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Preventing Peanut Panic

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For generations, peanuts have been a favorite, healthy American snack food (Planters, 2005). In recent years, however, peanuts increasingly have become a life-threatening nuisance. The profitable little protein-packed legume is responsible for about 160 to 200 deaths annually in the United States, and peanuts are responsible for causing 80% of all food allergy fatalities (Sampson, 2003; Scurlock, Lee, & Burks, 2005). It is estimated that 1.5 million Americans have peanut allergies and that hospital emergency departments treat about 30,000 cases of food-related allergic anaphylaxis per year (Sampson, 2003). The incidence of food allergies has been reported to be 1% to 2% in the general population and as high as 5% in children younger than 5 years, with 1.3% of all children exhibiting an allergic reaction to peanuts (Rous & Hunt, 2004).

The most common food trigger of anaphylaxis, as well as the most common overall trigger, is peanut allergy (Leung & Bock, 2003). Both American and British population studies of children cite an increasing prevalence of sensitization to peanuts (Chiu, Sampson, & Sicherer, 2001; Grundy et al., 2002; Sampson, 2000). In the United States, the prevalence of peanut allergy increased from 0.4% in 1997 to 1.0% in 2002 (Sicherer, Munoz-Furlong, & Sampson, 2003). Signifi-

cantly, peanut sensitivity on a standard skin prick test has increased by 55%, while allergic reactions increased by 95% in the past 10 years (Rous & Hunt, 2004). This increase may not be attributed solely to increasing sensitivity but also an increased awareness of allergies and improved reporting and management.

The increased incidence of allergies of all types has prompted speculation on their cause. Some scientists believe that the eradication of many infectious and communicable diseases may have affected the immune system. Others believe that improved hygiene practices also may alter the immune system and predispose individuals to the development of allergies later in life (Rous & Hunt, 2004). Why food allergies develop in one child and not another is yet unanswered, but the answer may lie in the composition of multiple genes (Long, 2002).

OUT-OF-HOME EXPOSURE TO PEANUT ALLERGEN

The increase in peanut allergy prevalence has led to heightened public and health care professional awareness, as well as demands for a rational plan for prevention of serious allergic reactions (Weisnagel, 2005). Initiation of "peanut-free" policies has become a common administrative solution to

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preventing full-blown anaphylactic reactions to peanut allergy, especially in schools and day-care settings. However, the interpretation of the meaning of “peanut-free” and enforcement of control measures are neither simple nor straightforward.

It is obvious that eliminating the threat of peanut exposure for an allergic child is a rational prevention for food-related anaphylaxis. Exposure to the peanut allergen is not easy to control in a pediatric population. Peanut butter sandwiches have been a staple of the typical school lunchroom, a primary setting for encouraging social eating habits. Children bring their brown bag lunches from home, frequently containing a “PB & J” sandwich, and often they pool their resources into a collective meal. However, what about the cupcake with peanut sprinkles or the potato chip fried in peanut oil? How much protein triggers a response, and can such a response be triggered from ingestion, a kiss, a handshake, or an airborne inhalation? Enforcing parental adherence, student compliance, and school response readiness demands knowledge based on hard data to avoid an overprudent protocol, cynicism, and apathy, as well as needless panic.

THE PATHOPHYSIOLOGY OF PEANUT SENSITIZATION AND ALLERGY

Peanuts contain 200 mg of seven identified proteins that serve as allergens (Sampson, 2002). The two dominant molecules in peanuts that contain allergic epitopes are Ara h 1 and Ara h 2, which are seed-storage glycoproteins; these are the cause of 95% of peanut allergy reactions (Long, 2002). The other five proteins are associated with only 50% of peanut allergic responses (Maleki et al., 2003).

Research also has shown that the type of peanut preparation contributes to the level of allergenicity (Beyer et al., 2001). Boiling

or frying peanuts reduces peanut protein allergens, whereas dry roasting enhances the three major peanut protein allergens.

Allergic reactions originate from highly specific binding between epitopes on the surface of the offending protein substance to regions of antigen-binding in cell-bound immunoglobulin E (IgE) (Long, 2002). In a pediatric research double-blind placebo-controlled peanut food challenge, the lowest dose that provoked an allergic response was 50 mg (about one sixth of a peanut), with the most common reactions being rhinitis and urticaria (Lack et al., 2003). However, newer studies report even lower allergic response doses that stimulate the immune system, resulting in a clinical reaction (Morisset et al., 2003; van Odijk et

persensitive individuals, touch or inhalation of allergens may produce the allergic response. The severity of the reaction depends on which and how many systems are involved and upon the amount of peanut protein exposure (Al-Muhesen, Clarke, & Kagan, 2003).

The first allergic response to peanuts usually occurs between the median age of 14 to 24 months (Sampson, 2002). Of persons having a first allergic response, 89% have reactions involving the skin, 42% have respiratory reactions, 26% have gastrointestinal reactions, and 4% have a cardiac reaction. Of the 1.3% of U.S. children who have had reactions to peanuts, 50% have involvement of one system; 30%, two systems; 15%, three systems; and 1%, four systems (Sicherer, Munoz-Furlong, Burks, & Sampson, 2001).

The IgE-mediated (late-phase) adverse reaction to peanuts can occur immediately or after a few hours.

al., 2005).

Millions of IgE antibodies circulate in the blood and bind to mast cells, and basophils present in the gastrointestinal tract, skin, and lungs. The food allergen binds to the food-specific antibodies on the basophils and mast cells, which in turn release mediators such as histamine, prostaglandins, and leukotrienes (Scurlock et al., 2005). These mediators promote the allergic manifestations that occur in the respiratory, gastrointestinal, cardiovascular, and cutaneous tissues, resulting from smooth muscle contraction, mucus secretion, edema, and vasodilation.

The IgE-mediated (late-phase) adverse reaction to peanuts can occur immediately or after a few hours. Most responses occur from ingestion, but in rare cases of hy-

Those with multiple system reactions and concomitant asthma have poorer clinical outcomes.

Seventy percent of first-time reactions occur at the first known exposure (Sicherer et al., 2001). Research has not yet established a conclusive link between peanut allergen exposure during intrauterine life or during breastfeeding and sensitization. Sibling and parent confirmed peanut allergy diagnoses validate a high risk for peanut allergy in subsequent children, suggesting that it would be prudent for mothers to avoid peanut ingestion during subsequent pregnancies and during breast feeding (Sicherer et al., 2001). Although cashews, pistachios, and walnuts originate from a different botanical family, in 25% to 35% of patients with an allergy to peanuts, an al-

lergic reaction to these tree nuts also will develop (Sampson, 2002). Cross-reactivity among members of the legume family also have been researched, including adverse reactions to soybeans, lupines, peas, and chickpeas (Wensing et al., 2003).

DIAGNOSIS

The diagnosis of peanut allergy should be made with care as the implications of changes to food avoidance and selection are important (Rangaraj et al., 2004). The history of the suspected allergy offers important clues to health professionals. Important historic information that parents can share with health professionals to help establish that a food allergy reaction has occurred includes the following: (a) the food suspected to initiate the reaction, (b) the quantity of the food ingested, (c) the length of time between ingestion and development of symptoms, (d) description of symptoms observed, (e) if these same symptoms developed when the food was eaten previously, (f) activities associated with food allergic reaction, and (g) length of time since the previous reaction (Scurlock et al., 2005).

Detection of peanut-specific IgE via a standard prick test or fluoroenzyme immunoassay confirms the diagnosis of peanut allergy (Scurlock et al., 2005). The standard prick test is a skin test that introduces a diluted extract of peanut proteins into the skin. A positive reaction occurs within 15 minutes in the form of a red bump similar in appearance to a mosquito bite. However, many children younger than 2 years of age have negative responses to the skin test even when they have a true peanut allergy. A positive skin test indicates the possibility of symptomatic reactivity to a food. While the positive predictive accuracy is less than 50%, a negative skin test confirms the absence of an IgE-mediated reaction with a

negative predictive accuracy greater than 95% (Scurlock et al.). Although the skin-prick test remains an excellent tool for excluding IgE-mediated food allergies, it is only suggestive of the presence of clinical food allergies.

The two primary blood tests used for peanut allergy testing are the radioallergosorbent test and the enzyme-linked immunosorbent assay (Scurlock et al., 2005; Wensing et al., 2003). As with skin testing, many false-negative results are reported.

There are two primary reasons for false-negative results. First, the prick test extract is very vulnerable to rapid deterioration, and peanut proteins disintegrate easily. Second, the quality of allergy testing equipment has less than 10% internal reproducibility, which is the World Health Organization standard (Elg, 2004). However, the newest in vitro measurement tests of serum food-specific IgE have a greater than 95% predictive value for food allergies, and the double-blind, placebo-controlled food challenge has become the reference standard and the most accurate means for the diagnosis of food allergy (Scurlock et al.).

A number of longitudinal studies have monitored children to determine the onset of sensitivity to peanuts and other allergens using IgE antibodies as predictive biomarkers for inhalant allergies. Food allergies had a negative predictive value of 90% in one study (Eysink et al., 2002). A second study concluded that utilizing the sum of antibody levels in combination with the number of positive allergies represents a more efficient diagnostic tool than the single IgE measure (Wickman, Soderstrom, Van HageHamsten, & Ahlstedt, 2005). Yet another study investigated children with known food and airborne allergies and used atopic dermatitis (rash) as a marker. It was found that many food allergies resolved over time but that peanut allergy was more

likely to persist, a fact that cannot be ignored (Gustafson, Sjoberg, & Foucard, 2003; Rangaraj et al., 2004).

CONTROLLING EXPOSURE

Elimination of peanut oils and food containing peanuts seems rational, yet with so many prepackaged and processed foods being used for meal preparations, the likelihood of exposure is great. Because food proteins aerosolize into vapors during cooking at high temperatures, even in well-ventilated cafeterias, concern has arisen about the possibility of respiratory exposure to peanut protein in the air and the triggering of a full-blown anaphylaxis in the school setting.

When airborne peanut protein exposure and reactions of children with known peanut allergies were explored, no allergic symptoms or anaphylaxis were observed when children allergic to peanuts were not aware of the airborne exposure (Simonte, Mofidi, & Sicherer, 2003). Interestingly, when aware of the exposure, symptoms of itchy eyes, sneezing, and runny nose resulted. In a research article by Perry et al. (2004), no peanut allergen was detected in the air after research subjects consumed peanut butter or unshelled peanuts or after shelling of peanuts occurred. Cutaneous reactions in the form of rash from exposure were consistent with the amount of skin exposure to the peanut protein. These exposures occurred from handshakes, kissing, and the wiping of eyes, nose, or oral mucosa contaminated with peanut allergen. In conclusion, the chance of a life-threatening anaphylactic reaction from airborne or cutaneous exposure was insignificant. Life-threatening exposures result most often through direct ingestion.

RECOMMENDATIONS AND GUIDELINES FOR PREVENTING PEANUT PANIC

Parents want to protect their children, and this desire extends

BOX. Parent handout

Signs and Symptoms of Food Allergy

- Timing
 - Symptoms may occur within minutes of exposure to several hours after ingestion
- Food-induced anaphylaxis includes:
 - Swelling of the lips, tongue, palate, and throat
 - Tingling, burning, itchiness in the mouth
 - Throat itchiness
 - Swelling around the eyes, itchiness, tearing
 - Nasal congestion
 - Feelings of tightening in the chest
 - Noisy, harsh, high-pitched breathing
 - Nausea, vomiting, or cramping abdominal pain within 1 to 2 hours
 - Diarrhea within 2 to 6 hours
 - Hives, wheals
 - Generalized redness of the skin
 - Chronic skin disorders like atopic dermatitis or eczema
 - Low blood pressure
 - Fast heart rate

Management of Peanut Allergies

- Avoidance and elimination from diet
 - If it is impossible to remove the food, then label offending foods with warning stickers
- Early recognition of symptoms
- Check all labels for the addition of peanuts
 - When eating away from home, request information on food content
 - Avoid buffets, ice cream parlors, unlabeled candies, and desserts
 - Check into types of oil in preparing food
- Highly processed peanut oils (acid-extracted and heat-distilled oils) do not contain peanut protein
- Cold-pressed, extruded peanut oils contain peanut protein
- Be wary of cross-contamination of food during cooking in restaurants through use of the same pan without adequate cleansing between uses
- Be knowledgeable about unanticipated exposures and work with hosting parents and teachers to make it a safe experience for all children
 - Holidays such as Halloween: tell the child to have a knowledgeable adult approve the treats before the child eats them
 - Birthday parties
 - School parties
- In school or day-care settings, have a clearly established emergency plan with medications that are easy to locate and accompanied by spoons or measuring cups marked for dosage)
 - Check drug label expiration dates on a regular basis
 - Have a photograph of the child near the first aid station with directions for care
- Parents, educators, child-care providers, and enabled children older than 7 years should be taught correct administration of emergency medications
 - Involve nursing and medical staff of facility or from the child's primary care medical home when possible
- Hand washing is essential for removal of allergens using soap and water, but not plain water or waterless antibacterial soap
- Wash tabletops and food areas with common household cleaners but not dishwashing liquid

*Intramuscular epinephrine and liquid
diphenhydramine should be easily available
with appropriate dosing clearly documented.*

into the school environment. Concerned parents should communicate with the school frequently and also educate their children about the importance of knowing what foods to avoid in the cafeteria. Children with peanut allergies must be constantly aware of potential hazards in their environment and should carry an EpiPen injection to treat food-induced anaphylaxis (Avery, King, Knight, & Hourihane, 2003). Research has revealed its underuse with food allergies (Kim, Sinacore, & Pongracic, 2005). Parents, children, and caregivers should be knowledgeable about correct use of the EpiPen. Written emergency plans for accidental ingestion of peanuts should be established and adequate doses of non-outdated medication should be available (Sampson, 2002). Intramuscular epinephrine and liquid diphenhydramine should be easily available with appropriate dosing clearly documented. Children should continue to be observed after remedial medications are administered because of the risk for a biphasic reaction (Sampson, 2003). In all situations, parents and emergency care physicians should be notified as well.

School staff and administration also must do their part. Because frequent hand washing with soap and water is so effective in controlling allergen exposure, soap and paper towel supplies should be maintained. A recent research study demonstrated that no evidence of peanut allergen remained on subjects' hands after washing with liquid soap and water compared with positive residual allergen on hands washed with plain water or waterless antibacterial soap (Perry, Conover-Walker,

Pomes, Chapman, & Wood, 2004). Carefully scrubbing down lunch tables with common household cleaning agents also removed peanut allergen, whereas using dishwashing liquid left some residue on 4 out of 12 tables (Perry et al.). If child care centers and schools prohibit the sharing of food and drinking straws, wash shared utensils, prohibit home-made birthday snacks where ingredients are unknown, and regulate dietary practices, the risk of allergen exposure will decrease. Having photographs of the allergic children posted near emergency supplies also is appropriate, alerting both regular and substitute personnel regarding allergic children and their emergency care supplies. These are all prudent strategies to minimize the school's exposure to legal liability while protecting the child from possible exposure to the peanut allergen, decreasing the risk of anaphylaxis, and assisting in preventing peanut panic.

CONCLUSION

Parents of children with strong allergic reactions to peanuts often fear their accidental exposure to potentially life-threatening doses of allergen outside the home. The parent handout in the Box offers evidence-based guidance to help parents recognize symptoms of a true peanut allergy, prepare their child and their child's school to handle allergic reactions, and alert parents to common hazards. Proactively educating parents helps them to ensure the safety of their child during out-of-home experiences through reasonable precautions.

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