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To cite this article: Laura A. Scott, Bethan I. Jones, Thomas R. Berni, Ellen R. Berni, Jane De Vries & Craig J. Currie (2019) Evaluation of the epidemiology of peanut allergy in the United Kingdom, Expert Review of Clinical Immunology, 15:12, 1333-1339, DOI: [10.1080/1744666X.2020.1693264](https://doi.org/10.1080/1744666X.2020.1693264)

To link to this article: <https://doi.org/10.1080/1744666X.2020.1693264>



Accepted author version posted online: 15 Nov 2019.
Published online: 27 Nov 2019.



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ORIGINAL RESEARCH



Evaluation of the epidemiology of peanut allergy in the United Kingdom

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ABSTRACT

Aims: To describe the epidemiology of peanut allergy (PA) in the UK over the last three decades.

Methods: PA patients were identified from the Clinical Practice Research Datalink between 1987 and 2015. Incidence and prevalence of PA were compared between 2000 and 2015. Prevalence and relative risk (RR) of atopic comorbidities, anaphylaxis, adrenaline prescriptions versus matched controls were calculated.

Results: Point prevalence of PA in the entire population and those <18 years increased from 31 to 202 and 116 to 635 per 100,000, respectively, between 2000 and 2015. Incidence increased from 8.6 to 18.2 per 100,000. Incidence in 2015 was 105 cases per 100,000 aged 0–4 years versus 13.4 per 100,000 aged 5+ years. Anaphylactic events affected 1.2% of the cases and 0.007% of the controls. The rate of adrenaline prescriptions was 5,910 per 100,000 person-years for PA patients. RRs for asthma, eczema and allergic rhinitis in PA patients versus controls were 4.5 (95% CI 4.2–4.8), 3.2 (3.1–3.4) and 2.6 (2.4–3.0), respectively.

Conclusions: The prevalence and incidence of PA increased markedly over the study period. PA was associated with atopic conditions and anaphylaxis. PA patients had increased adrenaline prescriptions.

ARTICLE HISTORY

Received 2 April 2019

Accepted 12 November 2019

KEYWORDS

Peanut; allergy; anaphylaxis; epidemiology; atopy

1. Introduction

Peanut allergy (PA) is an adverse, hypersensitive, immune response to peanuts [1] mediated by an immunoglobulin E (IgE) type I hypersensitivity reaction [2]. Peanuts are one of the most common causes of IgE-mediated reactions to food and food-induced anaphylaxis [3]. The severity of reactions to peanut can range from mild to severe and can vary between episodes [4]. Symptoms usually develop within minutes after the ingestion of a peanut. Fatal or near-fatal allergic reactions involve typically progressive lower respiratory symptoms, hypotension, and arrhythmias [5]. PA generally presents in early childhood, with the most common age of onset being 14 to 24 months of age in the UK [6], and the condition is rarely outgrown [7]. The current standard of care for PA is strict avoidance and the use of adrenaline following an accidental exposure [8].

Prevalence of PA is widely accepted to be increasing globally [9]. The reason for this increase is not fully understood, although the recent paradigm-shifting LEAP study in the UK found that early introduction of peanut in the diet was associated with a lower risk of developing PA, in contrast to previous advice that pregnant women and young children should avoid peanuts to reduce the risk of PA [10]. However, prevalence estimates vary due to factors such as differing age, geography, study design and method of diagnosis (self-reported, clinical history of previous reactions, positive skin prick test [SPT] or specific IgE food challenge) [11]. In a systematic review and meta-analysis of PA prevalence

studies in Europe between 2000 and 2012, Nwaru and colleagues reported that mean estimates of PA point prevalence ranged from 0.22% to 8.58% dependent on the method of diagnosis (0.22% to 1.74% when excluding prevalence of peanut IgE positivity alone, which is not typically sufficient for a diagnosis) [12]. Kotz and colleagues reported a rise in the all-ages prevalence of PA in the UK from 0.24 per 1000 in 2001 to 0.51 per 1000 in 2005 based on primary care clinician-recorded diagnoses in a population-based analysis [13]. In a more recent birth cohort study conducted in Hampshire (UK) as part of the European Commission-funded EUROPREVALL initiative, the cumulative incidence of IgE-mediated PA at 2 years of age was estimated to be 0.6%, based on diagnosis by double-blind, placebo-controlled food challenge [14].

Prevalence of food allergy has been shown to be significantly higher in children with atopic disorders. In a US study, an estimated 35% of the children with atopic dermatitis also had IgE-mediated food allergy [15]. In a study carried out over 5 years, children with a peanut anaphylaxis event were significantly more likely to have asthma compared to those with a non-anaphylactic reaction to peanut [16].

Fear of accidental exposures to peanut and potentially life-threatening reactions, as well as the burden of strict avoidance on daily activities and familial-social relations, can lead to impaired quality of life for both patients and their families [17,18].

Given the growing incidence and a wide range of prevalence estimates, the aim of this study was to generate updated prevalence and incidence rates for PA in the UK and to determine the rates of atopic comorbidity and anaphylaxis events and comparing the results to those without PA.

2. Methods

2.1. Data source

The data source used was the Clinical Practice Research Datalink (CPRD), a database that provides anonymous data from nearly 700 primary care practices in the UK, with over 20 years of data collected from 14 million individuals at the time of this study [19].

Data containing details on medical history, hospital referrals, test results, and other demographics, is recorded into a computerized database by primary care staff. Each diagnosis has a standardized associated Read code. Many studies have validated the Read code classification to give a high positive predictive value [20]. The data in CPRD are widely representative of the UK population in terms of age and sex, with practices spread throughout the whole of the UK [21]. Data on prescriptions are accurately recorded as they are automatically generated and documented by the practice software, with data quality markers provided by CPRD [22].

Patients are flagged as acceptable for research purposes if the patient has been permanently registered at the practice and records are consistent with regards to registration, age, sex, and event dates and from this are assigned an 'up-to-standard date', which corresponds to assessment of the completeness of their data. Approximately 60% of CPRD practices participate in a linkage scheme to secondary care data recorded in Hospital Episode Statistics (HES), where diagnoses are recorded using ICD-10 code classification. This study was granted CPRD Independent Scientific Advisory Committee approval (ISAC 17_116).

2.2. Patient selection and PA diagnosis

Patients diagnosed with PA between 1987 and 2015 were identified by diagnostic Read code from the CPRD database (See appendix). Patients with a positive radioallergen sorbent (RAST) test for peanut were included and those with a negative peanut RAST test were excluded, regardless of the presence of a diagnostic code. Patients were included if they were diagnosed between 1987 (start of CPRD records) and 2015. Prevalence and incidence rates were compared from 2000 onwards in 5-year increments, rather than from 1987 onwards. This was to ensure there was a large enough sample of diagnosed cases and controls to calculate these estimates.

The cohort index date was set for patients identified between 1987 and 2015 as the earliest of first electronic diagnosis of PA or first physician positive peanut-specific RAST or ICD-10 code for nonspecific food allergy or anaphylaxis (See appendix). If the cohort index date was prior

to the patient's registration date or up-to-standard date, then the index date was set to the latter of the two, with the end of CPRD follow-up being the patient's censor date. The index date reflects the first electronic recording of PA and not necessarily the true date of first PA diagnosis.

Cases were matched to controls at a ratio of 1:1 based on year of birth, sex, primary care practice, HES linkage status and year of registration to compare prevalence and incidence of atopic conditions and rates of anaphylaxis. Matched controls were only selected if they had no history of food allergies, to ensure that peanut-allergic patients were not included in the comparison group. The index date for the controls was set as the same as their respective cases.

The end of CPRD follow-up (the earliest of the patients' transfer-out date from the practice, date of death or the last data-collection date for the practice) was the censor date for both cases and controls.

2.3. Statistical analysis

Baseline characteristics were presented for all peanut-allergic cases and the prevalence of atopic comorbidities was calculated. Medians and interquartile range (IQR) were presented. Point prevalence was calculated at the mid-year point (1 July) of each year. The numerator was the number of patients registered at the mid-year point with an index date prior or equal to the mid-year point. The denominator was the number of patients who were up to standard and registered at the practice on the mid-year point. Patients aged ≥ 18 years were excluded from the point prevalence calculations for children and adolescents.

Incidence per year was calculated and subsequently split by age and gender in the year 2015, with the numerator representing newly diagnosed cases who were registered and up-to-standard each year. The denominator was the total follow-up in person-years for those patients who were eligible in the time periods.

Prevalence of relevant atopic comorbidities (eczema, asthma, and allergic rhinitis) at baseline was compared between cases and matched controls and p-values were derived from a chi-squared test. The rate of anaphylaxis events per 100,000 patient-years and the number of adrenaline prescriptions per 100,000 patient-years were calculated. For estimating the incidence of anaphylaxis two different denominators were used; firstly, peanut-allergic patient person time to understand the incidence of anaphylaxis within the peanut-allergic population and secondarily the full CPRD population person time.

Adrenaline prescriptions refer to the number of scripts prescribed in primary care, not the number of adrenaline autoinjectors administered in the case of an emergency. Incidence of anaphylaxis was calculated for each year from 1997 (start of HES records) to 2015 to determine if there was an increase in incident cases of anaphylaxis over time. It should be noted that anaphylaxis events within the PA cohort cannot be attributed definitively to peanut, given limitations of the dataset and high level of other allergic comorbidities in the cohort.

3. Results

3.1. Baseline characteristics

Fifteen thousand four hundred and eighty-three cases of PA were identified. Of these, 7,917 (51%) were males and 13,609 (87.9%) could be matched to controls and used for relevant analysis. The median age at index date for all cases was 13 (IQR = 5–25, Table 1).

3.2. Incidence and prevalence

The point prevalence of PA in the whole CPRD population increased each year over the study period (1987–2015) but, due to the lack of data in the early stages, rates were compared from 2000 onwards (Figure 1(a)).

Point prevalence increased from 31.3 (95% CI; 29.4–33.3) cases per 100,000 people in the whole CPRD population in 2000 to 78.2 (95% CI; 75.7–80.8) in 2005, 134.4 (95% CI; 131.2–137.8) in 2010 and 202.3 (95% CI; 197.6–207.1) per 100,000 in 2015. Incidence of PA increased from 8.6 (95% CI; 7.6–9.6) cases per 100,000 people in 2000 to 11.3 (95% CI; 10.3–12.3) in 2005, 15.2 (95% CI; 14.1–16.3) in 2010 and 18.2 (95% CI; 16.8–19.6) per 100,000 in 2015 (Figure 2).

In 2015, the age-specific incidence of PA was highest amongst those aged 0–4 years, with higher age-specific incidence in boys (127.6 (95% CI; 105.7–152.2) per 100,000) compared with girls (80.9 (95% CI; 63.4–101.4) cases per 100,000). As age increased, the age-specific incidence of PA amongst males decreased, where men aged ≥ 70 years old had an age-specific incidence rate of 2.0 (95% CI; 0.7–4.9) cases per 100,000 people. For females,

Table 1. Baseline characteristics for peanut allergy patients and their matched controls.

	Cases	Matched cases	Matched controls	p-Value
Patients, n (%)	15,483 (100)	13,609 (100)	13,609 (100)	
Male, n, %	7,917 (51.13)	7,000 (51.44)	7,000 (51.44)	1
Age (years), median, IQR	13 (5–25)	12 (4–24)	12 (4–24)	1
Age (years), mean, sd	17.07 (15.19)	16.06 (14.32)	16.06 (14.32)	1
Primary care contacts (prior 365 days), median, IQR	2 (0–6)	2 (0–6)	2 (0–4)	<0.0001

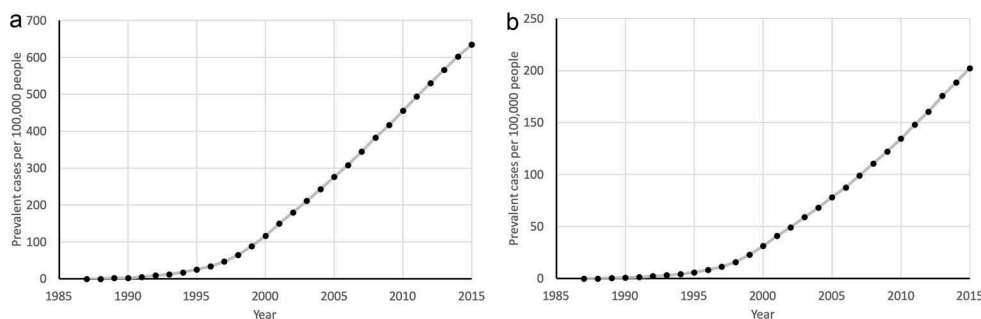


Figure 1. Prevalence of peanut allergy per 100,000 population (1987–2015) in the Clinical Practice Research Datalink. 1(a) Whole CPRD population 1(b) Those aged 17 years and younger.

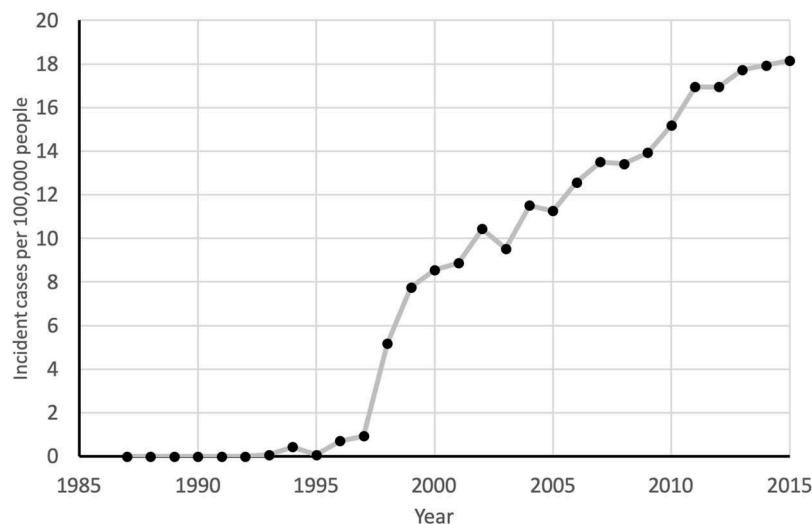


Figure 2. Incidence of peanut allergy per 100,000 population (1987–2015) in the Clinical Practice Research Datalink.

in general, the age-specific incidence of PA also decreased with increasing age, with the exception of the age group 20 to 24 years old, where there were 42.5 (95% CI; 30.9–56.6) cases per 100,000 people, a slight rise from the previous age group (Figure 3).

The point prevalence of PA in those aged <18 years increased each year from 2000 to 2015 (Figure 1(b)). Point prevalence increased from 116.4 (95% CI; 108.4–124.9) cases per 100,000 people aged <18 years in 2000 to 276.4 (95% CI; 266.0–287.1) in 2005, 455.3 (95% CI; 442.1–467.0) in 2010 and 634.7 (95% CI; 616.4–653.6) per 100,000 in 2015.

3.3. Anaphylaxis events and adrenaline prescriptions

There were 178 cases (1.2%) with a recorded anaphylaxis event on or after their index date, compared to 1 (0.007%) in the control, and a total of 194 anaphylaxis events in the cases compared to two events in the controls.

Incidence of anaphylaxis events was calculated for each year from 1997 to 2015. Using the peanut-allergic population as the denominator, incidence of anaphylaxis appeared to be relatively stable over the period 2000 to 2015 (Figure 4) with an incident rate ratio (IRR) of 7.5 (95% CI; 2.2–24.9) in the peanut-allergic population. In the previous years, there was a larger variation due to the small numbers in the denominator. When using the whole of the CPRD population as a denominator, there appeared to be a sharper increase in anaphylaxis incidence in the period post 2000 (Figure 5). IRR for the whole CPRD population was 3.5 (95%CI; 1.1–11.7).

The rate of anaphylaxis events was 236 per 100,000 person-years for cases compared to 2 per 100,000 person-years for controls. Sixty-five percent of the patients with PA <18 years old were prescribed adrenaline.

The median age for the first anaphylaxis event on or after index date was 15 years (IQR 6–25) for all PA cases. Out of the 178 cases with an anaphylaxis on or after index date, 99 were male and 79 were female. In the males, the median age of the first anaphylaxis for males was 13 (IQR 5–20) years compared to 19 (9–29) years in females. The rate of adrenaline

prescriptions was calculated at 5,910 per 100,000 person-years for all patients with a PA compared with 21 per 100,000 person-years for controls.

3.4. Comorbidities

Patients with PA had a higher prevalence of atopic conditions than controls. 30.8% of the cases had asthma compared to 6.9% of the controls ($p < 0.0001$), 28.8% of the cases had eczema compared to 8.9% of the controls ($p < 0.0001$) and 8.2% of the cases had allergic rhinitis compared to 3.1% of the controls ($p < 0.0001$, Table 2). The relative risk (RR) of each atopic condition was compared between cases and controls. The highest risk ratio was for asthma (RR = 4.5, 95% CI: 4.2–4.8), followed by eczema (RR = 3.2, 3.1–3.4) and allergic rhinitis (RR = 2.6, 2.4–3.0).

4. Discussion

The crude prevalence of PA in the UK in 2015 was 202 per 100,000 people in the whole CPRD population. There was a notable year-on-year increase in both rates of prevalence and incidence over the study period. Similar findings have been observed in other settings [13] and it has been postulated that this is due to both a genuine population increase in sensitivity to peanuts as well as an increasing awareness of the condition.

The increase in prevalence may reflect the increase in new cases being identified. Fortunately, PA still results in only a small number of fatalities, usually in young adults [23]. Thus, prevalence will increase as peanut-allergic patients generally survive to old age. In this study, we assumed that PA was a lifelong condition that did not resolve, as is commonly observed in clinical practice, although spontaneous remission has been reported in children younger than 5 years old [24].

A distinction must be drawn between age- and/or sex-specific values and crude population values when reviewing these epidemiological estimates. Despite an increase in prevalence of PA year-on-year, our overall prevalence

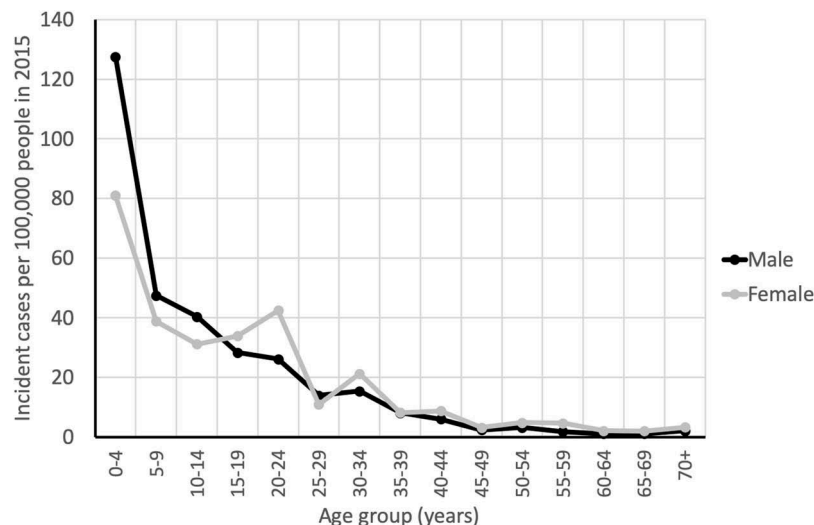


Figure 3. Incidence of peanut allergy per 100,000 population in 2015, split by age and gender from data from the Clinical Practice Research Datalink.

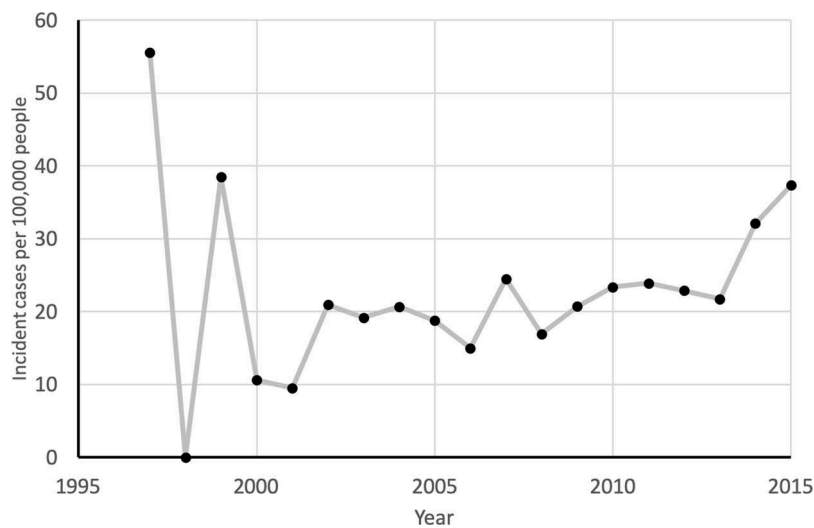


Figure 4. Incidence of anaphylaxis per 100,000 population over time from the Clinical Practice Research Datalink (total peanut-allergic patients – cases – as denominator).

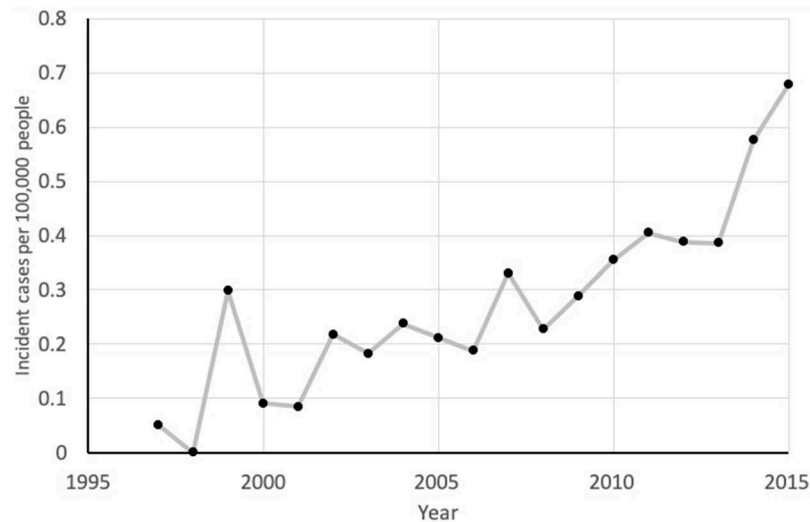


Figure 5. Incidence of anaphylaxis per 100,000 population over time from the Clinical Practice Research Datalink (CPRD patients as denominator).

Table 2. Prevalence of atopic diseases shown in peanut-allergic-matched population and their controls.

Atopic diseases	Matched cases (n = 13,609)	%	Matched controls (n = 13,609)	%	RR (95% CI)	p-Value
Asthma, n, %	4,190	30.8	932	6.8	4.5 (4.2–4.8)	<0.0001
Eczema, n, %	3,912	28.8	1,208	8.9	3.2 (3.1–3.4)	<0.0001
Allergic rhinitis, n, %	1,111	8.2	420	3.1	2.6 (2.4–3.0)	<0.0001

estimate appears to be lower than those reported in other studies [13,25].

Prevalence estimates could potentially be lower than real PA prevalence as the database used in this study is dependent on a general practitioner identifying PA and recording the diagnosis appropriately. Some cases of PA may have

been recorded as a more general diagnosis of ‘food allergy’ rather than use of the more specific diagnostic codes and actual cases of PA could be misclassified. However, our estimates may be less susceptible to selection bias than the studies using proactive methods. The study by Kotz and colleagues used a similar method to this study and

calculated the incidence and prevalence of PA from the year 2000 to 2005 [13]. The incidence in our study was slightly higher but largely comparable (0.08 versus 0.06 cases per 1,000 people in 2000 and 0.11 versus 0.08 in 2005). The small differences could be partly explained by our study using additional RAST and Read codes to diagnose PA. Patients with a low positive peanut-specific RAST test were included in the study, this does not necessarily confirm diagnosis, and this could have led to an overestimation of the prevalence of PA. However, in this study, only 34 patients were identified by a positive peanut RAST test with no record of a PA Read code. This methodology has also been applied to another PA related CPRD study [26]. Incidence calculations may have double counted patients in the year that they transferred practices. However, fewer than 10% of the practices in the UK contribute to CPRD; therefore, the chances of this happening are small. Moreover, the prevalence estimates from our study were similar to those using positive oral food challenges, with food challenge positive PA estimated by Nwaru et al. at 0.22% in the all-age population and by Grimshaw et al. at 0.6% in children aged 2 years [13,15].

In this study, the PA prevalence was higher for subjects aged <18 years compared with the whole CPRD population (635 versus 202 cases per 100,000 people, respectively). This is in line with the marked increase in incidence seen in young children over the last two decades, which has in turn led to a higher prevalence in children and young adults but not in the older adult population.

Rates of atopic diseases were significantly increased in the peanut-allergic population compared with matched controls, with asthma affecting 31% of the cases compared to 7% of the controls; eczema affecting 29% of the cases compared to 9% of the controls and allergic rhinitis affecting 8% of the cases compared to 3% of the controls. This is illustrative of the multitude of atopic diseases that are frequently associated with PA [27].

As with other studies, the incidence of PA was most common in males and in younger age groups. The most common age of onset reported in the literature is 14–24 months; however, our median age of peanut diagnosis was 13 years old. We would like to clarify that this is not age of onset and is age of first electronic recording of peanut allergy. The mean age of diagnosis was reported as 17 years of age, we believe that this may be due to young adults moving away from the family home and registering with a new practice, therefore, reporting their peanut allergy to the GP. Although most allergies are identified in childhood, anaphylaxis occurs mainly in young adults, as reflected in the age of our anaphylaxis cases. The median age of the first anaphylaxis event was 17 years old. This is unsurprising if it is assumed that parental vigilance is higher in the younger age groups and teenagers may be less vigilant about peanut avoidance. There were slightly more anaphylaxis events in males than females (99 versus 79, respectively) with males experiencing their first anaphylaxis at a younger age than females (medians of 13 and 19 years old, respectively). This is resonant with similar patterns reported in another study, where hospitalizations for anaphylaxis were more common in younger males than in females

[28]. Whilst rare, the overall incidence of anaphylaxis increased over-time as is shown in [Figures 4 and 5](#). This was due predominantly to an increase in the prevalence of PA, rather than an increased incidence in anaphylaxis in the PA population. Rates of anaphylaxis in this study are extremely low which may indicate underreporting or potential miscoding. Due to the lack of granularity in the accident and emergency dataset, it was not possible to exclusively identify anaphylaxis events from the diagnosis code 'Allergy (including anaphylaxis)'. The absence of coding anaphylaxis in ICD-10 has been recognized in a previous study and there have been promising improvements for ICD-11 [29–31].

Estimates of PA prevalence are generally higher based on studies using a proactive methodology such as self-reported studies and specialist center surveys compared to those based on electronically collated medical history [4]. Read codes that are noted in CPRD for PA may be recorded before or after a confirmed diagnosis based on the reporting physician's judgment.

As we were dependent upon a general practitioner diagnosing and recording PA appropriately within CPRD it is possible that some cases of PA were recorded as a generic 'food allergy' rather than using the specific Read code, leading to a lower than expected prevalence. It is not possible to quantify those missed because of this. When reporting the number of anaphylaxis events within the PA patients, we cannot be certain that the event is directly caused by the PA and not related to another allergy.

5. Conclusions

PA prevalence and incidence increased markedly over time and we provide contemporary estimates from a population-based sample, representative of the UK population. PA was associated with other atopic diseases such as asthma, eczema and allergic rhinitis which can cause additional health concerns and burdens for the patients involved. PA affects the younger population and leads to increased risk of anaphylaxis compared to the whole CPRD population. Due to the rise in incidence and prevalence of PA and the probable associated increased health-care utilization and family burden, new treatment methods are warranted to treat people with PA.

Acknowledgments

We would like to thank Amr Radwan, Andrea Vereda & Ben Skeel for their insight and helpful advice.

Funding

This paper was funded by Aimmune.

Declaration of interest

J de Vries is an independent consultant to Aimmune Therapeutics Inc. L Scott, BI Jones, TR Berni, and ER Berni are employed by, and CJ Currie is a director of Pharmatelligence, a research consultancy receiving funding from Aimmune Therapeutics Inc. for the submitted work. The authors have no other relevant affiliations or financial involvement with any organization

or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript apart from those disclosed.

Reviewer disclosures

Peer reviewers on this manuscript have no relevant financial or other relationships to disclose.

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- Tanno LK, Molinari N, Bruel S, et al. Field-testing the new anaphylaxis' classification for the WHO international classification of diseases-11 revision. *Allergy*. 2017 May;72(5):820–826.
- **Shows promising advancements into coding anaphylaxis.**

Appendix

Readcode	Description
SN58200	Peanut allergy
43Y1.00	Peanut RAST test
ICD	Description
L23.6	Allergic contact dermatitis due to food in contact with skin
T78.0	Anaphylactic shock due to adverse food reaction
T78.1	Other adverse food reactions, not elsewhere classified
T78.2	Anaphylactic shock, unspecified
T78.4	Allergy, unspecified
T80.5	Anaphylactic shock due to serum