Food Allergy — The Enigma and Some Potential Solutions

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ABSTRACT

Food allergy is one of the most common illnesses associated with ingestion of food. However, food allergy is often a loosely defined clinical phenomenon. An appeal is made for a stricter definition and more thorough diagnosis of food allergy. Food allergy should be a term reserved to describe those hypersensitive reactions to foods that have a demonstrated immunological basis. The incidence, symptoms and diagnosis of food allergy are discussed with some emphasis placed on subjects of current and continuing controversy. Several goals for future research programs in food allergy are stated, including (a) improvement of the food allergen extracts used in clinical diagnostic procedures, (b) development of adequate clinical procedures for diagnosis of delayed hypersensitivity reactions to foods and (c) development of methods for hypoallergenic processing of foods.

Food allergy remains an enigma within the area of food toxicology. Whereas many other aspects of food toxicology are drawing considerable research activity, food allergy continues to struggle for scientific attention. The lack of research activity on food allergy is not likely due to any lack of interest among food scientists. If not allergic to certain foods themselves, most food scientists are at least aware of the existence and importance of allergic reactions to foods through contact with allergic individuals, possibly even including their own families and friends. In addition, most food companies receive periodic complaints from consumers alleging allergic reactions to certain foods.

Multiple reasons exist for the dearth of research activities in food allergy. One major stumbling block that has limited progress and research in the field of food allergy is the lack of convenient and reliable methods for determining the allergenic activity of foods and food components. Usually a physician specializing in allergy must be consulted to perform such tests, since most of the available tests require use of consenting allergic patients. However, even with the assistance of a physician and his patients, the results of currently available tests are often not totally reliable. This unfortunate set of circumstances has almost certainly discouraged many food scientists from embarking on research programs in food allergy. In addition, many food scientists receive only minimal training in food allergy and consequently have limited knowledge and capabilities in this area. Meanwhile, physician-allergists have generally disdained food allergy because of their understandably meager knowledge of food chemistry and food processing and because of the greater incidence of allergies to pollens, molds, insect stings, etc. which has sparked research on those allergies. Consequently, these factors, along with the generally held attitude that not much can be done to eliminate food allergy anyway, have created an effective barrier to research in food allergy.

This review will attempt to remove some of the enigma associated with food allergy by (a) carefully defining and classifying food allergies, (b) discussing the types of food allergy and their incidence, (c) briefly describing the symptomology and clinical diagnosis of food allergy and (d) attempting to pinpoint some of the needs for future research in food allergy. Since it is impossible to discuss all relevant material, the reader is directed to several recent reviews for further information on clinical (32,33,32), chemical (1,10,45,54), immunological (17,42,44,58) and general (15,16,27,32,33,52) aspects of allergy and food allergy in particular.

A MORE PRECISE DEFINITION AND CLASSIFICATION

Even the definition of the term, food allergy, is fraught with difficulty. Food allergy is often loosely defined as any unpleasant, abnormal or heightened response of an individual to a food or food component. However, this broad definition would lead to the classification of Chinese Restaurant syndrome and those forms of milk intolerance caused by a genetic deficiency of the enzyme, lactase, as food allergies. Obviously these two syndromes are quite different from each other and also distinct from the classical types of food allergy, e.g. as manifested by hives following ingestion of a given food by a sensitive individual.

A more precise definition of food allergy can be achieved. Many allergists prefer to limit use of the term allergy to those types of hypersensitivity that have a proven immunological basis (10,27,32,33). Chinese Restaurant syndrome and lactase deficiency-induced milk intolerance have no known immunological basis and would not be classified as food allergies under this more precise definition. Unfortunately, techniques necessary to demonstrate an immunological basis for a food-induced reaction are neither readily available nor entirely foolproof. Many clinical allergists do not have direct access to some of the most reliable techniques. For certain types of food allergy, particularly delayed hypersensitivity, adequate tests remain to be developed. Finally, even the best methods are not totally accurate. Consequently, even if the more precise definition of food allergy is widely adopted, some confusion will continue.
until better methods become readily available. However, a good deal of the confusion surrounding food allergy could likely be eliminated if clinical allergists would uniformly apply the best of the available tests in the diagnosis of food allergy.

Adoption of the more precise definition of food allergy would lead to a better classification of the host of different conditions that comprise the unpleasant, abnormal and heightened responses of certain individuals to foods. Allergy would be reserved to describe those conditions with a proven immunological basis. Intolerance could be used to describe those conditions resulting from deficiencies or abnormalities in the enzymatic and/or biochemical mechanisms for metabolizing a normally non-toxic food component. Those conditions involving responses to a toxic chemical in a food would be classified as intoxications. Infection would be the classification for those conditions initiated by foodborne bacteria, viruses, rickettsia and parasites. Other conditions that defy classification in one of the other categories would simply be termed food sensitivities or idiosyncratic reactions.

Some allergists will undoubtedly argue with my definition of food allergy and classification of adverse reactions to food. Certain investigators have claimed that food allergy can be non-immunologic in its mechanisms, and thus not be recognized by the frequently used tests (15,16,19,20,59,64,67). According to this theory, individuals can have “masked” allergies and may be able to consume a certain food to which they are allergic without the occurrence of a clinically demonstrable immunological reaction. These contrary views seem to be supported mostly by testimonial case reports and appear to require further rigorous proof. Some scientists would prefer to include malabsorption diseases, inflammatory responses and enzyme deficiencies among the various forms of delayed or “occult” allergy (15,16).

TYPES AND INCIDENCE OF FOOD ALLERGY

Even if one decides to limit the definition of food allergy to conditions with a proven immunological basis, several distinct types of food allergy are known to exist. Often the types of food allergy are classified as immediate or delayed on the basis of the time between consumption of the food and the onset of adverse reactions (10,33). A more precise system of classification based on the type of immunological phenomenon observed has been described by Gell and Coombs (31) and has been widely adopted by other allergists (32,33,58). In this system, allergic reactions are grouped into four general types designated Types I through IV.

Immediate hypersensitivity-type allergic reactions appear to belong entirely within the Type I category. Type I reactions are often termed immediate hypersensitivity, reaginic hypersensitivity, anaphylactic hypersensitivity and atopy. Type I reactions are mediated by reaction of an allergen with a distinct type of immunoglobulin designated as IgE (some recent information has implicated IgG4 and IgD also) or sometimes by the older term, reagin. The molecular basis of IgE-mediated allergic reactions is fairly well understood and has been recently reviewed elsewhere (1,44). The observed symptoms of Type I hypersensitivity result from the release of pharmacologically active substances including histamine, serotonin, and others from mast cells as a consequence of IgE mediation. Type I reactions usually occur within several minutes to several hours following ingestion of the food.

Delayed hypersensitivity-type allergic reactions usually refer to the Type IV category (16,33). Type II and Type III reactions also exist (16,33), but have never been definitely documented in food allergies. Type II hypersensitivity reactions are produced by reaction of antibody with cell-bound antigen (allergen), followed secondarily by complement fixation. Examples of Type II reactions would be transfusion reaction and certain auto-immune diseases. Type III hypersensitivity reactions are caused by complement fixation which follows reaction and deposition of antigen-antibody complexes at some reaction site. An example of a Type III reaction would be serum sickness. Some forms of cow’s milk allergy may be similar to Type III reactions but further proof is needed (38). Type IV hypersensitivity reactions, also termed delayed or cellular hypersensitivity, are created by the reaction of certain sensitized cells usually lymphocytes with allergen. Type IV reactions may occur with foods. The molecular basis of Type IV reactions is poorly understood by comparison to Type I reactions. The ultimate effect of Type IV reactions is a cytotoxic or cell destruction phenomenon. Type IV reactions can involve small molecular weight chemicals, including some food additives (49,69) that apparently act like haptens and react with tissue proteins before exerting their effects (53). The onset of symptoms in Type IV reactions typically occurs 6-24 h following ingestion of food.

May and Bock (52) have argued convincingly that the separation of food allergies into immediate and delayed categories is arbitrary. Their argument centers on the realization that the interaction of the allergen with IgE or sensitized cells is surely immediate in all immune reactions. Consequently, the interval between ingestion and the onset of symptoms is likely to be affected by the quantity of food consumed, the degree of hypersensitivity, successive and concomitant exposures, threshold for complaints and other factors (52). All of these factors are unrelated to the type of immunological phenomenon (IgE-mediated or sensitized cell-mediated) taking place. Consequently, differential categorization of food allergies as Type I or Type IV reactions must be based on demonstrated immunological differences and not simply on the time between ingestion and onset of symptoms.

Since allergists cannot even agree on a definition for food allergy, accurate estimates of the incidence of food allergy are virtually impossible to obtain. Other reviewers have reported a wide range of incidence figures for food allergy including 0.3% (33), 20% (4), 20% (6), 25% (15)
and 60% (71) of the population. It is generally agreed that not more than 1% of the population suffers from immediate hypersensitivity (Type I reactions). The argument concerns delayed hypersensitivity (Type IV reactions). Breneman (15,16), who estimates that 25% of the population suffers from some type of allergic disease, concludes that 95% of these allergies fall into the delayed hypersensitivity category. However, he would include malabsorption diseases, inflammatory responses and enzyme deficiencies as forms of delayed hypersensitivity. This broad definition of the term food allergy would undoubtedly serve to inflate his incidence figures. At the opposite end of the spectrum, Golbert (33) estimates that the incidence of allergy among children is 0.3%. He discounts delayed hypersensitivity, which is difficult to demonstrate clinically, as extremely rare. Consequently, his figures rely almost entirely on the incidence of immediate hypersensitivity.

This reviewer will not even attempt to estimate the incidence of food allergy other than to concede that it probably lies somewhere between 0.3% and 25% of the population. Even determining the existence of allergic reactions to specific foods is difficult. One problem with such determinations is the number of testimonial reports of food allergy in the scientific literature. One example chosen randomly from many possible candidates follows. Rousseaux (64) described the case of an adult female who experienced giddiness which was exacerbated by honey and grapefruit. Honey and grapefruit are not usually considered allergenic foods and giddiness is not a normally described allergic symptom. Rousseaux (64) provided no immunological testing of this patient. Consequently, this case should be classified as food sensitivity but further investigation might reveal an allergic basis. This example should serve to illustrate the problem.

**SYMPTOMS OF FOOD ALLERGY**

A variety of symptoms, including cutaneous, gastrointestinal and respiratory manifestations, have been associated with Type I reactions. The most common cutaneous symptoms are urticaria (hives) and angioedema (swelling), although eczema or atopic dermatitis has also been described. Eczema may be a Type IV rather than a Type I symptom, although the literature is confusing on this point. The gastrointestinal symptoms frequently include vomiting, abdominal cramps, nausea and diarrhea. The occasional reports of steatorrhea (fatty fecal discharge), colic and stomatitis (inflammation of the mucous membranes of the mouth), and other gastrointestinal symptoms (6) are either rare or largely unsubstantiated (52). The respiratory symptoms include rhinitis (inflammation of the nasal membranes) and asthma most commonly, although respiratory symptoms tend to occur far more frequently in pollen allergies than with food allergies. Food-allergic individuals with Type I reactions usually experience no more than a few of these symptoms. Heiner's syndrome (8,38) should also be mentioned here, although it may not be manifestation of Type I reactions. Heiner’s syndrome was originally described in relation to cow’s milk allergy in infants and consists of poor weight gain, gastrointestinal and upper respiratory symptoms, recurrent pulmonary disease and iron deficiency anemia (8,38). Heiner’s syndrome involves antibodies other than IgE, and may be due to the accidental aspiration of milk (52).

The association of certain symptoms with food allergy has been somewhat controversial. The controversial symptoms have included common symptoms such as eczema or atopic dermatitis and asthma and uncommon symptoms such as otitis media and Meniere’s disease. Several recent studies have been performed on the association between these symptoms and food allergy. In a study of 134 subjects with atopic dermatitis, Bonifazi et al. (14) noted that only 45 had a clinical history of food sensitivity to eggs, milk or cod, while 79 showed clinical evidence of allergic sensitivity to these foods by virtue of the in vitro radioallergosorbent test (RAST). These workers concluded after monitoring IgE levels that atopic dermatitis was not necessarily attributable to specific sensitization by distinct food allergens, but was characterized by an excessive production of IgE (14). Similarly, Hammer (37) demonstrated that only 15 of 81 children less than 5 years old with atopic dermatitis suffered symptom exacerbation on oral provocation with cow’s milk and/or cereals (wheat, rye and oat mixture). In this study, initial serum IgE levels and evidence of specific antibodies by RAST did not distinguish a sensitive group. On the other side of the controversy, 14 of 20 infants with atopic dermatitis showed improvement on a diet eliminating cow’s milk, eggs, chicken and beef (7). High-risk infants with an allergic parent showed a lessened incidence of eczema if breast feeding was maintained for the first 12 weeks of life and milk products, eggs and fish were avoided for the first year (3,50). Some recent immunologic findings suggest that food-allergic persons with atopic dermatitis also have defective immune surveillance systems, including defective cell-mediated immunity, decreased T lymphocyte numbers and defective effector cell functions (26), all of which could lead to increased IgE levels and food allergen sensitivity. With asthma, food allergy seems to play a role in a consistent, though small, percentage of the affected individuals. A recent study with 147 asthmatic children and 250 asthmatic adults indicated that 15% of the children and 17% of the adults had food allergies, as demonstrated clinically by the skin test (29). Secretory otitis media and Meniere’s disease are rarely reported symptoms of food allergy. Estimates have been made that from 5 to 25% of the cases of secretory otitis media have an allergic component (62,63). Some of these individuals respond to elimination of certain foods from their diets (62), although more careful studies are needed to substantiate this relationship. The involvement of food allergy in Meniere’s disease is uncertain. Holloman (39) has discounted the importance of food allergy in

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Meniere's disease, but occasional reports continue to surface that implicate foods in the etiology of this condition (23).

A particularly serious manifestation of Type I hypersensitivity is anaphylaxis. Anaphylaxis is a severe, generalized shock reaction that can result in death. Fortunately, anaphylactic reactions to foods are rarely reported. Within the past year, anaphylactic reactions to cow's milk (60), sunflower seeds (55), shellfish (51) and gum tragacanth (21) have been reported. Severe systemic reactions are known to occur occasionally with the more frequently reported allergenic foods such as peanuts, fish and egg whites.

The manifestations of food-associated Type IV reactions are less clearly defined. Contact dermatitis associated with the handling of certain foods and food components by sensitive individuals has been demonstrated (9,49,65). Gastrointestinal, respiratory and other symptoms of allergy are difficult to confirm in Type IV reactions. Some allergists believe that Type IV reactions to foods are rare (33), while others would consider them to be common (15,16,19). Numerous symptoms have been ascribed to delayed hypersensitivity reactions but the evidence in many cases is sketchy. The possible association of certain behavioral disorders with Type IV reactions has been the subject of continuing conjecture. Speer (67) originally coined the term, allergic tension-fatigue syndrome, to describe a rather complex set of behavioral changes that he noted in certain allergic individuals. The fatigue symptoms included motor tension (tiredness and achiness), sensory fatigue (sluggishness, torpor) and mental fatigue (depression, paranoia etc.). The tension symptoms included motor tension resembling hyperkinesis and sensory tension (irritability, torpor) and mental fatigue (depression, paranoia etc.). Widespread support for the idea of a direct correlation between allergy and behavioral symptoms has never surfaced. Recently, Crook and others (19,20,59,62) have suggested that these behavioral symptoms may be manifestations of Type IV reactions to foods, although their reports have been largely testimonial. The suggestion has been made that hyperkinesis may have an allergic basis (19). One report links symptom exacerbation in an autistic child with certain foods (56). Recent studies have demonstrated that a small percentage of children with various behavioral disorders have food allergies and/or revert some symptoms upon withdrawal of certain foods (41,70).

Further support for an association between hyperkinesis and allergies comes from preliminary findings that suggested that cromolyn DSG, a drug that blocks histamine release from mast cells, had a positive behavioral effect on allergic hyperkinetic and autistic children (65). Considerable research is obviously needed to document further the manifestations of Type IV reactions to foods.

Numerous unusual symptoms have also been ascribed to food allergy. Since many of these symptoms were reported in testimonial style with no immunological evidence of allergy, they would mostly fall into the category of food sensitivity. That vast number of such reports eliminates the possibility of any thorough cataloguing of the symptoms or the suspect foods. Several recent reports have included anxiety, depression, agoraphobia, headache, aphthous ulcers, localized pain, chronic nausea, lethargy, giddiness, sweating, food craving, itch, general weakness, heatwaves, palpitations, blurred vision and a feeling of suffocation as symptoms of food allergy (24,54,64). The multiplicity of symptoms and a lack of general agreement among clinicians regarding which symptoms to definitely associate with allergic reactions have contributed significantly to the problems associated with correctly diagnosing food allergy.

CLINICAL DIAGNOSIS OF FOOD ALLERGY

Since this review is not intended to be clinically oriented, only limited comments on the clinical diagnosis of food allergy will be made. The reader is directed to several recent reviews for more complete discussions of this topic (32,33,52).

The diagnosis of food allergy remains a controversial subject. Numerous methods have been developed for the diagnosis of immediate hypersensitivity to foods, including subjective methods (clinical histories, diet diaries, and elimination-challenge studies), the more objective double-blind challenge studies, various cutaneous tests (intracutaneous test, direct skin test, and Prausnitz-Küstner or passive transfer test), and indirect, in vitro testing methods (pulse acceleration, leucopenic index, urinary proteose, eosinophilia, serum immunoglobulin levels, radioallergosorbent tests, cytotoxic reactions, leukocyte histamine release, lymphocyte transformation tests and intestinal biopsy). The pulse acceleration test, leucopenic index and urinary proteose test have fallen into general disfavor due to lack of accuracy. The other tests continue to be used to some degree in the diagnosis of Type I reactions to foods. Methods for the diagnosis of delayed hypersensitivity to foods are not generally available. Cutaneous Type IV reactions such as contact dermatitis can usually be confirmed by patch testing (58,66). Diagnosis of other manifestations of Type IV reactions usually relies on some type of elimination-challenge study. Since these responses are subjective, the methods are not wholly reliable.

The most readily available diagnostic procedures for food allergies for both Type I and Type IV reactions are the subjective methods of history-taking, elimination diets and challenge studies. Johnstone (43) suggests that despite the recent proliferation of indirect methods for diagnosis of Type I reactions, no marked improvement has been made on the use of careful histories and elimination diets. One recent study showed that food-sensitive patients remitted 70% of their allergic symptoms when placed on a chemically defined diet (40). Subsequent oral challenge studies allowed identification of the offending foods (40). The chemically defined diet was an elemental diet containing L-amino acids, glucose,
glucose oligosaccharides, and safflower oil (29). This diet caused remission of symptoms in 85% of the food-sensitive patients studied (40). The use of double-blind challenge studies in the diagnosis of food allergy has been recommended (32,52). Double-blind challenges may be extremely useful in the confirmation of clinical histories. May and Bock (52), using double-blind studies of children with positive histories, were able to provoke symptoms with foods in only 33% of children 3 to 16 years of age and 52% of children less than 3 years of age. The employment of a double-blind design in food challenge studies should improve the objectivity of such evaluations. Certainly in cases where immunological evaluation is not available, such as with certain suspected cases of delayed hypersensitivity, the use of double-blind challenge tests is the preferred procedure.

While the use of such subjective tests is considered by some allergists to be reasonably definitive in the diagnosis of food allergy, others have stressed the necessity of determining an immunological basis for food allergy (10,27,32,33). The most commonly used tests to demonstrate such an immunological basis are the skin test, the radioallergosorbent test (RAST), histamine release from leukocytes and the passive transfer test. These tests are limited mostly to the diagnosis of Type I reactions. The direct skin test involves the intracutaneous introduction of the food extract and observation for an immediate wheal-and-flare reaction (1,3,33). The RAST is an in vitro test using a small amount of patient's serum. The RAST is available only in certain laboratories due to the need for some specialized equipment and reagents. The RAST has been described in detail by Yunginger (73). The other tests have been performed on a more limited basis for a variety of reasons relating to difficulty, risk to the patient and comparative accuracy.

Unfortunately, the results of the skin test and the RAST do not always agree even though both tests are mediated by IgE (2,18,30,57). In addition, neither the skin test nor the RAST always agree with the clinical histories of the patients (2,13,14,37,52). The agreement is better for certain foods than for others (2). Several reasons may exist for the occasional disparate results between the tests and the clinical histories. As mentioned previously, clinical histories are not invariably accurate although the accuracy can be improved by double-blind challenge studies (52). While the skin tests and RASTs are both mediated by IgE, some technical differences exist. May and Bock (52) suggest that the RAST is less reliable because it depends on serum IgE, and IgE has a short half-life in the circulation. IgE fixed in the skin has a longer half-life, making the skin test somewhat more stable as an index of allergenicity (52). However, the RAST has been used effectively in numerous diagnostic laboratories (1,73). The skin test is not perfect either since it relies on the skin response to released mediators primarily histamine. The histamine responsiveness of the skin may vary between individuals and is not perfectly correlated with serum IgE levels. Other factors may shed some doubt on the reliability of either the skin test or the RAST in the diagnosis of food allergy. Both the skin tests and the RASTs require food extracts containing the allergen. The extracts used in clinical situations are usually rather crude preparations that can be obtained commercially. The standard practice seems to be to produce these food allergen extracts from fresh unprocessed foods. While the use of fresh material may have some validity with pollen allergens, it has dubious value in preparing food allergen extracts. For example, why prepare a peanut allergen extract from raw peanuts when the vast majority of consumers eat roasted peanuts, peanut butter or other processed forms of peanuts? This practice may represent convenience rather than logic. Some food allergens, such as the coffee bean allergen (47), are known to be destroyed by processing. The effect of processing on the allergenicity of most foods has not been investigated. However, use of unprocessed food extracts may lead to misdiagnosis of food allergy and a disparity between the skin tests or RASTs and the clinical histories. Fries (25) suggests that such misdiagnosis may occur rather frequently with allergy to chocolate.

Another factor adding to the difficulty in the diagnosis of food allergy is the lack of purified standards to use in the extracts. The presence of other substances in the crude extracts almost certainly confounds the diagnosis of food allergy on occasion by either inhibiting or promoting a positive response. Only a few food allergens have been isolated and identified. The most notable effort has been the purification of allergen M from cod (5,22). $\beta$-Lactoglobulin appears to be the most active milk allergen (11,34,35,46), although reactions to casein, $\alpha$-lactalbumin, and bovine serum albumin have also been observed (46). It should be noted that $\beta$-lactoglobulin and casein are stable to heat in excess of 100°C, while $\alpha$-lactalbumin and bovine serum albumin are heat-labile (48). The most active allergen in egg whites is the heat-stable protein, ovomucoid (12,72). Reactions to ovalbumin and lysozyme have also been noted to a lesser extent, while conalbumin seems to be non-allergic (72). The association of heat-stable proteins with allergenicity undoubtedly allows them to survive certain types of processing.

A further complicating factor in the diagnosis of food allergy is the effect of proteolytic digestion on the allergenicity of food proteins. Many food allergens are stable to proteolytic digestion (1,10) but some notable exceptions may exist. Spies et al. (68) demonstrated that with cow’s milk proteins, pepsin hydrolysates were more antigenic than the native proteins. Haddad et al. (36) recently confirmed this finding by showing that while only four of 10 milk-allergic patients had positive RASTs to $\beta$-lactoglobulin, 10 of 10 had positive RASTs to pepsin-trypsin digests of $\beta$-lactoglobulin. This result suggests that the clinical diagnosis of cow’s milk allergy could be improved by employing proteolytic digests in
the allergenicity tests. More research is needed to determine the allergenicity of proteolytic hydrolysates of other allergenic food proteins.

FUTURE RESEARCH NEEDS IN FOOD ALLERGY

Three areas of research on food allergy need to be investigated to provide some solutions to the continuing enigma surrounding food allergies.

(a) A need exists to improve the quality and reliability of the food allergen standards used in clinical diagnostic procedure. The ultimate reliability would be achieved with a series of highly purified food allergen preparations. However, purification of the numerous food allergens will be time-consuming work and the eventual cost may be prohibitive. Some increase in reliability might be obtained by simply preparing the crude extracts from processed foods.

(b) To substantiate the claims of Crook (19, 20), Breneman (15, 16) and others that delayed hypersensitivity or Type IV reactions to food occur with reasonably high frequency, better methods for diagnosis of non-cutaneous Type IV reactions are needed. A better understanding of the molecular mechanisms of delayed hypersensitivity may be prerequisite to the development of new methods.

(c) To completely destroy the old myth that nothing can be done about food allergy anyway, let me emphasize that the food industry is already performing some positive preventive measures. For example, green coffee beans are quite allergenic (47). Consequently, roasting serves as a hypoallergenic process (47). Substitution of soybean-based and other formulas for cow's milk has been another positive step in the formulation of hypoallergenic foods. Considerable research is needed on the allergenicity of various foods. Perhaps methods can be developed for the hypoallergenic processing of various foods.

REFERENCES

pound for canned or frozen products. The energy savings are based on a number of factors that distinguish “Gaspak” from canning and freezing. In transportation, for instance, there is no need to refrigerate “Gaspak” produce. In a similar vein, “Gaspak” products are not packed in water, as are those that are canned, where water constitutes as much as 40 percent of some can contents.

Frozen foods require the most energy in home cooking. Energy used in cooking “Gaspak” food is comparable to that used in cooking pre-thawed frozen foods.

Dr. Kramer says that if the “Gaspak” technology replaced only the canning and freezing of fruits and vegetables, the estimated energy savings would be the equivalent of about 25 million barrels of crude oil. The estimated energy savings of industrial plants now processing these foods would be 75 to 80 percent and reduced energy means reduced food processing costs, Dr. Kramer notes.