning sulphited foods and asthma. It is been reported that about one in nine people with asthma have a history of asthma worsened by drinking 'soft drinks' containing sulphur dioxide (68), although these individuals may not necessarily react after each ingestion of sulphited food (69)

Some patients with asthma are known to be sensitized to aspirin and aspirin-induced asthma is well recognized. Salicylate-free diets are prescribed, by some, to improve the asthma symptoms of these individuals. There are no published studies demonstrating the efficacy of this treatment, but anecdotal evidence suggests that it is helpful in some patients.

PRACTICE POINTS

- Consider adverse reactions to foods when taking patient history.
- Identification and avoidance of offending foods can lead to significant improvement in some individuals with asthma.
- Skin pricks tests and RASTs are not diagnostic tools for adverse reactions to food in asthma.
- Dietary manipulation is the cornerstone of diagnosis and treatment
- Multidisciplinary team of physician, dietitian and nurse provides the best patient care.

RESEARCH AGENDA

- Development of a simple, laboratory diagnostic test.
- Development of more accurate, objective markers of response.
- Investigations into mechanisms in adverse responses to food in asthma.
- Epidemiology of adverse reactions to food in asthma.

Nutrient intake

NUTRIENTS

Magnesium

Magnesium is a known, mild bronchodilator (70) and increased dietary magnesium has been shown to have a beneficial effect on lung function, airway responsiveness and wheezing in the U.K. population (71). In the latter study the average daily intake of magnesium in healthy adults was established from a semi-quantitative food frequency food questionnaire and a 100 mg day^{-1} increase in magnesium intake was found to be independently associated with a 27.7 ml higher FEV_1 and reduced airway reactivity to methacholine responsiveness, after adjusting for daily intake of calcium, vitamin C, smoking, occupation and social class. From this it might therefore be inferred that a low magnesium intake could be involved in the aetiology of asthma, although this remains to be proven—association does not imply causality. However, at the severe end of the spectrum, there is some evidence which might support the role of magnesium in asthma. Patients with brittle asthma show a lower magnesium intake (72) (233 mg day^{-1}) compared to those with non-brittle asthma (277 mg day^{-1}) and healthy adults (276 mg day^{-1}). While this difference did not achieve statistical significance, the number of subjects in each group whose dietary intakes of magnesium were less than the reference nutrient intake (RNI), was significantly higher in brittle asthma compared to in non-brittle asthma. Together, these findings suggest a possible role for magnesium in asthma, which merits further research. Indeed, an investigation into the effect of short-term change in dietary magnesium intake in moderate asthma (73) showed that a high magnesium intake was associated with improvement in symptom scores but not in objective measures of airway reactivity. At present the evidence is lacking for recommending alterations in magnesium intake in asthma.

Sodium

Observations of the effect of dietary sodium on asthma (13) seem to suggest that sodium deficient diets might be linked with reduction in non-specific bronchial reactivity, although this finding may have arisen due to confounding between the intakes of sodium and magnesium (67). Several studies have investigated the relationship between sodium intake and asthma and have demonstrated small adverse effects of increased sodium intake on bronchial reactivity, but not on clinical symptoms. Hence there is not, at present, any justification for recommending an alteration in sodium intake in asthma.

Selenium

It has been hypothesized (7) that deficiency of selenium, acting through a lowered activity of gluthathione peroxidase, may be implicated in the pathogenesis of asthma. A

case control study control study conducted in New Zeland (7) demonstrated a near two and near six-fold increase in risk of asthma in subjects with the lowest range of whole blood selenium concentrations and glutathione peroxidase activity respectively. A Swedish (8) double-blind, placebo-controlled study of selenium supplementation showed significant general clinical improvement in the selenium supplemented group as compared with the placebo group, but this improvement could not be validated by significant changes in the separate parameters of lung function and airway hyperresponsiveness. Similar findings, from a study in the Slovak republic (75), of reduced concentrations of plasma selenium in patients with asthma compared to that of controls, would also lend support to this line of thought. Conversely, in severe, brittle asthma (72) dietary intake of selenium did not relate to asthma severity. As with magnesium and sodium, there is insufficient evidence as yet to recommend dietary supplementation with selenium in asthma.

Antioxidant vitamins

It has also been suggested, quite logically, that reduced intakes of the antioxidant vitamins A, C and E might be important in the pathogenesis of asthma and there is some supporting evidence for this. This has been examined in regard to fruit and vegetable consumption. The relationship between lung function and the reported frequency of consumption of winter fresh fruit and fruit juice was studied (76) among 1502 life-long non-smokers and 1357 current smokers aged 18–69 years. The mean FEV_1 among those who never drank fruit juice and ate fresh fruit less than once a week in winter, was lower than for other subjects after adjusting for age, sex, height, smoking, region of residence and socio-economic group by about 80 ml. More specifically to asthma, demonstration that in adults the lowest intakes of vitamin C were associated with a more than five-fold increased risk of bronchial reactivity, has provided evidence that antioxidants may have a modulatory effect in asthma (77). This would be consistent with the hypothesis (78) that the observed reduction in antioxidant intake in the U.K. diet over the last 25 years has been a factor in the increase in the prevalence of asthma over the same period. This concept was taken a step further by the MORGEN study (79), which investigated the relationships between the antioxidant vitamins C, E and β-carotene and the presence of respiratory symptoms and level of lung function. In this large study (6555 adults) a high intake of vitamin C or β-carotene was associated with higher lung function but did not appear to protect against respiratory symptoms (cough, phlegm, productive cough, wheeze and shortness of breath). It remains to be seen whether the current healthy eating advice 'Take 5' (i.e. 5 portions of fruit and vegetable daily) will have any impact in this country in patients with asthma. However, a New Zealand study (80) has recently shown increasing fruit and vegetable consumption leads to an increase in plasma concentrations of vitamin C and α and β-carotene. Further studies are necessary to examine whether an increase in plasma levels impacts on disease state, as for a number of antioxidant vitamins, plasma levels only poorly correlate with overall antioxidant status.

Vitamin C

Vitamin C is the major antioxidant substance in the airways where it may act to protect against both endogenous and exogenous oxidant molecules (10). Studies conducted into the possible relationship between vitamin C and lung function suggest a short-term protective effect of vitamin C on airway responsiveness (77,81–82) but it has not yet been shown whether consistent supplementation with vitamin C would confer a positive effect on objective measures of lung function or symptoms.

Vitamins A and E

There are considerably fewer studies on vitamin A and E with respect to respiratory disease and lung function. Dietary vitamin A levels have been shown to be inversely associated with airway obstruction in a U.S. study using national survey data (83), while a further U.S. study found that a decreased level of serum retinol predicted an increased risk of airflow obstruction 5 years later (9). However, it was not clear whether the low serum retinol levels affected the disease and its rate of progression, or whether the disease resulted in low serum levels through its effect on appetite and dietary intake. In a study of a group of elderly subjects (12), dietary intake of vitamin E was positively correlated with lung function—for every extra

PRACTICE POINTS

- Consider sub-optimal intake of nutrients when taking patient history.
- Dietary assessment of patient's food intake should be routine in severe asthma.
- Supplementation with nutrients thought to be important in asthma, has not shown clinical efficacy.
- Multidisciplinary team of physician, dietitian and nurse provides the best patient care.

RESEARCH AGENDA

- Studies of supplementation with nutrients thought to be important is asthma need to be conducted.
- Objective markers of sub-optimal nutrient intake need to be validated.
- Validity of dietary data should always be considered when assessing nutrient intake and the diet/disease relationship.

TABLE 3. Dietary intake of vitamins A and E in patients with brittle asthma

Nurtient	RNI	Median dietary intake		Median dietary intake		Median dietary intake	
		Brittle asthma	(IQR)	Non-brittle asthma	(IQR)	Healthy adults	(IQR)
Vit.A	600 μg day^{-1}	522·5	(270.0–798.0)	869·5*	(667·5–1189·5)	706.5**	580·0–987·5)
Vit.E	3 mg/day (SI)$^{-1}$	3·4	2·0–3·9	4·6^	(3·3–6·3)	4·5^	(3·0–6·4)

brittle asthma : non-brittle asthma, *$P=0·01$; brittle asthma : non-brittle asthma, ^$P=0·005$;
brittle asthma : healthy adults, **$P=0·04$; brittle asthma : healthy adults, ^ ^$P=0·007$.
IQR: interquartile range; RNI: reference nutrient intake; SI: safe intake.

1 mg increase in vitamin E in the daily diet, FEV_1 increased by an estimated mean of 54 ml.

In asthma, there has been little work in general asthmatic populations. A case control study (72) in brittle asthma demonstrated significantly lower intakes of vitamin A in brittle asthma compared both to non-brittle asthma and healthy adults. In the same study, dietary intakes of vitamin E were also found to be significantly lower than in subjects with non-brittle asthma and healthy adults ($P=0·005$ and 0·007 respectively), with significantly more subjects with brittle asthma whose intakes were sub-optimal compared to subjects with non-brittle asthma and healthy adults (Table 3). Further evidence is needed before recommendations can be made for dietary supplementation with vitamin A and E.

Summary

Adverse reactions to food in asthma are now sufficiently well documented to be regarded as real and to demonstrate that recognition can result in significant improvement at an individual level. There is no clear idea of the true prevalence of adverse responses to foods in asthmatic populations. Relying on skin prick tests to determine food allergy is inaccurate and unwise, and the consequent widespread use of extreme food avoidance measures in individuals with modest asthma is not to be advised. Some foods can easily be recognized as causes of acute severe responses such as peanuts and shellfish but these, while potentially causing life-threatening attacks, are unlikely to be the cause of long-term destabilization of asthma. Conversely, where intolerance of staple foods such as milk/milk products and wheat is recognized, dietary avoidance is important in stabilizing asthma. This usually means that a major change in dietary habits is required, which is often difficult to undertake consistently. This can be reinforced by the observation that some patients, known to be allergic to a specific food stuff, can 'get away with' eating the forbidden food if their asthma is going through a good period, whereas they daren't try the food if their asthma is at the time poorly controlled. Whether such occasional doses of allergen maintain the adverse immune response in these patients or whether it is of no import in the long run is not known. It does, however, identify the need to balance the other needs of the patient who may have been advised to avoid a food which is a great favourite and who may feel that an occasional treat is well worth the potential risk!

Sub-optimal nutrient intake and the resultant reduced antioxidant activity may enhance asthmatic inflammation consequently contributing to bronchial hyperreactivity, but prospective studies of supplementation therapy will be needed to confirm this. A recent review of nutrition and asthma (84) concluded, 'It is clear from a review of the existing data that there is no proven role for nutritional therapy in the management of asthma. Until more definitive studies are completed, the use of nutritional supplements for the treatment of asthma cannot be recommended.' There is no published evidence to 1999 to suggest that this conclusion can be altered.

In both areas, food intolerance and nutrient intake, the research agendas suggested need to be taken up. In food intolerance, the development of simple laboratory diagnostic tests would perhaps encourage clinicians to investigate the phenomenon in patients where they are currently put off from doing so by cumbersome dietary manipulations. They may even be more convinced of the phenomenon itself if more accurate, objective markers of response could be found and the mechanisms of the response were understood. Epidemiological studies would help demonstrate something of the scale of food intolerance and, as a consequence, encourage appropriate resources to be made available for further research and treatment. In the study of the effects of sub-optimal intake of nutrients thought to be important in lung health, and, conversely, of their supplementation, there is much to be pursued. However, important considerations concerning the validity of dietary intake data, and the availability of and use of validated objective biological markders of dietary intake, should be weighed in order to avoid false conclusions being drawn

about the diet–asthma relationship. Currently, in asthma, available data are insufficient to implicate any dietary factor as causal (85).

In spite of the limitations of knowledge outlined above, it is the opinion of the authors that in caring for the patient with asthma, consideration should be given to adverse reactions to food and sub-optimal intakes of nutrients when taking the patient history since one or both factors may be impacting on day to day management of the disease; and that a multidisciplinary team approach, of physician, dietitian and nurse, provides the best patient care.

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