
Availability of the epinephrine autoinjector at school in children with peanut allergy

Moshe Ben-Shoshan, MD*; Rhoda Kagan, MD, FRCPC†; Marie-Noël Primeau, MD, FRCPC*; Reza Alizadehfar, MD, FRCPC*; Nina Verreault, MD, FRCPC*; Joyce W. Yu, MD, FRCPC*; Nathalie Nicolas, MD, MSc‡; Lawrence Joseph, PhD§; Elizabeth Turnbull‡; Claire Dufresne¶, Yvan St. Pierre, MSc‡; and Ann Clarke, MD, MSc, FRCPC‡||

Background: Peanut allergy accounts for most severe food-related allergic reactions, and accidental exposures are frequent. Delayed administration of epinephrine and the allergic individual's failure to personally carry epinephrine contribute to fatal outcomes.

Objectives: To describe epinephrine autoinjector availability at school and to determine factors that might affect autoinjector availability in children allergic to peanut.

Methods: Two hundred seventy-one children with peanut allergy living in Quebec were queried about their autoinjector. Logistic regression models were used to select factors associated with device availability.

Results: Four of 271 children diagnosed as having peanut allergy were not prescribed autoinjectors. Forty-eight percent of the children did not carry the autoinjector with them at school. In 78.0% of those, the autoinjector was located in the nurse's or another school office, which was staffed by a full-time nurse only in 18.5%. Of all the respondents, those administered epinephrine for a previous reaction (odds ratio [OR], 2.7; 95% confidence interval [CI], 1.3–5.7), older children (OR, 1.1; 95% CI, 1.0–1.2), and those living only with their mother (OR, 3.4; 95% CI, 1.0–11.0) were more likely to carry the autoinjector with them at school. Of children 7 years or older, those who experienced a severe reaction were more likely to carry their autoinjector (OR, 3.3; 95% CI, 1.4–8.1).

Conclusions: Almost 50% of children allergic to peanut might experience a delay in anaphylaxis treatment due to limited access to their device. More education is required regarding the importance of a readily available autoinjector.

Ann Allergy Asthma Immunol. 2008;100:570–575.

INTRODUCTION

Peanut allergy is a major health problem. It accounts for most severe food-related allergic reactions, tends to manifest early in life, does not usually resolve, and can be induced by trace quantities in highly sensitized people.^{1–9} In addition, it has recently been suggested that the prevalence of peanut allergy is increasing and affects 0.8% to 1.5% of British and North American children.^{10–13}

Avoidance of peanut is often difficult. Although peanut is ubiquitously used in the food industry and may cross-contaminate foods and utensils, its presence is not always clearly specified on product labels or in restaurants.^{14,15} Consequently, the risk of accidental exposure is considerable, and

the annual incidence of accidental exposure has been reported to vary between 14.3% and 50%.^{7,16,17} If such an exposure does occur, the principal treatment of an acute allergic reaction is prompt administration of epinephrine.² Hence, individuals with a food allergy should always be prescribed an epinephrine autoinjector^{2,18,19} at the appropriate dose,²⁰ be advised that it must always be readily accessible,²¹ and be shown how to correctly operate it.^{18,22}

Given that the risk of accidental exposure is significant and that epinephrine administration is the principal therapy, we sought to estimate the proportion of children allergic to peanut who carry their epinephrine autoinjector with them at school (referred to as “autoinjector availability”) and to identify factors that might affect this availability.

METHODS

Patient Selection

This study was approved by the Research Ethics Board of the McGill University Health Center. Children aged 4 to 18 years with peanut allergy were recruited from the Montreal Children's Hospital Allergy Clinic. Their medical records were reviewed to confirm the eligibility criteria (defined in the following subsection) and to abstract information on family history of atopy, initial and most severe reactions to peanut (if applicable), and previous use of epinephrine.

Affiliations: * Division of Pediatric Allergy and Clinical Immunology, McGill University Health Center, Montreal, Quebec, Canada; † Division of Allergy and Clinical Immunology, North York General Hospital, Toronto, Ontario, Canada; ‡ Division of Clinical Epidemiology, McGill University Health Center, Montreal, Quebec, Canada; § Department of Epidemiology and Biostatistics, McGill University, Montreal, Quebec, Canada; ¶ Association Québécoise des Allergies Alimentaires, Montreal, Quebec, Canada; || Division of Allergy and Clinical Immunology, McGill University Health Center, Montreal, Quebec, Canada.

Disclosures: Authors have nothing to disclose.

Funding Sources: This study was supported by The Foundation of the McGill University Health Center.

Received for publication October 25, 2007; Received in revised form November 29, 2007; Accepted for publication December 3, 2007.

Children with peanut allergy were also recruited from the Association Québécoise des Allergies Alimentaires (AQAA), a lay organization of people and parents of children with food allergy, through advertisements placed in the association newsletters and at annual association conferences. Interested individuals were asked to consent to a release of medical information from their treating physician. If this information confirmed that the individual fulfilled the eligibility criteria for peanut allergy, they were then enrolled in the study.

Establishing the Diagnosis of Peanut Allergy

Children were eligible for study enrollment if either of the following criteria were fulfilled: (1) the child had a convincing history of an allergic reaction to peanut and a positive skin prick test (SPT) reaction to peanut or a peanut specific IgE level of at least 0.35 kU/L or (2) the child had no history of peanut ingestion or an uncertain clinical history of peanut allergy and either a positive SPT reaction and a peanut specific IgE level of at least 15 kU/L or a positive SPT reaction and positive food challenge with peanut or a positive food challenge only. Age at diagnosis was either the age at which the child had his or her first allergic reaction to peanut or, in the case of children who had never been exposed to peanut, the age at which the diagnosis was made by a physician after confirmatory diagnostic testing.

Clinical History

A convincing history of peanut allergy was defined as a minimum of 2 mild symptoms or signs or either 1 moderate or 1 severe symptom or sign occurring within 120 minutes after peanut contact or ingestion. The severity of the reaction was defined as follows: (1) mild: pruritus, urticaria, flushing, or rhinoconjunctivitis¹; (2) moderate: angioedema, throat tightness, change in voice, coughing, difficulty breathing (other than wheeze), nausea or vomiting, or abdominal pain¹; and (3) severe: wheezing, stridor, cyanosis, or circulatory collapse.¹

Diagnostic Tests

Peanut protein SPT. The SPTs were performed using commercial extracts. For those recruited from the Montreal Children's Hospital, extracts were supplied by either Hollister-Stier Laboratories LLC (Spokane, Washington) or ALK-Abelló (Hørsholm, Denmark). An SPT reaction was defined as positive if the greatest diameter of the wheal was at least 3 mm greater than that of the negative control (saline) at the time of the child's initial evaluation for peanut allergy.²³

Peanut specific IgE level. The serum level of peanut specific IgE was measured using the CAP system fluoroenzyme immunoassay (Pharmacia Diagnostics, Uppsala, Sweden). Given that individuals with a peanut specific IgE level of at least 15 kU/L have at least a 95% likelihood of experiencing an allergic reaction to peanut on exposure,²⁴⁻²⁸ we defined patients who had never been exposed to peanut or who had an uncertain clinical history and a peanut specific IgE level of at least 15 kU/L as being allergic to peanut without requiring a food challenge. However, a patient with a convincing clinical

history of an allergic reaction to peanut and a peanut specific IgE level of at least 0.35 kU/L was also considered to be allergic because, in local clinical practice, it is believed that the risk of such a patient reacting on peanut ingestion is so high that a challenge is seldom performed.²⁹

Oral food challenges to peanut. Oral food challenges to peanut were conducted under medical supervision according to previously published protocols.^{29,30} Either open or single-blinded food challenges were performed at the discretion of the treating physician on the basis of the available clinical history.

Participant questionnaires. Eligible participants with peanut allergy were invited to complete a questionnaire. Informed consent was obtained from the parent or child. French questionnaires were developed by translation and back-translation. Reminders were sent to families who had not returned the questionnaire within 2 weeks of the mailing. Parents were queried on demographics, duration of breastfeeding, other atopic disorders, and the frequency, characteristics, and treatment of any accidental exposure to peanut in the preceding year. Full details on accidental exposure are described elsewhere.¹⁷ We could not conduct medical record reviews for children recruited from the AQAA; thus, we asked for details regarding the initial and most severe allergic reactions to peanut and epinephrine use on the participant questionnaire for these children only. One year after enrollment in the study, a follow-up questionnaire was administered to all the participants in which they were queried on the epinephrine autoinjector, including prescription of the autoinjector, availability of the device at school, type of physician prescribing initially and on last renewal, interval between diagnosis of peanut allergy and prescription, and use of epinephrine during any accidental exposure in the previous year.

Statistical Analysis

Descriptive statistics, including means, ranges, and standard deviations, were generated for all the variables. Univariate and multivariate logistic regression analyses were used to estimate the associations between the availability of the epinephrine autoinjector at school or day care and potential predictive factors. Comparing univariate and multivariate results allowed us to investigate possible confounding factors. We evaluated the association between the availability of the autoinjector and the patient's age, sex, parental education (high school, college, or university), parental employment status, living arrangement of family (single vs nonsingle parents), atopic features of the patient or his or her parents, accidental exposures in the year before study entry and through study follow-up, severity of the initial and most severe reactions (mild, moderate, and severe), the use of epinephrine during any of these exposures, whether the child attended a school or day care that allowed peanut, prescription of epinephrine at the initial reaction, type of physician prescribing initially and at the last renewal, interval between diagnosis and autoinjector prescription, possessing an outdated autoinjector, and awareness of the expiratory date of

the device. All these variables were measured at study entry unless otherwise stated.

This analysis was conducted for all the children. Because children younger than 7 years might be too young to assume responsibility for carrying their own autoinjector, the association between device availability and the potential predictors mentioned in the previous paragraph was also analyzed for children who were at least 7 years old.

RESULTS

Two hundred twenty-six of 298 patients (75.8%) with peanut allergy from the Montreal Children's Hospital and 50 of 57 (87.7%) from the AQAA responded to the follow-up questionnaire. The overall response rate was 77.7%. Five of 276 patients (1.8%) reported that the allergy had resolved. There were no meaningful differences between respondents and nonrespondents (Table 1).

Four of 271 patients were not prescribed epinephrine autoinjectors, although they were diagnosed as being allergic to

peanut. Forty-eight percent of the children did not carry the autoinjector with them at school. This percentage varied according to the child's age: 70.0% (95% confidence interval [CI], 50.6%–85.3%) of those aged 5 or 6 years, 43.8% (95% CI, 31.4%–56.7%) aged 6 or 7 years, 44.4% (95% CI, 30.9%–58.6%) aged 8 or 9 years, 54.7% (95% CI, 41.7%–67.2%) aged 10 or 11 years, and 35.8% (95% CI, 23.1%–50.2%) 12 years or older did not carry the autoinjector. In 78.0% of children who did not carry the autoinjector themselves, it was located in the nurse's office or another school office (Fig 1). Only 18.5% of these offices had a full-time nurse, 64.1% had a part-time nurse, and 17.4% had only a nurse-on-call.

Autoinjectors were initially prescribed by allergists in 52.4% (95% CI, 46.3%–58.6%) of children, by family physicians in 24.0% (95% CI, 19.0%–29.6%), by emergency department physicians in 10.5% (95% CI, 7.1%–14.8%), by pediatricians in 10.1% (95% CI, 6.8%–14.4%), and by other physicians in 3.0% (95% CI, 1.3%–5.8%). On last renewal,

Table 1. Characteristics of Responders and Nonresponders

Characteristics	Responders (n = 271)	Nonresponders (n = 79)
Age at baseline, y		
Mean (SD) [95% CI]	8.1 (2.9) [7.7–8.4]	8.2 (3.3) [7.5–9.0]
Range	3–16	4–15
Age at diagnosis, mean (SD) [95% CI], y	3.0 (2.5) [2.7–3.3]	2.5 (1.6) [2.1–2.8]
Disease duration, mean (SD) [95% CI], y	5.1 (3.2) [4.7–5.5]	5.8 (3.7) [4.9–6.6]
Sex, % boys [95% CI]	63.1 [57.1–68.9]	62.8 [51.1–73.5]
Ethnic background of child, % white [95% CI]	93.0 [89.3–95.7]	84.6 [74.7–91.8]
Personal atopic history, % [95% CI]		
Atopic dermatitis	49.1 [43.0–55.2]	43.0 [31.9–54.7]
Asthma	57.2 [51.1–63.2]	59.5 [47.9–70.4]
Allergic rhinitis	41.3 [35.4–47.4]	45.6 [34.3–57.2]
Other food allergies	49.4 [43.3–55.6]	53.2 [41.6–64.5]
≥1 atopic comorbidity	88.9 [84.6–92.4]	88.6 [79.5–94.7]
Family atopic history, %, [95% CI]	(n = 220)	(n = 71) ^a
Atopic dermatitis	14.5 [10.2–19.9]	15.5 [8.0–26.0]
Asthma	22.7 [17.4–28.8]	22.5 [13.5–34.0]
Allergic rhinitis	6.8 [3.9–11.0]	5.6 [1.6–13.8]
Other food allergies	12.3 [8.2–17.4]	4.2 [0.9–11.9]
≥1 atopic comorbidity	40.0 [33.5–46.8]	35.2 [24.2–47.5]
Initial reaction to peanut, % [95% CI]		
None	11.4 [7.9–15.8]	11.4 [5.3–20.5]
Mild	30.3 [24.8–36.1]	30.4 [20.5–41.8]
Moderate	47.6 [41.5–53.7]	44.3 [33.1–55.9]
Severe	10.7 [7.3–15.0]	13.9 [7.2–23.6]
Attending school permitting peanuts, % [95% CI]	18.9 [14.4–24.2]	18.7 [10.6–29.3]
Age of parents, mean (SD) [95% CI], y		
Mother	39.3 (5.2) [38.7–39.9]	38.8 (5.9) [37.5–40.2]
Father	41.2 (5.9) [40.5–41.9]	41.5 (6.3) [40.1–43.0]
Parental education and work status, mother/father, %		
Completed high school	12.6/16.9	26.3/28.0
Completed college education	21.9/22.6	22.4/13.3
Completed university education	63.3/56.4	51.3/53.3
Employed at baseline	68.6/87.8	70.5/87.2

Abbreviation: CI, confidence interval.

^a Cohort members with a complete family atopic history.

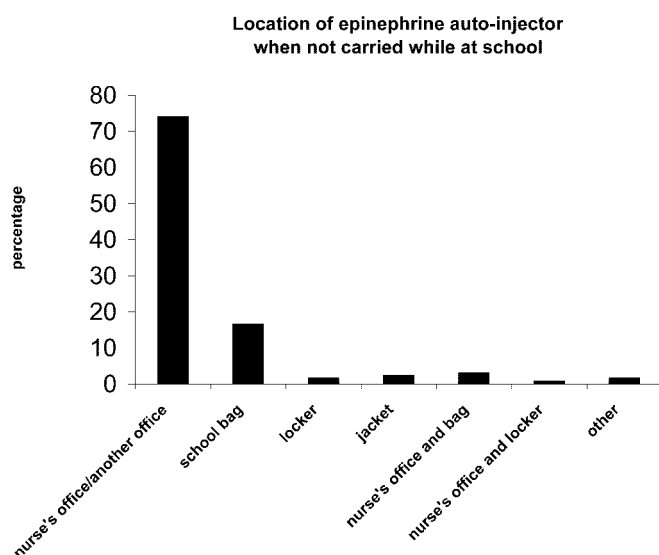


Figure 1. Location of epinephrine autoinjector when not carried while at school.

these percentages changed to 30.0% (95% CI, 24.5%–35.8%), 47.2% (95% CI, 41.1%–53.4%), 0.7% (95% CI, 0.1%–2.7%), 15.4% (95% CI, 11.3%–20.2%), and 6.7% (95% CI, 4.0%–10.4%), respectively. Although all the parents reported that the autoinjector had been renewed, 10.5% did not know the expiratory date of the device. However, of those who knew the expiratory date, the device had already expired in 3.3% at the time of the questionnaire. In 10.1% of cases, the autoinjector was not prescribed immediately after the diagnosis of peanut allergy, and in approximately 3.0%, it was given more than 6 months after diagnosis.

Of all the respondents, those who were administered epinephrine for a previous reaction (odds ratio [OR], 2.7; 95% CI, 1.3–5.7), older children (OR, 1.1; 95% CI, 1.0–1.2), and those living only with their mother (OR, 3.4; 95% CI, 1.0–11.0) were more likely to carry the autoinjector with them at school. Of those 7 years or older (77.9% of all respondents), those who had experienced a severe reaction were more likely to carry their autoinjector with them (OR, 3.3; 95% CI, 1.4–8.1).

No conclusive association was found between the availability of the device and the other variables mentioned in the “Statistical Analysis” subsection. With the present sample size, 95% CIs for all estimated ORs tended to be very wide, so these results were inconclusive rather than truly negative.

DISCUSSION

We conducted the largest study on the self-reported availability of the epinephrine autoinjector in children with peanut allergy. The results of this study suggest that approximately 50% of children with peanut allergy might experience a delay in the treatment of anaphylaxis while at school as a result of limited access to their device. Previous use of epinephrine,

older age, living only with the mother, and a previous severe reaction were identified as predictors of the availability of the device. We could not identify other potential predictors, potentially because of the sample size. Furthermore, more than 85% of the parents had completed at least a college education. These findings may have differed if the sample had been of a lower socioeconomic status. It is likely that we would have observed a lower rate of autoinjector prescription and availability if we had not required that children have an allergist-confirmed diagnosis of peanut allergy for inclusion in the study.

Previous studies have shown that food-induced anaphylaxis in patients aware of their allergy occurs most frequently at sites considered to be safe (ie, home, school, workplace, and hospital).³¹ A delay in the treatment of anaphylaxis may result from the caregiver’s failure to recognize anaphylaxis, reluctance or inability to treat, or failure to locate the epinephrine autoinjector.^{32–37} Furthermore, the allergic patient’s failure to personally carry epinephrine has been identified as the main contributor to a fatal outcome.^{37,38} In anaphylactic events that occur in schools, it has been reported that school personnel were unaware of the child’s allergy in 24% of cases and that epinephrine was almost always administered a mean of 10 minutes after symptom onset by a nurse in the office where the epinephrine was stored.³⁹ Keeping the device in a nurse’s office that is not staffed by a nurse on a daily basis, as is the case for many children in this study, would likely further delay administration of epinephrine during an anaphylactic reaction.

It is not surprising that we found that previous use of epinephrine is associated with carrying the autoinjector at school because previous experience likely increased the parents’ and children’s awareness of the importance of carrying the device. However, in contrast to other studies^{36,40} that suggest that teenagers are risk-seeking and are often reluctant to acknowledge their allergy and carry an autoinjector, in the present study, older children were more likely to carry their autoinjector. Also, although other studies suggest that children living in 2-parent households tend to have fewer problems,⁴¹ the present results suggest that children who live with their mother only are more likely to carry their autoinjector. It is likely that socioeconomic status, rather than household structure, is more predictive of outcome and, as noted earlier, this population was highly educated. Finally, the observed association between reaction severity and autoinjector availability while at school is of concern, and children and caregivers must be reminded that even those who have experienced less severe reactions must always carry their autoinjector.^{1,2,4,5}

Another factor that might be associated with less-than-optimal anaphylactic treatment is the relatively low rate of initial prescription of an autoinjector by the physicians most likely involved in the short-term management of allergy. In Canada, most patients first consult a pediatrician or a family or emergency department physician when they experience an allergic reaction. Given that in this study less than 50% of

patients were first prescribed an autoinjector by this type of physician, we anticipate that many such physicians are referring these patients to an allergist for a confirmatory diagnosis and prescription of an autoinjector. Hence, these patients are experiencing an unnecessary and potentially life-threatening delay in receiving their autoinjectors. Although only 10% of patients in this study indicated that they were not immediately prescribed an autoinjector after being diagnosed as having peanut allergy, we believe that these patients are actually considering the time of confirmatory diagnostic testing and not of first symptoms. If they considered the latter, many more would likely indicate that they experienced a delay.

Although it is desirable that physicians most often involved in the management of anaphylaxis prescribe autoinjectors, research has shown that their knowledge regarding autoinjectors is often suboptimal.^{42–44} Among pediatricians, only 18% were familiar with and able to demonstrate correct use of an autoinjector, and only 18% to 22% of general practitioners correctly answered questions regarding anaphylaxis therapy.^{18,45,46} Among junior and senior medical staff at a tertiary pediatric hospital, only 2% correctly demonstrated autoinjector administration; in 37%, the administration would not have delivered epinephrine to the patient.⁴⁷ Consultation with an allergy specialist has been reported to increase the competency of patients with the autoinjector.⁴⁸ Although the knowledge of Canadian pediatricians and family and emergency department physicians regarding use of the autoinjector has not been formally evaluated, we anticipate that it may warrant improvement.

There is no consensus on the appropriate age at which a child might assume responsibility for carrying his or her own autoinjector. The American Academy of Pediatrics, in collaboration with the National Association of School Nurses and other organizations, developed a list of student and parental factors to consider when entrusting a student to self-carry an autoinjector at school.⁴⁹ Based on these recommendations, 35 states have enacted self-carry laws for epinephrine, allowing students with anaphylaxis to carry and use prescribed medications at school. Although the actual age at which an allergic child could assume full responsibility for an autoinjector may not be clearly defined, one should assume that even if the child does not know how or when to administer the epinephrine, self-carrying would serve to increase accessibility of the device to those who are trained to recognize and treat severe allergic symptoms. Furthermore, in cases when no nurse is available, a child 7 years or older is likely to be the best person to use the device.

The Canadian Society of Allergy and Clinical Immunology, in collaboration with Anaphylaxis Canada, the AQAA, and the Asthma and Allergy Information Association, has developed new guidelines on school-based policies for anaphylaxis that are being promoted throughout Canada. These guidelines include recommendations for creating allergy-safe schools and day care centers and adopting an anaphylaxis emergency plan for each child at risk for anaphylaxis and training staff on the recognition and management of anaphy-

laxis. The guidelines recommend that children old enough to understand the proper use of the autoinjector (usually by grade 1 or 2) should carry it themselves⁵⁰ and that additional autoinjectors should be kept in easily accessible locations in the school.

Ensuring availability and correct use of epinephrine autoinjectors is crucial to preventing fatal allergic reactions in allergic children. Developing educational programs for parents, other caregivers, and health professionals and establishing guidelines or legislation that will promote accessibility and appropriate use of autoinjectors will contribute to minimizing the risk of fatal anaphylactic reactions.

REFERENCES

- Hourihane JO, Kilburn SA, Dean P, Warner JO. Clinical characteristics of peanut allergy. *Clin Exp Allergy*. 1997;27:634–639.
- Sampson HA, Mendelson L, Rosen JP. Fatal and near-fatal anaphylactic reactions to food in children and adolescents. *N Engl J Med*. 1992;327:380–384.
- Yocum MW, Khan DA. Assessment of patients who have experienced anaphylaxis: a 3-year survey. *Mayo Clin Proc*. 1994;69:16–23.
- Sicherer SH, Furlong TJ, Munoz-Furlong A, Burks AW, Sampson HA. A voluntary registry for peanut and tree nut allergy: characteristics of the first 5149 registrants. *J Allergy Clin Immunol*. 2001;108:128–132.
- Sicherer SH, Burks AW, Sampson HA. Clinical features of acute allergic reactions to peanut and tree nuts in children. *Pediatrics*. 1998;102:e6.
- Tariq SM, Stevens M, Matthews S, et al. Cohort study of peanut and tree nut sensitisation by age of 4 years. *BMJ*. 1996;313:514–517.
- Bock SA, Atkins FM. The natural history of peanut allergy. *J Allergy Clin Immunol*. 1989;83:900–904.
- Skolnick HS, Conover-Walker MK, Koerner CB, et al. The natural history of peanut allergy. *J Allergy Clin Immunol*. 2001;107:367–374.
- Hourihane JO'B, Kilburn SA, Nordlee JA, et al. An evaluation of the sensitivity of subjects with peanut allergy to very low doses of peanut protein: a randomized, double-blind, placebo-controlled food challenge study. *J Allergy Clin Immunol*. 1997;100:596–600.
- Sicherer SH, Munoz-Furlong A, Sampson HA. Prevalence of peanut and tree nut allergy in the United States determined by means of a random digit dial telephone survey: a 5-year follow-up study. *J Allergy Clin Immunol*. 2003;112:1203–1207.
- Kagan RS, Joseph L, Dufresne C, et al. Prevalence of peanut allergy in primary-school children in Montreal, Canada. *J Allergy Clin Immunol*. 2003;112:1223–1228.
- Grundy J, Matthews S, Bateman B, Dean T, Arshad SH. Rising prevalence of allergy to peanut in children: data from 2 sequential cohorts. *J Allergy Clin Immunol*. 2002;110:784–789.
- Rona RJ, Keil T, Summers C, et al. The prevalence of food allergy: a meta-analysis. *J Allergy Clin Immunol*. 2007;120:638–646.
- Vadas P, Perelman B. Presence of undeclared peanut protein in chocolate bars imported from Europe. *J Food Prot*. 2003;66:1932–1934.
- Furlong TJ, DeSimone J, Sicherer SH. Peanut and tree nut allergic reactions in restaurants and other food establishments. *J Allergy Clin Immunol*. 2001;108:867–870.
- Ewan PW, Clark AT. Efficacy of a management plan based on severity assessment in longitudinal and case-controlled studies of 747 children with nut allergy: proposal for good practice. *Clin Exp Allergy*. 2005;35:751–756.
- Yu JW, Kagan R, Verreault N, et al. Accidental ingestions in children with peanut allergy. *J Allergy Clin Immunol*. 2006;118:466–472.
- Sicherer SH, Forman JA, Noone SA. Use assessment of self-administered epinephrine among food-allergic children and pediatricians. *Pediatrics*. 2000;105:359–362.
- Sampson HA. Managing peanut allergy. *BMJ*. 1996;312:1050–1051.
- Macdougall CF, Cant AJ, Colver AF. How dangerous is food allergy in childhood? the incidence of severe and fatal allergic reactions across the

- UK and Ireland. *Arch Dis Child*. 2002;86:236–239.
21. Sampson HA, Munoz-Furlong A, Bock SA, et al. Symposium on the definition and management of anaphylaxis: summary report. *J Allergy Clin Immunol*. 2005;115:584–591.
22. Kapoor S, Roberts G, Bynoe Y, et al. Influence of a multidisciplinary paediatric allergy clinic on parental knowledge and rate of subsequent allergic reactions. *Allergy*. 2004;59:185–191.
23. Eigenmann PA, Sampson HA. Interpreting skin prick tests in the evaluation of food allergy in children. *Pediatr Allergy Immunol*. 1998;9:186–191.
24. Sampson HA, Ho DG. Relationship between food-specific IgE concentrations and the risk of positive food challenges in children and adolescents. *J Allergy Clin Immunol*. 1997;100:444–451.
25. Sampson HA. Utility of food-specific IgE concentrations in predicting symptomatic food allergy. *J Allergy Clin Immunol*. 2001;107:891–896.
26. Sampson HA. Update on food allergy. *J Allergy Clin Immunol*. 2004;113:805–819.
27. Roberts G, Lack G. Diagnosing peanut allergy with skin prick and specific IgE testing. *J Allergy Clin Immunol*. 2005;115:1291–1296.
28. Rance F, Abbal M, Lauwers-Cances V. Improved screening for peanut allergy by the combined use of skin prick tests and specific IgE assays. *J Allergy Clin Immunol*. 2002;109:1027–1033.
29. Pucar F, Kagan R, Lim H, Clarke AE. Peanut challenge: a retrospective study of 140 patients. *Clin Exp Allergy*. 2001;31:40–46.
30. Bock SA, Sampson HA, Atkins FM, et al. Double-blind, placebo-controlled food challenge (DBPCFC) as an office procedure: a manual. *J Allergy Clin Immunol*. 1988;82:986–997.
31. Eigenmann PA, Zamora SA. An internet-based survey on the circumstances of food-induced reactions following the diagnosis of IgE-mediated food allergy. *Allergy*. 2002;57:449–453.
32. Patel BM, Bansal PJ, Tobin MC. Management of anaphylaxis in child care centers: evaluation 6 and 12 months after an intervention program. *Ann Allergy Asthma Immunol*. 2006;97:813–815.
33. Bansal PJ, Marsh R, Patel B, Tobin MC. Recognition, evaluation, and treatment of anaphylaxis in the child care setting. *Ann Allergy Asthma Immunol*. 2005;94:55–59.
34. Mehl A, Wahn U, Niggemann B. Anaphylactic reactions in children: a questionnaire-based survey in Germany. *Allergy*. 2005;60:1440–1445.
35. Sicherer SH, Furlong TJ, DeSimone J, Sampson HA. The US Peanut and Tree Nut Allergy Registry: characteristics of reactions in schools and day care. *J Pediatr*. 2001;138:560–565.
36. Bock SA, Munoz-Furlong A, Sampson HA. Fatalities due to anaphylactic reactions to foods. *J Allergy Clin Immunol*. 2001;107:191–193.
37. Osuchukwu CI, Alfaham MA, Rangaraj S, et al. Parental questionnaire on EpiPen provision, education and subsequent usage. *J Allergy Clin Immunol*. 2001;107:S272.
38. Wuthrich B. Lethal or life-threatening allergic reactions to food. *J Invest Allergol Clin Immunol*. 2000;10:59–65.
39. McIntyre CL, Sheetz AH, Carroll CR, Young MC. Administration of epinephrine for life-threatening allergic reactions in school settings. *Pediatrics*. 2005;116:1134–1140.
40. Sampson MA, Munoz-Furlong A, Sicherer SH. Risk-taking and coping strategies of adolescents and young adults with food allergy. *J Allergy Clin Immunol*. 2006;117:1440–1445.
41. Schor EL. Family pediatrics: report of the Task Force on the Family. *Pediatrics*. 2003;111:1541–1571.
42. Singh J, Aszkenasy OM. Prescription of adrenaline auto-injectors for potential anaphylaxis: a population survey. *Public Health*. 2003;117:256–259.
43. Hughes JL, Stewart M. Self-administration of epinephrine in children: a survey of current prescription practice and recommendations for improvement. *Ulster Med J*. 2003;72:80–85.
44. Grouhi M, Alshehri M, Hummel D, Roifman CM. Anaphylaxis and epinephrine auto-injector training: who will teach the teachers? *J Allergy Clin Immunol*. 1999;104:190–193.
45. Ferreira MB, Alves RR. Are general practitioners alert to anaphylaxis diagnosis and treatment? *Allerg Immunol (Paris)*. 2006;38:83–86.
46. Hayman GR, Bansal JA, Bansal AS. Knowledge about using auto-injectable adrenaline: review of patients' case notes and interviews with general practitioners. *BMJ*. 2003;327:1328.
47. Mehr S, Robinson M, Tang M. Doctor: how do I use my EpiPen? *Pediatr Allergy Immunol*. 2007;18:448–452.
48. Arkwright PD, Farragher AJ. Factors determining the ability of parents to effectively administer intramuscular adrenaline to food allergic children. *Pediatr Allergy Immunol*. 2006;17:227–229.
49. Wheeler L. Guidance offered on self-administration of medication at school. *AAP News*. 2005;26:8.
50. Allen M, Binkley K, Chad Z, et al. *Anaphylaxis in Schools and Other Child Care Settings*. Ottawa: Canadian Society of Allergy and Clinical Immunology; 2005:1–59.

Requests for reprints should be addressed to:
Moshe Ben-Shoshan, MD
Claremont 418
app 57
Montreal, H3Y 2N2, Canada
E-mail: daliamoshebs@gmail.com