# Characteristics of childhood peanut allergy in the Australian Capital Territory, 1995 to 2007

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**Background: It is unknown whether clinical features of peanut allergy have changed in the past decade alongside possible increasing prevalence.**

**Objective: The clinical features of peanut allergy over 13 years were examined with regard to age of onset, sex distribution, severity, and incidence.**

**Methods: Retrospective study of 778 patients (age 4 months to 66 years) diagnosed with peanut allergy at a community-based specialist allergy practice in the Australian Capital Territory.**

**Results: Most peanut allergy (90%) developed by age 72 months. In this group, there were no significant time-dependent changes in sex distribution, reaction severity, or age of first reaction (mean/median 12/15.1 months). Later age of first reaction was associated with an increased risk of anaphylaxis in the overall population ($P < .01$) and in those with onset by 72 months, in whom risk increased by 22.7% (CI, 3.3-45.7) for every additional year of age ($P < .02$). Asthma was associated with increased risk of anaphylaxis (odds ratio, 1.9; $P < .001$). In children with peanut allergy, 22% experienced anaphylaxis with first exposure and 30% with anaphylaxis had preceding milder reactions. The estimated minimum incidences of peanut allergy and sensitization by age 72 months for children born in the Australian Capital Territory in 2004 were 1.15% and 1.53%, respectively (by end December 2007), compared with 0.73% and 0.84% for those born in 2001.**

**Conclusion: Although most characteristics of peanut allergy have changed little over the period of the last 13 years (onset age, sex, comorbidity, severity), later onset was associated with greater risk of anaphylaxis. Our data are consistent with a rise in incidence. (J Allergy Clin Immunol 2009;123:689-93.)**

***Key words:*** *Food allergy, peanut allergy, anaphylaxis, epidemiology, time trends*

*Abbreviations used*
ACT: Australian Capital Territory
AE: Atopic eczema
FA: Food allergy
PA: Peanut allergy
PAA: Peanut anaphylaxis
PS: Peanut sensitization
SPT: Skin prick testing
TNA: Tree nut allergy

Although there is evidence that peanut allergy (PA) prevalence has increased in young children from the United Kingdom and the United States,[1,2] there are no reliable data on current prevalence in Australia. In the absence of repeated population estimates using objective measures of assessment, evidence that PA might be more common in Australia is indirect, derived from surrogate markers such as changing service demand and increased hospital admission rates for anaphylaxis.[3,4] The primary aim of this study was to examine the clinical features of confirmed PA by using data derived from a single center providing half the regional ambulatory allergy-related services in the Australian Capital Territory (ACT), focusing on children aged 0 to 72 months. Importantly, a diagnosis of PA was defined as documented history of reaction after exposure to peanut together with a positive skin prick test (SPT) and was differentiated from sensitization alone. The relative stability of the ACT population offered the opportunity to estimate the age-adjusted incidence of PA, and to examine evidence for time-related changes in incidence adjusted for birth year.

## METHODS

### Study population

The study was undertaken in the ACT, an inland urban city of 2358 $km^2$ within rural South-Eastern Australia (~324,000 inhabitants[5]). Birth origin (2006 Census data[6]) includes Australia (74.7%), Western Europe (7%), Asia (6.7%), southern and eastern Europe (3.3%), the Americas (1.5%), Sub-Saharan Africa (0.8%) and Africa, and the Middle East (0.6%). ACT medical facilities service the local metropolitan population and surrounding regional areas. The characteristics were analyzed of patients referred to an ACT-based mixed adult/pediatric specialist allergy/immunology medical practice between January 1995 and December 2007. The practice is the largest regional outpatient allergy practice, providing approximately half the local ambulatory specialist allergy services, with the closest services outside the ACT being Sydney or Melbourne, 300 and 800 km away, respectively. Referrals were received from general medical practitioners, accident and emergency

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departments, and pediatricians. Population data were obtained from the Australian Bureau of Statistics.[5] Patients were divided into 3 groups for analysis: (1) all with PA (used to examine comorbidity, severity trends, and relationship between age of first reaction and severity), (2) all children with PA aged 0 to 72 months (used to examine age of first peanut reaction), and (3) all ACT-resident children aged 0 to 72 months (used to calculate age-adjusted and population-adjusted incidence rates). Age-adjusted and population-adjusted incidence rates were calculated using the ACT-resident patient data adjusted for the number of live births per year. To minimize the potential for bias introduced by delays in seeking medical review or waiting list lengths, data were expressed according to birth year. The Human Research and Ethics committee (Calvary Bruce/Calvary John James Private Hospitals) approved the study.

## Patient evaluation

The primary indication for undertaking SPT was a history of likely food allergy (FA). Secondary indications for testing were another FA (where peanut had not yet been ingested or tolerated), children with atopic eczema (AE), or siblings of a child with documented FA for whom parents were concerned about possible FA. SPT with peanut, egg, milk, soy, prawn, fish, sesame seed, and all available tree nut extracts were performed unless the patient regularly consumed and tolerated these foods. Glycerinated allergen extracts (Hollister Stier, Spokane, Wash) and histamine 10 mg/mL positive control (Hollister Stier) were purchased from EBOS Australia (Sydney). SPT was performed on the volar aspect of the forearm using metal lancets (Stallergens, Antony, France) according to standard guidelines.[7] A positive SPT was defined as a wheal size of at least 3 mm greater than a negative control (saline) at 15 minutes.[7]

## Diagnostic criteria

Food sensitization was defined by the presence of a positive SPT. FA was diagnosed if there was a history of acute systemic allergic reaction within 2 hours of known food exposure, combined with a positive SPT to the relevant food. The severity of systemic allergic reactions was classified and analyzed as described by Brown[8]: mild (skin and subcutaneous tissue involvement only), moderate (features suggestive of respiratory, cardiovascular or gastrointestinal involvement: dyspnea, wheeze, chest or throat tightness, nausea, vomiting, abdominal pain, dizziness, sweating) or severe (cyanosis, hypotension, confusion, collapse, loss of consciousness, incontinence). When more than 1 adverse food reaction occurred, severity was classified according to the worst ever reaction. A diagnosis of anaphylaxis was assigned if either of the first 2 criteria of the 2005 National Institute of Allergy and Infectious Disease/Food Allergy and Anaphylaxis Network Symposium definition was fulfilled.[9] Specifically, patients were labeled as having anaphylaxis if their symptoms included respiratory symptoms of noisy or difficulty breathing (dyspnea, bronchospasm/wheeze, cyanosis/hypoxia) and/or cardiovascular compromise in the setting of acute allergic symptoms. Asthma, AE, and allergic rhinitis were defined as previously described.[10,11]

## Data analysis

Demographic and diagnostic data were entered prospectively into searchable databases (MediMouse, Practice Innovators, Sydney; Blue Chip Clinical Research Module, Health Communication Network, Sydney; Microsoft Access, Microsoft Corporation, Redmond, Wash). Data (and accuracy) were analyzed and verified retrospectively in 2008. Poisson regression was used to examine incidence trends; linear regression for log-transformed age data; logistic regression for severity; and $\chi^2$ analysis for associations between diagnostic groups. Analyses were performed by using Stata Release 9 (Statacorp, College Station, Tex). CIs were expressed at the 95% level.

# RESULTS

## Patient characteristics

A total of 18,028 distinct patients were assessed between 1995 and 2007; 2203 were diagnosed with FA. A total of 946 patients had peanut sensitization (PS); 751 were confirmed as having PA based on clinical criteria confirmed by SPT, and 195 had a positive SPT alone (all children <72 months of age when evaluated; mean, median, and range of SPT to peanut were 6, 5, and 3-14 mm). Most (86%) of these 195 children had other FA but had not yet consumed peanut. Parents were advised to avoid peanut and undertake a hospital challenge at school age if accidental exposure had not occurred by that time. A total of 27 of 195 of these children developed PA between 1995 and 2007, twenty spontaneously and 7 with deliberate challenge, yielding a total of 778 with PA. Of the 778 individuals with PA, 486 were aged 0 to 72 months and 292 were aged 7 to 66 years at the time of diagnosis. Peanut anaphylaxis (PAA) occurred in 264 of 778 (34%) patients with PA, representing 145 of 486 aged 0 to 72 months and 119 of 292 aged 7 to 66 years, respectively. In children aged 0 to 72 months with PA, comorbidities included AE (63.2%); asthma (20.0%); allergic rhinitis (16.2%); and FA to hen's egg (36.0%), cow's milk (9.9%), tree nuts (9.9%), seafood (3.7%), soy (1.3%), wheat (1%), banana (0.9%), or sesame seed (0.5%). The average time between the first spontaneous PA reaction and assessment was 20 months (median, 15; range, 1-70).

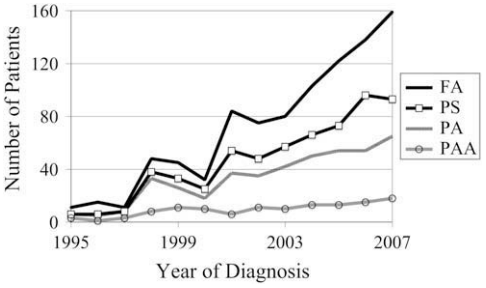


**FIG 1.** FA and PA diagnostic time trends. The number of patients diagnosed with FA, PA, PAA, and PS increased more than 10-fold between 1995 and 2007. Data are shown according to year of diagnosis.

## Time trends for peanut allergy diagnosis

The number of new FA and PA diagnoses (and PS) in the overall population increased more than 10-fold between 1995 and 2007 (Fig 1). Similar trends were seen when analysis was restricted to children aged 0 to 72 months (data not shown).

## Age of first peanut allergic reaction

The age of first PA reaction was recorded in 762 of 778 patients. These occurred at age less than 6 months (4.4% of patients), less than 24 months (68%), less than 72 months (90%), less than 10 years (95%), and less than 20 years (97.1%). The remaining 21 patients aged 21-66 years (median 42 years) developed late-onset PA despite earlier tolerance. Most were atopic (18 of 21), with intercurrent problems of oral allergy syndrome (7), or allergy to honey bee venom, sesame seed, or seafood (1, 1, or 3, respectively). The timing of first PA reaction in children (in whom onset occurred by 72 months of age) was analyzed using log-linear regression, adjusted by birth year. To minimize the potential for bias to older age of onset (because these patients were deliberately avoiding peanut), the 27 children identified with PS (but without known PA) were excluded from this analysis. In the remaining patients, there was no time-related change in mean/median age of

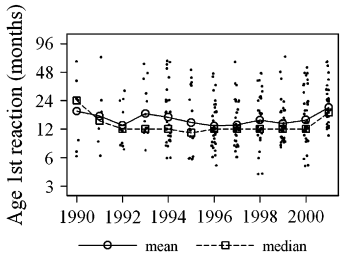


**FIG 2.** Age of first PA reaction. The age of first allergic reaction to peanut was analyzed in patients with first reaction by age 72 months using linear regression of age (log-transformed) against birth year. (Data from beyond 2001 are not shown, because patients born since that time were yet to reach the age of 72 months at the end of December 2007).

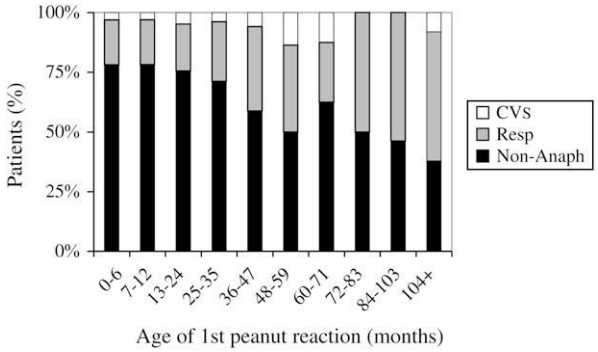


**FIG 3.** Relationship between reaction severity and age. There was a significant correlation between age of first peanut reaction and severity of worst ever reaction ($P < .001$, ordinal logistic regression). When patients with onset by 72 months were analyzed separately, the risk (odds) of anaphylaxis increased by 22.7% (CI, 3.3-45.7) for every additional year of age ($P < .02$). *CVS*, Cardiovascular collapse, *Resp*, respiratory difficulty; *Non-Anaph*, nonanaphylactic.

first peanut reaction between those born in 1990 to those born 2001 (median 12 months; geometric mean 15.5 months [CI 14.2-16.1]; Fig 2). Data from beyond 2001 is not shown, as patients born after that time had not reached the age of 72 months by December 2007.

## Severity of PA

Severity of PA in the entire PA population (778 patients) was graded as anaphylaxis or nonanaphylaxis (264 and 514, respectively; 33.9% had anaphylaxis) and further classified as mild, moderate, or severe by Brown classification (405/328/45 cases). Of these patients, 169 of 778 (22%) experienced anaphylaxis with first peanut exposure, 95 of 609 (16%) progressed from milder reactions to anaphylaxis over time, and 95 of 264 (36%) of those with PAA had preceding milder reactions. There was a significant correlation between age of first peanut reaction and severity of worst ever reaction ($P < .01$, ordinal logistic regression). When patients with onset by 72 months were analyzed separately, the risk (odds) of anaphylaxis increased by 22.7% (CI, 3.3-45.7) for every additional year of age ($P < .02$; Fig 3).

In 468 children aged 0 to 72 months with PA (either initially or later reclassified from PS to PA with follow-up), severity was graded as anaphylaxis or nonanaphylaxis in 145 (31%) and 323 (69%) cases, respectively. Severity was mild, moderate, or severe by Brown classification in 276, 165, and 27 cases, respectively. Of these children, 22% experienced PAA with first exposure, 9% progressed from milder reactions to anaphylaxis over time, and 30% with PAA had preceding milder reactions. No significant time-dependent changes in reaction severity using the Brown classification were observed (whether analyzed by birth or diagnosis year; data not shown). There was no significant difference in severity between the sexes. Current asthma was associated with increased risk of anaphylaxis overall (odds ratio, 1.9; $P < .001$) and respiratory difficulty (odds ratio, 1.6; $P = .03$) but not vascular collapse ($P = .3$).

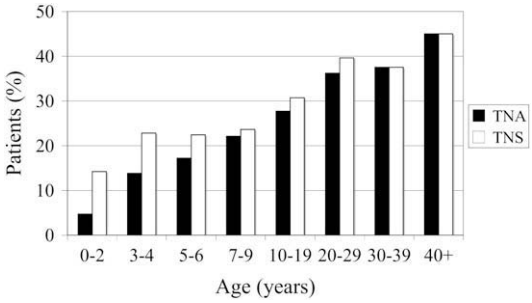


**FIG 4.** TNA and sensitization in patients with PA. In patients with PA, the proportion of those with coexistent TNA and tree nut sensitization *(TNS)* increased with age at time of diagnosis, from 4.7% (14.2%) of children aged less than 2 years to 45% (45%) of those aged 40 years or greater, respectively.

## Other observations

In patients with PA, the proportion of those with coexistent tree nut allergy (TNA) and tree nut sensitization increased with age at time of diagnosis, from 4.7% and 14.2%, respectively, of children age less than 2 years to 45% and 45%, respectively, of those age 40 years or greater (Fig 4). In the 54 patients with resolution of PA (31 male), age of first peanut reaction was less than 24 months in 45 (83%) and less than 72 months in all, and severity of worst ever reaction was Brown category mild (41, 76%), moderate (13, 24%) or severe (1).

## Estimating PA incidence in ACT children

Between 1995 and 2007, the total ACT population grew 6% from 304,805 to an estimated 324,200, whereas the number of children aged 0 to 72 months decreased from 26,991 to 25,283. The number of live births to ACT resident mothers increased slightly from 4415 to 4479 between 1995 and 2006 (mean, median, and range, 4196, 4086, and 3858-4400). There was no substantial change in ACT-based specialist allergy/immunology services between 1995 (77 specialist hours/week) and 2007 (78 hours/week).

Between 1995 and 2007, the total numbers of ACT children age 0 to 72 months diagnosed with FA, PA, and PS were 741, 348, and 465, respectively. Similar to these findings for the total referred population, the number of new diagnoses of FA, PA, and PS in ACT children increased between 1995 and 2007 (data not shown). To minimize the potential for bias introduced by delays in parents seeking medical review or length of waiting lists, data were re-expressed according to birth year. This facilitated calculation of the minimum incidence of confirmed FA, PA, and PS by the age of 72 months, matching the birth year of ACT patients to the number

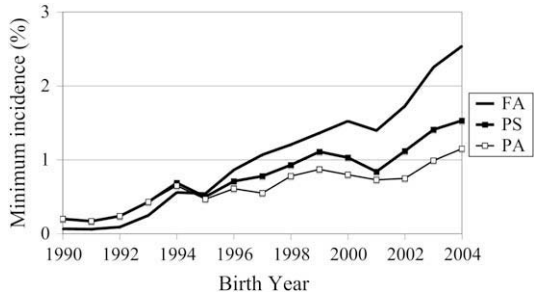


**FIG 5.** Minimum incidence of PA in ACT children. The minimum incidence of age-adjusted FA, PA, and PS by age 72 months were calculated for ACT-resident children, matching the birth year of patients to the number of live births to ACT-resident mothers for each year.

of live births in the ACT each year (Fig 5). Assuming no net population shifts of children with PA, the minimum incidence of PA and PS in ACT residents by age 72 months was 0.73% and 0.84%, respectively, for those born in 2001. Diagnoses since that time have facilitated even higher estimates of FA, PA, and PS incidence for those born in subsequent years (even though these children were not yet age 72 months by the end of 2007), with estimates of 2.5%, 1.15%, and 1.53%, respectively, for those born in 2004 so far.

## DISCUSSION

The primary aim of this study was to determine whether the characteristics of PA had changed over a 13-year period in a single center providing approximately half the regional specialist ambulatory allergy services. The ACT was considered to be a unique location for such a study given its relative isolation from allergy/immunology services in other regions (making it likely that most patient data would be captured locally), and its large and stable population facilitating comparison of clinical characteristics of PA over time. It was also anticipated that a minimum incidence of PA could also be estimated, and that evidence for time-related increases (as observed in the United States and the United Kingdom[1,2]) might be examined. Although it could be argued that the retrospective nature of this study and time gaps between peanut reactions and data collection might have biased estimates of severity and age of onset, the unchanging severity of reactions (whether assessed by birth year or evaluation year) and the contemporaneous recording of age of first peanut reaction (rather than data collection by later patient surveys) make this unlikely to be a significant confounder. Further, although we were limited to examining a subset of patients seeking medical assessment (rather than a random selection of the entire population), population sampling was substantial, with 1.8% of the entire ACT population age 0 to 72 months assessed from 2006 to 2007.

Consistent with other studies,[12] we found that most PA presented in the first decade, current asthma was associated with increased risk of anaphylaxis, a third of PAA was preceded by milder reactions, anaphylaxis often occurred unpredictably with first exposure, and the prevalence of associated TNA increased with age.[13] Furthermore, age of first reaction to peanut did not change, despite many parents reporting that they were attempting to delay exposure. This finding is contrary to a recent US report describing a trend to younger age of first peanut reaction from a median age of 19 months (born 1988-1999) to 12 months (born 2000-2005[14]). The robustness of our findings is underpinned by the assessment of a large number of patients with confirmed PA in a single center using identical diagnostic criteria, a low degree of missing data (6%), and data analysis by logistic regression to examine time trends. Interpretation of the US study[14] is limited by the use of historical cohorts from 2 different centers and diagnosis of PA using *in vitro* data alone in a third of patients, which correlates closely but imperfectly with clinical reactivity.[15] If the age of first peanut reaction is indeed different between the United States and Australia (and not related to differing methodology), potential explanations may include differences in consumption, accidental exposure, prevalence of peanut products, or perhaps introduction practices.

Importantly, our data showed that later age of first reaction to peanut was associated with increased severity of worst ever reaction, whether the entire population of patients with PA was examined or only those with onset by age 72 months. This stands in stark contrast to reports suggesting that older patients are at lower risk of anaphylaxis because their allergy is driven by cross-reactive responses to pollen allergens.[16] Our findings raise the interesting possibility that delayed introduction of allergenic foods (as advised in some prevention guidelines) might increase the risk of more serious reactions, although in the absence of long-term follow-up data, it is not possible to draw firm conclusions at this time.

Given the relative stability of the ACT population, we also attempted to estimate the local incidence of PA by age 72 months, adjusting by birth year to eliminate the potential bias introduced by waiting lists or time to specialist evaluation. We adopted a deliberately conservative approach by requiring a convincing history of a systemic allergic reaction after exposure in combination with SPT confirmation. The calculated minimum incidence of 1.15% for those born in 2004 is very likely an underestimate, because this figure does not include children assessed in other allergy local units or by general pediatricians, those who had not sought medical/specialist attention for their allergy, those yet to be exposed to peanut, or those avoiding it because PS had been identified. Unfortunately, we were unable to make useful comparisons with older Australian studies because of different methodologies employed. The Melbourne Atopy Cohort, for example, followed 620 "high risk" children (parental history of allergic disease) recruited antenatally to age 2 years, and assessed FA by SPT and limited challenge. PA was estimated in this study to affect 1.9% of children, but the data were derived from a selected population of high-risk infants, and their applicability to a general population remains unclear.[17] A more recent survey study of more than 9000 children aged less than 6 years attending ACT child care centers and preschools estimated the prevalence of PA to be 2.3% without significant change between 2003 and 2006, but results were unconfirmed by expert questioning of respondents, allergy testing, or challenge.[18]

The dramatic increase in the number of new diagnoses of PA in our clinic raised the possibility that incidence had also increased. The trend to increased incidence could not be explained as an artifact of changing ACT demographics, birth rates, or availability of specialist allergy/immunology services. The absence of changing reaction severity also makes it unlikely that these observations were an artifact of altered health-seeking behavior of parents presenting with children with milder allergic symptoms, although it is conceivable that such factors might (in part) have contributed to the 10-fold increase in new diagnoses compared to the more modest 2.5-fold increase in calculated

incidence between those born 1995 (0.47%) and 2004 so far (1.15%). Given that similar upward trends for FA diagnosis were observed in local hospital units (Dr C. Hawkins, Department of Clinical Immunology, The Canberra Hospital, personal communication, October 2008), it is unlikely that changes in referral patterns between allergy services in the ACT explain the findings. Nevertheless, one potential caveat to interpretation is the possibility that there was a shift from provision of allergy services by general pediatricians in the region, to provision of allergy services by allergy/immunology specialists. Taken together with recent increases in FA-related anaphylaxis hospitalization rates in Australian children,[3,4] however, our data are consistent with observations in the United Kingdom and the United States[1,2] and suggest that PA is becoming more common in Australian children as well.

This study is unable (nor designed) to explain the reasons for changing PA incidence. The minor 9% increase in Australian peanut consumption between 1995 to 1999 and 2005 to 2008,[19] for example, is too small to explain the dramatic changes observed in this study. Despite earlier evidence that early soy exposure may play a role,[20] the low rates of coreactivity to peanut and soy in this study and recent evidence that this link could be explained by preferential use of soy formula in families and infants with cow's milk allergy[21] make this an unsatisfactory explanation. Other hypotheses that have recently been reviewed include the hygiene hypothesis, topical sensitization, cesarean section, antacid medication use by infants, food processing methods, vitamin D exposure, alterations in dietary fat consumption, and perhaps even delayed introduction of allergenic food.[22-25]

The rapid increase in demand for FA assessment in Australia poses a public health issue that has already affected medical resources and waiting lists in the public and private sectors in Australia.[26] An increase in PA incidence will only serve to fuel that demand, is likely to result in a cohort effect for higher prevalence in older individuals (in whom anaphylaxis and mortality is more likely), and may require a reassessment of the relatively low risk of fatal outcomes in young children if the underlying problem is more common. Until the results of a controlled study comparing avoidance with early introduction of peanut on the risk of PA development are available (http://www.leapstudy.co.uk/index.html), it remains speculative whether public health measures to avoid or delay exposure to allergenic food[27-30] designed to prevent FA might (in part) have contributed to its recent rise.[24,25]

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**Clinical implications: Although the characteristics of peanut allergy have changed little over the past decade, later age of onset of peanut allergy was associated with a greater risk of anaphylaxis.**

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