The Role Of Foods In Asthma
G Borok, J Loots

Citation

Abstract

Objective: To determine whether certain foods can provoke asthma and nasal symptoms in adults.

Design: A randomised-groups design, case controlled, open food challenge crossover study on adult asthmatics randomised into control or diet groups. The control group continued with their medication while diet group was placed on an elimination and rotation diet programme (E&RP) for 6 weeks, to identify foods provoking symptoms. Those not symptom free, were investigated for inhalant allergens.

Setting: Patients recruited by media, underwent lung function evaluation, to measure Forced Expiratory Volume in 1 second (FEV1) of whom 102 patients supported the diagnosis of asthma. Subsequently, 27 of the 51 patients in the control group elected to cross over to the diet programme. Follow up period to be at three monthly intervals and extended over one or two years if possible.

Results: The initial mean FEV1 of 67.84% in control group, compared to the 69.94% diet group was not statistically different (p = 0.49). After 6 weeks, an increase of 21% of the predicted FEV1 value, in the diet group compared to the 1.83% of the control group, was highly significant (p<0.0001). Twenty-seven patients of control group who crossed over to the E&RP, resulted in a further increase of 20.36% in the mean FEV1 value. After 6 weeks, foods found to provoke asthma in 63% of patients and in 27% not asymptomatic, inhalants were investigated and found to be an added factor. In 8%, not responding to the diet, inhalants were the only factor. In two patients no factors identified. Medication of controls did not vary appreciably, whereas 63% of diet group needed no medication and 31% required less at the end of the trial. The most common triggers were fruits (apples, bananas, citrus) in 29%, followed by proteins (milk, fish, beef) in 26%, grains (wheat, corn) in 21%, vegetables (potato, tomato) in 12%, nuts in 2% and processed foods played a minor role in only 10%. Nasal symptoms, present in 64 of the 78 (82%), who completed the diet programme, was relieved in 43 (67%), improved in 17 (27%) by avoiding foods and in the 4 not helped by the diet, 3 were due to inhalants.

Medication dosage recorded weekly for first 6 weeks, then 3 monthly intervals for 1-2 years if possible.

Conclusion: Open food challenge technique, showed foods provoked asthma in adult asthmatics. Inhalants played a minor role. Identification of foods provoking asthma should be investigated when treating asthma.

INTRODUCTION

The World Health Organisation report on Allergic Rhinitis and Asthma (ARIA), indicates that asthma and rhinitis should be considered as one illness, i.e. One airway, one disease. This is supported by other publications indicating that, nasal symptoms occur in 75% of asthma cases. A recent editorial in the Journal of Asthma concluded that if foods do play a role in the causation of asthma and allergic rhinitis, it is by no means a universal phenomenon and occurs only in a minority of patients. This concurs with the views of Warner, who believes that foods rarely precipitate asthma in children. However other authors, have shown that foods do indeed play a role. As effective inhaled medications especially corticosteroids are available, some feel dietary sensitivity or food intolerances need not be investigated. Besides the increasing cost of treating asthma worldwide, the enormous costs are compounded by a significant
decrease in the quality of life, despite modern inhalation and oral therapy are still often absent from school and work. Moreover, the worldwide mortality from asthma is rising in developed countries. Laboratory-based food allergy tests often produce either negative or difficult to interpret results. Double-Blind-Placebo-Controlled-Food-Challenges (DBPCFC), the gold standard for diagnosis, do not always produce positive results, yet subsequent open food challenge may do so in several cases. Skin prick and CAP-RAST tests are less accurate for food allergy diagnosis than for inhalants. Practitioners who treat asthmatics are aware of difficult or “brittle” cases and should question all patients about possible food triggers. Confirmation may be established by the open food challenge technique. If an elimination diet can identify foods that on avoidance reduce symptoms and improve lung function results and on re-exposure lead to the recurrence of the same symptoms then a diagnosis of food intolerance can be made. The purpose of this study was to demonstrate that avoidance of foods identified as aggravating wheezing and dyspnoea in asthmatics, using an open challenge, elimination and rotation diet programme (E&RP) can lead to relief of symptoms and on re-exposure, a recurrence. The technique is simple and can easily be carried out in the consulting rooms, with the aid of a spirometer and the elimination diet. Simultaneously, if originally present, nasal symptoms may also be relieved.

SUBJECTS AND METHODS

EXPERIMENTAL PROTOCOL AND DESIGN

Consent for a randomised-groups design, case controlled, open food challenge, using the E&RP, cross-over study on adult asthmatics, was obtained from the Ethics Committee of the South African Medical Association (SAMA). The same protocol was accepted by the ethical committee of the University of the Witwatersrand (RAND).

SUBJECTS

Asthmatic adult volunteers, recruited by media advertisements, all underwent the initial screening requirements of the trial.

LUNG FUNCTION

Volunteers were accepted into the trial when the Forced Expiratory Volume in 1 second (FEV1), measured on a spirometer (Micro Loop, Micro Medicals, Kent, England) satisfied the conditions of the protocol, which were:

- a) If the initial FEV1 was less than 75% of predicted value.
- b) or if the initial was greater than 75%, a fall of more than 15% of the FEV1 predicted value, after exercise using Jones method and returned to normal after salbutamol inhalation.
- or after exercise or if too distressed to exercise, the FEV1 predicted value improved by 15% or more following salbutamol inhalation.

When the above requirements were met the volunteers were randomised by JML into Control (C) and Diet (D) groups, according to a statistically evaluated, randomising table. A flowgram of lung function, using the FEV1 as the standard criterion was measured:

NASAL SYMPTOMS

The incidence of nasal symptoms and improvement, amongst these asthmatics, such as sneezing, itching, pre and post nasal drip, congestion and throat clearing was recorded. The improvement of the above symptoms was recorded daily after each meal and noted at each weekly visit for the six weeks of the trial and follow up periods.

CLINICAL HISTORY

Each volunteer was informed to which group he/she had been randomised and the purpose of the particular group was explained. Each volunteer completed a questionnaire detailing severity and frequency of symptoms of rhinitis and asthma and dosage of current medication. Informed consent was signed by all. The questionnaire was again completed after 6 weeks, the period of the trial and again after 6 weeks of the cross-over period.

CONTROL GROUP

This group was seen weekly for 6 weeks when the lung function (FEV1), current symptoms and the dosage and frequency of medication used, were recorded. They were informed to carry on with their usual therapy. Only after 6 weeks the control group was given the information and
option to go onto the E&RP. Their symptoms and medications during the two periods were compared, with each person then acting as his or her own control.

**DIET GROUP**

Each patient completed an extra food questionnaire detailing their usual diet, and if any foods had affected him/her in any way. The principle and technique of the E&RP was explained to each volunteer and given a menu on a diet sheet, and instructions detailing the requirements of the diet.

**THE ELIMINATION AND ROTATION DIET (E&RP)**

The diet involved eating one or two different types of foods in their natural state at each meal. Each day a different food of the food types were to be eaten, such as grains for breakfast, fruits for lunch and a protein with a vegetable for supper. Foods were to be bought fresh or frozen and eaten raw or either cooked, steamed, baked, micro-waved or grilled but not fried. No processed foods (bread, pizzas, confections, sauces, cold drinks, coffee) were allowed. During the first week only water was allowed to be drunk and salt the only condiment added. The specific diet was prescribed on a tabulated menu-form, according to the likes and dislikes of each individual, with the food to be eaten at each meal for each day of the week for the 6-week period. No food item being repeated in the week. (Figure 1)

After each meal, any symptoms present were recorded and graded by the patient, as to severity, as well and dose of medication needed. Any food that provoked symptoms was replaced by another food, the following week, until the patient was symptom free. Patients were seen weekly and any further queries regarding the requirements of the diet were addressed. Over the six weeks other beverages, condiments and other foods, including processed foods were added and if any provoked symptoms were replaced. After 6 weeks those who were not completely relieved of symptoms then had inhalants investigated, to ascertain the role any inhalant played in each volunteer.

It must be noted that all patients were ambulant. One patient was on a monthly injection of cortisone, treated by his physician, 40 were on inhaled and 4 on oral corticosteroid therapy. All were on various long and short acting bronchial dilatation therapy. As asthma decreased or disappeared, both long and short acting bronchodilators were reduced or stopped completely. Those on inhaled corticosteroid therapy were tapered gradually. The dosage and frequency of medicines with the degree of relief were also recorded, on the diet sheet. Patients were requested to avoid any bronchodilator medication, on the morning of exercise.
testing. The follow up period was to be at three monthly intervals for one or two years, if possible.

**STATISTICAL ANALYSIS**

The mean change in the FEV1 predicted values between the control and trial groups, pre- and post- trial, was calculated using a paired Student-t test.

**RESULTS**

102 asthmatics, 51 patients in both control and diet groups (63 females and 39 males) completed the requirements of the protocol. The mean age of the Control group was 48.2 years and duration of asthma 32 years and the Diet group was 43.3 years and duration of symptoms 21 years. The control group showed an insignificant improvement of 1.83% of the FEV1, p = 0.49 compared to the highly significant 21% of the diet group, p<0.0001. (TABLE 1)

**Table 1:** The number, gender, mean age and duration of the asthma, and the FEV1% predicted value before and after the 6 weeks of the trial period. Initially, there was no statistical difference of the FEV1 between control and diet groups (p = 0.47). After 6 weeks the diet group showed a highly significant improvement, 21% (p = 0) compared to control group’s 1.83%. The 27 controls who crossed over to the diet programme showed a highly significant increase of 20.36% of the FEV1 predicted value.

**Figure 3**

**Table 2:** The incidence of the various foods that provoked the symptoms of asthma as identified by the elimination and rotation diet and subsequent avoidance over a period of six weeks and follow up of 24 -36 months.

**CROSS OVER TO DIET PROGRAMME BY CONTROLS.**

Twenty-seven patients (17 females and 10 males) in the Control group crossed over to the E&RP programme and experienced a mean improvement of 20.36% in the FEV1 which was highly significant p<0.0001. (TABLE 1)

**OFFENDING FOODS**

Of the 78 patients who completed the diet programme, foods alone provoked episodes of wheezing and cough in 49 (63%), foods combined with inhalants in 21 (27%) and 6 (8%) of the 8 (10%) who did not respond to the diet, improved after avoiding inhalants. In two patients (2%) no trigger, neither food nor inhalant, was identified.

Fruits were identified as the triggers in 83 (29%) instances, followed by proteins in 74 (26%), grains, in 61(21%), vegetables in 35 (12%), nuts in 5 (2%) and processed foods in only 27 (10%). Contrary to expectation, preservatives, colourants and so-called “processed foods” played a minor role. Chocolate was the trigger in 6 instances, wine in 4, fruit juices in 3 (orange juice in 2, guava juice in 1), while, ice cream, honey, coffee and tea and beer, accounted for 2 patients each. Custard cake, guava roll, tomato sauce, chicken and beef burger affected only one person each. Four patients, who had adhered to the diet for the 6 weeks, rewarded themselves by indulging in their favourite food (honey in 2, and a chicken burger and beefburger in one each) and reported an immediate onset of asthma. Some patients reacted to only one food, while others from two six, different foods. (TABLE 2)

**Figure 4**

**INHALANTS AS TRIGGERS**

Nineteen of the 78 patients, not adequately improved after 6 weeks on the diet, then had inhalants investigated. Perfumes, soaps, shampoos, pets, cigarette smoke, exhaust fumes, work situations, shopping centres, industrial sites, time of day and season of year were eliminated where applicable. The only laboratory test used, was when symptoms were related to being at home, to exclude house dust mite sensitivity. Inhalant factors included, cigarette smoke in 12 instances,
(15%) (8 passive and 4 active), house mite and dog hair in 4 (8%) each, cat hair in 3 (3%), perfumes and sprays in 2 (2%) each. Printers ink, car exhausts, damp walls due to a leaking roof, grass cuttings, saw dust, industrial area, smog, rain storm and grass fire affected one patient each. Multiple factors were involved in those reacting to both foods and inhalants.

NASAL SYMPTOMS. SECONDARY OUTCOME MEASURE

Nasal symptoms, such as sneezing, itching, excess mucous and nasal congestion occurred in 77 (75%) of the 102 asthmatics. Amongst the control group these symptoms occurred in 36 (70%). Only 2 improved when given increased doses of corticosteroid nasal sprays.

Amongst the 78 patients who completed the diet programme, avoiding the offending foods, nasal symptoms present in 64 (82%), cleared in 43 (67%), improved in 17 (27%) while 4 showed no change. Loss of sense of smell, taste and hearing also improved. Of the 4 (6%) who had not improved 3 were subsequently shown to react to dogs, cats and cigarette smoke. In one no cause was found. (Table 3).

Figure 5

Table 3: The incidence of sinusitis in the subjects of the present study before and after the 6 week trial period. Changes in the sensory modalities of taste, smell and hearing are shown as well. Tinnitus in 1 was relieved. The sight in one improved remarkably.

<table>
<thead>
<tr>
<th>SYMPTOM</th>
<th>NUMBERS</th>
<th>RELIEVED</th>
<th>IMPROVED</th>
<th>NO CHANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>SINUSITIS</td>
<td>64 (82%)</td>
<td>43 (68%)</td>
<td>17 (27%)</td>
<td>4 (6%)</td>
</tr>
<tr>
<td>Loss of sense smell</td>
<td>42 (67%)</td>
<td>26 (57%)</td>
<td>14 (33%)</td>
<td>4 (10%)</td>
</tr>
<tr>
<td>Loss of sense taste</td>
<td>31 (46%)</td>
<td>17 (55%)</td>
<td>12 (39%)</td>
<td>2 (6%)</td>
</tr>
<tr>
<td>Diminished hearing</td>
<td>7 (11%)</td>
<td>5 (71%)</td>
<td>2 (29%)</td>
<td></td>
</tr>
<tr>
<td>Tinnitus</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

SMOKING

Smoking played a minor role. Of the 102 asthmatics more than half, 69 (68%) had never smoked. While 21 (20%) had stopped smoking between 2 and 27 years previously. Only 12 still smoked. In patients with FEV1 predicted values less than 50%, 9 had never smoked while 7 had stopped smoking more than 2 years previously and only 1 was an active smoker. In eight patients passive cigarette smoke was the trigger of their wheezing. (Table 4)

Figure 6

Table 4: The incidence of smoking in this trial was surprisingly low. Of the 102 asthmatics more than half, 69 (68%) had never smoked while 21 (20%) had stopped smoking between 2 and 27 years previously and 12 (12%) still smoked. In the 17 patients with an FEV1 of less than 50%, 9 had never smoked while 7 had stopped smoking more than 2 years previously and only 1 was an active smoker. In eight patients passive cigarette smoke was the trigger of their wheezing. (Table 3).

MEDICATION

As symptoms were relieved, patients on inhaled long acting bronchodilators were asked to reduce the dosage from two puffs twice a day (bd) to one puff bd, then once daily and tapered off to alternate days, every second or third day and ceased when free of symptoms for four days. Similarly for short acting inhaled bronchodilators and then only when necessary. When free of bronchodilator therapy for a week, oral or inhaled corticosteroid therapy, was tapered off, over three to four weeks. Progress in reduction of medication, was noted at each visit and follow up, for one or more years.

Of the 78 patients who were on various inhaled bronchodilator medications only 17 needed such therapy occasionally, when they inadvertently ate a processed food, containing the trigger or when they wanted to test the identified food, their favourite. (Table 5) Similarly of the 67 patients on oral or inhaled corticosteroids only 8 needed this therapy after avoiding the trigger foods. Four of them though free of symptoms were advised by their physicians not to stop this therapy. (Table 6)
The Role Of Foods In Asthma

Figure 7
Table 5: Indicating the doses and number of patients on inhaled bronchodilators before and after the E&RP. After avoiding the offending food/s, the 16 patients who occasionally needed a bronchodilator, are those who inadvertently ate a processed food that contained the trigger of their symptoms, not previously identified, during the follow up period.

<table>
<thead>
<tr>
<th>INHALED BRONchodilATORS</th>
<th>BEFORE</th>
<th>AFTER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
<td>Dose</td>
<td>No</td>
</tr>
<tr>
<td>Salmeterol</td>
<td>25 mcg, 2 puffs bd</td>
<td>2</td>
</tr>
<tr>
<td>Formoterol</td>
<td>12 mcg, 2 puffs bd</td>
<td>0</td>
</tr>
<tr>
<td>Fenoterol</td>
<td>50 mcg, 1 puff bd</td>
<td>7</td>
</tr>
<tr>
<td>Salbutamol</td>
<td>100 mcg, 2 puffs bd</td>
<td>14</td>
</tr>
<tr>
<td>Fenoterol</td>
<td>100 mcg, 1 puff bd</td>
<td>5</td>
</tr>
<tr>
<td>Ipratropium</td>
<td>100 mcg, 1 puff bd</td>
<td>2</td>
</tr>
<tr>
<td>Ipratropium 100 Salbutamol</td>
<td>1 puff bd</td>
<td>2</td>
</tr>
<tr>
<td>Fenoterol (Ipratropium 40 mg)</td>
<td>1 puff qd</td>
<td>2</td>
</tr>
</tbody>
</table>

The one patient who received cortisone by injection monthly did not need further injections.

Nebuliser inhalation therapy used by 10 patients became redundant in all but 2. Five on fenoterol 1.25 nocte, 4 on Ipratropium bromide and 1 on Fenotrol/Ipratropium were free of therapy except 2 who needed fenoterol pm. Sodium cromoglycate, by inhalation, was used by two.

On follow up after 6 months, though improved and free of symptoms, four of the above were advised by their physician/s not to stop taking their previous cortisone medication, to which they complied.

Antihistamine use by 8 on Loratidine 10 mg daily, 3 on Cetirizine HCL 6 mg daily, 1 on Ketotofin 1 mg bd and another on Oxatomide 30 mg bd, including 2 patients on Singulair 10 mg inhalations, was unnecessary after the diet.

By avoiding foods identified by the E&RP, a statistical improvement of 21.00% of the predicted FEV1 value in the diet group compared to controls who were on their previous medication ( p<0.0001). Forty nine (63%) of the 78 asthmatics in the diet group, discontinued all medications, while 21 (27%) were taking less after six weeks. The diet did not help 8 (10%), 6 (8%) of whom afterwards, reacted to inhalants. Only two patients did not benefit.

**FOLLOW UP STUDIES AFTER 2-3 YEARS**

After two to three years, 66 of the original 78 persons on the diet protocol, were traced. Patients were encouraged to return to normal diets but to exclude offending foods. Forty nine (74%) patients were symptom free, without medication, 9 patients (14%) needed bronchodilators, when inadvertently they had eaten offending food(s). Five patients (8%) could not resist eating identified offending foods, (favourite foods), inhaling bronchodilators before eating them. Two patients (3%) were unwilling to forego their favourite food, (beer and toasted cheese) and resumed medication. One would not remove her pet cat and one would not stop his woodwork hobby. Two (3%) pensioners could only afford bread and cheese, their triggers and required medication. Four (6%) patients, though symptom free, were advised by their physicians to continue with their corticosteroid therapy, which they did.

During the follow-up of two to three years two patients, who had recorded the lowest FEV1 readings of 12 and 16%, had died of causes unrelated to asthma. Their quality of life, prior to death, had improved because of freedom from asthma symptoms, aided by reduced doses of inhaled corticosteroids. Generally absenteeism was much less, they were able to do much more work, without medication, but some remained on reduced doses of inhaled corticosteroids.

**DISCUSSION**

The fact that two thirds of the study subjects, who avoided...
The Role Of Foods In Asthma

eating the foods that were identified as aggravating their asthma and nasal symptoms, for a period of up to three years indicates that the open food challenge elimination technique is a practical and useful tool to identify these foods.

Fruits and vegetables (apples, citrus, bananas, avocados and potatoes) were the most common triggers, followed by proteins (mainly dairy products, meat and fish), grains (mainly wheat and maize) and the least common causes were, processed foods. (Table 2). Inhalants, accepted as causing asthma, provoked symptoms in only 8% of patients while in combination with a food in a further 27%.

Foods are known to cause asthma symptoms, especially sea foods. Such reactions may be allergic or due to chemicals found naturally in foods. Foods containing caffeine, theobromine and vanillic acid stimulate the sympathetic nervous system leading to bronchial dilation. Recent studies indicate that neuropeptides released by parasympathetic stimulation lead to bronchial constriction. The exquisite sensitivity of asthmatic lungs to endogenous food mediators of anaphylaxis, such as histamine, prostaglandins and leukotries may cause the bronchial constriction.

Salicylates, benzoates, sulphites and tartrazines, used as preservatives and to enhance the taste of foods, occur naturally in honey, milk, beef, tomato and walnuts, previously shown to provoke asthma, provoked asthma symptoms in this study. It is noteworthy that other food additives such as monosodium glutamate and aspartates, which occur naturally in foods, are known provocative factors for asthma. Vasoactive amines, such as tyramine, histamine, phenylethylamine and serotonin which occur naturally in banana, pineapple, avocado, plum and tomato may cause food reactions, which foods did so in this trial.

A diet rich in fruits and vegetables may provide significant amounts of these compounds when consumed on a daily basis. Interestingly, the most common food to trigger asthma in this study, was apple. Apples contain several natural chemicals of which salicylates is one, which are known to cause asthma in susceptible patients.

Physical factors such as exercise and cold that had previously triggered asthma in many of the trial patients did not to do so when offending foods, identified by the E&RP were avoided. Cold weather on patients, previously admitted to hospitals in winter, were symptom free, during the last two winters. Exercise induced asthma, present in two patients did not occur when wheat was avoided. Once airway reactivity is enhanced, other common triggers of asthma, such as cold air or drinks, exercise, or other environmental factors, readily precipitate acute episodes of asthma.

The incidence of nasal symptoms, in 77 of 102 of these patients (75%) is similar to other documented studies and supports the suggestion of the W H O guidelines on Allergic Rhinitis and Asthma (ARIA), that asthma and rhinitis should be considered as one illness. The mucosa which lines the nasal passages, sinuses, nose-phyarynx, bronchi and bronchioi is continuous and may be affected by the same trigger factor.

Identifying and avoiding inhalants as the sole cause leads to under-diagnosis of the cause of asthmatic symptoms. Once both food and inhalant factors are identified the incidence of relief of respiratory symptoms rises to about 80% in asthmatics. Their daily quality of life is improved, decreasing the costs of hospital admissions and medication needed. They will be emotionally happier without the daily fear of not having their bronchodilator therapy with them. I hypothesise that COPD may be avoided in asthmatics early in their history as repeated severe attacks will be avoided, preventing bursting of a certain number of alveoli with each acute attack, not to mention the increased pressure during each exhalation against the ongoing chronic bronchial constriction.

The DBPCFC technique, the gold standard to identify foods provoking respiratory symptoms is not difficult to perform but takes up a lot of time in the laboratory or a specialised unit, yielding low positive results though others have shown better results. The incidence of foods provoking asthma can be confirmed by the open food challenge technique. It is not clear how foods provoke asthma symptoms, though anaphylaxis can be caused by sea foods and nuts. Late phase reactions are difficult to associate with a food reaction as the food may have been eaten up to 48 hours previously. The elimination diet programme resolves this problem as only one or two foods are eaten at a specific meal and no food is repeated in the week. (Figure 1) Food/s provoking symptoms after a meal are replaced each week, and if no relief occurs the food/s of the previous meal are replaced in a retrograde manner, weekly, until relief is established, establishing the time for the reaction to occur after the particular food involved.

In this study 64% of patients, who adhered to the requirements of the diet, were relieved of their asthma as well as their nasal symptoms, and in addition a further 27% when inhalants are identified and avoided. Their quality of
The Role Of Foods In Asthma

life was improved and work absenteeism appeared to be reduced and work output increased. This indicates that the open food challenge elimination programme may have a practical role to play of identifying an ingested cause of asthma and rhinitis, in situations, where double blind facilities are not available. Any patients not completely relieved of their symptoms could then have inhalants investigated as causative factors.

Pollens are accepted as causing asthma. Pollens grow into plants with leaves, roots, trees and their products, which may contain some of the same chemicals as in the pollen. Fruits were the main triggers of asthmatic symptoms in this trial. Do fruits not contain the same chemicals as their pollens, which are accepted as causing asthmatic symptoms? Besides fruits and vegetables the next common trigger was protein, milk and beef being the most often. Milk and beef are derived from cows that eat grass. Grass chemicals will be in their milk and beef we eat, gain plant life triggering the asthma.

ACKNOWLEDGEMENTS

The trial was made possible by a grant from the BEARE Foundation for medical research, Durban, South Africa.

References

3. Corren J. Allergic Rhinitis and asthma: how important is the link? Allergy Clin Immun. 1997;99:781-786
10. Kaplan I. The elusive link in incurable bronchial asthma. SAMJ. 1967;1123-1127
12. Onorata J, Merland N,Terral C, Michel F B, Bousquet J. Placebo-controlled double-blind food challenge in asthma. J. Allergy & Clinical Immunology.1986;78:1139-1146. Food diets should not be prescribed for all patients reporting an adverse reaction or positive skin scratch or RAST test with food allergens.
31. Bousquet J, Merland N, Terral C, Michel FB, Bousquet J. Placebo-controlled double-blind food challenge in asthma. J. Allergy & Clinical Immunology.1986;78:1139-1146. Food diets should not be prescribed for all patients reporting an adverse reaction or positive skin scratch or RAST test with food allergens.
The Role Of Foods In Asthma

Author Information

G. Borok

J. M. Loots