Background: Detailed data on fatal anaphylaxis are limited, with national anaphylaxis fatality data for the United Kingdom and food-induced anaphylaxis fatality data for the United States. Time trends for anaphylaxis fatalities are not available. Objective: We examined causes, demographics, and time trends for anaphylaxis fatalities in Australia between January 1997 and December 2005 and compared these with findings for anaphylaxis admissions.

Methods: Data on anaphylaxis deaths and hospital admissions were extracted from a national database. Death certificate codes were analyzed to determine the likely cause and associated comorbidities.

Results: There were 112 anaphylaxis fatalities in Australia over 9 years. Causes were as follows: food, 7 (6%); drugs, 22 (20%); probable drugs, 42 (38%); insect stings, 20 (18%); undetermined, 15 (13%); and other, 6 (5%). All food-induced anaphylaxis fatalities occurred between 8 and 35 years of age with female preponderance, despite the majority of food-induced anaphylaxis admissions occurring in children less than 5 years of age. Most insect sting–induced anaphylaxis deaths occurred between 35 and 84 years almost exclusively in male subjects, although bee sting–induced admissions peak between 5 and 9 years of age with a male/female ratio of 2.7. However, most drug-induced anaphylaxis deaths occurred between 55 and 85 years with equal sex distribution similar to drug-induced anaphylaxis admissions. There was no evidence of an increase in death rates for food-induced anaphylaxis, despite food-induced anaphylaxis admissions increasing approximately 350%. In contrast, drug-induced anaphylaxis deaths increased approximately 300% compared with an approximately 150% increase in drug-induced anaphylaxis admissions.

Conclusion: The demographics for anaphylaxis deaths are different to those for anaphylaxis presentations. Anaphylaxis mortality rates remain low and stable, despite increasing anaphylaxis prevalence, with the exception of drug-induced anaphylaxis deaths, which have increased. (J Allergy Clin Immunol 2009;123:434-42.)

Key words: Anaphylaxis, fatalities, admissions, prevalence, time trends, Australia

Detailed data on fatal anaphylaxis is limited, with only 2 studies of anaphylaxis deaths from all causes derived from national mortality reporting systems. In the United Kingdom (UK) national statistics from a dedicated registry of anaphylaxis deaths allowed an estimated prevalence of 1 death per year per 3 million population. Pumphrey reported 202 anaphylaxis fatalities in the UK over a 10-year period (1992-2001), with 88 (44%) attributed to drugs, 47 (23%) to insect stings, 45 (22%) to foods, 18 (9%) to possible foods, and 4 (2%) to other causes. In Australia it was recently reported that between 1997 and 2004, there were 86 deaths in which anaphylaxis was certified as the underlying or associated cause of death. However, detailed analyses of the causes of anaphylaxis were limited, with the majority (72%) of deaths classified as unspecified anaphylaxis, and demographic characteristics were not examined. Although it was suggested that there are approximately 1500 anaphylaxis deaths in the United States (US) each year, no national statistics have been published for anaphylaxis deaths. A review of the coronal autopsy database in a New Zealand city (Auckland) hospital identified 18 anaphylaxis deaths between 1985 and 2005. Ten (56%) were due to drugs or intravenous contrast, 4 (22%) to insect stings, 2 (11%) to food, and 2 (11%) to undetermined causes. In these previous studies, time trends for fatal anaphylaxis were not examined.

Foods are an important cause of anaphylaxis fatalities. A careful search for fatal cases of food-induced anaphylaxis in the UK identified 48 deaths over a 7-year period between 1999 and 2006. A US voluntary registry of fatal food-induced anaphylaxis maintained by the American Academy of Allergy, Asthma & Immunology and the Food Allergy and Anaphylaxis Network recorded 32 cases of fatal food-induced anaphylaxis between 1994 and 1999 and a further 31 cases between 2001 and 2006. Consistent risk factors for food-induced anaphylaxis deaths identified from the reported UK and US series include active asthma, age of 11 to 30 years, peanut or tree nut allergy, unavailability or delayed use of adrenaline, and previous severe reactions. Of note, peanut- and tree nut–induced anaphylaxis deaths were more common in the US (81%) compared with the UK (38%).

Although several studies have reported increasing rates of hospital admissions and general practice presentations for anaphylaxis worldwide, there is a paucity of data on time trends for rates of anaphylaxis fatalities. Interestingly, the rate of fatal food-induced anaphylaxis appeared to remain stable over the last decade in both the UK and the US; however, this requires formal study, and data on time trends for other causes of fatal anaphylaxis are not available. Information on time trends for anaphylaxis admissions assessed by allergen trigger is also lacking.

The objective of this study was to examine in detail the causes and demographics of deaths from anaphylaxis in Australia, as
well as to compare time trends in anaphylaxis fatalities and hospital admissions. Such information will further clarify the subjects at greatest risk of fatal anaphylaxis and improve understanding of anaphylaxis and fatal anaphylaxis.

METHODS

The cause or causes of death in Australia are recorded on death certificates by medical practitioners or in coroners’ reports and collated by each state. Causes of death are amalgamated, validated, and coded by the Australian Bureau of Statistics and then entered into a National Mortality Database maintained by the Australian Institute of Health and Welfare (AIHW).

Data were extracted from the AIHW database from January 1997 through December 2005, and the following International Classification of Diseases, Tenth revision (ICD-10), death codes were assigned: anaphylactic shock due to adverse food reactions (T78.0); anaphylactic shock, unspecified (T78.2); anaphylactic shock due to serum (T80.5); and anaphylactic shock due to adverse effects of drugs properly administered (T80.5). In addition, data on deaths with the following allergy-associated codes were retrieved: other adverse food reactions, not elsewhere classified (T78.1); angioedema (T78.3); and allergy, unspecified (T78.4). The data are expressed in absolute numbers and tabulated into 5-year age groups and different sexes.

Further details regarding the cause of death were sought by reviewing all deaths codes listed in the deidentified cases within each of the above ICD-10 codes to determine whether a specific cause could be identified. Using this approach, we identified a subgroup of insect sting–induced anaphylaxis and sought further death codes for the following: toxic effects of contact with honeybees, wasps, and hornets (X23) and toxic effects of contact with other venomous arthropods, including jumper ants (X25). On review of the causes of deaths in X23 and X25, we deduced that these deaths were similar to the subgroup of insect sting–induced anaphylaxis and added them to our analysis.

The causes and contributors of death were analyzed, and the deaths were grouped according to ICD-10 codes, likely cause, and associated comorbidities.

Further details were obtained from the National Coroner Inquiry System for the food-induced anaphylaxis deaths to determine the causative food allergen or allergens because this information was not detailed in the ICD-10 codes. We did not obtain details of the coroner’s inquiries for the other deaths because the data were not complete. Additional information on food-induced anaphylaxis deaths was extracted from media interviews of families with anaphylaxis deaths.12

Hospitalization data were obtained from the National Hospital Morbidity Database, which is also held at the AIHW. Data were available in 1-year periods from July 1 to June 30 for each year. The data spanned 2 different versions of ICD coding: International Classification of Diseases, Ninth revision, Clinical Modification (ICD-9-CM) for admissions from 1993 through 1997 and International Classification of Diseases, Tenth Revision, Australian Modification (ICD-10-AM) from 1998 onward.

All hospital admissions from July 1994 to June 2005 in which the principal diagnosis corresponded to the above death codes were included (ICD codes: anaphylactic shock due to adverse food reactions [ICD-9-CM 995.6; ICD-10-AM T78.0]; anaphylactic shock, unspecified [ICD-9-CM 995.0; ICD-10-AM T78.2]; anaphylactic shock due to serum [ICD-9-CM 995.4; ICD-10-AM T80.5]; and anaphylactic shock due to adverse effects of drugs properly administered [ICD-10-AM T88.6, no separate classification for drug anaphylaxis in ICD-9-CM]). Detailed information on the causative food allergens was available through the AIHW between July 1994 and June 1998 only.

The AIHW anaphylaxis death and hospital admission data were grouped into food-induced anaphylaxis and non–food-induced anaphylaxis, with the latter being the combination of drug-induced anaphylaxis, unspecified anaphylaxis, and anaphylaxis to serum. Age-standardized rates (ASRs) for death and hospital admission were calculated by standardizing to the age distribution of the Australian population in mid-2001 obtained from the Australian Bureau of Statistics.13 Age-adjusted Poisson regression models were used to investigate time trends. Results are presented as rate ratios and 95% CIs.

RESULTS

There were 112 anaphylaxis fatalities in Australia during the 9-year study period. Based on the Australian 2001 population census, this equates to 0.64 anaphylaxis deaths per million population per year.

Of these 112 fatalities, 7 (6.3%) were attributed to food anaphylaxis (T78.0), 22 (19.6%) to drug anaphylaxis (T88.6), and 83 to unspecified anaphylaxis (T78.2). Detailed examination of the death codes for the latter group enabled us to deduce the allergen cause in the majority of cases. Of the 83 deaths, 20 (17.9%) were due to insect sting–induced anaphylaxis, 42 (37.5%) to probable drug-induced anaphylaxis, 15 to an undefined cause, and 6 were related to either a medical or surgical procedure (Fig 1). There were no fatalities from anaphylaxis to serum (T80.5).

A further 46 deaths had nonspecific allergy codes (other adverse food reaction, n = 4; allergy unspecified, n = 19; or angioneurotic edema, n = 23) listed in the death certificates as significant contributors of death. The death codes for these cases included a variety of medical comorbidities, which are the likely causes of death. We could not confidently conclude that allergy was a major contributor, and no allergens were clearly identified. These deaths were thus excluded from the subsequent analysis.

Food-induced anaphylaxis fatalities and admissions

All 7 food-induced anaphylaxis fatalities occurred in age groups 5 to 35 years (female predominance, 5/7 cases; Fig 2, A), with all but 1 subject being older than 10 years (Table I). Where data were available, all (5/5) had a previous food-induced allergic reaction. Peanut was the offending allergen in 3 cases, and fish was the offending allergen in 1 case (no information in 1 case and undetermined in 2 cases). All (5/5) had active asthma, and although we do not have information on the adequacy of asthma control, at least 1 subject had poorly controlled asthma that was untreated and managed by a homeopath. Four of 6 subjects received adrenaline administered by the local doctor, ambulance paramedics, or in the hospital, and 2 did not receive adrenaline (information regarding treatment was not available in 1 case). Finally, all (6/6) had eaten food prepared outside the usual residence (Table I). Details of the severity of the previous reaction and the temporal relationship from the eventual fatality were not available. Nevertheless, it was deduced that the previous food-induced reactions were not anaphylactic reactions because none of the subjects had a personal adrenaline autoinjector, which in Australia would usually be prescribed after anaphylaxis.
There were a total of 5007 food-induced anaphylaxis hospital admissions between July 1994 and June 2005 with 2 age peaks, one in the 0- to 4-year age group and one in the 15- to 29-year age groups (Fig 3). Overall, equal numbers of male and female subjects were admitted for food-induced anaphylaxis; however, male subjects outnumbered female subjects (1.5:1) less than 15 years of age, whereas female subjects outnumbered male subjects (1.4:1) older than 15 years (Fig 3). Although age-specific

**FIG 1.** Causes of anaphylaxis deaths. There were 112 deaths between 1997 and 2005 in Australia. Causes are shown.

**FIG 2.** Anaphylaxis fatalities. A, Absolute number of anaphylaxis deaths by cause and age group. B, Anaphylaxis death rates by cause and age group. All but 1 food-induced anaphylaxis death occurred in the 10- to 35-year age groups (1 death at 8 years), most insect sting–induced anaphylaxis deaths occurred between 35 and 84 years, and most drug-induced induced anaphylaxis deaths occurred between 60 and 85 years.
Hospitalization rates were highest in the 0- to 4-year age group (9.4/100,000 population), age-specific fatality rates peaked in the 10- to 35-year age groups (Fig 2, B).

Information on the causative food allergen was only available between July 1994 and June 1998 (ICD-9-CM). Peanut was the most common (23%) food allergen, followed by fish (18%), crustaceans (16%), tree nuts (16%), eggs (9%), and milk (8%). Peanut-, milk-, egg-, and tree nut–induced anaphylaxis accounted for the bulk of admissions in the 0- to 4-year age group, whereas peanut-, crustacean-, fish-, and tree nut–induced anaphylaxis accounted for the majority in the 15- to 30-year age groups (data not shown).

**Non–food-induced anaphylaxis fatalities and admissions**

There were a total of 105 non–food-induced anaphylaxis fatalities between 1997 and 2005: 22 drug induced, 42 probable drug induced, 20 insect sting induced, 15 of undetermined cause, and 6 relating to either a medical or surgical procedure (Fig 1).

There were a total of 9721 non–food-induced anaphylaxis admissions between June 1994 and June 2005: unspecified anaphylaxis, 6565 (68%) cases; drugs, 3019 (31%, data available June 1998 to June 2005) cases; serum/after infusion/transfusion or therapeutic injection, 137 (1%) cases.

**Insect sting–induced anaphylaxis fatalities**

Most (85%) of the 20 insect sting–induced anaphylaxis fatalities occurred in adults 35 to 84 years old (age group range, 15-84 years); (Fig 2) with male preponderance (19/20 [95%] cases). Thirteen (65%) were due to honeybee, wasp, and hornet stings; 6 (30%) to other arthropods, including Jumper-Jack ants; and 1 to a nonvenomous arthropod. We do not have details of the fatal sting event, including the specific insect and location of the sting, previous insect sting reactions, or whether adrenaline was administered for the fatal reaction. A review of comorbidities showed that 3 subjects had ischemic heart disease or dysrrhythmia, and 1 had active asthma. We were unable to obtain separate

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Death year</th>
<th>State</th>
<th>Age group</th>
<th>Sex</th>
<th>Allergen</th>
<th>Asthma</th>
<th>Previous reaction</th>
<th>Implicated food</th>
<th>Location</th>
<th>Adrenaline (time from anaphylaxis to adrenaline)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1999</td>
<td>NSW</td>
<td>15-19 y</td>
<td>M</td>
<td>Peanut</td>
<td>Yes</td>
<td>Yes</td>
<td>Satay sauce</td>
<td>Workplace</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>2000</td>
<td>VIC</td>
<td>5-10 y</td>
<td>F</td>
<td>Peanut</td>
<td>Yes</td>
<td>Yes</td>
<td>Satay sauce</td>
<td>Father’s home, parents separated</td>
<td>Yes (20 min, intravenous adrenaline administered by paramedics)</td>
</tr>
<tr>
<td>3</td>
<td>2002</td>
<td>NSW</td>
<td>10-14 y</td>
<td>M</td>
<td>Peanut</td>
<td>Yes, poorly controlled</td>
<td>Yes</td>
<td>Peanut butter</td>
<td>School camp</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>2002</td>
<td>VIC</td>
<td>30-34 y</td>
<td>F</td>
<td>Fish</td>
<td>DNA</td>
<td>Yes</td>
<td>Prawns (from a fish/seafood platter)</td>
<td>Friend’s house</td>
<td>Yes (time not documented, administered by paramedics)</td>
</tr>
<tr>
<td>5</td>
<td>2002</td>
<td>NSW</td>
<td>30-34 y</td>
<td>F</td>
<td>DNA</td>
<td>DNA</td>
<td>DNA</td>
<td>DNA</td>
<td>On the road</td>
<td>Yes (time not documented, administered by hospital emergency department)</td>
</tr>
<tr>
<td>6</td>
<td>2003</td>
<td>NSW</td>
<td>10-14 y</td>
<td>F</td>
<td>Undetermined</td>
<td>Yes</td>
<td>DNA</td>
<td>Consumed Chinese restaurant take out and (later) bun from bakery</td>
<td>On the road</td>
<td>Yes (time not documented, but administered by local doctor)</td>
</tr>
<tr>
<td>7</td>
<td>2004</td>
<td>SA</td>
<td>30-34 y</td>
<td>F</td>
<td>Undetermined</td>
<td>Yes</td>
<td>DNA</td>
<td>Consumed Chinese takeout with rice and pork</td>
<td>On the road</td>
<td>Yes (time not documented, but administered by local doctor)</td>
</tr>
</tbody>
</table>

**FIG 3.** Food-induced anaphylaxis admissions between 1994 and 2005 categorized by age group and sex.
data on insect sting–induced anaphylaxis admissions because there was no specific code for insect stings within the ICD-10-AM anaphylaxis codes.

**Drug-induced anaphylaxis fatalities and admissions**

The 22 drug-induced and 42 probable drug-induced anaphylaxis deaths were combined for analysis. Most deaths (73%) occurred in adults 55 to more than 85 years of age (age group range, 10-85 years; Fig 2) with similar numbers of male and female subjects. The implicated drugs are shown in Fig 4. Further analysis showed that all penicillin-induced deaths occurred between 60 and 74 years of age, whereas cephalosporin-induced deaths occurred between 35 and 74 years of age. Because of the limitation of the database, we were not able to differentiate patients who were admitted for the primary problem of drug-induced anaphylaxis from those who experienced drug-induced anaphylaxis caused by treatment for other comorbidities. Significant comorbidities included ischemic heart disease or dysrhythmia in 21 subjects, obstructive airway disease in 11 subjects (5 with asthma, 5 with chronic obstructive pulmonary disease, and 1 with emphysema), mastocytosis in 1 subject, and hypogammaglobulinemia in 1 subject.

There were a total of 3019 drug-induced anaphylaxis hospital admissions between July 1998 and June 2005. Male subjects outnumbered female subjects (1.4:1) younger than 15 years, whereas female subjects outnumbered male subjects (1.8:1) older than 15 years (Fig 5, A). Overall, there were more female subjects admitted for drug-induced anaphylaxis than male subjects (1.5:1). The age-specific hospitalization rates were highest for the 55- to 84-year age group (3.8/100000 population), which is similar to the peak for drug-induced anaphylaxis deaths (Fig 2, B).

**Time trends for anaphylaxis admissions and deaths**

Between 1994-1995 and 2004-2005, Poisson regression models showed strong evidence of an increase in the rate of admissions for food-induced anaphylaxis over time ($P < .0001$; Fig 6, A). The estimated rate ratio, the multiplicative increase of the rate per year, was 1.12 (95% CI, 1.11-1.13), which is equivalent to an estimated increase of 350% over the 11 years.
There was evidence that rates of food-induced anaphylaxis admissions are increasing more rapidly in some age groups than in others ($P = .0002$; Fig 7, A). The rate of admissions in the 0- to 4-year age group was estimated to increase 1.04 (95% CI, 1.02-1.06) times more rapidly than in the older age groups.

Admission rates for peanut- and crustacean-induced anaphylaxis appeared to be increasing the most rapidly over time, with more modest rate increases estimated for cow’s milk. Peanut- and crustacean-induced anaphylaxis admissions were estimated to increase 1.28 (95% CI, 1.00-1.63; $P = .05$) and 1.27 (95% CI, 0.98-1.65; $P = .07$) times more quickly than milk-induced anaphylaxis admissions, respectively. Among 0- to 4-year-olds, peanut-induced anaphylaxis admissions increased more rapidly than those to other foods (rate ratio compared with milk, 1.35; 95% CI, 0.96-1.91; $P = .08$).

There was also strong evidence of an increase in the rate of non–food-induced anaphylaxis admissions over time, with an estimated multiplicative rate increase of 1.08 per year (95% CI, 1.08-1.09; $P < .0001$), which is equivalent to an estimated 150% increase between 1998 and 2005 (Fig 6, B), with more modest increases in unspecified anaphylaxis and anaphylaxis to serum. Data on insect sting–induced admissions were not available separately for comparison because these were coded as unspecified anaphylaxis.

In contrast to the considerable increases in rates of admission for food-induced and non–food-induced anaphylaxis, there was no evidence of an increase in the rate of all anaphylaxis fatalities ($P = .204$; rate ratio, 1.03; 95% CI, 0.96-1.11; Fig 6, C). However, among non–food-induced anaphylaxis deaths, the rate of insect sting–induced anaphylaxis deaths was estimated to have decreased at a multiplicative rate of 0.79 per year (95% CI, 0.66-0.95; $P = .013$), which is equivalent to an estimated 88% decrease over 9 years, whereas drug-induced anaphylaxis deaths were estimated to have increased by 300% over the same period (rate ratio, 1.13; 95% CI, 1.03-1.25; $P = .013$; Fig 6, D).

**DISCUSSION**

**Anaphylaxis deaths: Causes and demographics**

This is the first detailed analysis of national statistics for anaphylaxis fatalities from all causes in Australia. The only other detailed analysis of national data for fatal anaphylaxis is the UK
series reported by Pumphrey. Our data estimate the rate of anaphylaxis fatality in Australia to be 0.64 deaths per million population per year, which is higher than the UK series estimate of 0.33 anaphylactic deaths per million population per year. Our data indicates that the relative number of deaths to admissions was 1:1000 for food-induced anaphylaxis and 11:1000 for non-food-induced anaphylaxis.

The causes of fatal anaphylaxis in our series were predominantly drug and probable drug (57%), followed by insect sting (18%). Foods were an uncommon causative agent (6%). Because there were no separate ICD codes for latex allergy, latex-induced anaphylaxis fatalities would be coded as unspecified anaphylaxis (T78.2) or could also be miscoded as anesthetic/medical or surgical procedure/radiological causes.

Compared with the UK series, fatalities in Australia were less likely to be due to foods and more likely to be due to drugs. In contrast, our findings were similar to those reported in the Auckland hospital series. Possible reasons for these disparities between the UK, Australia, and New Zealand include population differences in ecologic exposures, dietary exposures, or both and differences in methodology for data retrieval. Our data were obtained from a mandatory national mortality database that uses ICD-10 death codes to categorize all anaphylaxis-induced deaths. The advantage of this approach is the lower probability of missing data, allowing more accurate epidemiology and time-trend results; however, caveats include the limitations of coding, with a significant proportion coded as unspecified anaphylaxis, and time lag for data entry. The strength of the UK series data is that they are extracted from a dedicated anaphylaxis deaths registry. This allowed for greater in-depth analysis of risk factors for anaphylaxis deaths but would be dependent on passive reporting and might thus be less complete. The Auckland series was derived from a single hospital’s forensic department database, which might not be representative of the national data. Collation of data could be improved by a dedicated anaphylaxis death registry that is managed alongside the national mortality database and by review of the anaphylaxis ICD codes to better classify anaphylaxis deaths and admissions.

**Risk factors for fatal anaphylaxis**

Anaphylaxis deaths from the various causes occurred in distinct age groups and with different sex distributions. All but 1 food-induced anaphylaxis death occurred in children and young adults 10 to 35 years of age with a preponderance of female subjects, whereas most insect sting–induced anaphylaxis deaths occurred between 35 and 84 years and almost exclusively in male subjects, and most drug-induced anaphylaxis deaths occurred...
between 55 and 85 years with equal sex distribution. These differing demographics can assist in the identification of those at the highest risk of fatality from a specific allergy.

For both food-induced and non–food-induced anaphylaxis, we identified similar risk factors for death, as previously reported in UK, US, and Australian studies.\(^4\),\(^7\)-\(^10\),\(^14\)-\(^16\) Risk factors for food anaphylaxis were age 10 to 35 years, active asthma, peanut allergy, ingestion of food prepared outside of the subject’s residence, and delayed administration of adrenaline. Risk factors for drug-induced anaphylaxis fatalities were age 55 to 85 years, presence of respiratory or cardiovascular comorbidities, and antibiotics or anesthetic agents. Risk factors for insect sting–induced anaphylaxis deaths were age 35 to 84 years and male sex.

Comparison of demographics for anaphylaxis fatalities and admissions

It is important to note the different demographics for anaphylaxis admissions and fatalities. Although most admissions for food-induced anaphylaxis were in male subjects younger than 5 years, all deaths occurred between 8 and 35 years and predominantly in female subjects (Fig 3). Similarly, although the highest rates of bee sting–induced admissions (which account for 75% of all insect sting–induced admissions) are in male subjects 5 to 9 years of age,\(^5\) most insect-induced anaphylaxis deaths occurred in 30- to 84-year-old male subjects. In contrast, drug-induced anaphylaxis deaths followed similar demographics to admissions, with both occurring in the 55- to 85-year age group (Fig 5). These findings illustrate the different demographics for nonfatal anaphylaxis and fatal anaphylaxis and emphasize the need to consider both when developing management plans for patients.

Time trends in anaphylaxis admissions and fatalities

ASRs of hospital admissions for food-induced and non–food-induced anaphylaxis increased 350% and almost 230%, respectively, in the 11 years from 1994-1995 to 2004-2005. For food-induced anaphylaxis admissions among 0- to 4-year-olds, the increase was most prominent for peanut–induced anaphylaxis, with more modest increases observed for cow’s milk–induced and egg–induced anaphylaxis. This is consistent with a recent Melbourne study\(^1\),\(^8\) that found peanut (18%) was the leading cause of food anaphylaxis in children.

In contrast to these increasing prevalence rates for anaphylaxis admissions, particularly for peanut- and tree nut–induced anaphylaxis, the ASRs for food-induced and non–food-induced anaphylaxis deaths have remained stable between 1997 and 2005. Our data support the observation that the number of deaths per year in the UK remained unchanged over a decade.\(^4\) These disparate trends in anaphylaxis fatalities and admissions suggest that anaphylaxis management has been largely successful. The slight dip in anaphylaxis fatalities in Australia in 2003 (Fig 6, C and D) could be related to increased community awareness after the highly publicized death of a teenager from peanut-induced anaphylaxis in 2002, new food labeling legislation that required listing of common allergens as an ingredient in any quantity or form from December 2002, and greater availability of adrenaline autoinjectors, which became subsidized under the Australian Pharmaceutical Benefits Scheme from November 2003. Indeed, there was an exponential increase in prescriptions for adrenaline autoinjectors after this.\(^1\),\(^9\) It is also possible, although less likely, that the increase in anaphylaxis admissions does not reflect a true increase in prevalence but rather an increase in community anxiety, awareness, and recognition of anaphylaxis.

Interestingly, we estimated an 88% decrease in ASRs for insect sting–induced anaphylaxis deaths over the 9-year study period. This is in contrast to the stable time trends in age-specific admission rates for contact with honey bees, wasps, and hornets (ICD 10-AM) between 2002 and 2005.\(^1\),\(^7\) Possible explanations include the widespread use of insect venom immunotherapy since the mid-1980s, recent increased availability of adrenaline autoinjectors,\(^1\),\(^9\) and a decrease in the rural population in Australia.

Of concern, the ASRs for drug-induced anaphylaxis deaths increased 300%, double the increase in drug-induced anaphylaxis hospital admissions (150%). This might reflect the fact that most deaths from drug-induced anaphylaxis occur with the first known exposure to the drug.\(^2\) Early identification of those at risk of drug-induced anaphylaxis and improved management of this condition, similar to recent efforts in food allergy management, are urgently needed to reverse this trend. Possible strategies include improvement of drug allergy diagnostics, use of pharmacogenetics to identify at-risk subjects, specific clinics for assessment and management of drug allergy, and integration of national drug allergy alert systems.

Conclusion

The demographics for fatal anaphylaxis differ from those for anaphylaxis presentations and are specific to the anaphylaxis allergen trigger. Mortality rates from food- and insect sting–induced anaphylaxis in Australia have remained low and decreased, respectively, but drug-induced anaphylaxis fatalities are on the increase. Further studies toward early identification of patients at risk of fatal drug-induced anaphylaxis are required.

We thank Mr Robert van der Hoek, Population Health Unit, AIHW, for extracting the national death data and Ms Helen Messinis, Administration Officer, National Coroners Information System, Victorian Institute of Forensic Medicine, for extracting the coroner’s reports on food-induced anaphylactic deaths.

Clinical implications: Food-induced anaphylaxis death rates in Australia remained stable despite increasing prevalence of food-induced anaphylaxis admissions, suggesting that management strategies have been successful. The increasing drug-induced anaphylaxis fatalities are of concern.

REFERENCES