EXTERNAL SCIENTIFIC REPORT

Literature searches and reviews related to the prevalence of food allergy in Europe

CFT/EFSA/NUTRI/2012/02[1]

University of Portsmouth

ABSTRACT

In 2011, the European Food Safety Authority (EFSA) received a mandate from the Food Safety Authority of Ireland (FSAI) to review the available scientific data on the prevalence of each food allergy in Europe, to derive threshold concentrations for each allergen in foods when possible, and to review the analytical methods available for the detection/quantification of food allergens. This report presents the findings of a series of systematic reviews of the literature related to these aims. Systematic searches of relevant bibliographic databases and the grey literature were conducted, studies were selected for inclusion according to pre-specified criteria, relevant data was extracted from all included studies, and the quality of included studies assessed. The first systematic review examined the literature on the prevalence of food allergy (IgE-mediated and non-IgE mediated) in different regions of the World and in individual European countries for different age groups in relation to each of the following food allergens: milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame. For each of these allergens changes in prevalence trends over time were also examined. Additionally, emerging food allergens in different European countries were identified. Of the 7333 articles identified by the searches, data from 92 studies was included, 52 of which reported on studies conducted within Europe. The second systematic review examined the effects of food processing on the allergenicity of foods in relation to each of the following food allergens: milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame. From 1040 articles identified by the searches, 25 studies were included in this review. The final systematic review examined the evidence regarding the new analytical methods available to analyse/detect the food allergens considered in the previous systematic reviews in processed foods. From 1475 articles identified by the searches, 84 studies were included.

KEY WORDS

food allergy, prevalence, population study, systematic review, allergen, allergenicity

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SUMMARY

In 2011, the European Food Safety Authority (EFSA) received a mandate from the Food Safety Authority of Ireland (FSAI) to review the available scientific data on the prevalence of each food allergy in Europe, to derive threshold concentrations for each allergen in foods when possible, and to review the analytical methods available for the detection/quantification of food allergens. Hence, EFSA commissioned this research project, the objectives of which were to carry out a series of systematic reviews of the literature reviews. This project followed systematic review methodology: systematic searches of relevant bibliographic databases and the grey literature were conducted; studies were selected for inclusion according to pre-specified criteria; relevant data was extracted from all included studies; and the quality of included studies assessed.

The first systematic review examined the literature on the prevalence of food allergy (IgE-mediated and non-IgE mediated) in different regions of the World and in individual European countries for different age groups in relation to each of the following food allergens: milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame. For each of these allergens changes in prevalence trends over time were also examined. Additionally, emerging food allergens in different European countries were identified.

Of the 7333 articles identified by the searches, 92 articles were included in this systematic review, 52 of which reported on studies conducted within Europe, presenting data for the following countries: Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Iceland, Sweden, Italy, Netherlands, Norway, Portugal, Spain, Sweden, Turkey, United Kingdom and Estonia. In the included studies, the prevalence of food allergy was assessed using a variety of methods of diagnosis, and prevalence data has been presented in this report accordingly. Fifty-seven studies utilised questionnaire or interview methods to assess the prevalence of either self-reported allergy and/or clinician-diagnosed allergy. Twenty-five studies presented data on sensitisation to foods, measured by either skin prick testing and/or serum-specific IgE testing. Some studies (27) combined information from self-reports of adverse reactions with the results of skin prick or serum-specific IgE testing to present the prevalence of allergy to a specific food. Only 21 of the included studies utilised food challenges to determine the prevalence of food allergy. Of the included studies, 55 were considered to have utilised a method of diagnosis at high risk of bias, 11 used a sampling method considered to be at high risk of bias (the sampling method was unclear in 16 studies) and seven failed to consider reasons for non-response and/or explore withdrawal/loss-to-follow-up (for 69 studies this was unclear). Worldwide milk/dairy was the most common allergen examined (by 40 European studies and 29 non-European), followed by egg (35 European studies, 26 non-European), fish/shellfish/molluscs (34 European studies, 27 non-European) and peanut (27 European studies, 26 non-European). The least examined allergens were celery (four European studies, one non-European), mustard (one European study) and lupin (no studies).

Although some allergens were widely studied, such as milk, peanut and fish/shellfish/molluscs, the systematic review revealed that there are many gaps in the evidence base for the prevalence of allergies to some individual foods (e.g. lupin and celery). Moreover, there are gaps in the evidence base related to the prevalence of food allergies in specific age groups and countries. An important issue is that many studies focus on the prevalence of self-reported rather than challenge-proven food allergy. Even in studies utilising food challenges there was a huge variety in the approach taken, which hinders comparisons across allergens, age groups and countries. For example, in many studies aspects of the challenge protocol were unclear and several studies utilising food challenges did so as part of an algorithm drawing upon other information (e.g. sensitisation data, symptom reports) to diagnose food allergy and such algorithms differed between studies. Time trends are particularly difficult to describe based upon the current evidence base given the lack of studies utilising similar methodologies with comparable age groups in the same country.

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The second systematic review examined the effects of food processing on the allergenicity of foods in relation to each of the following food allergens: milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame. This review was concerned with studies that used food challenges to assess changes in the allergenicity of foods processed using a wide variety of methods. From 1040 articles identified by the searches, 25 studies were included in this review. The included studies investigated the allergenicity of the following reported allergens: celery (one study), wheat (one study), egg (six studies), hazelnut (two studies), milk and dairy (14 studies) and peanut oil (one study).

The majority of studies focussed on the effect of heat; commonly boiling, roasting or baking. The exceptions were the studies investigating hydrolysis and fractioning of milk for infant milk formulas and one study investigating the effect of maturation time for cheese production for those with allergy to the additive lysozyme (from egg) or milk allergens. There were no included studies investigating the effect of using egg or milk as fining agents within the wine making industry. Additionally, although a large number of studies were carried out on peanut allergy no studies were identified that challenged participants with two forms of peanut, for example raw and roasted. However, we did find one study that investigated the allergenicity of crude versus refined peanut oil.

Most studies utilized a cross-over design where each participant underwent challenge to two forms of the food. The order in which the participants were allocated to the challenge with each type of food was determined randomly for only a small proportion of studies. The remaining cross-over studies used a non-random order, usually because the participants were challenged to the food considered least allergenic first since the studies were designed to investigate whether a diet including extensively heated egg or milk could lead to increased tolerance rather than the effect of processing on allergenicity. In all cases, data was extracted only for those participants who were challenge positive to one or more of the forms of the food being examined. Studies did not tend to include a high proportion of participants with severe allergy. In the large majority of studies that carried out a double-blind placebo-controlled food challenge the challenge procedure (for example the method of masking (and its validity), the method of generating the random sequence, the ratio of active to placebo challenge and the way in which the sequence was concealed from the participants and the study personnel) was not clearly reported.

The evidence suggests that the allergenicity of foods can be altered by food processing. However, although there are trends for certain foods, for example, that extensive heating of egg, milk, celery, and to some extent hazelnut, reduces allergenicity, this reduction will not be experienced by all people with that allergy. The included studies were small and not representative of the wider allergic population. More high quality research is required to determine if certain types of processing increase allergenicity, especially for foods such as peanut where this is suggested by the in vitro research evidence. It would be useful to identify groups of people more likely to tolerate certain types of processed foods, so that more specific diagnostic challenges can be accessed and lead to individualised management strategies.

The final systematic review examined the evidence regarding the new analytical methods available to analyse/detect the food allergens considered in the previous systematic reviews in processed foods. The review set out to include studies investigating extraction and detection of the food/proteins in a food matrix of relevance to the real world setting. Studies investigating food matrixes spiked with allergen were included. From 1475 articles identified by the searches, 84 studies were included.

This review revealed that there are a large number of studies that have investigated the effectiveness of assays for detecting allergens in foods published since 2004. The foods with the most research conducted was tree nuts, followed by peanut, milk and dairy and egg. For most allergens there are tests developed that can detect down to 10µg/ml. However the food matrix used could affect the performance of the extraction processes and assays. There was variability in the types of experiments

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carried out, the format and statistical analysis of the data presented and in specific techniques such as the method of spiking and in the source of extracts used to validate the assay in the studies retrieved for this review. In a large proportion of studies there was a potential high risk of bias for at least one item. There are a range of criteria that could be used to validate assays and ensure that there is consistent quality control across institutions. We focused on the accuracy as determined by the percentage recovery of a spiked sample and the limit of detection of each allergen within a suitable food matrix; this is just one aspect of quality control. The limit of detection reported by some of these studies showed that the values reported by manufacturers are not always achieved in practice. Reasons for variation could be the type of matrix used, for example manufacturers may report the sensitivity of the assay when the allergen standard is diluted in assay buffer rather than being within a complex food matrix. Before funding or adopting an assay and extraction procedure it is recommended that all key quality and validation data are reviewed in accordance with the relevant standards and that each laboratory carry out their own validation experiments to assess the performance of the assay within their specific context. The organisations providing guidance for quality assurance are discussed.

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BACKGROUND

In 2011, the European Food Safety Authority (EFSA) received a mandate from the Food Safety Authority of Ireland (FSAI) to review the available scientific data on the prevalence of each food allergy in Europe, to derive threshold concentrations for each allergen in foods when possible, and to review the analytical methods available for the detection/quantification of food allergens. In order to address this mandate, the EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA) will update its opinion, published in 2004[2], relating to the evaluation of allergenic foods for labelling purposes which provides the scientific basis for the identification of foods, food components and food ingredients which may trigger allergic reactions in susceptible individuals, as well as an overview on the prevalence of food allergy, on the setting of threshold concentrations/minimal eliciting doses for individual food allergens, and on the analytical methods for the detection/quantification of these food allergens in raw and processed foods.

OBJECTIVES

The objectives of the contract resulting from the present procurement procedure are the collection, collation and analysis of published and unpublished data related to:

1. The prevalence of food allergy (IgE-mediated and non-IgE mediated) in different regions of the World (e.g. North America, Canada, Australia and New Zealand) and primarily in individual European countries for different age groups in relation to each of the following food allergens: milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame.

2. The natural history of food allergy to each allergen listed above (changes in prevalence and/or severity with age) and on changes in prevalence trends over time at a population level, whenever available.

3. The most prevalent (emerging) food allergies in different European countries (i.e. food allergens other than those listed above) and changes in sensitisation patterns where known or emerging.

4. The effects of food processing on the allergenicity of foods in relation to each of the following food allergens: milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame; and on the new analytical methods available to analyse/detect these food allergens in processed foods.

To achieve these objectives the contractor should carry out comprehensive literature searches to identify and retrieve all related information/data published in peer-reviewed journals and should make reasonable efforts to identify and retrieve unpublished data. The data retrieved should be further analysed following well-accepted methodologies and criteria in order to identify relevant scientific data. The information should be transferred in a concise way to EFSA including the full list of references used for each single food allergen. References not considered pertinent should be listed and a reasoning why these references were not considered pertinent should be provided, in both raw and processed foods.

[2] EFSA (European Food Safety Authority), 2004. Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission relating to the evaluation of allergenic foods for labelling purposes. The EFSA Journal 32, 1-197

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TERMS OF REFERENCE

This contract was awarded by EFSA to: University of Portsmouth

Contractor: Dr Elizabeth Bartle, University of Portsmouth Higher Education Corporation

Contract title: Literature searches and reviews related to the prevalence of food allergy in Europe.

Contract number: CFT/EFSA/NUTRI/2012/02

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INTRODUCTION AND OBJECTIVES

In order to address the four objectives we have brought together a team of academics with expertise in the field of food allergy research and systematic reviews. The overall approach was a series of systematic reviews of the literature, using the following stages:

Stage 1. Conduct a comprehensive and systematic search of the (published and unpublished) literature to identify all potentially relevant studies.

Stage 2. Screen all identified studies against pre-specified eligibility criteria for their relevance to the objective.

Stage 3. For all included studies, extract data relevant to the objective (using pre-specified data collection forms).

Stage 4. For all included studies, assess the validity of the findings (using pre-specified quality assessment criteria).

Stage 5. Synthesise the results of the included studies (as appropriate) and present the characteristics and findings.

These literature reviews would adhere to the nomenclature for food allergy as specified by the World Allergy Organisation and so will not include non-allergic food hypersensitivity (i.e. where immunologic mechanisms have not been implicated).

The objectives are to carry out systematic literature reviews:

1. on the prevalence of food allergy (IgE-mediated and non-IgE mediated) in different regions of the World (e.g. North America, Canada, Australia and New Zealand) and primarily in individual European countries for different age groups in relation to each of the following food allergens: milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame;

2. and for each allergen listed above to present changes in prevalence trends over time at a population level for specific age groups, whenever available;

3. to identify emerging food allergens in different European countries (i.e. food allergens other than those listed above, where there is a significantly high prevalence) and present the prevalence and changes in prevalence with time, whenever available;

4. (4a) on the effects of food processing on the allergenicity of foods in relation to each of the following food allergens: milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame;

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5. (4b) on the new analytical methods available to analyse/detect these food allergens in processed foods.

The methods and the results for objectives 1-3 are reported in the same section as they share the same search strategy. The methods and the results for objectives 4a and 4b are presented separately as the search strategies and the assessment criteria are distinct.

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1. THE PREVALENCE OF FOOD ALLERGY IN DIFFERENT REGIONS OF THE WORLD AND INDIVIDUAL EUROPEAN COUNTRIES (OBJECTIVES 1-3)

1.1. Materials and Methods

1.1.1. Literature search strategy

1.1.1.1. Bibliographic databases and grey literature searching

We searched the following databases: Web of Science including Social Science Citation Index Expanded (1970-present), Social Sciences Citation Index (1970-present), Conference Proceedings Citation Index Science (1990-present), Book Citation Index Science (2005-present), and PubMed.

Searches of conference proceedings were carried out using the Conference Proceedings Citations Index in which studies reported in the proceedings of a comprehensive range of allergy conferences (including the World Allergy Congress, the Annual meeting of the American Academy of Asthma, Allergy and Immunology and the Congress of the European Academy of Allergy and Clinical Immunology) can be identified.

Grey literature was sought via direct contact with a list of topic experts and examination of the lists of awards made by known funders of research in the field (see Box 1). To ensure thoroughness, a snowball approach was taken, whereby the experts were asked whether they knew of any others working in fields directly related to the objectives whom we should contact.

Box 1. Topic experts and known funders of research in the field.

Dr Katie Allen	Dr Scott Sicherer
Professor S Hasan Arshad	Dr Bodo Niggemann
Professor Peter Burney	Professor Ulrich Wahn
Dr Kirsten Beyer	Professor Jonathan Hourihane
Professor Gideon Lack	Dr Graham Roberts
Dr Montserrat Fernandez Rivas	Professor Susan Prescott
Professor Hugh Sampson	

1.1.1.2. Search terms and Boolean operators

Specific search strategies were tailored for the requirements of each database. In order to identify all relevant articles, no language or date restrictions were employed and searches were not limited by study type. The team evaluated the sensitivity of the search strategy by checking that the search results included studies on this topic known by experts within the field.

In PubMed the terms were searched for in the title and abstract fields and using MeSH terms where appropriate. In Web of Science the terms were searched for in the 'Topic Search' field (which includes title, abstract and keywords). Within groups of terms the terms were combined using OR, the groups of terms themselves were then combined in the following manner: #1 AND #2 AND #3.

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Table 1.1: Search terms for the prevalence of food allergy (objectives 1, 2 and 3)

Topics	Search terms[3]	Search terms for PubMed	Search terms for Web of Science
Group 1. Prevalence			
Prevalence	Prevalence, point prevalence	prevalence[Tiab] OR "point prevalence"[Tiab] OR prevalence[MeSH Terms]	prevalence OR "point prevalence"
Incidence	Incidence, cumulative incidence	incidence[Tiab] OR "cumulative incidence"[Tiab] OR incidence[MeSH Terms]	incidence OR "cumulative incidence"
Natural history	Natural history	"natural history"[tiab] OR ((change[tiab] OR changes[tiab]) AND (severity[tiab] OR prevalence[tiab]) AND time[tiab])	"natural history" OR ((change OR changes) AND (severity OR prevalence) AND time)
Group 2. Food		food[Tiab]	food
Milk and dairy	Milk, lactose, dairy, butter, cream, infant formula, cheese, yoghurt, petit filous, casein, whey	milk[Tiab] OR milk[MeSH Terms] OR lactose[MeSH Terms] OR lactose[Tiab] OR dairy[Tiab] OR butter[Tiab] OR cream[Tiab] OR "infant formula"[Tiab] OR cheese[Tiab] OR yoghurt[Tiab] OR "petit filous"[Tiab] OR casein[Tiab] OR whey[Tiab]	milk OR lactose OR dairy OR butter OR cream OR "infant formula" OR cheese OR yoghurt OR "petit filous" OR casein OR whey
Egg	Egg, eggs	egg[Tiab] OR eggs[Tiab]	egg OR eggs
Cereals	Cereal, gluten, wheat, rye, barley, oats, spelt, kamut	cereals[MeSH Terms] OR cereal[Tiab] OR cereals[Tiab] OR glutens[MeSH Terms] OR glutens[Tiab] OR gluten[Tiab] OR wheat[Tiab] OR rye[Tiab] OR barley[Tiab] OR oats [Tiab] OR oat[Tiab] OR spelt[Tiab] OR kamut[Tiab]	cereal OR cereals OR gluten OR glutens OR wheat OR rye OR barley OR oats OR oat OR spelt OR kamut
Peanut	Peanut, arachis	peanut[Tiab] OR arachis[Tiab]	peanut OR arachis

[3] As indicated in technical offer and updated in light of kick-off meeting (e.g. expanded the range of terms included for specific types of fish and shellfish)

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Topics	Search terms[3]	Search terms for PubMed	Search terms for Web of Science
Nuts	Nut, almond, hazelnut, walnut, cashew, pecan, macadamia, pistachio, beechnut, filbert, tree nuts	nuts[MeSH Terms] OR nuts[Tiab] OR nut[Tiab] OR almond[Tiab] OR almonds[Tiab] OR hazelnut[Tiab] OR hazelnuts[Tiab] OR walnut[Tiab] OR walnuts[Tiab] OR cashew[Tiab] OR cashews[Tiab] OR pecan[Tiab] OR pecans[Tiab] OR macadamia[Tiab] OR macadamias[Tiab] OR pistachio[Tiab] OR pistachios[Tiab] OR beechnut[Tiab] OR beechnuts[Tiab] OR filbert[Tiab] OR filberts[Tiab]	nuts OR nut OR almond OR almonds OR hazelnut OR hazelnuts OR walnut OR walnuts OR cashew OR cashews OR pecan OR pecans OR macadamia OR macadamias OR pistachio OR pistachios OR beechnut OR beechnuts OR filbert OR filberts
Celery	Celery	celery[tiab]	celery
Crustaceans	Crustacean, crab, lobster, shrimp, prawn, crayfish, shellfish, langoustine	crustacean[MeSH Terms] OR crustacea[Tiab] OR crustacean[Tiab] OR crustaceans[Tiab] OR crab[Tiab] OR crabs[Tiab] OR lobster[Tiab] OR lobsters[Tiab] OR shrimp[Tiab] OR shrimps[Tiab] OR prawn[Tiab] OR prawns[Tiab] OR crayfish[Tiab] OR shellfish[MeSH Terms] OR shellfish[Tiab] OR langoustine[Tiab] OR langoustines[Tiab]	crustacea OR crustacean OR crustaceans OR crab OR crabs OR lobster OR lobsters OR shrimp OR shrimps OR prawn OR prawns OR crayfish OR shellfish OR langoustine OR langoustines
Fish	Fish, pollock, carp, cod, mackerel, salmon, tuna, shark, sea bass, swordfish, hake, sole, megrim, sardines, halibut, anchovy, catfish, trout	fishes[MeSH Terms] OR fish[Tiab] OR pollock[Tiab] OR carp[Tiab] OR cod[Tiab] OR mackerel[Tiab] OR salmon[Tiab] OR tuna[Tiab] OR shark[tiab] OR “sea bass”[tiab] OR swordfish[tiab] OR hake[tiab] OR sole[tiab] OR megrim[tiab] OR sardine[tiab] OR sardines[tiab] OR halibut[tiab] OR anchovy[tiab] OR anchovies[tiab] OR catfish[tiab] OR trout[tiab]	fish OR pollock OR carp OR cod OR mackerel OR salmon OR tuna OR shark OR “sea bass” OR swordfish OR hake OR sole OR megrim OR sardine OR sardines OR halibut OR anchovy OR anchovies OR catfish OR trout
Molluscs	Mollusc, oyster, snail, squid, mussels, clams, abalone, octopus, scallop	mollusca[MeSH Terms] OR mollusc[Tiab] OR molluscs[Tiab] OR oyster[Tiab] OR oysters[Tiab] OR snail [Tiab] OR snails[Tiab] OR squid[Tiab] OR mussel[Tiab] OR mussels[Tiab] OR clam[Tiab] OR clams[Tiab] OR abalone[tiab] OR octopus[tiab] OR scallop[tiab] OR scallops[tiab]	mollusc OR molluscs OR oyster OR oysters OR snail OR snails OR squid OR mussel OR mussels OR clam OR clams OR abalone OR octopus OR scallop OR scallops
Soy	Soy, soya, soybean	soy[Tiab] OR soybeans[MeSH Terms] OR soybean[Tiab] OR soybeans[Tiab] OR soya[Tiab]	soy OR soybean OR soybeans OR soya
Lupin	Lupin, lupinus-albus	lupinus[MeSH Terms] OR lupin[Tiab]	lupin
Mustard	Mustard	"mustard plant"[MeSH Terms] OR mustard[Tiab]	mustard

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Topics	Search terms[3]	Search terms for PubMed	Search terms for Web of Science
Sesame	Sesame	sesamum[MeSH Terms] OR "sesame"[Tiab]	sesame
Group 3. Allergy			
Allergy	Hypersensitivity, allergy, immunology, sensitivity, intolerance, anaphylaxis, adverse reaction	hypersensitivity[MeSH Terms] OR hypersensitivity[Tiab] OR allergy[Tiab] OR "allergy and immunology"[MeSH Terms] or immunology[Tiab] OR sensitivity[Tiab] OR intolerance[Tiab] OR anaphylaxis[MeSH Terms] OR anaphylaxis [Tiab] OR “adverse reaction”[Tiab]	hypersensitivity OR allergy OR immunology OR sensitivity OR intolerance OR anaphylaxis OR “adverse reaction”

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1.1.1.3. Management of search results

Search results were managed using reference management software (EndNote) and duplicates removed. Search results were then imported into EPPI Reviewer 4 (systematic review software) prior to screening for relevance. English language versions of articles were obtained via the British Library's document supply service (the British Library holds more than 500,000 articles translated into English). Where articles were not available, translation services were used. Searches were updated prior to data analysis/synthesis.

1.1.1.4. Specific search strategy for identifying articles related to the prevalence of emerging allergens

It was anticipated that many of the articles which report the prevalence of food allergy to common allergens such as peanut and milk, would do so in the context of a larger study that screened participants for adverse reactions to a number of (or, in some cases, to any) foods. Hence, for such studies data was presented for allergens other than those listed in Objective 1. These studies were identified by the search strategy outlined in Section 1.1.1.2. Nevertheless, there may also be some smaller studies which have specifically explored the prevalence of allergens that have the potential to be 'emerging'. Hence, within the main search strategy the term 'food' was included to identify articles which might be reporting the prevalence of allergy to foods other than those specifically listed in Objective 1.

1.1.1.5. Specific search strategy for identifying the clinical reactivity to emerging allergens

For the key emerging allergens identified, we have also reported information on clinical reactivity and reports of severe reactions. If available, this was sourced from challenge data provided within the relevant articles. However, if no challenge data was presented in the prevalence studies (i.e. they present sensitivity data only) we searched for smaller observational studies, particularly case reports of anaphylaxis.

1.1.1.6. Specific search strategy for identifying the prevalence of allergy to any food

In addition to the key objectives, we also sought to summarise the prevalence of allergies to any food. Since this was not part of the original objectives, only those studies already included in the review were identified for screening.

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Table 1.2: Search terms to identify articles related to the clinical reactivity of emerging allergens

Topics	Search terms[4]	Search terms for PubMed	Search terms for Web of Science
Group 1. Clinical reactivity	Anaphylaxis, asthma, oedema, odema.	Anaphylaxis[MeSH Terms] OR anaphylaxis[Tiab] OR asthma[Tiab] OR oedema[Tiab] OR odema[Tiab]	Anaphylaxis OR asthma OR oedema OR odema.
Group 2. Emerging allergens	This will be a list of emerging food allergens identified for objective 3 (with specific search terms as described in Table 1.1).	The search terms provided will be adapted for use in PubMed.	The search terms provided will be adapted for use in Web of Science.
Group 3. Allergy	Hypersensitivity, allergy, immunology, sensitivity, intolerance,	hypersensitivity[MeSH Terms] OR hypersensitivity[Tiab] OR allergy[Tiab] OR "allergy and immunology"[MeSH Terms] or immunology[Tiab] OR sensitivity[Tiab] OR intolerance[Tiab]	hypersensitivity OR allergy OR immunology OR sensitivity OR intolerance OR anaphylaxis OR “adverse reaction”
Group 4. Case reports	Case report, case study, case history	“case report”[Tiab] OR “case study”[Tiab] OR “case history”[Tiab] OR “case reports”[MeSH]	“case report” OR “case study” OR “case history”

1.1.2. Study selection general approach

All identified articles were screened for inclusion in the review as follows. Firstly, the titles and abstracts of all identified articles were screened for potential relevance by one review author (a team approach was taken whereby references were divided amongst the review team for screening). At this stage, articles were excluded if, for example, they were obviously unrelated to the topic of the review (e.g. Diagnostic value of D-dimer in outpatients with suspected deep venous thrombosis receiving oral anticoagulation); the sample was inappropriate for the scope of the review (e.g. Prevalence of soy protein hypersensitivity in cow's milk protein-sensitive children in Korea) or because they did not present primary research (e.g. Gastrointestinal allergy to food: a review). An inclusive approach was taken, whereby if the review author was unsure of the potential relevance of an article it was marked as ‘potentially eligible’. The full-text of all potentially eligible studies was then retrieved and assessed against the criteria outlined in section 1.1.3. If the review author was unsure about the eligibility of the paper for inclusion in the review, the paper was discussed with another review author. Reasons for exclusion were recorded.

1.1.3. Study selection specific approach: objectives 1-3

1.1.3.1. Types of studies

We have included population-based cross-sectional studies and cohort studies examining the prevalence of food allergy (IgE-mediated and non-IgE mediated). To be included all studies must have presented an identifiable point (or period) in time at which the prevalence of food allergy was measured.

[4] As indicated in technical offer and updated in light of kick-off meeting

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1.1.3.2. Types of participants

We included participants of all age groups from any country. Studies that did not present region or country-specific data were excluded from the review. Studies must have been population based, using either a fixed cohort or an appropriate sampling strategy. Studies conducted in a clinical setting (e.g. a survey of the prevalence of specific food allergies in current patients at an allergy clinic) or in selected patient groups (e.g. measuring the prevalence of food allergy in patients with asthma) were excluded since they do not provide information about the general prevalence of food allergies.

1.1.3.3. Types of outcome measure

Objectives 1 and 2 are interested in one outcome - the prevalence of food allergies (IgE and/or non-IgE mediated) to any one of the following allergens: milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame. Objective 3 is interested in the prevalence of food allergies to emerging allergens. Hence, all studies reporting the prevalence of food allergies to specific allergens were eligible for inclusion in the review.

Studies employing at least one of the following methods of diagnosis to determine the prevalence of allergies to one or more of the above food allergens were eligible for inclusion in the review for Objectives 1-3:

- Self-reported food allergy
- Clinical history of adverse reactions to foods and positive SPT (for IgE-mediated food allergy)
- Clinical history of adverse reactions to foods and positive serum-specific IgE (for IgE-mediated food allergy)
- Clinical history of adverse reactions to foods and positive food challenge (open or double-blind placebo-controlled: for IgE and non-IgE mediated food allergy, allowing for delayed reactions in the case of non-IgE mediated food allergy)

Studies which presented data regarding sensitisation as determined by the following methods were also eligible for inclusion in the review for Objective 1:

- Positive SPT
- Positive serum-specific IgE

Studies that did not present separate prevalence data for individual allergens were excluded from the review.

1.1.4. Study selection specific approach for identifying the prevalence of allergy to any food

All included studies were screened for the inclusion of data for the prevalence of allergy to any food. The methods and outcome measures used to identify this data needed to meet the criteria outlined for Objectives 1-3 (Section 1.1.3) to be eligible for inclusion in the review.

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1.1.5. Data collection general approach

As described in the technical offer, data extraction and management was facilitated by the EPPI Reviewer software (EPPI Centre, 2011), which has been developed to aid the management of systematic reviews. The software facilitates the following activities: reference management, study classification/screening, data extraction and retrieval, collaborative working (i.e. allocation of screening and comparison of screening decisions), data analysis and reporting.

As has been piloted for articles related to the prevalence of peanut allergy, we used data collection forms developed in EPPI Reviewer to extract relevant data for objectives 1-3 according to predetermined criteria. The following was extracted for **all** included studies:

1. General information: Authors' contact details, research funder, year(s) study conducted, country/countries in which conducted.
2. Methods: Study design (cross-sectional or cohort study, and for cohort studies additional information regarding at what ages articles have reported), type of food allergy considered (IgE mediated, non-IgE mediated or both), food(s) assessed (including potential emerging allergens), method of diagnosis (to include additional information with regard to the procedure, e.g. whether extracts or prick-to-prick method has been used for skin prick testing), sampling strategy (e.g. local or general population, random or non-random) and sample characteristics (e.g. age group, ethnic background, response rate, withdrawal).
3. Outcomes [for ease of reporting, this data has been recorded in a Microsoft Excel spread sheet]: Information on reported outcomes and relevant data (percentage prevalence, raw data and confidence intervals; presented by allergen, year of study, method of diagnosis and age).

Additional information was collected if reported by a study, as follows:

- Where a study has reported the prevalence of sensitisation to a food (indicated by either a positive skin prick test or serum-specific IgE test), and where relevant (e.g. in the case of wheat and grass) and reported by the study, data was recorded regarding cross-reactivity. Where such data was relevant but not reported, this was also recorded.
- Objective 3 (emerging allergens): Where studies have been sought which provide evidence regarding the clinical reactivity of emerging allergens, information regarding the nature of reactions reported was extracted. This included information regarding the symptoms of the reaction, the time between ingestion and reaction and the treatment required.

Where there was ambiguity in the reporting of results, all efforts were made within the given timeframe to contact the study authors to provide additional information.

Upon completion of data collection, those studies included in the review were exported from EPPI Reviewer into EndNote reference management software. Where available in electronic format (and when compliant with copyright and data sharing rules), the full-text of articles not currently accessible within EFSA's current subscriptions have been provided within the EndNote file.

1.1.6. Data collection specific approach for emerging allergens (Objective 3)

Objective 3 is interested in the prevalence of allergies (IgE and/or non-IgE mediated) to any emerging allergens. Emerging allergens have been defined as any allergen other than: milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame that has either increasing prevalence or was reported to have a significant prevalence in at least one country in Europe.

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It was anticipated that articles which report the prevalence of food allergy to common allergens such as peanut and milk, do so in the context of a larger study which has screened participants for adverse reactions to a number of (or, in some cases, to any) foods. Hence, for such studies data has often been presented for allergens other than those listed in Objective 1. Additional studies may also have been identified which have examined the prevalence of less common allergens that have the potential to be defined as 'emerging'. Data from such studies have been included. All studies have been screened on the criteria outlined in Section 1.1.3. and data has been collected in accordance with section 1.1.4. Prevalence data has been extracted for all foods reported in a report of a study, in order to identify those allergens which may be considered 'emerging'.

1.1.7. Data collection specific approach for allergy to any food

In addition to the key objectives, data has also been collected and reported related to the prevalence of allergies to any food. This data was collected only from, and in the same manner as, the studies included within the systematic reviews conducted for objectives 1-3.

1.1.8. Assessing the quality of included studies

Studies were assessed as being at low or high risk of bias on the basis of three quality criteria (Table 1.3). The first related to the risk of bias of the diagnostic method employed by the study. In studies utilising more than one method of diagnosis, the risk of bias of the highest quality method was judged. The second criterion related to the method of sampling, in particular, whether the sample utilised the whole population (for example, all consecutive births), a random sample or a non-random sample. The third criterion related to whether the study had explored the reasons for non-response (in cross-sectional studies) or withdrawal/loss o follow-up (in cohort studies).

Table 1.3: Quality assessment criteria

Quality assessment criteria	Diagnostic method	Sampling strategy: method	Reasons for non-response or withdrawal/loss to follow-up
Low risk of bias	• Food challenges (open or double-blind) with or without clinical history • Sensitisation (skin prick test and/or serum-specific IgE) with clinical history	• Whole population • Random	Yes
High risk of bias	• Sensitisation (skin prick test and/or serum specific IgE) without clinical history • Clinical history alone • Clinician diagnosed • Self-report	• Non-random	No

1.1.9. Data synthesis and presentation

1.1.9.1. General approach

Our general approach to the synthesis of data was as follows. For all objectives a narrative approach was taken, presenting data in tables reporting the mean and, where possible, the confidence intervals. Confidence intervals were calculated for proportions using Wilson's correction for continuity. Where

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raw data was not presented in the article, confidence intervals have been presented as per the article or marked 'unknown' if not reported.

Europe has been defined geographically rather than by membership of the European Union. Key characteristics of the included studies have been presented (Table 1.4) , including (but not limited to) information about study design (e.g. cohort study), country studied, allergens assessed and the method of diagnosis. Information has also been presented regarding the quality of the evidence (Table 1.6).

1.1.9.2. Objectives 1 and 3

As described in the technical offer, in addition to the approach described above, for Objective 1 and 3, for each allergen we have presented a table which maps the data (percentage prevalence and 95% confidence intervals, where possible) according to country and then by age (this has been grouped however is meaningful dependent upon the approach taken by the included studies). The prevalence data has been presented by method of diagnosis, and information has also been included on the year, country and age group for which data is being presented, and on whether the study assessed IgE-mediated allergy, non-IgE-mediated allergy or both (it is important to note that this was assessed across the whole study rather than by individual food; where a study provides only self-report data and has not distinguished between symptoms typical of either IgE and non-IgE mediated reactions this has been classified as examining both IgE and non-IgE mediated allergies although it has been noted that the presence or absence of IgE was not tested for). Data has additionally been narratively reported for the prevalence of allergy to any food both across Europe and for countries outside of Europe.

1.1.9.3. Objective 2

In addition to the general approach, for each listed allergen (milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame) we have provided a narrative summary of changes in prevalence over time. We have discussed this by country and age group.

1.2. Results

1.2.1. Results of the search

After removal of duplicates 7323 references were identified with a further ten papers identified through the expert panel thereby totalling 7333. Of these 7145 were excluded based on the title and abstract. The full-text was obtained for 187 references (the full text could not be obtained for Wang 1990). After full text screening a further 99 studies were excluded. The flow chart and the reasons for exclusion are outlined in Figure 1.1. One of the most common reasons for exclusion was that the article reported data that was reproduced in another included paper, for example a conference abstract subsequently presented in a full journal article or a report of a subset of a population that was reported in full in another paper. Another reason for exclusion was that the study utilised an unsuitable design such as case-control or a case series within a clinic setting as the samples would not be representative of the general population. The excluded studies are presented in Table 1.33. After screening the full text 89 studies were included in the final systematic review (Figure 1.1).

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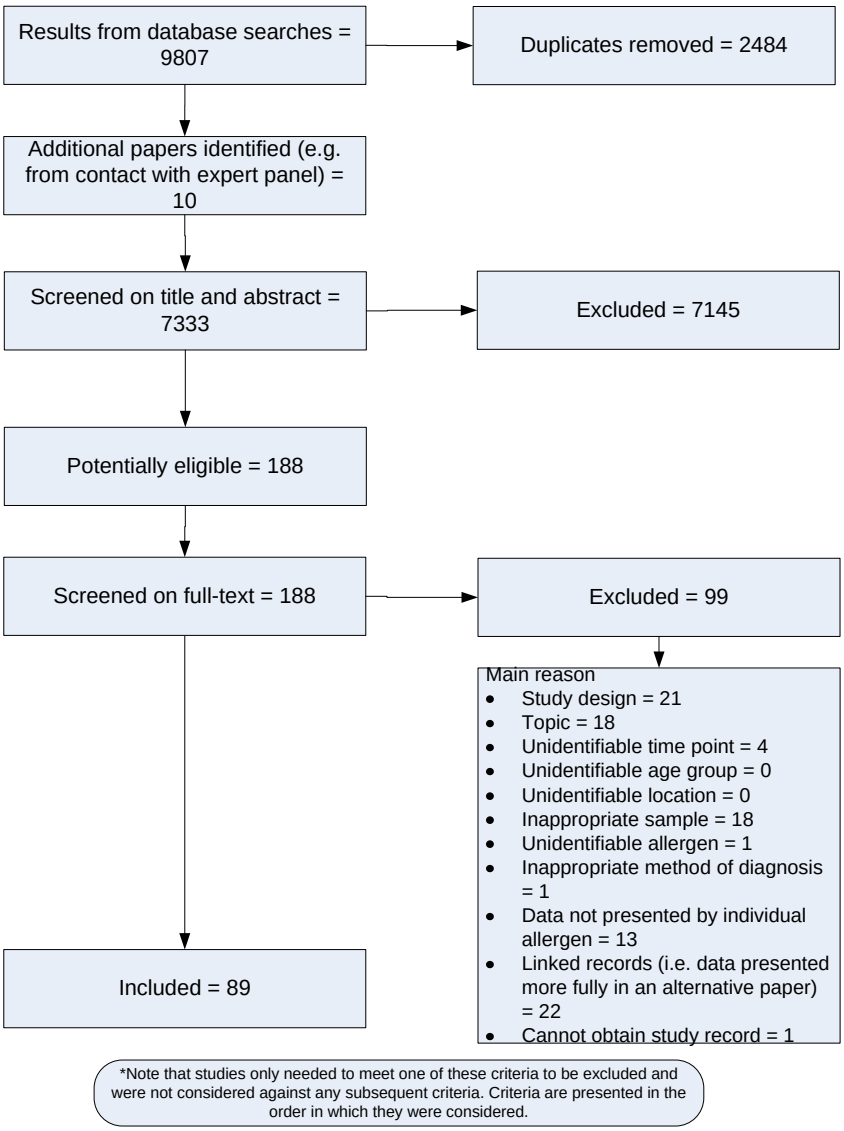

Figure 1.1.: Flowchart of search results and screening for all studies.

1.2.2. Included studies

We included 92 articles, 52 of which were conducted within Europe. Of these, five were based in Denmark, one in Estonia, three in Finland, three in France, three in Germany, two in Greece, one in Hungary, one in Iceland and Sweden combined, two in Italy, one in the Netherlands, two in Norway, one in Portugal, one in Spain, four in Sweden, six in Turkey, thirteen in the United Kingdom, and lastly one in Estonia (Table 1.4).

Of the 40 studies conducted outside of Europe, one was conducted in West Africa (Ghana), ten in Eastern Asia (China, Korea, Hong Kong, Japan, and Taiwan), one in South-Central Asia (India), four in South-East Asia (Philippines, Singapore, Thailand), two in the Middle East (Israel, United Arab Emirates), 18 in North America (Canada, USA), one in North-West South America (Colombia) and three in Australia. The key characteristics of these studies are shown for each country in alphabetical order (Table 1.4)

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The majority of studies (66) employed a cross-sectional design and 25 used a cohort design. Further information about the included studies are presented in a series of tables.The method of identifying food allergy is outlined in Table 1.4 and additional tables provide further information about the method utilised for questionnaire or interview based approaches (Table 1.7), sensitisation testing (Table 1.8), and food challenge (Table 1.9). Some studies presented the findings for more than one method of identification enabling comparison of methods as exemplified by Schäfer 2001 in Germany, Mustafayev 2010 in Turkey, Venter 2006 and Nicolaou 2010 in the UK and Woods 2002 in Australia. Many studies reported using a combination of methods within an algorithm; almost without exception this two or three step process was applied to food challenges where only those who either self-reported food allergy in a questionnaire or who had a positive clinical history were challenged.

Questionnaire or interview methods for assessing suspected food allergy were presented in 57 studies. The sensitivity and specificity of these questionnaire-based methods was not available for some of the studies (for example, Murrugo 2008 used a ten item questionnaire with no reference to validation) whereas some studies used tools that had undergone some pretesting (such as Ben Shoshan 2010, Sicherer 1999, 2002 and 2010 and others such as Martinez-Gimeno 2000 who used tools that had undergone rigorous validation ref http://isaac.auckland.ac.nz/, Table 1.7). Although we provide data under the headings self-reported, clinician diagnosed and clinical history it should be noted that there is overlap between these identification methods as some self-report questionnaires include questions on 'do you have doctor diagnosed allergies' and some of the 'clinical histories' were collected using a structured format questionnaire.

The IgE sensitisation of the entire study population was assessed using skin prick test for 19 studies and serum specific IgE in eight. In total 25 studies used either or both methods. Rates of sensitisation were consistently higher than rates of prevalence of food allergy. For example, Woods (2002) tested sensitisation to milk using a skin prick test and found sensitisation of 0.7% (95% CI: 0.2-2.1), but when this was combined with clinical history the rate was 0.0% (95% CI: 0.0-1.0); when testing for peanut sensitisation using SPT, Grundy 2002 reported a rate of 3.3% (95% CI: 2.4-4.5), but this fell to 0.7% (95% CI: 0.3-1.3) upon food challenge. Although sensitisation to food allergens has poor specificity for food allergy this measure does allow for comparisons between countries and over time.

Twenty-seven studies reported data on the prevalence of food allergy as determined by combining sensitisation data from the whole study population with self-reports of allergy, for example Tariq 1996, Orhan 2009 and Ostblom 2008a. In contrast, oral food challenges were usually carried out on a subset of the study population who reported allergy to a particular food or foods (via a questionnaire or clinical interview) and/or were sensitised to a specific food allergen (determined by SPT or SIgE). It is important to note, however, that in the majority of studies utilising food challenges, a subset of participants (typically individuals with a convincing clinical history of severe reactions, and clear elevated specific IgE and or skin prick test) were not challenged since it is unethical to do so. This aligns with the management of patients in practice, and these individuals were typically considered to be allergic and, for prevalence calculations, had been counted alongside those who experienced a positive oral food challenge.

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Table 1.4: Key characteristics of included studies

Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
Al-Hammadi (2010)	Cross-sectional study	2006	United Arab Emirates	6-9 years	Main list: Cereal (wheat), Eggs, Fish, Milk/dairy, Peanuts, Tree nuts Additional food(s) Fruit and/or vegetables	Both IgE and non-IgE	• Clinician diagnosed	7 years (±1.06)	Not reported	397
Altintas (1995)	Cross-sectional study	1992-1993	Turkey; Adana	Newborn	Main List: Eggs, Milk/dairy, (cow's milk)	Both IgE and non-IgE	•Clinician diagnosed	Not reported	0-2 years	1700
Arbes (2005)	Cross-sectional study	1988-1994	United States	All ages	Main list: Peanuts	IgE- only	• Positive skin prick test without clinical history	Not reported	6-19 years	10508
Arshad (2001)	Cohort study	1993-1994	United Kingdom; Isle of Wight	4 years	Main list: Cereals (wheat), Eggs, Fish (cod), Milk/dairy, Soy	IgE- only	• Positive skin prick test without clinical history	Not reported	4 years	981
Babu (2008)	Cross-sectional study	Not reported	India	5-60 years	Additional food(s): Eggplant, Aubergine	IgE only	• Self-report • Positive skin prick test without clinical history • Positive serum-specific IgE with clinical history	Mean 35.6 years (± 17.0)	Not reported	741

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
Bakos (2006)	Cross-sectional study	2004	Hungary	Elderly people mean age of 77 years	Main list: Celery, Cereals (wheat, rye) Crustaceans (crab), Eggs (egg yolk and egg white), Fish (cod), Milk/dairy (milk, casein), Peanuts, Sesame, Soy, Tree nuts (hazelnut, walnut, almond) Additional food(s): Apple, Banana, Carrot Orange, Potato, Tomato	IgE- only	• Positive skin prick test without clinical history • Positive serum-specific IgE without clinical history	Mean 77 years (± 9.3)	20-97 years	109
Ben-Shoshan (2009)	Cross-sectional study	2005-2007	Canada	5-9 years	Main list: Peanuts	IgE- only	• Other	Mean 7.1 years	5-9 years	5161
Ben-Shoshan (2010)	Cross-sectional study	2008-2009	Canada	All ages	Main list: Crustaceans, Fish, Peanuts, Sesame, Tree nuts	Both IgE and non-IgE-mediated	• Self-report • Clinician diagnosed • Clinical history	Not reported	Not reported	9667
Bjornsson (1996)	Cross-sectional study	1991-1992	Sweden	20-44 years	Main list: Cereals (wheat), Eggs Fish, Milk/dairy Peanuts, Soy	IgE- only	• Positive serum-specific IgE without clinical history	Not reported	20-44 years	1397
Bock (1987)	Cohort study	1980 - 1981	United States	< 3years	Main list: Cereals (corn, rice, wheat), Eggs, Milk/dairy, Peanuts, Soy Additional food(s): Chocolate	Both IgE and non-IgE	• Self-report • Other	Not reported	Birth-3 years	480

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
Branum (2009)	Cross-sectional study	2005-2006	United States	< 18 years	Main list: Crustaceans (shrimp), Eggs, Milk/dairy, Peanuts	IgE- only	• Positive serum-specific IgE without clinical history	Not reported	Not reported	3500
Brugman (1998)	Cross-sectional study	1993-1994	Netherlands	4-15 years	Main list: Fish, Crustaceans Milk/dairy, (cow's milk, Soy, Tree nuts, Peanuts Additional food(s): Additives and Colourings, Apple juice, Banana, Chocolate, Lemonade, Mayonnaise, Pork, Strawberry, Sugar, Tomato	Both IgE and non-IgE	• Self-report	Not reported	4-15 years	4400
Chen (2011)	Cross-sectional study	2009	China	<12 months	Main list: Cereals (wheat), Crustaceans (shrimp), Eggs (yolk and white), Fish, Milk/dairy, Peanuts, Soy Additional food(s): Carrot, Orange	IgE- only	• Positive skin prick test without clinical history • Positive open food challenge with clinical history	Not reported	0-12 months	497
Chen (2012)	Cross-sectional study	2009-2010	China	<2years	Main list: Eggs, Milk/dairy	Both IgE and non-IgE	• Other	Not reported	0-2 years	573
Connett (2012)	Cross-sectional study	2007-2008	Philippines Singapore Thailand	14-16 years	Main list: Fish	Both IgE and non-IgE	• Self-report • Clinical history	Not reported	14-16 years	19966

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								Age Mean (SD)	Age (Range)	Sample size
Dalal (2002)	Cross-sectional study	Not reported	Israel	<2years	Main list: Eggs, Fish, Milk/dairy Peanuts, Sesame, Soy, Tree nuts Additional food(s) Beef, Chicken, Chocolate, Garlic, Strawberry, Tomato	IgE- only	• Clinical history • Positive skin prick test with clinical history	Not reported	0-2years	9070
Eggesbo (1999)	Cohort study 2 maternity clinics in Oslo	1992-1993; 1993-1995	Norway	<24 months	Main list: Cereals, Eggs, Fish Milk/dairy, Peanuts Additional food(s) Chocolate Fruit and/or Vegetables	Both IgE and non-IgE	• Self-report	Not reported	Not reported	3366
Eller (2009)	Cohort study	1998-2005	Denmark	Followed up birth cohort at 3, 6, 9, 12, 18, 36 and 72 months of age	Main list: Eggs, Milk/dairy, Peanuts	Both IgE and non-IgE	• Positive open food challenge with clinical history	Not applicable (cohort study following up at defined ages)	Not applicable (cohort study following up at defined ages)	unknown
Emmett (1999)	Cross-sectional study	1995-1996	United Kingdom	15+ years	Main list: Cereals (wheat, flour, gluten), Eggs, Fish, Milk/dairy, Peanuts, Sesame, Soy, Tree nuts Additional food(s) Cheese, Chocolate, Fruit and/or Vegetables, Pulses	Both IgE and non-IgE	• Self-report • Clinical history	Not reported	Not reported	16420 (stage 1), 1253 (stage 2)

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								Age Mean (SD)	Age (Range)	Sample size
Falcao (2004)	Cross-sectional study	Not reported possibly 2000	Portugal	>39 years	Main list: Eggs, Fish, Milk/dairy Molluscs, (squid, octopus) Additional food(s): Chocolate, Kiwi, Meat (sausages, pork), Strawberry	Both IgE and non-IgE	• Self-report	Not reported	Not reported	659
Frongia (2005)	Cross-sectional	2003	Italy	12-24 months	Eggs, Milk/dairy	Both IgE and non-IgE	• Clinician diagnosed	Mean 18.5 years	12-24 months	4602
Gelincik (2008)	Cross-sectional study	Not reported (published in 2008)	Turkey	18+ years	Main list: Eggs, Hens, Milk/dairy Tree nuts Additional food(s): Banana, Chocolate, Eggplant, Garlic, Grape Mushroom, Peach, Pickle, Seafood, Spices, Strawberry, Tomato	Both IgE and non-IgE	• Self-report • Positive skin prick test with clinical history • Positive serum-specific IgE with clinical history • Positive DBPCFC with clinical history	Not reported	18+ years	11816
Gerrard (1973)	Cross-sectional study	Not reported	Canada	6-36 months	Main list: Milk/dairy	Both IgE and non-IgE	• Clinical history	Not reported	6-36 months	803
Greenhawt (2009)	Cross-sectional study	Not reported	United States	18+ years	Main list: Cereals (wheat), Eggs Fish, Milk/dairy, Peanuts Soy, Tree nuts Additional food(s): shellfish	IgE and non_IgE (but unclear)	• Self-report	Not reported	18+ years	513

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								Age Mean (SD)	Age (Range)	Sample size
Grundy (2002)	Cohort study	1999-2000	United Kingdom	3-4 years	Main list: Peanuts	IgE- only	• Self report • Positive skin prick test without clinical history • Positive open food challenge with clinical history	Mean 3.2 years	3-4 years	1246
Gupta (2011)	Cross-sectional study	2009-2010	United States	<18 years	Main list: Cereals (wheat), Crustaceans, Eggs, Fish Milk/dairy, Peanuts, Soy, Tree nuts Additional food(s): Strawberry	Both IgE and non-IgE	• Clinical history	Mean 8.5 years	0-17 years	10514
Haahtela (1980)	Cross-sectional study	Not reported	Finland	15-17 years	Main list: Fish	IgE- only	• Positive skin prick test without clinical history	Not reported	15-17 years	708
Host (2002)	Cohort	1985-2000	Denmark	0-15 years	Main list: Cow's milk	Both IgE and non-IgE	• Clinical history • Positive open food challenge with clinical history	Not reported	0-15 years	1749
Hourihane (2007)	Cross-sectional study	2003-2005	United Kingdom	3-6 years	Main list: Peanuts	IgE- only	• Positive skin prick test without clinical history • Positive	Not reported	3-6 years	1072

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
							DBPCFC with clinical history			
Hu (2010)	Cross-sectional study	1999 2009 (2 cross sectional studies)	China	<24 months	Main list: Cereals (wheat), Crustaceans (shrimp), Eggs, Fish, Milk/dairy Peanuts, Soy Additional food(s): Orange	Both IgE and non-IgE	• Positive skin prick test with clinical history • Positive open food challenge with clinical history	Not reported	0-24 months	382
Isolauri (2004)	Cross-sectional study	1990-1997	Finland	7, 27, 47 and 67 year olds	Main list: Cereals (Wheat), Eggs, Fish (cod), Milk,	IgE- only	• Positive serum-specific IgE without clinical history	Not reported	7-67 years	400
Julge (2001)	Cohort study	1994-1999	Sweden Estonia	<5 years	Main list: Eggs, Milk/dairy	IgE- only	• Positive skin prick test without clinical history • Positive serum-specific IgE without clinical history	Not reported	0-5years	222
Kagan (2003)	Cross-sectional study	2000-2002	Canada	5-9 years	Main list: Peanuts	IgE- only	• Other	Mean 7.4 (±1.2)	5-9 years	4339
Kajosaari (1982)	Cross-sectional study	1980-1981	Finland	1,2,3 and 6 years	Main list: Cereals (wheat), Eggs, Fish, Milk/dairy, Tree nuts Additional food(s) Apple, Chocolate, Citrus, Pea, Strawberry, Tomato	Both – not clearly specififed	• Self-report • Positive open food challenge with clinical history	Not reported	1-6 years	261

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
Katz (2010)	Cohort	2004-2006	Israel	0-2 years	Main list: Cow's milk	Both IgE and non-IgE	• Self-report • Positive open food challenge with clinical history • Positive skin prick test with clinical history	Not reported	0-2 years	13019
Keet (2012)	Cross-sectional study	2005-2006	United States	1-21 years	Main list: Eggs, Milk/dairy, Peanuts	IgE- only	• Positive serum-specific IgE without clinical history	Not reported	1-21 years	3550
Kilgallen (1996)	Cross-sectional study	Not reported	United Kingdom	<48 months	Main list: Eggs, Milk/dairy (milk and milk products) Additional food(s): Additives and colourings, Artificial colourings and e-numbers (sweets, soft drinks), Yoghurt	Both IgE and non-IgE	• Self-report	Not reported	0-48 months	600
Kim (2011)	Cohort study	2006-2007	Korea	<12 months	Main list: Crustaceans, Seafood Eggs, Milk/dairy, Soy Tree nuts, Peanuts Additional food(s): Fruit and/or Vegetables	IgE- only	• Clinician diagnosed	Not reported	0-12 months	1177
Krause (2002)	Cross-sectional study	1998	Greenland	5-18 years	Main list: Cereals (Wheat), Eggs, Fish, Milk/dairy, Peanuts, Soy	IgE- only	• Positive serum-specific IgE with clinical history	Not reported	7-15 years	1031

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								Age Mean (SD)	Age (Range)	Sample size
Kristjansson (1999)	Cross-sectional study	1994 - 1995	Iceland Sweden	18 months	Main list: Cereals, Crustaceans, Eggs, Fish, Milk/dairy, Peanuts, Soy, Tree nuts Additional food(s): Apple, Banana, Carrot, Cherry, Chicken, Chocolate, Lemon, Orange, Pea, Plum, Tomato	IgE- only	• Self report • Positive skin prick test with clinical history	Mean Icelandic children 18.8 years Mean Swedish children 19.3 years	18- 19 months	328
Kucukosmanoglu (2008 a)	Cross-sectional study	2002-2004	Turkey	8-18 months	Main list: Eggs	IgE- only	• Positive skin prick test without clinical history	Median12 months	8-18 months	1015
Kucukosmanoglu (2008 b)	Cohort study	2002 - 2003	Turkey	8- 18 months	Main list: Milk/dairy	IgE- only	• Positive skin prick test without clinical history • Positive serum-specific IgE with clinical history • Positive open food challenge with clinical history	Mean 12.5 months (± 2.5)	Not reported	1015
Kumar (2011)	Cohort study	Not reported	United States	Recruited 24 to 72 hours after birth	Main list: Eggs, Milk/dairy, Peanuts	IgE- only	• Positive serum-specific IgE without clinical history	Not reported	0-6 years	1104

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
				schedule d visits at 6-12 months, 2, 4 and 6 years						
Lack (2003)	Cohort study	1997-1998	United Kingdom	<38 months	Main list: Peanuts	IgE- only	• Clinical history • Positive skin prick test with clinical history • Positive DBPCFC with clinical history	Not reported	0-38 months	12090
Lao-araya (2012)	Cross-sectional study	2010	Northern Thailand	3-7 years	Main list: Cereals (wheat) Crustaceans (shrimp, crab),Eggs, Fish, Milk/dairy Molluscs, (squid, mollusc not specified) Additional food(s) Ant eggs, Beef, Chocolate, Coconut, Insect	IgE- only	• Self-report • Positive open food challenge without clinical history	Mean 5.3 (± 1.0)	3-7 years	452
Leung (2009)	Cross-sectional study	2006-2007	Hong Kong	2-7 years	Main list: Crustaceans, Eggs, Fish, Milk/dairy, Peanuts, Tree nuts Additional food(s): Beef, Chocolate, Citrus Fruit and/or Vegetables,	Both IgE and non-IgE	• Self-report • Other	Not reported	2-7 years	3677

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
					Orange, Banana, Lamb, Tomato					
Liu (2010)	Cross-sectional study	2005-2006	United States	All ages	Main list: Crustaceans (shrimp), Eggs (egg white), Milk/dairy, Peanuts	IgE- only	• Positive serum-specific IgE without clinical history • Other	Not reported	Not reported	8203
Marrugo (2008)	Cross-sectional study	Not reported	Colombia	1-83 years	Main list: Eggs, Milk/dairy Additional food(s): Additives and colourings, Alcohol, Fruit and/or vegetables, Meat, Seafood	Both IgE and non-IgE	• Self-report	Not reported	1-83 years	3099
Martinez-Gimeno (2000)	Cross-sectional study	Not reported	Spain	6-13 years	Main list: Eggs, Fish, Milk/dairy, Peanuts Additional food(s) Fruits, legumes	Both IgE and non-IgE	• Self-report	Not reported	6-13 years	5163
Morita (2012)	Cross-sectional study	2009 - 2010	Japan	Adults	Main list: Cereals (wheat)	IgE only	• Self-report • Positive skin prick test with clinical history • Positive serum-specific IgE with clinical history • Positive serum-specific IgE without clinical history	Not reported	24-93 years	935

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
Mortz (2005)	Cohort study	1995-1996	Denmark	14 years	Main list: Peanuts	IgE- only	• Positive skin prick test without clinical history • Positive serum-specific IgE without clinical history • Positive open food challenge with clinical history	Mean 14.1 years	14 years	862
Mustafayev (2012)	Cross-sectional study	2010	Turkey	10-11 years	Main list: Eggs, Fish, Milk/dairy Cow's milk, Peanuts, Tree nuts (pistachio, walnut, hazelnut)	IgE- only	• Self-report • Positive skin prick test without clinical history • Positive open food challenge with clinical history	Not reported	10-11 years	6963
Nicolaou (2010)	Cohort study	2003	United Kingdom	8 years	Main list: Peanuts	IgE- only	• Positive skin prick test without clinical history • Positive serum-specific IgE without clinical history • Other	Not reported	8 years	1029

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
Obeng (2011)	Cross-sectional study	2006-2008	Ghana	5-16 years	Main list: Cereals (wheat) Crustaceans (shrimp) Eggs, Fish, Milk/dairy Peanuts, Soy Additional food(s): Apple, Avocado, Banana, Beans, Carrot, Cassava, Coconut, Cocoyam, Corn, Kontomire, Mango, Melon, Millet, Okro, Orange, Palm nut, Pawpaw, Pineapple, Potato, Nutmeg, Rice, Sorghum, Sweet potato, Water yam	Both IgE and non-IgE	• Self-report	Not reported	5-16 years	1431
Oh (2004)	Cross-sectional study	1995-2000	Korea	6-12 years and 12-15 years	Main list: Cereals (wheat), Eggs, Fish, Milk/dairy (cow's milk), Peanuts, Soy Additional food(s): Apple, Banana, Beef, Buckwheat, Chicken, Peach, Pork, Seafood, Tomato	Both IgE and non-IgE	• Self-report	Not reported	6-12 years and 12-15 years	27425
Orhan (2009)	Cross-sectional study	2006	Turkey	6-9 years	Main list: Eggs, Fish, Milk/dairy, Peanuts, Tree nuts (hazelnut, walnut) Additional food(s): Banana, Beef, Black Pepper, Chickpea, Cocoa,	IgE only	• Self-report • Positive skin prick test with clinical history • Positive DBPCFC with clinical history	Not reported	6-9 years	2739

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
					Corn, Kiwi, Potato, Strawberry, Tomato					
Osborne (2011)	Cohort study	2007-2010	Australia	11-15 months	Main list: Crustaceans, Eggs, Milk/dairy, Peanuts, Sesame	Both IgE and non-IgE	• Self-report • Clinical history • Positive skin prick test without clinical history	Not reported	11-15 months	2768
Ostblom (2008 a)	Cohort study	1999-2000	Sweden	4 years	Main list: Cereals (wheat), Eggs (egg white), Fish (cod), Milk/dairy, Peanuts, Soy Additional food(s): Banana, Chocolate, Citrus, Pea, Stone fruit	Both IgE and non-IgE	• Self-report • Positive serum-specific IgE with clinical history • Positive serum-specific IgE without clinical history	Not reported	4 years	2563
Ostblom (2008 b)	Cohort study	1995-2004	Sweden	1, 2, 4 and 8 years (same cohort)	Main list: Cereals (wheat),Eggs, Fish, Milk/dairy, Peanuts, Soy, Tree nuts	Both IgE and non-IgE	• Self-report • Clinician diagnosed	Not reported	1, 2, 4 and 8 years	3104
Osterballe (2005)	Cohort study	2001-2002	Denmark	Group 1: 3 years, Group 2: <3 years, Group 3: Children > 3 years,	Main list: Cereals (wheat), Crustaceans (shrimp), Eggs, Fish (codfish), Milk/dairy, Peanuts, Soy Additional food(s): Additives and colourings Fruit and/or V egetables	Both IgE and non-IgE	• Positive open food challenge with clinical history • Positive DBPCFC with clinical history • Other	Group 1,2 and 3 median age 3, 0.7, 7.6 and 33.7 years respectively	Group 1: 3 years, Group 2: 0.1 - 2 years, Group 3: 4-22 years,	936

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
				Group 4: Adults					Group 4: 21-58 years	
Osterballe (2009)	Cohort study	2001-2002	Denmark	22 years	Main list: Cereals (wheat), Crustaceans (shrimp), Eggs, Fish (cod) Milk/dairy, Molluscs, (octopus), Peanuts, Soy Additional food(s): Additives and colourings	Both IgE and non-IgE	• Self-report • Other	Not reported	22 years	843
Pereira (2005)	Cohort study 2 birth cohorts used: 1991-1992 and 1987-1988	2002-2003	United Kingdom	11 and 15 years	Main list: Cereals (wheat), Crustaceans (shellfish), Eggs, Fish, Milk/dairy, Peanuts, Tree nuts Additional food(s): Additives and colourings	Both IgE and non-IgE	• Self-report • Positive skin prick test without clinical history	Not reported	11 and 15 years	757 11 year olds 775 15 year olds
Pyrhonen (2009)	Cross-sectional study	2001-2009	Finland	1-4 years	Main list: Cereals (wheat, barley, rye, oat, maize, rice, millet/buckwheat) Eggs, Fish, Milk/dairy, Peanuts, Tree nuts Additional food(s): Chocolate, Citrus, Fruit and/or vegetables, Apple, Pear, Cherry, Peach, Banana, Strawberry, Tomato	Both IgE and non-IgE	• Self-report • Clinician diagnosed	Not reported	1-4years	853

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
Rance (2005)	Cross-sectional study	2002	France	2-14 years	Main list: Crustaceans (shrimp), Eggs, Fish, Milk/dairy, Peanuts, Tree nuts Additional food(s): Kiwi	Both IgE and non-IgE	• Self-report	Mean 8.9 years (2.6)	2.5-14 years	2716
Ro (2012)	Cohort study	2002-2006	Norway	2 years	Main list: Eggs (egg white), Fish Milk/dairy, Peanuts, Tree nuts (hazelnut)	IgE- only	• Positive skin prick test without clinical history • Positive serum-specific IgE without clinical history	Mean 26.6 months	2 years	668 (although only 352 complete d testing)
Roberts (2005)	Cohort study	1998-2000	United Kingdom	7 years	Main list: Eggs, Fish (cod), Milk/dairy, Peanuts, Sesame, Soy, Tree nuts (cashew, almond, walnut, hazelnut, brazil nut, pecan)	IgE- only	• Positive skin prick test without clinical history	90 months (median)	Interquarti le range 89-91 months	6213 (main panel), approx. 2000 (subpane l)
Ronchetti (2008)	Cross-sectional study	2005-2006	Italy Rome	9-13 years	Main list: Cereals (wheat flour) Eggs, Milk/dairy Additional food(s): Tomato	Both IgE and non-IgE	• Positive skin prick test without clinical history • Other	Group 1: 12.6 years (± 0.89) Group 2: 8.71 years (± 1.41)	9-13 years	196

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
Saarinen (1999)	Cohort study	1994-1997	Finland	0-34 months	Main list: Cow's milk	Both IgE and non-IgE	• Self-report • Positive open food challenge with clinical history	6. 2.3	0-34 months	6209
Sai (2011)	Cross-sectional study	2008-2009	China	Not reported	Main List: Cereals, Crustaceans, Eggs, Fish, Milk/dairy, Molluscs, Soy Additional food(s): Beef, Chicken, Corn, Mushroom, Pork, Rice, Seafood, Tomato	IgG	Other	46.57± 7.91 years	Not reported	12766
Sakellariou (2008)	Cross-sectional study	2007	Greece	20-54 years	Main list: Eggs, Fish Additional food(s): Chocolate, Processed meats	Both IgE and non-IgE	• Self-report	Not reported	20-54 years	2003
Santadusit (2005)	Cross-sectional study	Not reported	Thailand	6 months - 6 years	Main list: Crustaceans (shrimp), Eggs (egg yolk and egg white), Fish, Milk/dairy, Molluscs (crab, mollusc, squid), Soy Additional food(s): Duck	Both IgE and non-IgE	• Self-report	Not reported	6 months - 6 years	656
Schafer (1999)	Cross-sectional study	1994	Germany	5-7 years	Main list: Eggs, Milk/dairy	Both IgE and non-IgE	• Positive skin prick test without clinical history	Not reported	5-7 years	1235

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								Age Mean (SD)	Age (Range)	Sample size
Schafer (2001)	Cross-sectional study nested case-control study	1997-1998	Germany	25-74 years	Main list: Celery, Cereals (flour), Crustaceans (crab), Eggs, Fish, Milk/dairy Peanuts, Soy, Tree nuts Additional food(s): Additives and colourings, Citrus, Fruit and/or vegetables, Meat, Pork, Seafood, Spices, Herbs Sugar, wine sparkling, Tomato	Both IgE and non-IgE	• Self-report • Positive skin prick test without clinical history	50.4% female had a median age of 50 years	25-74 years	4178
Schrander (1993)	Cohort study	1985-1989	The Netherlands	0-1 years	Main list: Cow's milk	Both IgE and non-IgE	• Self-report • Positive open food challenge with clinical history	Not reported	0-1years	1158
Shek (2010)	Cross-sectional study	2007-2008	Philippines Singapore	4-6 years, 14-16 years	Main list: Crustaceans, Peanuts, Tree nuts	Both IgE and non-IgE	• Self-report • Clinical history	Not reported	4-6 years, 14-16 years	11322
Sicherer (1999)	Cross-sectional study	1997	United States	All ages	Main list: Peanuts, Tree nuts	IgE- only (no SPT or SIgE)	• Clinical history	Not reported	Not reported	8049
Sicherer (2003)	Cross-sectional study	2002	United States	All ages	Main list: Peanuts, Tree nuts	IgE- only (no SPT or SIgE)	• Clinical history	Not reported	Not reported	1809
Sicherer (2004)	Cross-sectional study	2002	United States	All ages	Main list: Fish Additional food(s):	Both IgE and non-IgE	• Self-report • Clinical history	Not reported	All ages	4336

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
					shellfish (crustacean, mollusc)					
Sicherer (2010)	Cross-sectional study	2008	United States	All ages	Main list: Peanuts, Sesame, Tree nuts	IgE- only (no SPT or SIgE)	• Clinical history	Not reported	Not reported	13534
Soller (2012)	Cross-sectional study	2008-2009	Canada	All ages	Main list: Cereals (wheat), Eggs, Fish, Milk/dairy, Peanuts, Sesame, Soy, Tree nuts Additional food(s): Fruit and/or vegetables Shellfish	Both IgE and non-IgE	• Self-report	Not reported	Not reported	9667 from 10596 homes
Tariq (1996)	Cross-sectional study	Not reported possibly 1993 – 1994	United Kingdom	4 years	Main list: Peanuts, Tree nuts Peanut, Hazelnut, Cashew	IgE only	• Self report • Positive skin prick test with clinical history • Positive skin prick test without clinical history	Not reported	4 years	1218
Touraine (2002)	Cross-sectional study	2000-2001	France	5-17 years	Main list: Celery, Cereals (wheat) Crustaceans, Eggs, Fish Milk/dairy, Molluscs (oyster), Mustard, Peanuts, Sesame, Tree nuts (cashew, hazelnut, almond, pistachio) Additional food(s):	Both IgE and non-IgE	• Self-report	Not reported	5-17 years	1086

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
					Apple, Banana, Carrot, Cherry, Chocolate, Fruits, Garlic, Kiwi, Melon, Peach, Pear, Pork, Raspberry, Chestnut					
Venter (2006)	Cohort study	2003-2004	United Kingdom	6 years	Main list: Cereals (wheat),Eggs, Fish, Milk/dairy, Peanuts, Sesame, Tree nuts Additional food(s): Additives and colourings Strawberry	Both IgE and non-IgE	• Self-report • Positive skin prick test without clinical history • Positive DBPCFC with clinical history	Not reported	6 years	798
Venter (2008)	Cohort study	2002-2005	United Kingdom	Birth cohort	Main list: Cereals (wheat, corn), Eggs, Fish, Milk/dairy, Peanuts, Sesame Additional food(s): Additives and colourings, Kiwi, Pineapple	Both IgE and non-IgE	• Positive skin prick test with clinical history • Other	Not reported	1-3 years	891
Vierk (2007)	Cross-sectional study	2001	United States	≥18 years	Main list: Cereals (wheat/gluten) Crustaceans, Eggs, Fish, Milk/dairy, Peanuts, Soy, Tree nuts Additional food(s): Additives and colourings, Chocolate, Fruit and/or vegetables	Both IgE and non-IgE	• Self-report • Other	Not reported	Not reported	4482

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
Wan (2012)	Cross-sectional study	Not reported	Taiwan	6-8 years	Main list: Celery, Crustaceans (lobster), Milk/dairy (goat, cheese, casein) Molluscs (clam, squid, oyster. scallop, abalone, pacific squid, octopus), Tree nuts, Pistachio Additional food(s): Cacao, Fruits, Litchi, Garlic, Grape, Melon, Onion	IgE only	• Positive serum-specific IgE with clinical history	Not reported	6-8 years	1010
Woods (1998)	Cross-sectional study	1992-1994	Australia	20-44 years	Main list: Cereals (wheat products, bread/plain cereal), Eggs, Milk/dairy (milk, cheese, yoghurt, ice cream), Peanuts (Including peanut butter and coconut) Additional food(s): Additives and colourings, Alcohol, Chocolate, Fats/Oils, Fruits Dried, High fat foods, Meat and Poultry, Processed meats, Restaurant/takeaway meals, Sauces, Seafood, Spices, Herbs, Condiments, Sugar, Syrup and Jam, Tea/coffee, Vegetables	Both IgE and non-IgE	• Self-report	Not reported	20-44 years	669

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Study ID	Study design	Year conducted	Country (s)	Target age group	Allergens assessed	Type of food allergy	Methods of diagnosis employed	Sample characteristics		
								Age Mean (SD)	Age (Range)	Sample size
Woods (2002)	Cohort study follow up of European Community Respiratory Health Survey	1992-1994 (sub-sample in 1998)	Australia	20-44 years	Main list: Cereals (wholegrain wheat), Crustaceans (shrimp) Eggs, Milk/dairy, Peanuts	IgE mediated only	• Self-report • Positive skin prick test with clinical history • Positive skin prick test without clinical history	Not reported	20-44 years	457
Wu (2012)	Cross-sectional study	2004	Taiwan	All ages	Main list: Crustaceans (shrimp, crab), Eggs, Fish, Milk/dairy, Molluscs, Peanuts, Soy Additional food(s): Kiwi, Mango	Both IgE and non-IgE	• Clinician diagnosed	Not reported	<3 years 4-18 years >19	30018 (813 <3 years, 15169 4-18 years, 14036 >19 years)
Young (1994)	Cross-sectional study	Not reported	United Kingdom	Not reported	Main list: Cereals, Crustaceans, Eggs, Fish, Milk/dairy, Soy Additional food(s): Additives and colourings, Alcohol, Caffeine, Cheese, Chocolate, Citrus, Fruit and/or vegetables, Meat, Tomato	Both IgE and non-IgE	• Self-report	Not reported	Not reported	18880
Zannikos (2008)	Cross-sectional study	2007	Greece	7-13 years	Main list: Cereals, Wheat, Eggs Additional food(s): Chocolate, Fruits, shellfish	Both IgE and non-IgE	• Self-report	Not reported	7-13 years	3821

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								Age Mean (SD)	Age (Range)	Sample size
Zuberbier (2004)	Cross-sectional study	1999-2000	Germany	All ages	Main list: Celery, Cereals (barley, wheat, rye, flour, oat meal), Crustaceans (crab), Eggs (hen), Fish (herring, mackerel), Milk/dairy (cow's milk), Molluscs (mussels), Peanuts, Sesame, Soy, Tree nuts (hazelnut, walnut) Additional food(s): Apple, Apricot, Carob Carrot, Cherry, Grape, Guar gum, Nectarine, Peach, Pear, Plum, Potato (raw),Pork, Seeds (poppy)	Both IgE and non-IgE	• Positive skin prick test with clinical history • Positive DBPCFC with clinical history • Other	Not reported	0-80+ years	4093

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Table 1.5: Study designs of included studies

Study ID	Study design	Cross-sectional: utilising existing survey	Cohort: reported single or multiple time-points	Cohort: cohort utilised	Target age group or age at recruitment and follow up
Al-Hammadi (2010)	Cross-sectional study	No	N/A	N/A	6-9 years
Altintas (1995)	Cross-sectional study	No	N/A	N/A	Newborn
Arbes (2005)	Cross-sectional study	No	N/A	N/A	All ages
Arshad (2001)	Cohort study	N/A	Single time point	Isle of Wight 1989-1990	4 years
Babu (2008)	Cross-sectional study	No	N/A	N/A	5-60 years
Bakos (2006)	Cross-sectional study	No	N/A	N/A	Elderly people mean age of 77 years
Ben-Shoshan (2009)	Cross-sectional study	No	N/A	N/A	5-9 years
Ben-Shoshan (2010)	Cross-sectional study	No	N/A	N/A	All ages
Bjornsson (1996)	Cross-sectional study	Yes - European Community Respiratory Health Survey 1991-1992	N/A	N/A	20-44 years
Bock (1987)	Cohort study	N/A	Multiple time points	Fort Collins Youth Centre	Birth- 3years
Branum (2009)	Cross-sectional study	Yes - NHANES 2005-2006	N/A	N/A	< 18 years
Brugman (1998)	Cross-sectional study	No	N/A	N/A	2, 4 and 7 or 8 primary school and 2nd yr of secondary school

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Study ID	Study design	Cross-sectional: utilising existing survey	Cohort: reported single or multiple time-points	Cohort: cohort utilised	Target age group or age at recruitment and follow up
Chen (2011)	Cross-sectional study	No	N/A	N/A	<12 months
Chen (2012)	Cross-sectional study	No	N/A	N/A	0-2years
Connett (2012)	Cross-sectional study	No	N/A	N/A	14-16 years
Dalal (2002)	Cross-sectional study	No	N/A	N/A	0-2years
Eggesbo (1999)	Cohort study	N/A	Multiple time points	Population based cohort (2 maternity clinics in Oslo)	Birth, 12,18,24 months
Eller (2009)	Cohort study	N/A	Multiple time points	DARC 1998-1999	Birth, 3, 6, 9, 12, 18, 36, 72 months
Emmett (1999)	Cross-sectional study	No	N/A	N/A	≥15 years
Falcao (2004)	Cross-sectional study	No	N/A	N/A	>39 years
Frongia (2005)	Cross-sectional study	Yes - linked to ICONA 2003	N/A	N/A	12-24 months
Gelincik (2008)	Cross-sectional study	No	N/A	N/A	≥18 years
Gerrard (1973)	Cross-sectional study	No	N/A	N/A	6-36 months
Greenhawt (2009)	Cross-sectional study	No	N/A	N/A	18> years
Grundy (2002)	Cohort study	N/A	Single time point	Isle of Wight 1994-1996	3-4 years

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Study ID	Study design	Cross-sectional: utilising existing survey	Cohort: reported single or multiple time-points	Cohort: cohort utilised	Target age group or age at recruitment and follow up
Gupta (2011)	Cross-sectional study	No	N/A	N/A	<18 years
Haahtela (1980)	Cross-sectional study	No	N/A	N/A	15-17 years
Host (2002)	Cohort	N/A	Multiple time points	Odense University Hospital 1985-2000	0-15 years
Hourihane (2007)	Cross-sectional study	No	N/A	N/A	3-6 years
Hu (2010)	Cross-sectional study	Yes - repeated 1999 methodology in 2009	N/A	N/A	0-24 months
Julge (2001)	Cohort study	N/A	Multiple time points	Tartu women's clinic, Estonia	6 months, 1, 2 and 5 years
Kagan (2003)	Cross-sectional study	No	N/A	N/A	5-9 years
Kajosaari (1982)	Cross-sectional study	No	N/A	N/A	1,2,3 and 6 years
Katz (2010)	Cohort	N/A	Multiple time points	Assaf-Harofeh Hospital (Zerifin, Israel) 2004-2006	0-2 years
Keet (2012)	Cross-sectional study	Yes - NHANES 2005-2006	N/A	N/A	1-21 years
Kilgallen (1996)	Cross-sectional study	No	N/A	N/A	0-48 months
Kim (2011)	Cohort study	N/A	Single time point	Samsung Medical	0-12 months

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Study ID	Study design	Cross-sectional: utilising existing survey	Cohort: reported single or multiple time-points	Cohort: cohort utilised	Target age group or age at recruitment and follow up
				Centre 2006-2007	
Krause (2002)	Cross-sectional study	No	N/A	N/A	7-15 years
Kristjansson (1999)	Cross-sectional study	No	N/A	N/A	18 months
Kucukosmanoglu (2008a)	Cross-sectional study	No	N/A	N/A	8-18 months
Kucukosmanoglu (2008b)	Cross-sectional study	No	N/A	N/A	8-18months
Kumar (2011)	Cohort study	N/A	Single time point	Boston birth Cohort	2 years
Lack (2003)	Cohort study	N/A	Single time point	ALSPAC 1991-1992	38 months
Lao-araya (2012)	Cross-sectional study	No	N/A	N/A	3-7 years
Leung (2009)	Cross-sectional study	No	N/A	N/A	2-7 years
Liu (2010)	Cross-sectional study	Yes - NHANES 2005-2006	N/A	N/A	All ages
Marrugo (2008)	Cross-sectional study	No	N/A	N/A	1-83 years
Martinez-Gimeno (2000)	Cross-sectional study	Yes - Extension of the International Study of Asthma and Allergy in Children (ISAAC)	N/A	N/A	6-13 years
Morita (2012)	Cross-sectional study	No	N/A	N/A	Adults
Mortz (2005)	Cohort study	N/A	Single time point	TOACS	14 years

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Study ID	Study design	Cross-sectional: utilising existing survey	Cohort: reported single or multiple time-points	Cohort: cohort utilised	Target age group or age at recruitment and follow up
				1995-1996	
Mustafayev (2012)	Cross-sectional study	No	N/A	N/A	10-11 years
Nicolaou (2010)	Cohort study	N/A	Single time point	Manchester Asthma and Allergy Study 1995	8 years
Obeng (2011)	Cross-sectional study	No	N/A	N/A	5-16 years
Oh (2004)	Cross-sectional study	No	N/A	N/A	2 age groups, 6-12 year olds and 12-15 year olds
Orhan (2009)	Cross-sectional study	No	N/A	N/A	6-9 years
Osborne (2011)	Cohort study	N/A	Single time point	HealthNuts 2007	11-15 months
Ostblom (2008a)	Cohort study	N/A	Single time point	BAMSE 1994-1996	4 years
Ostblom (2008b)	Cohort study	N/A	Multiple time points	BAMSE 1994-1996	1, 2, 4 and 8 years (same cohort)
Osterballe (2005)	Cohort study	N/A	Single time point	DARC 1998-1999	Group 1: 3 years, Group 2: <3 years, Group 3: Children > 3 years, Group 4: Adults
Osterballe (2009)	Cohort study	N/A	Single time point	TOACS 1995-1996	22 years
Pereira (2005)	Cohort study 2 birth cohorts used: 1991-1992 and 1987-1988	N/A	Single time point	Isle of Wight 2002-2003	Birth cohort 1991-1992 – 11years Birth cohort 1987-1988 - 15 years

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Pyrhonen (2009)	Cross-sectional study	No	N/A	N/A	1-4 years
Rance (2005)	Cross-sectional study	No	N/A	N/A	2-14 years
Ro (2012)	Cohort study	N/A	Single time point	PACT 2002-2006	2 years
Roberts (2005)	Cohort study	N/A	Single time point	ALSPAC 1991-1992	7 years
Ronchetti (2008)	Cross-sectional study	No	N/A	N/A	9-13 years
Saarinen (1999)	Cohort study	N/A	Single time point	Recruited from Helsinki maternity hospital	0-.34 months
Sai (2011)	Cross-sectional study	No	N/A	N/A	Not reported
Sakellariou (2008)	Cross-sectional study	Yes EUROPREVALL	N/A	N/A	Not reported
Santadusit (2005)	Cross-sectional study	No	N/A	N/A	3 months- 6 years
Schafer (1999)	Cross-sectional study	No	N/A	N/A	5-7 years
Schafer (2001)	Cross-sectional study	No	N/A	N/A	25-74 years
Schrander (1993)	Cohort study	N/A	Single time point	Recruited from health care centres in Maastricht	0-1 years
Shek (2010)	Cross-sectional	No	N/A	N/A	4-6 years, 14-16 years

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	study				
Sicherer (1999)	Cross-sectional study	No	N/A	N/A	All ages
Sicherer (2003)	Cross-sectional study	No	N/A	N/A	All ages
Sicherer (2004)	Cross-sectional study	No	N/A	N/A	All ages
Sicherer (2010)	Cross-sectional study	No	N/A	N/A	All ages
Soller (2012)	Cross-sectional study	No	N/A	N/A	All ages
Tariq (1996)	Cross-sectional study	No	N/A	N/A	4 years
Touraine (2002)	Cross-sectional study	No	N/A	N/A	5-17 years
Venter (2006)	Cohort study	N/A	Single time point	Isle of Wight 1997-1998	6 year olds
Venter (2008)	Cohort study	N/A	Multiple time points	Isle of Wight 2001-2002	1,2,3 years
Vierk (2007)	Cross-sectional study	No	N/A	N/A	≥18 years

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Study ID	Study design	Cross-sectional: utilising existing survey	Cohort: reported single or multiple time-points	Cohort: cohort utilised	Target age group or age at recruitment and follow up
Wan (2012)	Cross-sectional study [5]	No	N/A	N/A	6-8 years
Woods (1998)	Cross-sectional study	No	N/A	N/A	20-44 years
Woods (2002)	Cohort study	N/A	Multiple time points	ECRHS	Not reported
Wu (2012)	Cross-sectional study	No	N/A	N/A	All ages
Young (1994)	Cross-sectional study	No	N/A	N/A	Not reported
Zannikos (2008)	Cross-sectional study	Yes EUROPREVALL	N/A	N/A	7-13 years
Zuberbier (2004)	Cross-sectional study	No	N/A	N/A	All

[5] Although suggests a cohort study it appears to be a cross sectional study in which there were stages of detection, i.e, questionnaire which led to further testing

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1.2.4. Quality of included studies

Table 1.6. presents the quality assessment for all included studies.

Table 1.6: Quality assessment of all studies

Study ID	(1) Method of diagnosis	(2) Sampling strategy: method	(3) Explored reasons for withdrawal/non-response
Al-Hammadi (2010)	High risk of bias	Low risk of bias	Unclear
Altintas (1995)	High risk of bias	Low risk of bias	Unclear
Arbes (2005)	High risk of bias	Unclear	Low risk of bias
Arshad (2001)	High risk of bias	Low risk of bias	Low risk of bias
Babu (2008)	Low risk of bias	Low risk of bias	Unclear
Bakos (2006)	High risk of bias	High risk of bias	Low risk of bias
Ben-Shoshan (2009)	Low risk of bias	Low risk of bias	Unclear
Ben-Shoshan (2010)	High risk of bias	Low risk of bias	Unclear
Bjornsson (1996)	High risk of bias	Low risk of bias	Unclear
Bock (1987)	Low risk of bias	Low risk of bias	High risk of bias
Branum (2009)	High risk of bias	Low risk of bias	Unclear
Brugman (1998)	High risk of bias	Unclear	Unclear
Chen (2011)	Low risk of bias	Low risk of bias	Unclear
Chen (2012)	Low risk of bias	Low risk of bias	Unclear
Connett (2012)	High risk of bias	Low risk of bias	Unclear
Dalal (2002)	Low risk of bias	Unclear	Unclear
Eggesbo (1999)	High risk of bias	Low risk of bias	Unclear
Eller (2009)	Low risk of bias	Low risk of bias	High risk of bias
Emmett (1999)	High risk of bias	Unclear	Unclear
Falcao (2004)	High risk of bias	Low risk of bias	Unclear
Frongia (2005)	High risk of bias	Low risk of bias	Low risk of bias
Gelincik (2008)	Low risk of bias	Low risk of bias	Unclear
Gerrard (1973)	High risk of bias	High risk of bias	Low risk of bias
Greenhawt (2009)	High risk of bias	Low risk of bias	Unclear
Grundy (2002)	Low risk of bias	Low risk of bias	Unclear
Gupta (2011)	High risk of bias	Low risk of bias	Unclear
Haahtela (1980)	High risk of bias	Low risk of bias	Low risk of bias
Host (2002)	Low risk of bias	Low risk of bais	Unclear
Hourihane (2007)	Low risk of bias	Low risk of bias	Unclear
Hu (2010)	Low risk of bias	Low risk of bias	Unclear
Julge (2001)	High risk of bias	Low risk of bias	Low risk of bias
Kagan (2003)	Low risk of bias	Low risk of bias	High risk of bias
Kajosaari (1982)	Low risk of bias	Low risk of bias	Unclear
Katz (2010)	Low risk of bias	Low risk of bias	High risk of bias

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Study ID	(1) Method of diagnosis	(2) Sampling strategy: method	(3) Explored reasons for withdrawal/non-response
Keet (2012)	High risk of bias	Low risk of bias	Unclear
Kilgallen (1996)	High risk of bias	Unclear	Unclear
Kim (2011)	High risk of bias	Unclear	Unclear
Krause (2002)	Low risk of bias	Low risk of bias	Unclear
Kristjansson (1999)	Low risk of bias	High risk of bias	Unclear
Kucukosmanoglu (2008a)	High risk of bias	Unclear	Unclear
Kucukosmanoglu (2008b)	Low risk of bias	High risk of bias	Unclear
Kumar (2011)	High risk of bias	High risk of bias	Unclear
Lack (2003)	Low risk of bias	Low risk of bias	Unclear
Lao-araya (2012)	Low risk of bias	Low risk of bias	Unclear
Leung (2009)	High risk of bias	Low risk of bias	Unclear
Liu (2010)	High risk of bias	Low risk of bias	Unclear
Marrugo (2008)	High risk of bias	Low risk of bias	Unclear
Martinez-Gimeno (2000)	High risk of bias	Low risk of bias	Unclear
Morita (2012)	Low risk of bias	High risk of bias	Unclear
Mortz (2005)	Low risk of bias	Unclear	Unclear
Mustafayev (2012)	Low risk of bias	Unclear	Unclear
Nicolaou (2010)	Low risk of bias	Low risk of bias	Unclear
Obeng (2011)	High risk of bias	Unclear	Unclear
Oh (2004)	High risk of bias	Low risk of bias	Unclear
Orhan (2009)	Low risk of bias	Low risk of bias	Unclear
Osborne (2011)	High risk of bias	High risk of bias	Unclear
Ostblom (2008a)	Low risk of bias	Unclear	Unclear
Ostblom (2008b)	High risk of bias	Unclear	Unclear
Osterballe (2005)	Low risk of bias	Low risk of bias	Unclear
Osterballe (2009)	High risk of bias	Low risk of bias	Unclear
Pereira (2005)	High risk of bias	Low risk of bias	Low risk of bias
Pyrhonen (2009)	High risk of bias	Low risk of bias	Unclear
Rance (2005)	High risk of bias	Low risk of bias	Unclear
Ro (2012)	High risk of bias	Unclear	Unclear
Roberts (2005)	High risk of bias	Low risk of bias	Unclear
Ronchetti (2008)	High risk of bias	Low risk of bias	Unclear
Saarinen (1999)		High risk of bias	High risk of bias
Sai (2011)	Low risk of bias	Low risk of bias	Unclear
Sakellariou (2008)	High risk of bias	Unclear	Unclear
Santadusit (2005)	High risk of bias	Low risk of bias	Unclear
Schafer (1999)	High risk of bias	High risk of bias	Unclear

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Study ID	(1) Method of diagnosis	(2) Sampling strategy: method	(3) Explored reasons for withdrawal/non-response
Schafer (2001)	High risk of bias	Low risk of bias	Unclear
Schrander (1993)	Low risk of bias	Low risk of bias	Low risk of bias
Shek (2010)	High risk of bias	Low risk of bias	Unclear
Sicherer (1999)	High risk of bias	Low risk of bias	Low risk of bias
Sicherer (2003)	High risk of bias	Low risk of bias	Low risk of bias
Sicherer (2004)	High risk of bias	Low risk of bias	Low risk of bias
Sicherer (2010)	High risk of bias	Low risk of bias	Unclear
Soller (2012)	High risk of bias	Low risk of bias	High risk of bias
Tariq (1996)	Low risk of bias	Low risk of bias	Low risk of bias
Touraine (2002)	High risk of bias	High risk of bias	Unclear
Venter (2006)	Low risk of bias	Low risk of bias	Low risk of bias
Venter (2008)	Low risk of bias	Low risk of bias	Unclear
Vierk (2007)	High risk of bias	Low risk of bias	Low risk of bias
Wan (2012)	Low risk of bias	Unclear	Unclear
Woods (1998)	High risk of bias	Low risk of bias	Unclear
Woods (2002)	Low risk of bias	High risk of bias	Unclear
Wu (2012)	High risk of bias	Low risk of bias	Unclear
Young (1994)	High risk of bias	Low risk of bias	High risk of bias
Zannikos (2008)	High risk of bias	Unclear	Unclear
Zuberbier (2004)	Low risk of bias	Low risk of bias	Unclear

(1) Low risk of bias = food challenges (open or double-blind) with or without clinical history or sensitisation (skin prick test and/or serum-specific IgE) with clinical history; High risk of bias = Sensitisation (skin prick test and/or serum specific IgE) without clinical history, clinical history alone, clinician diagnosed or self-report.

(2) Low risk of bias = whole population, random; High risk of bias = non-random.

(3) Low risk of bias = reasons for non-response or withdrawal/loss to follow-up explored; High risk of bias = reasons for non-response or withdrawal/loss to follow-up not explored.

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1.2.5. Further information about diagnostic procedures employed by all studies

Table 1.7: Further information about questionnaire-based methods employed by studies

Study ID	Self-report	Clinician diagnosed	Clinical history
Al- Hammadi (2010)	Not applicable	Questions regarding allergic disease and atopic family history were asked through a questionnaire for parents to complete. A child was considered to have food allergy or other allergic illness only if it was diagnosed by a physician.	Not applicable
Altintas (1995)	Not applicable	The diagnosis of cow's milk allergy was based on a) the presence of symptoms in response to a diet containing cow's milk, b) the disappearance of symptoms upon withdrawal of cow's milk, and c) at least two positive milk challenges.	Not applicable
Babu (2008)	A detailed case history was taken based on a structured questionnaire containing information regarding demographics, age at onset of disease and the present allergic status. In addition type of complaints, allergy to other foods and/or pollens and insects, and duration of onset of allergic symptoms after ingestion of the offending food were taken.	Not applicable	Not applicable

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Study ID	Self-report	Clinician diagnosed	Clinical history
Ben-Shoshan (2010)	A standardised questionnaire developed previously by Sicherer et al (1999; 2004) to determine the general population prevalence of peanut, tree nut, fish, and shellfish allergy in the United States, and modified it to incorporate questions regarding sesame allergy	Confirmed allergy only if one of the following was fulfilled: a) Convincing history of an IgE-mediated reaction attributed to food and physician confirmation of a positive SPT, serum food-specific IgE >0.35 kU/L or a positive food challenge. b) Never exposed to the food or had an uncertain history of an IgE-mediated reaction and physician confirmation of a positive SPT and a food-specific IgE above previously published thresholds (i.e., >15 kU/L for peanut and tree nut and >20 kU/L for fish) or a positive SPT and a positive food challenge or a positive food challenge alone	A convincing history of an IgE-mediated reaction to a specific food was defined as a minimum of 2 mild signs/symptoms or 1 moderate or 1 severe sign/symptom that was likely IgE-mediated and occurred within 120 minutes after ingestion or contact (or inhalation in the case of fish and shellfish). Reactions were classified as mild, moderate or severe based on the same criteria outlined for Ben-Shoshan 2010.
Bock (1987)	At each visit to the clinic, parents were asked to complete a dietary questionnaire that inquired about the infant's current diet and whether any adverse reactions to foods had been noted. The parents were also asked about any restrictions on the child's diet.	Not applicable	Not applicable
Brugman (1998)	A questionnaire on food hypersensitivity was mailed to parents. Once completed this was then checked by the school physician or nurse, where some aspects of the child's health were added based on school records of absence, medicinal use, medical treatment and overall health evaluation.	Not applicable	Not applicable
Chen (2011)	Not applicable	Not applicable	Information collected by questionnaire about medical history of adverse reactions to foods and risk factors, such as delivery, feeding pattern, family history of allergy, and other allergic co-morbidities

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Study ID	Self-report	Clinician diagnosed	Clinical history
Dalal (2002)	Not applicable	Not applicable	Information was obtained from patient medical records at the family health centre, and from the family health centre staff, including nurses and dieticians.
Eggesbo (1999)	The parents of infants were asked to complete a self-administrered questionnaire on the maternity ward. Further information was collected by postal questionnaire every 6 months until the child reached the age of two. The operational definition of the outcome, parentally perceived reactions to food, was based on the question 'does the child react to any food items?'. Possible symptoms were listed for parents to mark of what symptoms the child had experienced.	Not applicable	Not applicable
Emmett (1999)	Identification of food allergies suffered within the household.	Not applicable	Questions on source of diagnosis, doctor consultation, number of reactions, age at first reaction, type of contact with peanuts causing the reactions, amount of peanuts taken, symptoms occurring, medication taken, and hospitalisation if necessary
Falcoa (2004)	Participants completed a large questionnaire as part of an on-going health and nutrition survey of residents of Porto.	Not applicable	Not applicable
Frongia (2005)	Not applicable	An interview with the parents of the children was carried out with a healthcare professional, guided by a questionnaire. Food allergy was only included when it had been diagnosed by a doctor.	Not applicable

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Study ID	Self-report	Clinician diagnosed	Clinical history
Gelinick (2008)	An initial screening questionnaire contained two questions relating to foods, those who disclosed food-related complaints were called once more and a similar questionnaire was repeated. Those suspected of having a food allergy were invited for a personal investigation at the clinic.	Not applicable	Not applicable
Gerrard (1973)	Not applicable	Not applicable	Case histories were obtained by a nurse and included the following data: the age, marital status and ethnic origin of the parents; the prevalence in parents and siblings of a history of eczema, hay fever, urticaria, recurrent bronchitis, allergies to food/drugs, enuresis and recurrent headaches; and the attitudes of parents and siblings to milk. Additional medical records and follow up examinations were taken at each age.
Greenhawt (2009)	Questions asked about the occurence of a specific allergic reaction, the symptoms and foods attrituable to the reaction, emergency medications maintained.	Not applicable	Not applicable
Grundy (2002)	Parents of children completed a questionnaire asking information about past and current atopic symptoms on the basis of the ISAAC questionnaire.	Not applicable	Not applicable
Gupta (2011)	Not applicable	Not applicable	A convincing food allergy based on self report in conjunction with one or more of the following reaction symptoms: anaphylaxis, angioedema, coughing, other oropharyngeal symptoms, eczema, flushing, hives, low blood pressure, pruritus, trouble breathing, vomiting, or wheezing. A confirmed food allergy also included report of physician-diagnosis with serum-specific immunoglobulin E testing, skin prick testing, or an oral food challenge

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Study ID	Self-report	Clinician diagnosed	Clinical history
Host (2002)	Not applicable	Not applicable	The diagnosis of CMPA/CMPI was established by the following, generally accepted, criteria; definite disappearance of symptoms after each of two dietary eliminations of cow's milk and cow's milk products; recurrence of identical symptoms after one challenge; exclusion of lactose intolerance and coincidental infection
Kajosaari (1982)	Information was obtained from the mothers by questionnaire. The family history of atopy, the child's possible atopic symptoms and signs, duration of breast feeding, and the introduction age for fish, citrus and eggs were recorded. The history was confirmed and checked by telephone interviews whenever symptoms or signs of atopy were suspected.	Not applicable	Not applicable
Katz (2010)	Initial contact made by telephone interview in 95.8% infants and by questionnaire for the remaining 4.2%	Not applicable	Sixty-six infants were given diagnoses of IgE-cow's milk allergy, forty-eight fulfilled all criteria, including suggestive history of an immediate response, a positive SPT response, and a positive challenge result to cow's milk protein. Common symptoms of IgE-mediated cow's milk allergy were cutaneous reactions, including urticarias, angioedema and pruritus, followed by gastrointestinal and respiratory symptoms.
Kilgallen (1996)	An interview-assisted questionnaire was designed for use with parents. It contained four sections and covered the presence or absence of perceived food allergy, symptoms, foods implicated and infant feeding history.	Not applicable	Not applicable
Kim (2011)	Not applicable	Food allergy was defined as a convincing history of reproducible symptoms within 2 hours after ingestion of single food	Not applicable

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Study ID	Self-report	Clinician diagnosed	Clinical history
Kristjansson (1999)	A questionnaire was designed based on a questionnaire developed by the Allergology section of the Swedish Paediatric Association. It included 17 questions relating to the duration of breast feeding, food habits, symptoms relating to adverse food reactions, other manifestations of allergy and family atopic history.	Not applicable	Not applicable
Lack (2003)	Not applicable	Not applicable	Children identified up to 38 months old having peanut allergy, based on responses to questions about food avoidance and reactions to particular foods. Affected children were also identified from responses to questions on the questionnaire regarding previous hospitalizations and clinical investigations
Lao-araya (2012)	Parents were asked about the child's demographics, number of siblings, feeding history during infancy and the child's and family history of atopic disease.	Not applicable	Not applicable
Leung (2009)	Parents were asked about the occurrence and frequency of any AFR (adverse food reaction) in their children. 'Current' symptoms referred to symptoms in the past 12 months, whereas 'AFR ever' was defined as suffering from AFR in the subjects' life time	Not applicable	Not applicable
Marrugo (2008)	Questions were asked about personal data and occupation and personal history of atopic disease.	Not applicable	Not applicable
Martinez-Gimeno (2000)	Extension of the International Study of Asthma and Allergy in Children (ISAAC study) questionnaire.	Not applicable	Not applicable
Morita (2012)	Participants were screened for wheat allergy by a questionnaire-based examination.	Not applicable	Not applicable

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Study ID	Self-report	Clinician diagnosed	Clinical history
Mustafayev (2012)	Any person answering yes to the question ‘did your child have any allergic complaint after any food intake within the last year’ was contacted via telephone by a paediatrician trained in food allergy.	Not applicable	Not applicable
Obeng (2011)	The questionnaire included questions from the EuroPrevall study on the symptoms of adverse reactions to food (www.europrevall.org)	Not applicable	Not applicable
Oh (2004)	The Korean version of the ISAAC questionnaire was administered to the parents of the children and to the student themselves in middle schools.	Not applicable	Not applicable
Orhan (2009)	Questionnaire asking ‘Has your child ever had an adverse reaction to any food within two hours following consumption?’. If the parent responded 'yes' then a further series of questions were asked to gain information about the reaction.	Not applicable	Not applicable
Ostblom (2008a)	Any of the following parentally reported symptoms related to ingestion of a certain food were defined as food allergy: asthma, itchy eyes and/or runny nose, oedema of lips/eyes, urticaria, eczema or vomiting/diarrhoea	Not applicable	Not applicable
Ostblom (2008b)	Parents asked to report on any reactions to foods experienced by their child	Parental report of doctor diagnosed food allergy	Not applicable
Osterballe (2009)	A questionnaire with the main question: 'do you suspect hypersensitivity to foods and/or drinks?'	Not applicable	Not applicable
Pereira (2005)	Questionnaires were completed by the parent and child and where a current adverse reaction to any food was stated, they were asked to describe the symptoms that they experienced.	Not applicable	Not applicable

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Study ID	Self-report	Clinician diagnosed	Clinical history
Pyrhonen (2009)	The baseline questionnaire asked structured questions about the child's background and food allergy or hypersensitivity. Parents were asked to indicate, per food, whether they never perceived symptoms, never tasted the foods, parents perceived allergy, physician diagnosed allergy, symptoms occurred in last 12 months and symptoms occurred more than 12 months ago.	The definition of food allergy and food hypersensitivity was based on a diagnosis reached by a physician.	Not applicable
Rance (2005)	A standard, anonymous questionnaire asked 'Has your child ever had an allergic reaction to food?' If 'Yes' parents were asked additional questions about clinical and treatment data and the results of allergy tests.	Not applicable	Not applicable
Saarinen (1999)	For the first 8 weeks mothers asked to record daily feeding regime and return the records. Also completed a questionnaire on parental atopy.	Not applicable	Not applicable
Sakellariou (2208)	A survey was conducted in the context of EUROPREVALL.	Not applicable	Not applicable
Santadusit (2005)	A 16-item food allergy questionnaire was answered by parents. Families reporting adverse food reactions were invited to participate in further diagnostic investigations.	Not applicable	Not applicable
Schafer (2001)	A computer-assisted standardised interview asked whether participants had allergic reactions to foods and if so the type of reaction was recorded in detail. The reported reactions were catergorised according to reaction site, furthermore history and doctor's diagnosis were recorded.	Not applicable	Not applicable

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Study ID	Self-report	Clinician diagnosed	Clinical history
Schrander (1993	A standard form was used by the four health care doctors for entering data concerning family history, symptoms and dietary interventions.	Not applicable	Family history regarding atopic disease as well as possible food intolerance in first and second degree relatives was recorded. When present for more than two weeks the following complaints were considered suspect for the presence of cow's milk protein intolerance: Symptoms, gastrointestinal, respiratory and cutaneous manifestations. Symptoms crying/colic were considered when present for more than two hours per day.
Shek (2010)	Survey conducted using a structured questionnaire used in the US population (Sicherer et al. 2003).	Not applicable	Reactions considered convincing if organ systems were affected and symptoms were typical of allergic reactions (skin: hives and angioedema; respiratory system: trouble breathing, wheezing, and throat tightness; gastrointestinal system: vomiting and diarrhoea) occurring within 2 hours of ingestion.
Sicherer (1999)	Not applicable	Not applicable	Telephone script with computerized algorithms. Reactions considered "convincing" if organ systems were affected and symptoms typical of allergic reactions (skin system: hives and angioedema; respiratory system: trouble breathing, wheezing, throat tightness; gastrointestinal system: vomiting and diarrhoea) occurring within 1 hour of ingestion
Sicherer (2004)	Not applicable	Not applicable	Telephone script with computerized algorithms. Screening questions, to identify individuals, additional questions administered depending on responses and included those regarding, severe reactions, lifetime recurrence, seafood related medical history. Algorithms categorised people into no allergy, physician diagnosed (self reported), convincing allergy (levels 1-4) and probable allergy (levels 1-3).

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Study ID	Self-report	Clinician diagnosed	Clinical history
Sicherer (2010)	Not applicable	Not applicable	As reported for Sicherer et al. (1999)
Soller (2012)	A cross-sectional telephone interview asked if anyone in the household had a food allergy, and to which foods.	Not applicable	Not applicable
Tariq (1996)	Data was obtained on feeding, atopic disease, family history, parental smoking. Exposure to pets, housing conditions, and current illness from records. Questions about eating nuts were asked only at age 4 years.	Not applicable	Not applicable
Touraine (2002)	Questionnaire distributed to schools for parents to answer. The questionnaire asked 'Does your child have a food allergy?'. If answered yes, further information was gathered about the types of symptoms, and the presence of allergies to pollen, house dust mites and mould. Also asked about family atopic disease and any treatment received.	Not applicable	Not applicable
Venter (2006)	Parents completed a questionnaire, asking 'Does your child currently have a problem with any of the following foods? Milk, egg, peanut, tree nuts (e.g. almond, brazil), wheat, fish, sesame and other. If yes to any of the above foods, can you describe the problem'	Not applicable	Not applicable
Woods (1998)	Participants completed detailed second phase ECRHS questionnaire administered by a trained interviewer. The questionnaire covered respiratory symptoms during the last 12 months, history of asthma, home and work environment, allergic symptoms, smoking, demographics, medications and dietary information.	Not applicable	Not applicable

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Study ID	Self-report	Clinician diagnosed	Clinical history
Woods (2002)	Four questions relating to diet were asked in the ECRHS questionnaire. The first three gathered information on the amount of convenience-type food and drinks consumed, the fourth asked whether responders had ever suffered from any illness/trouble from food ingestion.	Not applicable	Not applicable
Wu (2012)	Not applicable	Self-administered questionnaire. Six reviewed and analysed questionnaire descriptions of symptoms and records of physicians' evaluations to distinguish food allergy from non-immunologic adverse food reactions. Cases diagnosed by clinicians and confirmed by positive laboratory tests were enrolled as definite cases. If symptoms occurred within minutes diagnosis was presumed to be food allergy on the basis of type I immediate hypersensitivity reaction. Non-allergic food hypersensitivity was usually characterized by a delayed reaction, occurring hours or even days after eating certain food. Allergic reactions did not depend on the amount of ingested food, whereas food intolerance worsened as more food was consumed.	Not applicable
Young (1994)	Questions were about perceived connection between food ingestion and allergic symptoms.	Not applicable	Not applicable
Zannikos (2008)	A survey was conducted in the context of EUROPREVALL.	Not applicable	Not applicable

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Table 1.8: Further information about skin-prick test and serum-specific IgE testing performed by studies

Study ID	Skin prick test				Serum Specific IgE	
	Method of determining positive test	Time to read response	Allergen for testing	Was cross-reactivity explored?	Method of determining positive test	Test used
Arbes (2005)	Wheal with mean diameter >3mm larger than the negative control	15 minutes	Extracts Only house dust mite, cat, and short ragweed allergens were standardized	Not reported	Not applicable	Not applicable
Arshad (2001)	Wheal with mean diameter >3mm larger than the negative control	15 minutes	Extracts Standardized extracts were used when available. All extracts were from Biodiagnostics (Reinbek, Germany) Histamine (0.1%) in phosphate buffered saline and physiologic saline as positive and negative controls, respectively	Not reported	Not applicable	Not applicable
Babu (2008)	Wheal with mean diameter >3mm	>15 minutes	Extracts Eggplant allergenic extracts, EE and EC, along with controls (positive: histamine dihydrochloride equivalent to 1 mg/mL histamine base, and	Not reported	Cut-off value twofold higher readings than those of normal subjects. Only those SPT positive tested	ELISA

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Study ID	Skin prick test				Serum Specific IgE	
	Method of determining positive test	Time to read response	Allergen for testing	Was cross-reactivity explored?	Method of determining positive test	Test used
			negative: 50% glycerinated PBS) were used for SPT			
Bakos (2006)	Wheal with mean diameter >5mm	Not reported	Extracts Lofarma, Milan, Italy	Not reported	0.35 kU/l (class 1) and above	Allergyscreen
Ben-Shoshan (2009)	Wheal size greater than negative control	<15 minutes	Extracts Glycerinated peanut extract supplied by ALK-Abello (Hørsholm, Denmark) Prick-to-prick Children with convincing or uncertain history having a negative SPT response with commercial extract, test repeated with crude extract (i.e., peanut butter)	Not reported	Peanut specific IgE >15 kU/L those never or rarely ingested peanut or had an uncertain history Peanut-specific IgE >0.35 kU/L for those with a convincing history	CAP FEIA
Bjornsson (1996)	Not applicable	Not applicable	Not applicable	Not applicable	> 0.35 kU/L for single allergens, only those with a positive reaction to the panel (fx5) were analysed for the single allergens	Pharmacia CAP

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Study ID	Skin prick test				Serum Specific IgE	
	Method of determining positive test	Time to read response	Allergen for testing	Was cross-reactivity explored?	Method of determining positive test	Test used
Branum (2009)	Not applicable	Not applicable	Not applicable	Not applicable	The range of detectable serum IgE levels was 0.35 to 1000 kU/L	ImmunoCAP 1000
Chen (2011)	Wheal with mean diameter >3mm larger than the negative control	15 minutes	Extracts GREER, Lenoir, NC, USA	Not reported	Not applicable	Not applicable
Dalal (2002)	Wheal with mean diameter >3mm	Not reported	Extracts Commercial extracts (Centre laboratories, Port Washington, NY, USA)	Not reported	Not applicable	Not applicable
Gelincik (2008)	Wheal with mean diameter >3mm	Not reported	Prick-to-prick	Not reported	Detection limit 0.35kU/L	Pharmacia CAP
Grundy (2002)	Wheal with mean diameter >3mm	15 minutes	Unclear	Not reported	Not applicable	Not applicable
Haahtela (1980)	Not reported	>15 minutes	Not reported	Not reported	Not applicable	Not reported
Hourihane (2007)	Wheal with mean diameter >3mm	15 minutes	Extracts ALK-Abello, Hungerford, UK	Not reported	Not applicable	Not applicable
Hu (2010)	Wheal with mean diameter >3mm	15 minutes	Extracts Glycerinated food extract supplied by Greer Company	Not reported	Not applicable	Not applicable

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Study ID	Skin prick test				Serum Specific IgE	
	Method of determining positive test	Time to read response	Allergen for testing	Was cross-reactivity explored?	Method of determining positive test	Test used
			(Taibei, China)			
Julge (2001)	Wheal with mean diameter >3mm	15 minutes	Prick-to-prick Solu-prick SQ, ALK-Abello, Horsholm, Denmark	Not reported	Detection level was 0.5 standardised units per ml (SU/mL) corresponding to approx 0.09 paper RAST units (PRU)	Magic Lite
Kagan (2003)	Wheal with mean diameter >3mm larger than the negative control	<15 minutes	Extracts SPT performed by standard technique. Lots of glycerinated extract from the same manufacturer used throughout the study. Prick-to-prick When SPT response was negative and clinical history convincing or uncertain, SPT was repeated with crude extract	Not reported	Peanut-specific IgE >15 kU/L assumed allergic without a DBPCFC	CAP FEIA
Katz (2010)	Wheal with mean diameter >3mm	20 minutes	SPTs were done to CMP, soy, a negative control, and histamine (1mg/mL; ALK-Abello, Port Washington, NY)			

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	Method of determining positive test	Time to read response	Allergen for testing	Was cross-reactivity explored?	Method of determining positive test	Test used
Keet (2012)	Not applicable	Not applicable	Not applicable	Not applicable	A specific IgE level of at least 0.35 kU/L to milk, egg, or peanut.	ImmunoCAP 1000
Krause (2002)	Not applicable	Not applicable	Not applicable	Not applicable	The cut off for a positive reaction was set at ≥0.7 kU/L	Pharmacia CAP
Kristjansson (1999)	Wheal with mean diameter >3mm	>15 minutes	Prick-to-prick	Not reported	Not applicable	Not applicable
Kucukosmanoglu (2008a)	Wheal with mean diameter >3mm	15 minutes	Extracts code no: 0145, STAL-LARGENES, France	Not reported	Not applicable	Not applicable
Kucukosmanoglu (2008b)	Wheal with mean diameter >3mm	>15 minutes	Extracts Standardised allergen extracts from whole CM extract (Hollister-Stier Laboratories USA)	Not reported	Not reported	Pharmacia CAP
Kumar (2011)	Not applicable	Not applicable	Not applicable	Not applicable	Sensitisation defined as sIgE ≥ 0.35 kUA/L at 2-year visit for egg white, cow's milk, peanut, soy, shrimp, walnut, wheat, and cod. Number of food sensitisations for each	ImmunoCAP at Quest Diagnostics (Madison, NJ)

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Study ID	Skin prick test				Serum Specific IgE	
	Method of determining positive test	Time to read response	Allergen for testing	Was cross-reactivity explored?	Method of determining positive test	Test used
					subject was categorised as 0 (reference) 1 or 2 or >= 3 foods. Peanut sIgE levels were dichotomised at >= 5 kUA/L, a level associated with greater likelihood of clinical reactivity among children 1 to 5 years. Milk and egg sIgE levels were dichotomised as 5 and 2 kUA/L, respectively, corresponding to 95th percentile positive predictive values for children <= 2 years of age. These cut off points, rather than those for children > 5 years of age (15 kVA/L for milk and 7 kUA/L for egg) were chosen due to the ages of children in the cohort. For assessment of cut off points, the control group included all children with levels	

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Study ID	Skin prick test				Serum Specific IgE	
	Method of determining positive test	Time to read response	Allergen for testing	Was cross-reactivity explored?	Method of determining positive test	Test used
					below the cut off points	
Lack (2003)	Wheal with mean diameter >3mm	15 minutes	Extracts Skin testing was performed with peanut (concentration, 1:20 [wt/vol] in 50 percent glycerol) (Soluprick, ALK-Abelló)	Not reported	Not applicable	Not applicable
Liu (2010)	Not applicable	Not applicable	Not applicable	Not applicable	The following 95% predictive levels have been proposed, based on positive predictive values for clinical reactivity: egg, 7 kU/L; milk, 15 kU/L; and peanut, 14 kU/L.1 Clinical studies determined that 95% predictive levels differ for young children (i.e., <2 years old): egg, 2 kU/L14; milk, 5 kU/L.15 There is a lack of data correlating outcomes of allergy for shrimp with IgE levels, and thus no well established IgE cut off point for likely shrimp	ImmunoCAP 1000

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Study ID	Skin prick test				Serum Specific IgE	
	Method of determining positive test	Time to read response	Allergen for testing	Was cross-reactivity explored?	Method of determining positive test	Test used
					allergy. Therefore, shrimp was treated in accordance with the typical patterns described, using a threshold of 5 kU/L.	
Morita (2012)	Wheal with mean diameter >3mm	>15 minutes	Extracts Wheat and bread extract	Not applicable	CAP > 0.35 kUA/L	ImmunoCAP
Mortz (2005)	Not applicable	Not applicable	Not applicable	Not applicable	A serum level > 0.35 kU/l (corresponding to class 1) for specific IgE was considered positive	CAP FEIA
Mustafayev (2012)	Wheal with mean diameter >3mm	Not reported	Prick-to-prick	Not reported	Not reported	Pharmacia CAP
Nicolaou (2010)	Wheal with mean diameter >3mm larger than the negative control	15 minutes	Extracts Hollister-Stier Laboratories	Not reported	sIgE ≥0.2 kUa/L	ImmunoCAP
Orhan (2009)	Wheal with mean diameter >3mm	15 minutes	Extracts SPT carried out with commercially available extracts of standard food allergens (Allergopharma,	Not reported	Not applicable	Not applicable

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Study ID	Skin prick test				Serum Specific IgE	
	Method of determining positive test	Time to read response	Allergen for testing	Was cross-reactivity explored?	Method of determining positive test	Test used
			Reinbek, Germany) Prick-to-prick Sensitisation to fresh fruits or vegetables or beef was tested using prick-to-prick testing			
Osborne (2011)	Wheal with mean diameter >3mm	Not reported	Extracts ALK, Madrid	Not reported	Not applicable	Not applicable
Ostblom (2008a)	Not applicable	Not applicable	Not applicable	Not applicable	An IgE antibody level ≥0.35 kUA/L was considered positive. Serum samples scoring positive for fx5® were further analyzed towards the individual allergens included in the mix	ImmunoCAP
Osterballe (2005)	Wheal with mean diameter >3mm larger than the negative control	15 minutes	Prick-to-prick Skin prick test was performed by the prick-prick technique using a selected panel of fresh unprocessed foods	Yes, and reported both primary allergy (allergy independent of pollen sensitisation) and secondary allergy (reactions to pollen related fruit and vegetables in pollen sensitised	Measurable specific IgE was classified as a positive test result (ML > 1.43 SU/ml, CAP > 0.35 kUA/l)	Pharmacia CAP Adults and siblings only. Magic Lite 3 year olds, adults and siblings

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Study ID	Skin prick test				Serum Specific IgE	
	Method of determining positive test	Time to read response	Allergen for testing	Was cross-reactivity explored?	Method of determining positive test	Test used
				individuals)		
Osterballe (2009)	Wheal with mean diameter >3mm	15 minutes	Prick-to-prick Skin prick test was performed with the suspected food (fresh unprocessed foods)	Primary food allergy defined as being independent of pollen sensitisation, whereas secondary food allergy was defined as reactions to pollen related fruits and vegetables in pollen allergic patients. Food allergy included both immunoglobulin E (IgE)- and non-IgE-mediated reactions.	Not applicable	Not applicable
Pereira (2005)	Wheal with mean diameter >3mm	>15 minutes	Not reported	Wheat and grass cross-reactivity in 72/80 15 year olds and 76/80 11 year olds	Not applicable	Not applicable
Ro (2012)	Wheal with mean diameter >3mm larger than the negative control	15 minutes	Extracts SPT allergen extracts were purchased from Soluprick® (ALKAbello, Copenhagen, Denmark) Prick-to-prick For SPTs to milk,	Not reported	The reference value for total IgE in two-year-old children, specified by the manufacturer, was 0–45 kU/L. The detection limit for sIgE tests was 0.1 kU/L. Concentrations of 0.35 kU/L or above were regarded as positive	Immulite 2000

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Study ID	Skin prick test				Serum Specific IgE	
	Method of determining positive test	Time to read response	Allergen for testing	Was cross-reactivity explored?	Method of determining positive test	Test used
			undiluted fresh skimmed milk was used.			
Roberts (2005)	Wheal with mean diameter >3mm	<15 minutes	Not reported	Not reported, although recognised as a potential limitation within the discussion	Not applicable	Not applicable
Ronchetti (2008)	Wheal with mean diameter >3mm	<15 minutes	Not reported	Not reported	Serum studies are mentioned in the methodology however no raw data has been presented	Not reported
Schafer (1999)	Wheal with mean diameter >3mm >2mm	>15 minutes	Not reported	Not reported	Not applicable	Not applicable
Schafer (2001)	Wheal with mean diameter >3mm >2mm	>15 minutes	Unclear	Not reported	Not applicable	Not applicable
Tariq (1996)	Wheal with mean diameter >3mm	>15 minutes	Not reported	Not reported	Not applicable	Not applicable
Venter (2006)	Wheal with mean diameter >3mm	15 minutes	Extracts Conducted with commercially prepared extracts of technically optimized standard allergens (Soluprick	Cross-reactivity was explored between grass and wheat. If a participant tested positive to wheat and grass but ate wheat without problem, this was	Not applicable	Not applicable

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Study ID	Skin prick test				Serum Specific IgE	
	Method of determining positive test	Time to read response	Allergen for testing	Was cross-reactivity explored?	Method of determining positive test	Test used
			SQ allergens-ALK Allergologisk Laboratorium A/S, Horsholm, Denmark) to a predefined panel of foods (milk, egg, wheat, cod fish, peanut and sesame) and to additional foods reported to be a problem. Prick-to-prick In the case of fruits and vegetables, prick-to-prick testing to the fresh product was conducted	defined as cross-reactivity and not reported as wheat allergy/sensitisation		
Venter (2008)	Wheal with mean diameter >3mm	Not reported	Not reported	Not reported	Not applicable	Not applicable
Wan (2012)	Not applicable	Not applicable	Not applicable	Not applicable	Not reported	ImmunoCAP RAST
Woods (2002)	Wheal with mean diameter >3mm	>15 minutes	Not reported	Not reported	Not applicable	Not applicable
Zuberbier (2004)	Wheal with mean diameter >3mm	Not reported	Prick-to-prick	Not reported	Not reported	Pharmacia CAP

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Table 1.9: Further information about food challenge procedures performed by studies

Study ID	Time-frame for monitoring reactions	Active and placebo food carriers	Dosing schedule	Method of determining positive test	Total food to be ingested	Additional information
Ben-Shoshan (2009)	See Kagan 2003	See Kagan 2003	See Kagan 2003	See Kagan 2003	See Kagan 2003	See Kagan 2003
Bock (1987)	Not reported	Not reported	Initial amount given was less than parents thought would produce a reaction, amounts then increased until a reaction was produced	Not reported	Not reported	Not reported
Chen (2011)	15-20 minute intervals	Not reported	Cow's milk challenge; a drop of ordinary formula/milk was put on the lips at first. If no reaction after 20 min, the dose increased stepwise (1.0, 2.0, 5.0, 10, 20, 40, 80–120/150 ml). In the egg challenge, boiled egg was used, and 1/16 of yolk/white (depending on the SPT or history, or else started with yolk then followed by white) were the starting dose and the amount doubled every 20 min (1/16, 1/8, 1/4, 1/2)	Observed for at least 2h after last dose. Parents reported any symptoms occurring within the 3 days after challenge. Food allergy confirmed if evidence of an unequivocal allergic reaction i.e. urticaria, angioedema, vomiting, diarrhoea, acute eczema flare up, or respiratory and cardiovascular symptoms during the challenge procedure. Parents of those demonstrating no reactions on test day were asked to the research paediatrician by phone daily for 3 days, if suspicious reactions were reported, the child should return to hospital immediately	Highest dose administered was the normal daily intake of the food in question, adjusted for the age of the children	Not reported

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Study ID	Time-frame for monitoring reactions	Active and placebo food carriers	Dosing schedule	Method of determining positive test	Total food to be ingested	Additional information
Chen (2012)	2 hours	Not reported	Not reported	Not reported	Not reported	Not reported
Eller (2009)	Not reported, although did classify late reactions as those >2 hours and monitored for late reactions via telephone interview	DBPCFC was performed with peanut, shrimp, cow's milk and hen's egg, all masked as previously described (see Osterballe, 2005). Vehicle foods were used as placebo reference	Administered in increasing doses according to guidelines	Not reported	Not reported	The standardized open food challenge was performed in all children 3 years of age. The double-blind placebo-controlled food challenge was performed in children >3 years of age
Gelincik (2008)	2-12 hours depending on patient history	Peppermint oil, pure cacao powder, cereal flakes, wheat flour, lemon juice, honey, sugar, mashed potato, milkshake, rice-pudding, carob, cinnamon and various vegetables	Not reported	Not reported	Not reported	Not reported
Grundy (2002)	Observed for 15 minutes for any symptoms, if no reaction an oral challenge (stage 2) was performed	Increasing amounts of peanut butter spread on bread or a flapjack biscuit that contained peanut were given	Offered a portion containing 0.25 g of peanut, then 0.5 g, 1 g, 2 g, and 4.25 g (total 8 g)	Not reported	8g	Not reported

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Study ID	Time-frame for monitoring reactions	Active and placebo food carriers	Dosing schedule	Method of determining positive test	Total food to be ingested	Additional information
Host (2002)	Varied within in age groups	Not reported	Not reported	Not reported	Not reported	Not reported
Hourihane (2007)	30 minutes	Peanut flour-based biscuits, prepared in Southampton by an experienced dietician (K.E.C.G.)	1 mg, 10 mg, 100 mg, 1 g, and 5 g	Identification of an objective allergic reaction with clinical signs or completion of the full challenge with no such signs up to 2 hours after the last dose	5g	Not reported
Hu (2010)	2 hours	Not reported	0.5, 1.0, 2.0, 5.0, 10, 20, 40, 80, 100, 150 mL	Respiratory rate, heart rate, blood pressure and any symptoms	Usual dietary weight	Not reported
Kagan (2003)	15-30 minutes	Peanut flakes served as the source of peanut, and cracker crumbs served as the placebo. The peanut and placebo were disguised with either applesauce or grape jelly, depending on preference	Challenges started with 10 mg of either peanut or placebo, if tolerated, the dose was increased to 25 mg, 50 mg, 100 mg, 250 mg, 500mg, 1 g, and 2.5 g	A challenge was considered positive if at least 2 of the mild manifestations described previously as characterizing a convincing clinical history (i.e.; involving only pruritus, urticaria, flushing, or rhinoconjunctivitis) or at least 1 of the moderate (i.e. involving angioedema, throat tightness, gastrointestinal complaints, or breathing difficulties (other than wheeze)) or severe manifestations occurred	If 2.5 g of peanut was tolerated, 14 g was administered in an open challenge	Not reported
Kajosaari (1982)	Not reported	Not reported	Not reported	Not reported	Not reported	Not reported
Katz (2010)	2 weeks	Not reported	Increasing doses of Materna infant formula, 1:10 diluted formula 1.0mL (2.7mg of CMP) up to 120mL (3.24g of	Not reported	Not reported	Not reported

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Study ID	Time-frame for monitoring reactions	Active and placebo food carriers	Dosing schedule	Method of determining positive test	Total food to be ingested	Additional information
			CMP) every 30 minutes			
Kucukosmanoglu (2008b)	Min 4 hours	Not reported	Not reported	Not reported	Not reported	Not reported
Lack (2003)	Not reported	Not reported	Use of graded doses until a reaction or 8 g of dry-weight equivalent had been consumed	Not reported	8g of dry-weight equivalent, followed by an open challenge with a peanut butter sandwich in the case of a negative result	Not reported
Lao-araya (2012)	Min 4hours	Not reported	Not reported	Not reported	Not reported	Not reported
Mortz (2005)	2 hours	Chocolate bar	0.16, 0.32, 1.28, 2.56, 5.12, 10.24, 30.50g	Followed EAACI guidelines	50g	Not reported
Mustafayev (2012)	20 minute intervals	Not reported	Not reported	Not reported	Not reported	Not reported
Nicolaou (2010)	30 minute intervals for open challenges. DBPCFC lasted 8 hours (including the 2-hour observation period after the administration of the final dose)	Peanut concealed in brownies for open challenges	10 mg, 100 mg, 1 g, and 5 g peanut protein for open and DBPC challenges	Challenge was considered positive after development of at least 2 objective signs i.e. skin rash, sneezing, vomiting, cough, wheeze, >20% fall in FEV1	5g	Sensory evaluation by individuals not participating in the study confirmed no differences between placebo and active brownies could be detected
Orhan (2009)	Negative DBPCFCs were followed by open challenges. Duration between a	A wide variety of foods were used to mask the active doses. All active and	15 minutes	DBPCFC were considered positive if a single or a combination of the clinical reactions, including cutaneous	The titrated doses used for hazelnut, peanut, walnut,	Not reported

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	negative DBPCFC and open challenge was 2 hours. In the open challenge, patients received a larger quantity of food (a meal-size portion for age)	placebo foods were as similar as possible in colour, flavour-taste, consistency, and texture so as not to be differentiated by the patients		(eruption, itching, rash, swelling), nasal (sneezing, itching, secretion, blockage), ocular (redness, itching, secretion), bronchial (cough, wheezing, shortness of breath), gastrointestinal (vomiting, diarrhoea), laryngeal (difficulty in swallowing, difficulty in speaking), cardiovascular (tachycardia, hypotension), and other (sweating, pallor, fainting, loss of consciousness) symptoms were noted	chickpea, and corn was of the same magnitude: 0.1, 0.3, 0.6, 1.5, 2.5, 5, and 15 g, in total 25 g of the respective food. The dose steps for cow's milk were 5, 10, 40, 75, and 150 mL, in total 280 mL; 0.1, 0.3, 0.6, 1.5, 2.5, 5, 15, and 25 g for hen's egg, kiwi fruit, banana, and tomato, in total 50 g; 1, 2, 5, 7, and 10 g for cocoa, in total 25 g; 1, 2, 7, 15, 25, and 50 g for beef and fish, in total 100 g; and 0.1, 0.3, 0.6, 1.5, 2.5, and 5 g for black pepper, in total 10 g	
Osterballe (2005)	The dose interval was 15 minutes. A positive	Codfish, hazelnut, peanut and walnut	The titrated doses of codfish were: 125,	Not reported	63.5g (unclear, but appears to be	The open controlled

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Study ID	Time-frame for monitoring reactions	Active and placebo food carriers	Dosing schedule	Method of determining positive test	Total food to be ingested	Additional information
	challenge was divided into immediate or late reactions. The immediate reactions were defined as a reaction taking place within 2 h after the last dose administered, whereas late reactions occurred between 2 and 24 h after the last dose. All participants with a positive outcome in food challenge were examined for late reactions by telephone interview and reported symptoms were subsequently verified/excluded by clinical examination	were masked in chocolate bars with basic ingredients of margarine, dark chocolate, salt, icing sugar, oat grains, soy flour, oat flour and mint. Cow's milk and hen's egg were masked in a coloured cup (with top) with basic ingredients of sugar, cocoa, vanilla sugar and oat drink (placebo).Challenge with additives comprised the same type of candy as suspected by the participants; i.e. containing natural dyes [carmine (E120), turmeric (E100), copper chlorophyll (E141)	250, 1000, 2000, 4000, 8000 and 23,750 mg of codfish, in total 39 g, whereas the doses used for hazelnut, peanut and walnut challenges were of the same magnitude: 80, 160, 640, 1280, 2560, 5120 and 15,200 mg, in total 25 g of the respective food. The titrated doses of cow's milk were: 5, 10, 40, 80 and 160 g of fresh cow's milk, in total 295 ml, whereas the dose steps for hen's egg were 11, 44, 250, 500, 1000, 2500, 5000 and 40,000 mg of pasteurized whole-egg (49,305 mg, approx one egg). OCFC were performed with the following dose steps: 0.5, 1, 2, 4, 8, 16 and 32 g		for all foods except additives, for which total dose on 90 g wine gum in children and 160 g wine gum in adults was given)	standardized food challenge was performed in all children <3 yr of age. The double-blind placebo controlled food challenge was performed in children older than 3 yr of age
Osterballe (2009)	A positive challenge was divided into immediate or late	Codfish and peanut were masked in chocolate bars with	The dose interval was 15 min	Not reported	Open controlled standardized food challenge	Open controlled standardized food challenge

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Study ID	Time-frame for monitoring reactions	Active and placebo food carriers	Dosing schedule	Method of determining positive test	Total food to be ingested	Additional information
	reactions. The immediate reactions were defined as a reaction taking place within 2 h after the last dose administered, whereas late reactions occurred between 2 and 24 h after the last dose of the food had been administered. All participants with a positive immediate reaction after food challenge were examined for late reactions by telephone interview and reported symptoms were subsequently evaluated by clinical examination	basic ingredients of margarine, dark chocolate, salt, icing sugar, oat grains, soy flour, oat flour and mint. Cow's milk and hen's egg were masked in a coloured cup (with top) with basic ingredients of sugar, cocoa, vanilla sugar and oat drink (placebo). Hen's egg challenge was performed with fresh pasteurized whole-egg and disguised according to Norgaard and Bindslev-Jensen			(OCFC) was performed with the following dose steps: 0.5, 1, 2, 4, 8, 16, 32 g, in total 63.5 g of octopus and shrimp. Oral challenge with additives was performed with the same type of wine gum (containing natural dyes) as suspected by the participants and a total dose of 160 g wine gum was given. The titrated doses of codfish were: 125, 250, 1000, 2000, 4000, 8000, 23,750 mg of codfish, in total 39 g, whereas the doses used for peanut and soy challenges were of the same	(OCFC) was performed with additives, octopus and shrimp as no standardized procedures for masking the culprit food in double-blind placebo-controlled food challenge (DBPCFC) were available. Double-blind placebo-controlled food challenge was performed with codfish, cow's milk, hen's egg, peanut and soy according to EAACI guidelines

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Study ID	Time-frame for monitoring reactions	Active and placebo food carriers	Dosing schedule	Method of determining positive test	Total food to be ingested	Additional information
					magnitude: 80, 160, 640, 1280, 2560, 5120, 15,200 mg, in total 25 g of peanut or soy. The titrated doses of cow's milk were: 5, 10, 40, 80, 160 g of fresh cow's milk, in total 295 ml. Dose steps for Hen's egg were 11, 44, 250, 500, 1000, 2500, 5000, 40,000 mg of pasteurized whole-egg (totally 49,305 mg, approximately one egg)	
Saarinen (1999)	Initially infants were fed cow's milk formula every 30 to 60 minutes. All those without symptoms were examined for delayed symptoms 5 days after challenge test.	Not reported	Cow's milk formula was given in quantities of 1,10,50 and 100 ml at intervals of 30 to 60 minutes	Challenge was considered positive if one or more of the following symptoms appeared; urticaria, exanthema, atopic dermatitis, vomiting, diarrhoea, wheezing or allergic rhinitis.	Maximum 100 ml	

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Study ID	Time-frame for monitoring reactions	Active and placebo food carriers	Dosing schedule	Method of determining positive test	Total food to be ingested	Additional information
Schrander (1993)	Not reported	Not reported	Challenges in patients with gastrointestinal symptoms were done with full amounts of milk. In children with an increased risk of anaphylaxis were performed with increasing amounts of milk. 5, 10, 30. 50, 100 ml.	A positive challenge was defined as the recurrence of the patients original complaints. Two positive elimination challenge tests after exclusion of lactose intolerance were consider diagnostic for cow's milk protein intolerance	100 ml	Not reported
Venter (2006)	1 day in hospital for immediate and 1 week at home for non-generalized late reactions	Not reported	Not reported	Not reported	One-day challenge protocols were based on the consumption of the equivalent of 8-10g of dried food, unless the history clearly indicated a different approach. If negative, the parent was asked to give the child further doses of the food at home. One week challenges were based on normal daily	Not reported

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Study ID	Time-frame for monitoring reactions	Active and placebo food carriers	Dosing schedule	Method of determining positive test	Total food to be ingested	Additional information
					consumption for the specific age group	
Zuberbier (2004)	Not reported	On one day a range of food additives known to cause non-allergic intolerance reactions were given in 13 capsules, on another day the same number of capsules filled with mannit and silcium dioxide were given as a placebo	Not reported	Not reported	Not reported	Food items were blinded by the nutritionist using Sinlac, orange flavour, carotine, cereal flakes and or pure cacao powder. Blinding was confirmed by tasting panels

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1.2.6. Results for Prevalence with age in different countries and regions

1.2.6.1. Celery allergy prevalence across Europe

Four studies reported the prevalence of celery allergy in a European country. These studies were published between 2001 and 2006 and reported prevalence data from France, Germany and Hungary. The studies assessed the prevalence of celery allergy in those aged 5 years or more.

Self reported celery allergy was presented in only one study, which found that 5.5% (95% CI: 4.3-7.1%) of 5 -17 year olds in France reported a problem with eating celery (Touraine 2002). Two studies performed SPT to celery and reported rates of sensitisation of 9.1% (95% CI: not reported) in adults in Germany (Schafer 2001), 11.1% (95% CI: 3.6-27.0%) in adults in Hungary (Bakos 2006) declining to 3.7% (95% CI: 1.2-9.7%) in elderly people in Hungary (Bakos 2006). One study (Bakos 2006) also assessed serum SIgE levels to celery in Hungarian adults and the elderly and reported sensitisation rates of 2.8% (95% CI: 0.2-16.2%) and 9.2% (95% CI: 4.7-16.6%) respectively (Bakos et al, 2006). One population-based study reported the prevalence of celery allergy based on a positive SPT and clinical history as 3.5% (95% CI: 2.9-4.2%) (Zuberbier 2004).

Aside from the self-report data, for which it is not clear whether the researchers detected delayed reactions to celery, the only study to assess the prevalence of allergy, rather than sensitisation, to celery appears to be detecting IgE-mediated allergies (since they have used skin prisk testing in combination with a positive clinical history). The majority of studies, however, assessed sensitisation to celery rather than allergy. Furthermore, there were no studies that reported the prevalence of celery allergy based on open or double blind food challenge.

1.2.6.2. Celery allergy prevalence of different regions of the world

Only one study investigating the prevalence of celery allergy could be identified in other regions of the world. A study conducted in Taiwan found that 1.8% (95% CI: 1.1-2.9%) of 6-8 year olds in Taiwan suffer from celery allergy based on positive serum SIgE level and a good clinical history (Wan 2012). Hence, at present only IgE mediated allergy has been investigated.

1.2.6.3. Cereals allergy prevalence across Europe

The prevalence data for cereal allergy was derived from 13 countries (22 studies), including Denmark, Finland, France, Germany, Greece, Greenland, Hungary, Iceland, Italy, Norway, Sweden, Turkey and the UK. The data was published from 1980 to 2009 and the age range of the participants ranged from birth – 97 years. The majority of the studies focused on wheat allergy, but a number of studies also reported data on rye, barley, oat, corn or mixed grains.

Mixed grains

Three studies presented the prevalence rates of self reported allergy to mixed grains or cereals. The rates reported ranged between 0.2% (95% CI: 0.1-0.5%) in 18 month olds in Norway (Eggesbo 1999) to 2.3% (95% CI: 1.5-3.7%) in one year olds in Finland (Pyrhonen 2009). Pyrhonen 2009 also report rates of clinician diagnosed cereal allergy at 1.1% (95% CI: 0.5-2.1%) of one year olds, 0.9% (95% CI: 0.4-1.9%) of 2 year olds and 2% of 3 (95% CI: 0.9-2.9%) and 4 (95% CI: 1.2-3.2%) year olds. All of these studies examined IgE and non-IgE mediated allergy, although only two of the three studies tested for the presence or absence of IgE; Eggesbo 1999 presented self-reported allergy and did not attempt to distinguish between IgE or non-IgE mediated reactions.

Rye, Barley and oatmeal

One study reported data on rye/barley allergy, one study reported on rye allergy only and one more on barley and oatmeal allergy. Self reported allergy to rye/barley ranged between 1.3 (95% CI: 0.7-2.4%)

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– 1.8% (95%CI:1.1-3.1%). Clinician diagnosed rye/barley allergy was reported for 1.3% (95% CI: 0.7-2.4%) of one year olds, 1.8% (95% CI: 1.0-3%) of 2 year olds, 2% (95% CI: 1.2-3.4%) of 3 year olds and 2.7% (95%CI:1.7-4.1%) of 4 year olds (Pyrhonen 2009). One study (Zuberbier 2004) diagnosed food allergy in Germany based upon a positive skin prick test and clinical history and reported the following prevalence rates: barley allergy in 2.2% (95% CI: 1.7-2.8%), rye allergy in 1.2% (95% CI: 0.8-1.6%) and oatmeal allergy in 1.2% (95% CI: 0.9-1.7%). The study presenting the prevalence of self-reported allergy did not attempt to distinguish between IgE or non-IgE mediated reactions. Pryhonen 2009 also reported prevalence rates that included both IgE and non-IgE-mediated allergy. The diagnostic methods utilised by Zuberbier 2004 detected IgE-mediated allergy only.

Corn

Two studies looked at the prevalence of corn allergy. A study conducted in Turkey (Orhan 2009) found that only 0.1% (95% CI: 0.0 - 0.4) of the study population of 6-9 year olds reported allergy to corn all of whom were sensitised to it. However, in all cases corn allergy was not confirmed by DBPCFC (95%CI:0-0.2%). This study detected IgE-mediated allergy only. In the UK, corn allergy was confirmed by DBPCFC in 0.1% (95%CI: 0.0-0.8%) of 1, 2 and 3 year old children, in a study that monitored patients for sufficient time to identify both IgE and non-IgE mediated allergy (Venter 2008).

Flour

Two studies reported prevalence rates of reactions to “flour” where the type of flour was unspecified, both of which were conducted in Germany. Schafer 2001 found that 0.7% of their study population (95%CI: not reported) reported symptoms upon ingestion of flour (which may have been either IgE or non-IgE mediated). Examining IgE and non-IgE mediated allergy separately, Zuberbier 2004 reported a prevalence, for all ages, of 0.5% (95% CI: 0.3-0.8) and 0.1% (95% CI: 0.0-0.3) respectively.

Wheat/Gluten

Twenty studies assessed the prevalence of self-reported wheat allergy/gluten sensitivity. The lowest rates of self-reported wheat allergy were presented for a group of 7-13 year olds in Greece (0.2% (95% CI: 0.0-0.5%)) (Zannikos 2008). The highest rates were reported by a Finnish study of 1-year-olds (2.1% (95% CI: 1.3-3.4%)) (Pyrhonen 2009). Clinician-diagnosed wheat allergy was reported by two studies. Prevalence rates of clinician-diagnosed wheat allergy were reported to be 0.3% (95% CI: 0.1-0.6%) of 1 year olds and 8 year olds in Sweden (Ostblom 2008b). A higher rate (3.4% (95% CI: 2.3-5%)) was reported for a group of 4 years olds in Finland (Pyrhonen 2009).

Sensitisation to wheat, as measured by SPT, was reported in seven studies and, as measured by specific IgE, in four studies. The lowest rate of sensitisation (determined via SPT) was 0% (95% CI: 0-0.6%) reported for 1 and 3 year olds in the UK (Venter 2008). The highest rate, 13.9% (95% CI: 5.2-30.3%), was reported by a Hungarian study of 20-69 year olds (Bakos 2006). Only three studies reported prevalence of wheat allergy based on a positive SPT and clinical history. Based on this method of diagnosis, the lowest prevalence of wheat allergy was reported in a group of 18 month-old children in Sweden (0% (95% CI: 0-1.4%)) and Iceland 0% (95% CI: 0-1.5%)) (Kristjansson 1999) and the highest rate, 1.2% (95% CI: 0.9-1.7%), was reported for all ages in Germany (Zuberbier 2004) . Only one study (Ostblom 2008b) reported a prevalence rate based on positive specific IgE levels and clinical history. Using this method in a Swedish population, the prevalence rate was 1.3% (95% CI: 1.0-1.9%) of 4 year olds (Ostblom 2008b). In the only study to combine clinical history with a positive OFC/DBPCFC outcome, Osterballe 2005 did not identify a single confirmed case of wheat allergy in any age group.

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A number of studies used other methods of diagnosing wheat allergy. Using atopy patch tests in an Italian population, Ronchetti 2008 reported the prevalence of wheat allergy to be 5.6% (95% CI; 3.0-10.1%) of 13 year olds and 6% (95% CI: 3.2-10.7%) of 9 year olds. Using a combination of history and SPT and/or OFC and DBPCFC, the prevalence of wheat allergy was 0.4% (95% CI: 0.1-1.2%) in one year olds, 0.3% (95% CI: 0.1-1.1) in two year olds, 0.2% (95% CI: 0.0-0.9%) in 3 year olds and 0.3% (95% CI: 0-1.0%) in six year olds in the United Kingdom (Venter 2008; Venter 2006). Of the studies that assessed the prevalence of wheat/gluten allergy in Europe, 17 assessed both IgE and non-IgE mediated wheat/gluten allergy (although four of these did not perform any tests to determine the presence or absence of IgE) and four IgE-mediated allergy only.

1.2.6.4. Cereals allergy prevalence in different regions of the world

A number of studies (N=14) have looked into cereal allergy outside of the EU. Studies were conducted in Australia, Canada, China, Ghana, Japan, Korea, Thailand, United Arab Emirates, and the United States.

Corn and Millet

One study looked at IgE-mediated cereal allergy in 5-16 year olds in Ghana and found reported allergy to corn in 0.2% (95% CI: not reported) of children and millet in 0.1% (95% CI: not reported) (Obeng 2011). In the United States, one study reported a prevalence rate for corn allergy (both IgE and non-IgE mediated) based on food challenges of 0.2% (95%CI:0-1.3%) in 0-3 year olds (Bock 1987).

Wheat

Nine studies looked at the reported prevalence of wheat allergy. The lowest rates were reported by a study conducted in Korea in a group of 6-12 year old children (0% (95%CI:0.0-0.1%)) (Oh 2004). The highest rate was found in the United States, where 2.3% (95% CI: 1.3-4.2%) of the adult study population reported having wheat allergy (Greenhawt 2009). One study, conducted in the United States, looked at the prevalence of a reported clinical diagnosis of wheat allergy across different age groups (Gupta 2011). The prevalence ranged between 0.3% for both 0-2 year olds (95% CI: 0.1-0.5) and 14-17 year olds (95% CI: 0.2-0.4). Two studies presented prevalence rates for clinician diagnosed wheat allergy. These were 0.1% (95% CI: 0-0.5%) for a group of 0-12 month olds in Korea (Kim 2011) and 0.5% (95% CI: 0.1-2.0%) for a group of 6-9 year olds in the United Arab Emirates (Al-Hammadi 2006).

Three studies measured the prevalence of sensitization to wheat by either SPT (n=2) or serum specific IgE levels (n=1). In an Australian adult population, the prevalence of sensitization to wheat was found to be 2.2% (95% CI: 1.1 - 4.1) (Woods 2002). A study conducted in 0-24 month olds in China in 1999 and 2009 reported wheat sensitisation rates (assessed by SPT) of 0.3% (95% CI: 0.0-2.1%) and 0.5% (95%CI:0.1-2.1%) respectively (Hu 2010). The prevalence of sensitisation to wheat in a group of 0-12 month olds in Japan, as determined by positive serum specific IgE levels, was 1.4% (95%CI: not reported) (Morita 2012).

Two studies used a positive SPT/specific IgE level in combination with clinical history to estimate the prevalence of wheat allergy. These studies reported prevalence rates of between 0% (95% CI: 0.0-0.1%) for Australian adults (Woods 2002) and 0.2% (95% CI: 0.0-0.9%) for Japanese adults (Morita 2012). A higher prevalence rate (1.2% (95% CI: 1.0-1.4%)) was reported by a Chinese study which utilised IgG levels to diagnose wheat allergy (Sai 2011). In the United States the prevalence of wheat allergy in 0-3 year olds has been found to be 0.2% (95% CI: 0-1.3%) when using food challenges (Bock 1987). Also in the United States, self-reports of clinician diagnosed wheat allergy yielded a prevalence rate of 0.5% (95% CI: 0.3-0.8%) (Vierk 2007).

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The minority of adverse reactions to wheat are considered to be IgE-mediated. Of the studies reporting the prevalence of wheat allergy outside of Europe, five assessed IgE-mediated allergy only, nine considered both IgE and non-IgE mediated allergy (of which seven did not perform tests to determine the presence or absence of IgE) and one assessed IgG-mediated allergy.

1.2.6.5. Egg allergy prevalence across Europe

The prevalence of egg allergy has been assessed in 17 countries (35 studies), including Denmark, Estonia, Finland, France, Germany, Greece, Greenland, Hungary, Iceland, Ireland, Italy, Norway, Portugal, Spain, Sweden, Turkey and the UK. The included studies were published between 1980 and 2012 and the age range of the participants ranged from birth – 97 years.

None of the included studies reported prevalence rates for egg allergy based on self-report or clinical history. Three studies focussed on clinician diagnosed egg allergy with the lowest prevalence figures seen in 8 year olds from Sweden (1.6% (95% CI: 1.2-2.1)) (Ostblom 2008) and the highest in 4 year olds from Finland (3.9% (95%CI:2.7-5.5%)) (Pyrhonen 2009).

Eleven studies reported sensitisation rates based on skin prick test results and six on specific IgE levels. In the younger cohorts (0-3 years old), sensitisation rates as determined by SPT ranged from 1.3% (95% CI: 0.7-2.3%)(Venter 2008) to 5.2% (95% CI :not reported) (Julge 2001). In this age group, rates determined by sIgE ranged between 4.2% (95% CI: not reported) (Julge 2001) and 20.6% (95% CI: not reported) (Julge 2001). In children older than 3 years, sensitisation rates as determined by SPT ranged from 0% (95% CI: not reported) (Julge 2001; Roncetti 2008) to 2.8% (95% CI: 1.9-3.9%) (Schafer 1999), and as determined by sIgE, from 0.4% (95% CI: 0.1-1.1%) (Krause 2002) to as high as 22.7% (95% CI: not reported) (Julge 2001). Sensitisation rates in adults ranged between 0.4% (95%: not reported) (Schafer 2001) and 1.9% (95% CI: not reported) (Schafer 2001) when SPTs were utilised. When sensitisation in adults was determined via sIgE testing to egg yolk, sensitisation rates were 0% (95% CI: 0-12%) in ages 20-69 years and 60-97 years (Bakos 2006).When the sIgE to egg white was tested, sensitisation rates were reported to be 2.8% (95% CI: 0.2-16.2%) in ages 20-69 years and 2.8% (95% CI: 0.7-8.4%) in ages 60 – 97 years (Bakos 2006).

Four studies based egg allergy prevalence rates on a good clinical history plus a positive SPT, and reported rates ranging from 0.1% (95% CI: 0-0.1%) in 18 year olds in Turkey (Gelincik 2008) to 1.5% (95% CI: 0.6 – 3.7%) in 18 month olds in Sweden (Kristjansson et al. 1999). Two studies based egg allergy prevalence rates on a good clinical history plus a positive serum specific IgE result. One was conducted in Sweden and reported the prevalence of egg allergy to be 0.6% (95% CI: 0.3-1.0%) (Ostblom 2008a). The other found the prevalence of egg allergy in Turkey to be 0.1% (95% CI: 0.0-0.1%)(Gelinicik 2008).

Several studies utilised food challenges (four used open food challenges and four DBPCFC), in combination with clinical history, to diagnose egg allergy. Based on open food challenge and a good clinical history the highest prevalence rate was 2.6% (95% CI: not reported) in 18 month old children in Denmark (Eller 2009). In contrast, based on DBPCFC and history the highest prevalence rate reported was 1.6% (95% CI: 0.1-3.4%) (Osterballe 2005) in 3 year old children also from Denmark. Five studies combined a variety of methods to determine egg allergy prevalence. Of these, the highest reported prevalence of egg allergy was a very high rate of 10.2% (95% CI: 6.5-15.5%) diagnosed using the atopy patch test in 13 year old children (Ronchetti 2008).

Egg allergy is classically considered as an IgE mediated food allergy. We tried to understand from the included studies if the symptoms related to egg were considered IgE or non-IgE mediated. In Europe, 24 studies covered both IgE and non-IgE medidated food allergies. Apart from the study by Venter et al.which clearly indicate the presence of IgE and non-IgE mediated egg allergy, it is very difficult to

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tell from the other papers if the egg allergy per se was IgE or non-IgE mediated or both. In fact 11 of the studies who indicated that they studied both IgE and non-IgE mediated allergies, did not perform any tests to determine the presence or absence of IgE. Ten studies focussed on IgE mediated egg allergy.

1.2.6.6. Egg allergy prevalence in different regions of the world

A number of studies outside of Europe have looked at the prevalence rates of egg allergy. The countries studies include Australia, Canada, China, Colombia, Ghana, Hong Kong, Israel, Korea, Taiwan, Thailand, United Arab Emirates and the USA. The studies were reported between 1998 and 2012 and included participants of all ages.

Twelve studies looked into the self-reported prevalence of egg allergy. The lowest prevalence rate, 0.1% (95% CI: not reported), was reported in 5-16 year olds from Ghana (Obeng 2011). The highest prevalence, 1.6% (95% CI: 0.7%-3.2%), was reported in a group of US adults (Greenhawt 2009). Two studies reported prevalence based on a reported clinical history of egg allergy ranging from 0.4% (95% CI: 0.3 – 0.5%) in 14-17 years olds in the US to 1.3% (95% CI: 0.9 – 1.7%) in 3-5 year olds (Gupta et al. 2011). Only three studies focussed on clinician diagnosed egg allergy with the lowest prevalence figures seen in adults from Taiwan (0.3% (95% CI: 0.2-0. 4%)) (Wu 2012) and the highest in 6-9 year olds from the UAE (3% (95%CI:1.8-5.7%)) (Al-Hammadi 2010).

Four studies reported sensitisation rates based on skin prick test results and four as determined by specific IgE levels. High rates of sensitisation to egg, as measured by SPT, are reported with, for example, a sensitisation rate of 11.8% (95% CI: 10.6-13.0) in 12-15 month olds in Australia (Osborne 2011) and 16.2% (95% CI: 12.8-20.4%) in 0-24 month olds in China (Hu 2010). Sensitisation rates as measured by serum specific IgE levels ranged between 2.1% (95% CI: not reported) in 20-39 year olds in the US (Liu 2010) and 21% (95% CI: 18.7-23.6%) in 6 months – 6 year olds in the US (Kumar 2011).

Only two studies based egg prevalence rates on a good clinical history plus a positive SPT. Dalal 2002 found a prevalence for egg allergy of 0.5% (95% CI: 0.3-0.6%) in 0-2 year olds in Israel and Woods 2002 reported a rate of 0.2% (95% CI: 0.0-1.4%) in 26-50 year olds in Australia. No study based egg allergy prevalence rates on a good clinical history plus a positive serum specific IgE result or a positive DBPCFC and a good clinical history. However, three studies based a diagnosis of egg allergy on a positive OFC plus history. Chen 2011 reported egg allergy prevalence rates of 0.5% in 0-12 month olds in China. A different study in the same country reported prevalence rates of 2.9% (95% CI: 1.4-5.6%) in 0-24 month olds in 1999 and 5% (95% CI: 3.2-7.7%) in 0-24 month olds in 2009 (Hu 2010). Osborne 2011 reported a prevalence of 9% (95% CI: 7.9-10.0) in 12-15 months olds in Australia.

Five studies utilised other methods to diagnose egg allergy. The methodologies varied widely from using a combination of history, sensitisation status and/or food challenges, to less credible methods such as IgG levels. Many studies conducted on egg allergy outside of Europe utilised questionnaire based methods to determine the prevalence of egg allergy, which in some cases focussed on IgE mediated allergy, but did not confirm a history of immediate type symptoms with specific IgE or SPT. Only three studies reported on IgE and non-IgE mediated egg allergy, and one of these studies did not determine the presence of IgE, with 20 studies reporting on IgE mediated food allergies and 8 of these not testing for the presence of IgE. One study used IgG testing.

1.2.6.7. Fish and Shellfish prevalence across Europe

There were 34 studies which looked at the prevalence of fish and shellfish allergies in Europe (the countries studied were Denmark, Finland, France, Germany, Greece, Greenland, Hungary, Iceland,

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Norway, Portugal, Spain, Sweden, The Netherlands, Turkey and the United Kingdom). Data was published between 1980 and 2012. The prevalence of seafood allergy was assessed in participants from 6 months to 97 years. Prevalence rates based on self-reported allergy were presented in 22 studies; sensitisation rates were assessed in eight studies using skin prick tests and five studies using serum SIgE tests; sensitisation plus clinical history was obtained in four studies and seven studies adopted open and/or double blind food challenges.

IgE-mediated allergy was considered in 11 studies (Arshad 2001; Bakos 2006; Bjornsson 1996; Haahtela 1980; Kajosaari 1982; Krause 2002; Kristjansson 1999; Mustafayev 2012; Orhan 2009; Ro 2012; Roberts 2005). The methods adopted by these studies included skin prick and specific IgE tests to assess sensitisation, and food challenges and/or self-reported allergy where only IgE-associated symptoms were considered a positive indication of allergy. In the remaining 23 studies both IgE-mediated and non-IgE mediated allergy were included in the reported prevalence figures.

The highest self-reported prevalence of fish allergy was found in Finland, with 7% (95% CI: not reported) (Kajosaari 1982) of parents of 1 year olds reporting that their child had an adverse reaction to fish. A similar prevalence rate, 6.9% (95% CI: 6.2-7.6%) (Martinez-Gimeno 2000), was found in 6-13 year olds in a Spanish population. The lowest rate was found in Denmark where only 0.2 % (95% CI: 0-1%) (Osterballe 2009) of 22 year olds reported an adverse reaction to fish, however this study only asked about an allergy to cod, and so the neglect of other fish species could account for the low prevalence. Studies reporting prevalence of clinician diagnosed allergy or a diagnosis based on clinical history of fish allergy ranged from 0.2% (95% CI: 0-0.9%) (Pyrhonen 2009) of 1 year olds in Finland and 0.2% (95% CI: 0.1-0.4%) (Ostblom 2008 b) of 1 year olds in Sweden to 1.0% (95% CI: 0.5-2.0%) (Pyrhonen 2009) of 4 and 5 year olds in Finland.

Looking at sensitisation, the highest prevalence of fish sensitisation as detected by skin prick tests was seen in Finland, with 2.7% (95% CI: 1.7-4.2%) (Haahtela 1980) of 15-17 year olds being sensitised. The lowest rates were found in the UK where 0% (95% CI: 0-0.3%) (Roberts 2005) of 7 year olds had a positive skin prick test to cod, and in Hungary where 0% (95% CI: 0-4.2%) (Bakos2006) of 60-97 year olds showed sensitisation to cod on a SIgE test. When sensitisation plus a convincing clinical history was obtained, the highest rate for fish allergy was 0.6% (95% CI: 0.1-2.5%) (Kristjansson,1999) was reported in Iceland at 18 months of age. The lowest rate was found in Turkey in 0.2% (95% CI: 0.1-0.5%) of 6-9 year olds (Orhan 2009). Four of the studies that adopted open and/or double-blind food challenges to diagnose fish allergy reported 0% prevalence to fish, however one study in a Finnish population found a prevalence of 1% (95% CI: not reported) of 6 year olds (Kajosaari,1982).

With regard to crustacean allergy, the prevalence of self-reported crustacean-related adverse food reactions ranged from 0.3% (95%CI: 0.1-1.0%) of 11 year olds in the UK (Pereira, 2005) to 5.5% (95%CI: 4.3-7.1%) of 5-17 year olds in France (Touraine, 2002). Sensitisation rates for crustacean allergy were similar in Germany 1.9% (95% CI: not reported) based on skin prick tests (Schafer, 2001) and Hungary 1.8% (95% CI: 0.3-7.1%)) based on SIgE testing (Bakos 2006. Only one study, conducted in Denmark, reported challenge proven prevalence data for crustacean allergy, which found a prevalence of 0% (95% CI: 0.0-2.0%) in 0-22 year olds and 0.3% (95% CI: 0.1-1.0%) in individuals 22 years or older (Osterballe, 2005).

Where mollusc allergy is concerned, only three studies collected data on self-reported mollusc-related adverse reactions in Europe, with the highest prevalence reported in France where 1.5% (95% CI: 0.9-2.4%) of 5-17 year olds reported an allergy to oysters (Touraine 2002) and the lowest prevalence in Denmark, with only 0.4% (95% CI: 0.1-1.1%) of 22 year olds self-reporting an allergy to octopus (Osterballe 2009). Prevalence of allergy to mollusc, as diagnosed using positive SPT and convincing

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clinical history, was presented by only one study, conducted in Germany, which reported a prevalence of 0% (95% CI: 0.0-0.2) for mussel allergy (Zuberbier 2004). There were no studies in Europe that adopted food challenges to confirm mollusc allergy.

1.2.6.8. Fish and Shellfish prevalence in different regions of the world

Twenty-seven studies looked at the prevalence of fish and shellfish allergy across the rest of the world. Two studies have been conducted in Australia, one in Canada, three in China, one in Colombia, one in Ghana, one in Hong Kong, one in Israel, two in Korea, eight in South-East Asia, two in Taiwan, one in the United Emirates, and the rest of the studies were all conducted in the USA. Data was published between 1998 and 2012 with participant ages ranging from 0- 83 years of age. Self-reported allergy was presented in 16 studies, 10 studies combined clinical history with a clinician diagnosed seafood allergy, seven studies measured sensitisation rates, with a further three studies also taking into account a convincing clinical history as well as sensitisation. Only two studies adopted food challenges to confirm suspected allergy.

IgE-mediated allergy was considered in 11 studies (Ben-Shoshan 2009; Branum 2009; Chen 2011; Dalal 2002; Greenhawt 2009; Kim 2011; Lao-araya 2012; Liu 2010; Osborne 2011; Wan 2012; Woods 2002). The methods adopted by these studies included skin prick and specific IgE tests to assess sensitisation, and food challenges and/or self-reported allergy where only IgE-associated symptoms were considered a positive indication of allergy. In the remaining 16 studies both IgE-mediated and non-IgE mediated allergy were included in the reported prevalence figures.

The highest prevalence of self-reported fish-related adverse reactions was seen in adults in the United States (2.7% (95%CI:1.6-4.7%)) (Greenhawt 2009) compared with 0.6% (95% CI: 0.4-0.8) (Ben-Shoshan 2010) of adults in Canada. In children in Canada, 0.2% (95% CI: 0.0-0.4%) (Ben-Shoshan 2010) self-reported a fish allergy, which lowered to 0% (95% CI: not reported) (Ben-Shoshan 2010) confirmed with a clinician diagnosed fish allergy. The highest prevalence of clinician diagnosed fish allergy in Non-European countries is 2.8% (95% CI: 1.5-5.1%) (Al-Hammadi 2010) seen in 6-9 year olds in the United Arab Emirates; the lowest prevalence rates were reported in 0-2 year olds in Israel (0% (95% CI: 0-0.1%) (Dalal 2002) and 0-5 year olds in the United States (0% (95% CI: 0.0-0.5%) (Sicherer 2004).

Two studies measured sensitisation, reporting prevalence ranges from 0.2% (95% CI: 0.0-1.3%) (Chen 2011) in 0-12 month olds to 0.8 % (95% CI: 0.2-2.5%) (Hu 2010) of 0-2 year olds both in China. In Israel, 0% (95% CI: 0.0-0.1%) (Dalal 2002) prevalence of fish allergy was found in 0-2 year olds when a convincing clinical history plus sensitisation was the method of diagnosis. Open food challenges were performed in 3-7 year olds in Thailand, revealing a 0.2% (95% CI: 0.0-1.4%) prevalence of allergy to fish (Lao-araya, 2012).

With regard to shellfish allergy, self-reported shellfish allergy varied from a very low rate of 0.1% (95% CI: not reported) in 5-16 year olds in Ghana (Obeng 2011) to a very high rate of 24.5% (95% CI: not reported) in adults in China (Sai 2011). The lowest prevalence for clinician diagnosed shellfish allergy was 0.1% (95% CI: 0.0-0.1%) (Ben-Shoshan 2010), for under 18 year olds in Canada, and the highest prevalence based on a convincing clinical history was seen in Singapore, with 5.2% (95% CI: 4.5-6.1%) of 14-16 year olds suggesting a positive shellfish allergy (Shek 2010). Based on a positive skin prick test, crustacean sensitisation was 0% (95% CI: 0-1.6%) in 0-2 year olds from China (Hu 2010) compared with 17.3% (95% CI: 15.1-19.8%) of Taiwanese 6-8 year olds sensitised to lobster, determined using serum specific IgE testing (Wan 2012). Despite the large number of studies looking at the prevalence of shellfish allergy based on self reports of adverse reactions, convincing clinical history and a clinician diagnosis, only one study was found to perform

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open food challenges to crustaceans, reporting a prevalence between 0.2% (95% CI: 0.0-1.4%) for crab and 0.9 (95% CI: 0.3-2.4%) for shrimp in 3-7 year olds in Thailand (Lao-araya 2012).

Self-reported mollusc allergy was found to be 0.2% (95% CI: 0.0-1.4) for 3-7 year olds in Thailand (Lao-Araya 2012). In Taiwan, mollusc allergy defined by a clinician diagnosis varied from 0.1% (95% CI: 0.0-0.8) in under 3 year olds to 1.5% (95% CI: 1.3-1.7) in adults (Wu 2012). Sensitisation, as determined by serum-specific IgE testing, has been reported for 6-8 year old children in Taiwan for scallop (24.9% (95% CI: 22.2-27.7%)) and abalone (25.1% (95% CI: 22.4-27.9%)) sensitised to mollusc (abalone) (Wan 2012). There were no studies conducted outside of Europe reporting data on challenge proven mollusc allergy.

Six studies reported seafood allergy, which one can only presume to include both fish and shellfish allergy, with the highest prevalence rate found in Colombia with 4% (95% CI: 3.3-4.7%) of all ages self-reporting an allergy (Marrugo 2008). The lowest prevalence was seen in Korea, with 0.4% (95% CI: 0.3-0.4%) of 6-12 year olds self-reporting a seafood allergy (Oh 2004). In addition, one study from China reported high prevalence of allergy to fish 11.2% (95% CI: 10.7-11.8%) crab 24.5% (95% CI: 23.8-25.3%) and shrimp 10.0% (95% CI: 9.5-10.6%) (Sai 2011) however data was calculated by IgG measurements, which do not report allergy. Furthermore, it was not clear how the clinical history was taken. Hence, caution should be taken when interpreting these findings.

1.2.6.9. Fruit allergy prevalence across Europe

A large number of studies (n=14) reported on fruit and in some cases vegetable allergies. Within Europe, the countries where the studies were performed include: Denmark, Finland, France, Germany, Greece, Hungary, Iceland, Norway, Portugal, Spain, Sweden, The Netherlands, Turkey and the UK. The data was published from 1982 to 2012 and the age range of the participants ranged from birth – 97 years.

A large variety of fruits have been studied including: a mixture of fruit and vegetables (n=13), apple (n=5), citrus/orange fruits (n=11), strawberry (n=6), kiwi (n=3), pear (n=3), apricot (n=1) , cherry (n=2) , grape (n=2), nectarine (n=1), peach (n=4), plum (n=2), banana (n=8), and pineapple (n=1). A number of these fruits have been implicated in Oral Allergy Syndrome (pear, apple, cherry and peach) and banana has been shown to cross react with latex, although this is outside the remit of this report.

The highest rate of citrus fruit allergy, 11% (95%CI: not reported), was reported using a self-report method in a sample of 3 year old children in Finland (Kajosaari 1982). In the same study, using open food challenges, the prevalence of citrus fruit allergy was 2% (95%CI: not reported) in 6 year old children. This was the only study to use food challenges to diagnose citrus fruit allergy in a paediatric sample). Only two studies used food challenges, reporting a prevalence of 2% (95% CI: not reported) in 6 year olds in Finland (Kajosaari 1982) and 0% (95% CI: 0.0-0.1%) of adults in Turkey (Gelincik 2008).

Strawberry allergy was examined in six studies. Similar to the pattern for citrus fruits, the highest rates were presented for young children in Finland: 7% (95% CI: not reported) at age 1, 4% at age 2 and 7% at age 3 years, however all were measured using self-report methods (Kajosaari 1982). Lower rates of self-reported strawberry allergy were reported for adults in Turkey (0.7% (95% CI: 0.5-0.8%)), which translated to a 0% (95% CI: 0.0-0.1%) prevalence when diagnosis was made using DBPCFC (Gelincik 2008). Similarly low rates 0% (95% CI: 0.0-0.2%) were reported in children in Turkey using DBPCFC (Orhan 2009).

Kiwi fruit, which is sometimes cited as the "15th" major allergen was found to have a 0.8% (95% CI: 0.5-1.0%) allergy prevalence in a sample children in France, using a self report method (Rance 2005).

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The only other studies examining kiwi allergy prevalence were both conducted with children in Turkey. One study identified the prevalence of self-reported kiwi allergy as 0.3% (95% CI: 0.1-0.6%) decreasing to 0.1 % (95% CI: 0-0.4%) when a DBPCFC method was employed (Orhan 2009). More recently, a prevalence of 0.1% (95% CI: 0.0-0.8%) was also found using open food challenges (Mustafayev 2012).

It is difficult to truly distinguish IgE mediated from non-IgE mediated food allergies or even chemical intolerances in fruit induced reactions. Nineteen studies in Europe report to have studied both IgE and non-IgE mediated reactions but only ten of these reported to have performed SPT or specific IgE tests and it was not clear if these tests have been performed to the fruit in question. Only two studies have reported IgE mediated reactions and both have utilised SPT/specific IgE testing, but it was once again not clear if these tests have been carried out the fruit in question.

1.2.6.10. Fruit allergy prevalence in different regions of the world

There were a total of 16 studies conducted in countries outside Europe that reported fruit allergy prevalence rates. The countries where the studies were performed include Australia, Canada, China, Colombia, Ghana, Hong Kong, Israel, Korea, Taiwan, United Arab Emirates and the United States. The data was published from 1987 to 2012 and the age range of the participants ranged from birth – 44 years.

In addition to the fruits that were reported as allergens in Europe (orange, apple, banana, pineapple, peach, grape, kiwi, strawberry and “fruits” not specified), additional fruit allergies were reported in these non-European countries. These were pawpaw, mango and melon in Ghana (Obeng 2011); mango, melon and litchi in Taiwan (Wu 2012), “fruit juice” in USA (Bock 1987) and “dried fruit” in Australia (Woods 1998). Conversely, cherry, plum and apricot were reported as causing adverse reactions in Europe, but not in countries outside.

Food challenges were rarely used, with the majority of studies reliant on self-report methods. The highest prevalence rate was 10.8% (95% CI: 8.3-14%), which was reported to fruit juice in a study of one year old children in the United States (Bock 1987), which converted to a 7.9% (95% CI: 5.7-10.8%) rate of “probable or convincing allergy” using a combination of SPT, sIgE, clinical history and food challenge. Indeed, this study was the only one to use food challenges as a method of diagnosis. Skin prick testing was only used by two further studies; Chen 2011 (orange) and Dalal 2002 (strawberry) and sIgE by one study (Wan 2012) (lychee, melon and grape), perhaps reflecting the lack of valid diagnostic tests available for fruit allergens.

As with the studies from Europe, it is very difficult to say with certainty if the fruit-related reactions were IgE mediated or not. Three studies reported on both IgE and non-IgE mediated reactions, but only one study tested for the presence of IgE and it is not clear if the test were performed to the particular fruit. Thirteen studies reported on IgE mediated reactions but only five of these tested SPT/Specific IgE, once again it was not clear if the reactions were IgE mediated or not.

1.2.6.11. Milk/dairy allergy prevalence across Europe

In total, forty studies looked at the prevalence of cow’s milk allergy in Europe. The studies were from Denmark, Estonia, France, Finland, Germany, Greenland, Hungary, Iceland, Ireland, Italy, Norway, Portugal, Spain, Sweden, The Netherlands, Turkey and the United Kingdom. Data was published between the years 1982 and 2012 and included all age groups.

Twenty-two studies reported prevalence rates based on self (or parentally) reported allergy. The highest self-reported rate of cow’s milk allergy was 21% (95% CI: 19.9-22.1%), in a large Spanish

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study of 6-13 year old children (Martinez-Gimeno 2000). The lowest rate of parentally reported cow's milk allergy was 0% (95% CI: 0-6.1%) in Ireland (Kilgallen 1996), however this was in a study of infants aged 0-6 months, which is an age at which symptoms may not yet have fully manifested. The same study reported a 0% (95% CI: 0-3.1%) prevalence of parentally reported allergy to yoghurt at age 24-36 months old. The lowest self-reported prevalence was found in a large study of adults in Turkey (0.2% (95%CI: 0.2-0.4%)) (Gelinicik 2008).

Seventeen studies reported sensitisation rates; seven using sIgE, ten using SPT only and three using both SPT and sIgE. The highest rate of positive sIgE was 25.8% (95% CI: not reported) in 2 year old children in Estonia (Julge 2001), although very surprisingly 0% (95% CI: not reported) of this sample had positive skin prick tests, which was the lowest reported level of positive SPT overall. The lowest rate of positive sIgE in adults was 1.0 (95% CI: 0.0–5.5) (Isolauri 2004). The lowest rate of positive sIgE in children was 0.5% (95% CI: 0.2-1.2%) in a study of children aged 5-18 years from Greenland (Krause 2002). The highest rate of positive SPT in adults was 14.7% (95% CI: 8.9-23.0%) in Hungary (Bakos 2006) and in children 3.9% (95%CI: 2.9-5.2%), in a study of German children aged 5-6 years (Schafer 1999).

Prevalence of milk/dairy allergy as determined by sensitisation (SPT or specific IgE) plus clinical history was reported in six studies. The only study to do so in an adult population reported a rate of 0.1% (95%CI:0.0-0.3%) based on SPT and history (Zuberbier 2004). A prevalence of 0.2% (95% CI: 0.0-0.8%) was reported in 8-18 month old infants in Turkey using specific IgE testing and history (Kucukosmanoglu 2008b). One study assessed the prevalence in older children, aged 4 years old, finding a prevalence of 1.8% (95% CI: 1.3-2.4%) (using specific IgE testing and history) in Sweden (Ostblom 2008a).

Twelve studies used either open or double blind food challenges. The highest rates of challenge-proven cow's milk allergy was 2.3% (95% CI: 1.5-3.3%) in a Dutch study of infants (Schrander 1993). The lowest prevalence rate reported was 0.0% (95% CI: 0.0-4.2%), in a study of <3 year old children in Denmark (Osterballe 2005). In an adult population from Turkey, one study reported a prevalence of 0.0% (95%CI: 0-0.4%) using history and DBPCFC (Gelincik 2008). The highest rate in adults was 0.2% (95% CI: 0.1-1.0%) in a study conducted in Denmark (Osterballe 2005). One study used atopy patch testing (Ronchetti 2008) and reported a prevalence rate of 4.1 % (95% CI: 1.9-8.2%) in 13 year old children.

Milk allergy is by far the most difficult food allergy to classify in terms of IgE and non-IgE mediated symptoms. It is the clinically most complex food allergy seen in young children with many of them suffering from both IgE and non-IgE mediated symptoms. Twenty-nine studies reported symptoms of both IgE and non-IgE mediated cow's milk allergy with only 14 studies confirming the presence of IgE by SPT or specific IgE testing. Nine studies reported rates of IgE mediated cow's milk allergy only and these studies have all utilised SPT or specific IgE tests.

1.2.6.12. Milk/dairy allergy prevalence in different regions of the world

Twenty-nine studies looked at the prevalence of cow's milk allergy outside Europe. This included studies from Australia, Canada, China, Colombia, Ghana, Hong Kong, Israel, Korea, Taiwan, Thailand, United Arab Emirates and United States of America. Data was published between the years 1973 and 2012 and included all age groups.

Fourteen studies reported prevalence rates based on self (or parentally) reported allergy. The highest rates in children and adults were both reported in studies from the USA; 13.1 % (95% CI: 10.3-16.6%) for a group of one year olds (Bock 1987) and 10.5% (95%CI: 8.1-13.6%) in a study of adults (Greenhawt 2009). The lowest parentally reported prevalence rate was 0.2% (95% CI: not reported) in

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a study of 5-16 year old children in Ghana (Obeng 2011).The lowest self reported rate in adults was 1.9% (95% CI: 1.56-2.21%) in a Canadian study (Soller 2012).

Eight studies reported sensitisation rates; four using sIgE and four using SPT. No studies measured both sIgE and SPT. A study conducted in the US reported the highest rate of positive sIgE in adults (4.9% (95% CI: not reported)) and children (22% (95% CI: not reported)) (Liu 2010). The only study that measured SPT in adults (Woods 2002), reported a sensitisation rate of 0.7% (95% CI: 0.2-2.1%). In children, the lowest sensitisation rate using SPT was 2.7% (95% CI: 1.5-4.7%) in China (Chen 2011) and the highest 6.5% (95%CI: 4.4-9.6%), also in China (Hu 2010).

Sensitisation plus clinical history was reported as a method of diagnosis in only three studies, the first of which reported a 0% (95% CI: 0-1.0%) prevalence rate in a sample of adults in Australia. A similar prevalence, 0.3% (95% CI: 0.2-0.5%), was reported by a study of children aged 0-2 years old in Israel (Dalal 2002). A study of children aged 6-8 years in Taiwan reported much higher prevalence rates of between 6.2-14.5% using sIgE plus clinical history (Wan 2012).

Three studies used open food challenges to determine the prevalence of milk/dairy allergy. A study of 3-7 year olds in Thailand reported the lowest prevalence rate, 0% (95% CI: 0-1.1%), of confirmed milk allergy (Lao-araya 2012). The other two studies (Hu 2010, Chen 2011) were both conducted in infants in China and reported prevalence rates of 3.5% (95% CI: 2-5.9%) and 1.3% (95% CI: 0.5-2.9%) respectively.

Bock 1987 reported a prevalence of 5% (95% CI: 3.3-7.4%) in one year old children using a combination of history, SPT and oral food challenge to determine a diagnosis of “probable or confirmed” food allergy. Similarly, Chen 2012 reported a prevalence of 3.5% (95% CI: 2.2-5.4%) in children under 2 years old in China, using a combination of clinical history and/or SPT and/or oral food challenge and/or elimination diet. A study of 0-2 year old children in Israel reported a lower prevalence rate of 1.1% (95% CI: 0.9 – 1.2), also using a combined method of a clear history, SPT and/or food challenge (Katz 2010).One study used IgG tests to diagnosis cow’s milk allergy, reporting a very high prevalence rate of 24.5% (95% CI: 23.8-25.3%) in adults in China (Sai 2011), although as noted before prevalence data from IgG testing should be interpreted with caution.

The picture of IgE vs. Non-IgE mediated cow’s milk allergy is very different in the rest of the world than what is reported in Europe. Twenty one studies reported on IgE mediated cow’s milk allergy, with eight studies not confirming the presence of IgE by appropriate tests. Only five studies reported on symptoms of both IgE and non-IgE mediated cow’s milk allergy and three of these did not test for the presence of IgE. One study used IgG tests for diagnosis.

1.2.6.13. Mustard allergy prevalence across Europe

There was only one study which examined the prevalence of allergy to mustard. This was conducted in a French population of 5-17 year olds, 3% (95% CI: 2.1-4.3%) of which self-reported adverse reactions to mustard (no distinction was made between likely IgE or non-IgE mediated reactions; Touraine 2002).

1.2.6.14. Mustard allergy prevalence in different regions of the world

There were no studies on mustard allergy in other regions of the world.

1.2.6.15. Peanut allergy prevalence across Europe

The peanut allergy prevalence data was derived from 11 countries, including Denmark, France, Germany, Greenland, Hungary, Iceland, Norway, Sweden, The Netherlands, Turkey and the UK. The

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data was published from 1996 to 2012 and the age range of the participants ranged from birth – 97 years.

Fifteen studies looked into the self-reported prevalence of peanut allergy. The lowest prevalence rate, 0% (95% CI: 0-1.5%) was reported in 18 month olds from Iceland (Kristjansson 1998). The highest was reported for a group of 15-17 year olds from France ((15% (95%CI:13-17.3%)) (Touraine 2002). Two studies, both conducted in the UK, reported prevalence based on a clinical history of peanut allergy. This ranged from 0.2% in 0-14 year olds (Emmett 1999) to 0.4% in 4-6 year olds (Lack 2003) to 0.5% in those older than 15 years (Emmett 1999). Only one study focussed on clinician diagnosed peanut allergy with the lowest prevalence figures seen in 1 year olds from Sweden (0.2% (95% CI: 0.1-0.4)) (Ostblom 2008) and the highest from 8 year olds in the same study (4% (95%CI:3.4-4.8)) (Ostblom 2008).

Thirteen studies reported prevalence of peanut sensitisation based on skin prick test results and seven determined by serum-specific IgE levels. In the younger cohorts (0-3 years old), the rates of positive SPT ranged from 0.4% (95% CI: 0.0-1.2%) (Venter 2008) to 2.8% (95% CI: 1.5-5.3%) (Ro 2012). In the older children (>3 years) positive SPT results ranged from 0.7% (95% CI: 0.5-1.0%) (Mustafayev 2012) to 5.1% (95% CI: 3.8-6.8%) (Nicolaou 2010). In adults, the sensitisation rates determined by positive SPT were between 6.4% (95% CI: 2.8-13.2%)(Bakos 2006) and 6.8% (95% CI:not reported) (Schafer 2001). Similarly, for specific IgE levels, in the younger cohorts (0-3 years old) only one study from Norway determined specific IgE levels in younger children reporting a rate of sensitisation of 3.4% (95% CI: 1.9 – 6%) (Ro 2012). In the older children (>3 years) prevalence of sensitisation to peanut ranged between 2.6 % (95% CI: 1.8 – 3.8%) (Krause 2002) and 12.2% (95% CI: 9.7 – 15.2%) (Nicolaou). In adults sensitisation rates to peanut were reported between 0% (95% CI: 0 – 12.0%) (Bakos 2006) and 3.1% (95% CI: 2.3 – 4.2%) (Bjornsson 1996).

Six studies based peanut allergy prevalence rates on a good clinical history plus a positive SPT. The prevalence rates determined using this method ranged from 0.0% in 18 month olds in Iceland (95% CI: 0-1.5%) (Kristjansson 1999) and 18 years olds in Turkey (95% CI: 0.0 – 0.1%) (Gelincik 2008) to 0.6 (95% CI: 0.4-1.0%) in a whole population in Germany (Zuberbier 2004). Only one study based peanut allergy prevalence rates on a good clinical history plus a positive serum specific IgE result. This study was conducted in Sweden and found a prevalence of 2.4% (95% CI: 1.9-3.1%) (Ostblom 2008a)

Five studies used open food challenge and a good clinical history and eight studies used a good clinical history plus DBPCFC to diagnose peanut allergy. Based on OFC and a good clinical history the highest prevalence rate was 1.4% (95% CI: 0.9-2.3%) reported in 3-4 year olds children (Grundy 2002) and based on DBPCFC and history the highest prevalence rate reported was 2.8% (95% CI: 1.8-3.8%) in 3-6 year old children (Hourihane 2007). Both of these studies were conducted in the UK. Four studies utilised a good clinical history plus positive SPT, and/or a positive food challenge (either OFC or DBPCFC) to determine prevalence rates. The highest rate was reported by Nicolaou 2010 in 8 year old children as 1.9% (95%CI:1.2-2.9%).

Peanut allergy is classically considered to be an IgE-mediated allergy. Of the studies examining the prevalence of peanut allergy in Europe, 15 assessed both IgE and non-IgE mediated peanut allergy (although in two of these studies, they did not perform tests to determine the presence or absence of IgE) and 13 IgE-mediated allergy only.

1.2.6.16. Peanut allergy prevalence in different regions of the world

A number of studies outside of Europe have looked at the prevalence rates of peanut allergy. The countries included Australia, Canada, China, Ghana, Hong Kong, Israel, Korea, Philippines,

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Singapore, Taiwan, United Emirates and the USA. The studies were published between 1987 and 2012 and included all ages.

Nine studies looked into the self-reported prevalence of peanut allergy. The lowest prevalence rate (0.1% (95% CI: 0.1-0.2%)) was reported in 12 – 15 year olds from Korea (Oh 2004) and the highest in an adult group from the US (8.4% (95% CI: 6.2%-11.2%)) (Greenhawt 2009). Seven studies, mostly questionnaire based studies from the US and Canada, reported prevalence based on a reported clinical history (in some cases with reported history of a clinician diagnosis) of peanut allergy ranging from 0.1% (95% CI: 0.0 – 0.2%) in 0-2 years olds in Israel (Dalal 2002) to 2.8% (95% CI: 2.3 – 3.4%) in 3-5 year olds in the US (Gupta et al. 2011). Only three studies focussed on clinician diagnosed peanut allergy with the lowest prevalence figures reported in a study of adults from Canada (0.3% (95%CI:0.18-0.34)) (Ben-Shoshan 2010) and the highest in a study of 6-9 year olds from the UAE (2.3% (95%CI:1.1-4.4)) (Al-Hammadi 2010).

Five studies reported data on skin prick test results and four studies determined specific IgE levels. In the younger cohorts (0-3 years old), the rates of positive SPT ranged from 0.3% (95% CI: 0.0-2.1%) (Hu 2010) to 6.4% (95% CI: 5.5-7.3%) (Osborne 2011). Woods 2002 reported figures of 5.7% (95% CI: 3.8-8.3%) in adults in Australia and Arbes 2005 a figure of 8.6% (95% CI: 8.1-9.2%) in all ages in the US, indicating the lack of studies of using SPT data in countries outside of Europe. Different age cut-offs were used to describe sensitisation rates to peanut allergens measured by specific IgE levels, but Kumar 2011 report a very high sensitisation rate of 13.5% (95% CI: 11.6-15.7%) in children under 6 years in the US. The highest reported sensitisation rates in adults were 8.7% (95% CI: not reported) in 20 – 39 year olds in the US (Liu 2010). This study also reported a sensitisation rate of 7.6% (95%CI:not reported) for all ages (Liu 2010).

Only two studies based peanut prevalence rates on a good clinical history plus a positive SPT. Dalal 2002 found no peanut allergy in 0-2 year olds in Israel and Woods 2002 reported a rate of 0.4% (95% CI: 0.1-1.8%) in 26-50 year olds in Australia. In the only study outside of Europe to utilise food challenges to assess the prevalence of peanut allergy Osborne 2011 2.9% (95% CI: 2.2-3.5) of 12-15 month olds had peanut allergy, based on open food challenges. Four studies utilised other methods to diagnose peanut allergy. These studies have used varied methodologies which makes them difficult to compare, but the prevalence rates reported range between 0.3% in an elderly US population (Liu 2010) up to 2.7% (95% CI: not reported) in 6-19 year olds in the US.

Peanut allergy is classically considered to be an IgE-mediated allergy. Of the studies examining the prevalence of peanut allergy outside of Europe, two assessed both IgE and non-IgE mediated peanut allergy. The remainder of the studies assessed IgE-mediated allergy only, although of these nine did not utilise SPT or SIgE to determine the presence or absence of IgE and one did not clearly define how they determined that the allergy was IgE-mediated.

1.2.6.17. Sesame allergy prevalence across Europe

Studies looking at the prevalence of sesame allergy in Europe were from four countries: France, Germany, Hungary and the United Kingdom. Eight studies from Europe were reported between 1999 and 2008 and all ages were studied.

Self-reported sesame allergy was investigated in three studies, with the highest prevalence seen in France where 1.5% (95% CI: 0.9-2.4%) of 5-17 year olds self-reported an adverse reaction (Touraine 2002). The lowest rate was found in the United Kingdom where, across all age groups, 0% (95% CI: 0.0-0.1%) self-reported sesame allergy (Emmett 1999). Sensitisation to sesame measured by SPT was reported in four studies. Roberts 1999 reported the lowest rate of sensitisation, 0.1% (95% CI: 0.0-0.5%), in 7-year-old children in the UK and Venter 2008 the highest, 1.4% (95% CI: 0.7-2.7%), in 3

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year olds also from the UK. Only one study determined specific IgE levels to sesame and found 0% (95% CI: 0.0-4.2%) of the 60-97 year olds in Hungary investigated were sensitised (Bakos 2006). In Germany, a population based study reported prevalence rates based on a positive skin prick test plus a convincing clinical history of 2.2% (95% CI: 1.7-2.7%) (Zuberbier 2004). In the United Kingdom, two studies challenged those with suspected sesame allergy reporting prevalence of between 0.1% (95% CI: 0.0-0.8%) in 6 year olds (Venter 2006) and 0.6% (95% CI: 0.2-1.4%) in 3 year olds (Venter 2008). Pereira 2005 performed a DBCPCFC to sesame in a 15-year old on the IOW, who did not have a positive result (not shown in table).

For those studies examining the prevalence of sesame allergy in Europe, six looked at both IgE and non-IgE mediated allergy (one of which did not utilise SPT or SIgE to determine the presence or absence of IgE) and two IgE-mediated allergy only.

1.2.6.18. Sesame allergy prevalence in different regions of the world

Only four studies that investigated the prevalence of sesame allergy could be identified in other regions of the world, these were from Australia, Canada, Israel and the United States. Studies were reported between 2002 and 2011 and all ages were studied. Self reported sesame allergy was investigated in a Canadian study, with the highest prevalence reported in children under the age of 18 years 0.2% (95% CI: 0.0-0.4%) (Ben-Shoshan 2010) and the lowest rate in adults 0.1% (95% CI: 0.0-0.1%) (Ben-Shoshan 2010). Sensitisation (determined by skin prick test) to sesame was observed in 1.6% (95% CI: 1.2-2.1%) of 12-15 month olds in Australia (Osborne 2011). Three studies looked at a clinical history of sesame allergy and reported figures ranging from 0% (95% CI: 0.0-0.1%) in the US (Sicherer 2010) to 0.2% (95% CI: 0.0-0.4%) in Canada (Ben-Shoshan 2010) and 0.2% (95% CI: 0.1-0.3%) in Israel (Dalal 2002). Two studies reported prevalence rates for sesame allergy based on open food challenges, with a study conducted in the UK reporting prevalence rates of 0.6% (95% CI: 0.2-1.4) in 3 year olds and 0.1% (95% CI: 0-0.8) in 6 year olds (Venter 2008), and a study conducted in Australia reporting a rate of 0.7% (95% CI: 0.4-1.0) in 12-15 month olds (Osborne 2011). For those studies examining the prevalence of sesame allergy outside of Europe, two looked at both IgE and non-IgE mediated allergy (one of which did not utilise SPT or SIgE to determine the presence or absence of IgE) and two IgE-mediated allergy only.

1.2.6.19. Soya allergy prevalence across Europe

There were 15 studies that looked at soya allergy prevalence across Europe. The countries included Denmark, Germany, Hungary, Iceland, Sweden, The Netherlands and the United Kingdom. The data was reported from 1994 to 2008 and all ages were included.

Eight studies reported prevalence based on self-reported soya allergy with the highest prevalence reported by a study conducted in Sweden in 4 year olds (1.2%, 95% CI: 0.8-1.7%) (Ostblom 2008a) and the lowest prevalence reported by a study conducted in the United Kingdom, with 0% (95% CI: 0.0-0.1%) of those older than 15 years self-reporting an adverse reaction to soya (Emmett 1999). Only one study reported the prevalence of clinician diagnosed soya allergy, which found the following prevalence rates: 0.2% (95% CI: 0.1-0.4%) in 1 year olds and 0.8% (95% CI: 0.5-1.2%) in 4 and 8 year olds in Sweden (Ostblom 2008a).

Four studies reported sensitisation data based on a positive skin prick test with the highest sensitisation rate reported by a Hungarian study of 20-69 year olds (8.3%, 95% CI: 2.2-23.6%) (Bakos 2006) and the lowest in a group of 7 year olds from the United Kingdom, with only 0.2% (95% CI: 0.0-0.7%) having a positive skin prick test to soya. Four studies used serum-SIgE tests and reported sensitisation rates ranging from 2.1% (95% CI: 1.4-3.0%) in a group of 20-44 year olds in Sweden (Bjornsson 1996) to 3.7% (95% CI: 1.2-9.7%) in 60-97 year olds in Hungary (Bakos 2006). When a

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convincing history was combined with sensitisation, prevalence of soya allergy ranged from 0% (95% CI: 0.0-1.4%) in 18 month olds in Sweden (Kristjansson 1999) to 1.6% (95% CI: 1.1-2.1%) of 4 year olds, also in Sweden (Ostblom 2008a). Only one study performed a double-blind placebo-controlled food challenge, reporting 0% prevalence to soya in 0-22 year olds (Osterballe 2009). For those studies examining the prevalence of soya allergy in Europe, ten looked at both IgE and non-IgE mediated allergy (two of which did not utilise SPT or SIgE to determine the presence or absence of IgE) and five IgE-mediated allergy only.

1.2.6.20. Soya allergy prevalence in different regions of the world

There were 13 studies conducted in Canada, China, Ghana, Israel, Korea, Taiwan, Thailand and the United States. The data was published from 1987 to 2012 and all ages were included. Seven studies presented data for self-reported adverse reactions to soya with the lowest rate found in Korea; affecting in 0.1% (95% CI: 0.1-0.2%) of 12-15 year olds (Oh 2004). The highest rate was reported by Bock 1987, with 2.7% (95% CI: 1.2-4.2%) of 0-3 year olds in the United States reporting soya allergy. Four studies reported the prevalence of soya allergy based on clinical history and/or clinician diagnosis soya allergy with 0% (95% CI: 0.0-0.2%) of 0-2 year olds in Israel (Dalal 2002) and 0.6% (95% CI: 0.4-0.8%) of 11- 13 year olds in the United States (Gupta 2011) diagnosed with soya allergy. One study reported sensitisation based on skin prick test data, with sensitisation rates varying from 0.5% (95% CI: 0.1-2.1%) in 0-2 year olds in 2009 to 1% (95% CI: 0.3-3.1%) of 0-2 year olds in 1999 (Hu 2010). One study combined clinical history and sensitisation reporting a prevalence of soya allergy of 0% (95% CI: 0.0-0.1%) in 0-2 year olds in Israel (Dalal 2002). No studies outside of Europe used food challenges to confirm soya allergy. One study measured IgG levels reporting a prevalence of 7.2% (95% CI: 6.6-7.7%) of adults in China (Sai, 2010) however caution should be applied to these results as IgG is not a true and accurate measure of food allergy. For those studies examining the prevalence of soya allergy outside of Europe, 11 looked at both IgE and non-IgE mediated allergy (seven of which did not utilise SPT or SIgE to determine the presence or absence of IgE), four IgE-mediated allergy only and one IgG-mediated only.

1.2.6.21. Tree Nuts allergy prevalence across Europe

The tree nut prevalence data was derived from 11 countries, including Finland, Germany, Greenland, Hungary, Iceland, Norway, Spain, Sweden, The Netherlands, Turkey and the UK. The data was published from 1982 to 2009 and all ages were included. The discussion will divide the results into all nuts- unspecified, hazelnuts, walnuts, almond, pistachio nuts, brazil nuts, cashew nuts and pecan nuts.

All nuts unspecified

Studies where the particular nut(s) studied were not reported have mainly focussed on self-reported "nut" allergy. The lowest rate of self-reported nut allergy was in Turkey amongst a group of adult respondents (0.1% (95% CI: 0-0.6%) (Gelincik 2008) and the highest rates were reported in Spain amongst 6-13 year olds (6.9% (95% CI: 6.2-7.6%)) (Martinez-Gimeno 2000).

Only one study (conducted in Sweden) reported results based on SPT and a clinical history, which found that no parents reported their 18 month old to have a "nut" allergy (Kristjansson 1999). In addition, one study looked at clinician diagnosed nut allergy and found that 0.1% (95% CI: 0-0.6) of one year olds, 0% (95% CI: 0-0.6%) of two year olds, 0.5% (95%CI: 0.2-1.4%) of three year olds and 0.4% (95% CI: 0.1-1.2%) of four year olds in Finland suffered from a "nut" allergy (Pyrhonen 2009). For those studies examining the prevalence of unspecified tree nut allergy in Europe, all ten looked at both IgE and non-IgE mediated allergy, six of which did not utilise SPT or SIgE to determine the presence or absence of IgE.

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Hazelnuts

Nine studies have examined the prevalence of hazelnut allergy. The lowest rate of reported hazelnut allergy was amongst 6-9 year olds in Turkey (0.3% (95% CI: 0.1-0.6%)) (Orhan 2006) and the highest rates amongst 10-11 year olds, also in Turkey (1.5% (95%CI:1.2-1.8%)) (Mustayev 2012). Sensitisation to hazelnut was tested by using SPT in five studies, with the lowest rates reported by Roberts 2005 in a 7-year-old cohort in the UK (0.1% (95%CI:0-0.5%)) and the highest in a group of of 25-74 year olds in Germany (11.3% (95% CI: not reported)) (Schafer 2001). Only one study measured specific IgEs to hazelnut in an adult and elderly population in Hungary and found rates of 0.0% – 9.7% with the highest figures seen in the 60 – 97 year group (Bakos 2006). When prevalence rates were determined by combining SPT and a good clinical history (four studies), the lowest rates were reported for those older than 18 years in Turkey (0% (95% CI: 0-0.1%)) (Gelincik 2008) and the highest rates in Germany in a whole population (5.9% (95% CI: 5.1-6.8%), Zuberbier 2004). Utilising a good clinical history, positive SPT and/or a positive OFC/DBPCFC, Venter 2008 found that 0.1% (95% CI: 0.0-0.2%) of 3 year olds suffer from a hazelnut allergy in the UK. For those studies examining the prevalence of hazelnut allergy in Europe, four looked at both IgE and non-IgE mediated allergy and five IgE-mediated allergy only.

Walnut allergy

Only six studies in Europe investigated self-reported rates of adverse symptoms to walnut, sensitisation to walnut or prevalence of walnut allergy. In terms of sensitisation, Roberts 2005 found that 0.5% (95% CI: 0.3-1%) of 7 year olds in the UK have a positive SPT to walnut (Roberts 2005). Bakos 2006 found that 3.7% (95% CI: 1.2-9.7%) of 60 – 97 year olds in Hungary showed sensitisation to walnut measured by specific IgE levels (Bakos 2006). A study conducted in Germany found that 1.4% (95% CI: 1.1-1.8%) of respondents were diagnosed with a walnut allergy based on history and SPT and 1.0% (95% CI: 0.7-1.4%) based on history and a positive DBPCFC outcome (Zuberbier 2004).

Three studies from Turkey investigated walnut allergy. Orhan 2009 found that, in a group of 6-9 year olds, 0.3% (95% CI: 0.1-0.6%) reported a problem on ingestion of walnut, 0.1% (95% CI: 0.0-0.3%) were diagnosed with a walnut allergy based on a good clinical history and positive SPT, and 0.0% (95% CI: 0-0.2%) were diagnosed based on DBPCFC and a good clinical history. A further study found that 1.2% (95% CI: 1.0-1.5%) of 10 -11 year olds in Turkey reported a problem on ingestion of walnut and reported prevalence of 0.4% (95% CI: 0.1-1.2%) based on an OFC and a good clinical history (Mustayev 2012). Gelincik 2008 reported that 0.1% (95% CI: 0.1-1.2%) of adults suffered from walnut allergy based on DBPCFC outcome and a good clinical history (Gelincik 2008). For those studies examining the prevalence of walnut allergy in Europe, two looked at both IgE and non-IgE mediated allergy and four IgE-mediated allergy only.

Almond

Five studies in Europe investigated almond allergy. Ostblom 2008a reported that 3.8% (95% CI: 3.1-4.7%) of 4 year olds in Sweden reported problems with almond. In terms of sensitisation, Venter 2008 determined that 0.3% (95% CI: 0.0-1.2%) of 3 year olds in the United Kingdom had a positive SPT to almond and that 0.2% (95% CI: 0.0-0.9%) of 3 year olds had either a positive SPT with a good clinical history and/or a positive OFC/DBPCFC outcome. Also in the UK Roberts 2005 found that 0.5% (95% CI: 0.2-0.9%) of 7 year olds are sensitised to walnut. Bakos 2009 found that no 60-97 year olds in Hungary had positive specific IgE levels to almond. Furthermore, in a study conducted in Iceland and Sweden, the prevalence rates for almond allergy in 18 months old were reported to be 0% (95% CI: 0-1.4%) and 0% (95%CI:0-1.5%) respectively based on skin prick test and history (Kristjansson 1999). For those studies examining the prevalence of almond allergy in Europe, three looked at both IgE and non-IgE mediated allergy and two IgE-mediated allergy only.

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Pistachio
Only one study in Europe investigated pistachio allergy which reported that 0.8 % (95% CI: 0.6-1.1%) of 10-11 year olds in Turkey reported a problem on ingestion of pistachio (Musatayev 2012). This study examined IgE-mediated allergy only.

Pecan
Pecan allergy was only reported in one study in Europe, indicating that 0.2% (95% CI: 0.0-0.5%) of 7-year-olds in the UK are sensitised to Pecan (Roberts 2005). This study examined IgE-mediated allergy only.

Brazil
Sensitisation and allergy to brazil nut was reported in only two, UK-based, studies, one of which examined both IgE and non-IgE mediated allergy and the other IgE-mediated allergy. In younger children Venter 2008 reported 0.3% (95% CI: 0.0-1.2%) of 3 year olds and 0.2% (95% CI: 0.0-0.9%) of 3 year olds to be sensitised to brazil nut. In older children, Roberts 2005 found that 0.5% (95% CI: 0.3-1%) of 7-year-olds in the UK are sensitised to brazil nut.

Cashew
Three studies from the UK (one of which examined both IgE and non-IgE mediated allergy and the other two IgE-mediated allergy) found that 0.2% (95% CI: 0.0-1.0) of 3 year olds (Venter 2008), 0.1% (95% CI: 0.0-0.2%) of 4 year olds (Tarik 1996) and 0.4% (95% CI: 0.2-0.8%) of 7-year olds (Roberts 2005) are sensitised to cashew nut. One study confirmed cashew nut allergy by food challenge in 0.1% (95% CI: 0.0-0.2%) of 3 year olds (Venter 2008).

1.2.6.22. Tree Nuts allergy prevalence in different regions of the world

Nine studies conducted in non-European countries investigated tree nut allergies with the majority of studies coming from the US and Canada. The studies were published between 1997 and 2008 and included all ages.

All nuts unspecified
Self-reported allergy to “nuts” was reported in three studies. The lowest rates were reported in 22 – 44 year olds in Australia (0.6% (95% CI: 0.2-1.6%) (Woods 1998) and the highest rates reported in 4-6 year olds in Singapore (4.7% (95% CI: 4.1-5.4%)) (Shek 2010). Prevalence of “nut” allergy based on clinical history was reported in four studies with the lowest rates from the US in those under 18 years old (0.2% (95% CI: 0.1-0.3%)) (Sicherer 1997, Sicherer 2002) and the highest in those over 18 years old (1.6% (95% CI: 1.4-1.9%)) (Sicherer 1997). One study from Korea reported the prevalence of clinician diagnosed nut allergy in 0-12 months old children to be 0.7% (95% CI: 0.3-1.4%) (Kim 2011). For those studies examining the prevalence of unspecified tree nut allergy outside of Europe, two looked at both IgE and non-IgE mediated allergy and four IgE-mediated allergy only (although two did not test for the presence or absence of IgE).

Pistachio allergy
Only one study conducted outside of Europe reported (IgE mediated) prevalence to a particular tree nut. Wan 2012 reported that 2.2% (95% CI: 1.4-3.3%) of 6-8 year old Taiwanese children suffer from pistachio allergy based on history and a positive specific IgE level.

1.2.6.23. All other foods, allergy prevalence across Europe

Unsurprisingly, allergies to numerous less common foods have been reported in the literature in a wide number of countries, both in and outside Europe and at all ages. In Europe, 27 studies looked at the prevalence of allergy to “other foods”. The less common food allergens that were reported

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included (but were not limited to): vegetables, such as peas, tomato, spinach, eggplant and carrot, in addition to chocolate, garlic, honey, pork, black pepper, pickle, cocoa, potato, sugar, chicken and beef. Generic terms such as "colourings" "additives "junk food" and "soft drinks" were also reported as allergens in the titles of journal articles. The majority of such studies used self-report methods to determine prevalence. SPT plus clinical history was used by only two studies reporting prevalence of allergies to carob, carrageen and guar gum in adults in Germany (all < 1%) (Zuberbier 2004) and to pea of 0% (95% CI: 0.0-1.5%) in 18 month old infants in Iceland (Kristjansson 1999).

Only seven studies used food challenges when reporting prevalence rates (Gelincik 2008; Mustafayev 2012; Orhan 2009; Osterballe 2005; Venter 2006; Venter 2008; Zuberbier 2004). It is difficult and perhaps illogical to combine and summarise these studies due to the heterogenity of the allergens and populations studied. However, with the exception of Zuberbier 2004, who reported a 1.8% (95% CI: 1.4-2.4%) prevalence of challenge proven allergy to "vegetables" (n = 3156), the other six studies all reported prevalence rates of less than 0.5%. For those studies examining the prevalence of other food allergies in Europe, 25 looked at both IgE and non-IgE mediated allergy (11 of which did not test for the presence or absence of IgE) and three IgE-mediated allergy only.

1.2.6.24. All other foods, allergy prevalence in different regions of the world

Outside of Europe, 16 studies looked at the prevalence of allergy to "other foods". The studies were conducted in Australia, Canada, China, Colombia, Ghana, Hong Kong, India, Israel, Korea, Taiwan, Thailand, United Arab Emirates and the United States. Unusual allergens that were reported in countries outside of Europe that were not reported in Europe included cassava, cocoyam, sorghum and okra in Ghana (Obeng 2011); perilla seeds and buckwheat in Korea (Kim 2011; Oh 2004); duck in Thailand (Santadusit 2005); and monosodium glutamate in Australia (Woods 1998). The majority of the studies relied on self-report measures as a means of diagnosis, with none of the studies using food challenges. Sai (2011) used IgG as a measure of food allergy, and Leung (2009) reported self-reported clinician-diagnosed prevalence of allergy to several foods. For those studies examining the prevalence of other food allergies outside of Europe, three looked at both IgE and non-IgE mediated allergy (one of which did not test for the presence or absence of IgE), twelve IgE-mediated allergy only (seven of which did not test for the presence or absence of IgE) and one IgG-mediated allergy only.

1.2.6.25. Prevalence of allergy to any food across Europe

We have reviewed all the included European studies in our systematic review to identify those studies which have reported on rates of diagnosed food allergy based on objective measures including clinician diagnosed food allergy/good clinical history plus supporting test or those who had either an open (OFC) or double blind placebo controlled food challenges (DBPCFC). We were able to identify a total of eight studies carried out in Denmark, Finland, Germany, United Kingdom and Turkey.

Denmark

Eller 2009 investigated food allergy in Danish children aged 0-6 years and reported that 3.6% of children suffered from any food allergy by 6 years based on OFC or DBPCFC (95% CI: 2.3 – 5.4%). Self-reported FA to any food by the age of 6 years was 11.6% (95% CI: 9.2-14.5). The main foods implicated were milk, egg and peanut. Osterballe 2005 reported OFC/DBPCFC confirmed FHS in young adults in Denmark as 1.7% (95% CI: 1.1 – 2.95%). Self-reported FHS was 19.6% (95% CI: 17.0-22.4). The most common allergenic food was peanut followed by additives, shrimp, codfish, cow's milk, octopus and soy.

Looking at young children (0-3 year olds), older siblings and parents of the young children, OFC/DBPCFC-confirmed FHS was 2.4% (95% CI: 1.8-3.2) in the whole population studied and 1.6%

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(95% CI: 0.9-2.6) in the children (Osterballe 2005). Breaking the point prevalence figures into specific age groups the data was: 2.3% (95% CI: 1.3-4.0) at the age of 3 years, 0.0% (95% CI: 0.0-3.3) in those under 3 years, 1.0% (95% CI: 0.3-2.9) in those children over 3 years and 3.2% (95% CI: 2.3-4.5) in the adults. The point prevalence of reported FHS in this study was: 13.0% (95% CI: 11.6-14.7) in all of those studied, 14.1% (95% CI: 12.0-16.5) in the adults and 11.9% (95% CI: 10.0-14.1) in children of all ages. The most common allergenic foods were hen's egg affecting the children 3 years of age and peanut in the adults. Codfish and shrimp allergies were seen in the adults but not in the children.

Germany

Zuberbier 2004 conducted a whole population study in the Germany. The point prevalence of adverse reactions to food confirmed by DBPCFC tests in the Berlin population as a mean of all age groups was 3.6% (95% CI: 3.0-4.2%) and 3.7% in the adult population (18-79 years, 95% CI: 3.1-4.4%). Two and a half percent were IgE-mediated and 1.1% non-IgE-mediated. In the children (0-17 years), the prevalence of all FHS was 4.2%; IgE-mediated was 3.5% (95% CI: 2.4-5.1%) and non-IgE-mediated was 0.7% (95% CI: 0.3-1.6%). Foods most commonly identified by oral challenges were apple, hazelnut, soy, kiwi, carrot and wheat. The self-reported lifetime prevalence of any adverse reaction to food in the Berlin population (mean age 41 years) was 34.9%.

Turkey

Gelincik 2008 reported FHS based on DBPCFC in adults (>18 years) in Turkey as 0.1% (95% CI: 0.05– 0.18). Adding those with non-allergic FA, the figures were 0.3% (95% CI: 0.2 – 0.4). The foods most commonly implicated in the reactions were tomato, cocoa and egg. The lifetime prevalence or self-reported FA and NAFA of all ages reported in the paper was 9.5% (95% CI: 8.9-10.0). Orhan 2009 reported DBPCFC confirmed FA in 6-9 year old Turkish children as 0.8% (95% CI: 0.5 – 1.1). Using a positive SPT and a clear history as the diagnostic end point, the recorded prevalence was 1.8% (95% CI: 1.3-2.3). The most common allergenic foods were beef, cow's milk, cocoa, egg and kiwi. Self-reported food allergy in this group was 5.7% (95% CI: 4.8-6.6). In another study focusing on IgE-mediated FA only, Mustayev 2012 reported a prevalence rate of 0.1% (9/6963; 95% CI: 0.1-0.3) in adolescents in Turkey. The most common foods involved in allergic reactions were walnut and beef, followed by egg, peanut, spinach, kiwi, cheese, hazelnut and peach. A total of 2.2% (152/6963; 95% CI 1.9-2.6) of parents reported a food related problem.

United Kingdom

Pereira 2005 studied 11 and 15 year old children in the UK. In the 11 year old cohort FHS confirmed by DBPCFC was 0.1% (95% CI: 0-0.7%) and OFC-confirmed FHS: 1.0% (95% CI: 0.5-2.0%). In the 15 year old cohort, DBPCFC-confirmed FHS was 0.5% (95% CI: 0.2-1.4%) and OFC-confirmed FHS 1.1% (95% CI: 0.5-2.1%). Using a positive SPT and/or a good clinical history or a positive food challenge as diagnostic end point, the figures were (at 11 years) 2.3% (95% CI: 1.5-3.6) based on a clear clinical history and/or OFC or 1.4% (0.8-2.5) based and/or a clear clinical history or DBPCFC. At 15 years, based on a clear clinical history and/or OFC, the rates were 2.2% (95% CI: 1.4-3.6) or 2.1% (95% CI: 1.3-3.4) based on a clear clinical history and/or DBPCFC. Among the 11-year-olds, the foods most commonly implicated in FHS were peanuts, tree nuts, egg, milk, shell fish, gluten, green beans, cheese, kiwi, tomato, and additives. Among the 15-year-olds, the foods implicated were peanut, tree nuts, gluten, wheat, shellfish, egg, milk, and additives. Self-reported rates of FHS were 11.6% (95% CI: 9.5-14.1%) at 11 years and 12.4% (95% CI: 10.3-15.0%) at 15 years.

Venter et al studied a birth cohort age 1-3 years and a separate cohort at the age of 6 years. The prevalence of FHS defined by a positive OFC was 2.8% (95% CI: 1.9-4.1) at 1 year, 1.0% (95% CI: 0.6-2.0) at two years and 0.8% (95% CI: 0.4-1.6) at 3 years. FHS diagnosed using a positive DBPCFC was 1.3% (95% CI: 0.8-2.3) at one year, 0.1% (95% CI: 0.0-0.7) at two years and 0.0% at 3 years. Using a clear clinical history and/or a positive OFC/DBPCFC as diagnostic end point the figures were

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for OFC: 3.0% (95% CI: 2.1-4.3) at one year, 2.5% (95% CI: 1.5-3.7) at two years and 3.0% (95% Ci: 2.1-4.4) at 3 years. For DBPCFC this was 2.7% (95% CI: 1.8-3.9) at one year, 2.1% (95% CI: 1.3-3.3) at two years and 2.9% (95% CI: 2.0-4.2) at 3 years. Using open food challenge and a good clinical history, the cumulative incidence of FHS was 6.0% (95% CI: 4.6-7.7). Based on DBPCFC and a good clinical history, the cumulative incidence was 5.0% (95% CI: 3.7-6.5). Overall, 33.7% of parents reported a food-related problem. The main foods implicated in the allergic reactions were milk, egg and peanut.

Looking at different group of children recruited at 6 years of age, based on open food challenge and/or suggestive history and skin tests, the prevalence of FHS was 2.5% (95% CI: 1.5-3.8). Based on double-blind challenges, a clinical diagnosis or suggestive history and positive skin tests, the prevalence was 1.6% (95% CI: 0.9-2.7). Self-reported prevalence of FHS was 11.8% (95% CI: 9.6-14.2) in this cohort. Milk, peanut and wheat were the key food allergens amongst those with positive challenges.

1.2.6.26. Prevalence of allergy to any food in different regions of the world

Very few studies outside of Europe used food challenge outcome as the final diagnostic point to determine the prevalence of food allergy. Looking at the studies we have identified from our systematic review a total 4 studies have reported on overall food allergy based on food challenge.

China

Chen 2011 studied the prevalence of FA in 0-1 year old children in Chongqing, China and found an overall prevalence of challenge-proven FA of 3.8% in infants (95% CI: 2.5-5.9%). The main foods implicated were egg and milk. Among the parents, 9.3% (46/ 497; 95% CI: 6.9-12.2) reported that their child had adverse food reactions. Looking at the prevalence of FA in 0-2 year olds, Chen 2012 reported an overall prevalence of challenge-proven FA of 5.9% (95% CI: 4.9-7.2%). The most common food allergy was to egg, but cow’s milk, shrimp and fish were also common allergens. Hu 2010 reported on FA in 1999 and 2009 in China and reported that food allergy prevalence increased significantly from 3.5% (11/314; 95% CI: 1.9-6.4) in 1999 to 7.7% (31/401; 95% CI: 5.4-10.9) in 2009 (p= 0.017). The main foods implicated were egg and milk and the rates did not change over the 10 year period. Reported rates of FA was 13.7% (43/314; 95% CI: 10.2-18.1) in 1999 and 16.7% (67/401; 95% CI: 13.3-20.8) in 2009.

Thailand

The study by Lao-araya 2012 focused on IgE mediated food allergy only. The prevalence of IgE-mediated FA confirmed by OFC was 1.1% (95% CI: 0.4-3.0). The five main allergens reported were shrimp, cow's milk, fish, chicken eggs, and ant eggs. Forty-two children (9.3%; 42/452; 95% CI: 6.9-12.4) were reported to have FA.

United States

The study reported by Bock 1987 is one of the first papers reporting FHS based on oral food challenge outside of Europe and the only one ever from the US. Bock 1987 showed that of the 501 children enrolled into the study, 37 (7.7%; 95%CI: 5.6-10.6) were diagnosed with FHS by means of either OFC or DBPCFC. However, 27.7% (95% CI: 23.8-32.0) were thought to have symptoms produced during food ingestion, due to parental reported problems. The most common foods implicated in the allergic reactions were egg and milk.

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Table 1.10: Celery allergy prevalence in European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Touraine (2002)	France	2000-2001	5-17 years	celery/carrot	Both IgE and non-IgE mediated (no SPT or SIgE)	5.5† (4.3-7.1) n=1086	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	celery	Both IgE and non-IgE mediated	-	-	-	-	-	3.5 (2.9-4.2) n=3156	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	celery	Both IgE and non-IgE mediated	-	-	-	9.1† (nr) n=nr	-	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	celery	IgE mediated only	-	-	-	11.1† (3.6-27.0) n=36	2.8† (0.2-16.2) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	celery	IgE mediated only	-	-	-	3.7† (1.2-9.7) n=109	9.2† (4.7-16.6) n=109	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.11: Celery allergy prevalence in non-European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Wan (2012)	Taiwan	Not Reported	6-8 years	celery	IgE mediated only	-	-	-	-	-	-	1.8 (1.1-2.9) n=1010	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

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Table 1.12: Cereals allergy prevalence in European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Osterballe (2005)	Denmark	2000-2001	< 3 years	wheat	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0 (nr) n= 111	-	0† (0.0-4.2) n=111
Osterballe (2005)	Denmark	2000-2001	3 years	wheat	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0 - 1) n=486	0 † (0 - 1) n=486
Osterballe (2005)	Denmark	2000-2001	3-22 years	wheat	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0 (0 - 2) n=301	0 (0 - 2) n=301
Osterballe (2009)	Denmark	2001-2002	22 years	wheat	Both IgE and non-IgE mediated	0.8† (0.4 - 1.8) n=843	-	-	-	-	-	-	-	-	-
Osterballe (2005)	Denmark	2000-2001	>22 years	wheat	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0 - 0.5) n=936	0.1 † (0 - 1) n=936
Pyrhonen (2009)	Finland	2001-2009	1 year	barley/rye	Both IgE and non-IgE mediated (no SPT or SIgE)	1.5 † (0.9-2.7) n=853	-	1.3 † (0.7 - 2.4) n=853	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	2 years	barley/rye	Both IgE and non-IgE mediated (no SPT or SIgE)	1.8 † (1.0-3.0) n=852	-	1.8 † (1.0-3) n=852	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	3 years	barley/rye	Both IgE and non-IgE mediated (no SPT or SIgE)	1.3 † (0.7-2.4) n=784	-	2 † (1.2-3.4) n=784	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	4 years	barley/rye	Both IgE and non-IgE mediated (no SPT or SIgE)	1.8 † (1.1-3.1) n=819	-	2.7 † (1.7 - 4.1) n=819	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	1 year	cereals (oat/maize/rice/ millet/buckwheat)	Both IgE and non-IgE mediated (no SPT or SIgE)	2.3 † (1.5-3.7) n=853	-	1.1† (0.5 - 2.1) n=853	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	2 years	cereals (oat/maize/rice/ millet/buckwheat)	Both IgE and non-IgE mediated (no SPT or SIgE)	2 † (1.2-3.3) n=852	-	0.9 † (0.4-1.9) n=852	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	3 years	cereals (oat/maize/rice/ millet/buckwheat)	Both IgE and non-IgE mediated (no SPT or SIgE)	1.2 † (0.6-2.3) n=784	-	2 † (0.9 - 2.9) n=784	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Pyrhonen (2009)	Finland	2001-2009	4 years	cereals (oat/maize/rice/millet/buckwheat)	Both IgE and non-IgE mediated (no SPT or SIgE)	1.5 † (0.8 - 2.6) n=819	-	2 † (1.2-3.2) n=819	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	1 year	wheat	Both IgE and non-IgE mediated (no SPT or SIgE)	1 (nr) n=261	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	1 year	wheat	Both IgE and non-IgE mediated (no SPT or SIgE)	2.1 † (1.3-3.4) n=853	-	1.6 † (0.9 - 2.8) n=853	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	2 years	wheat	Both IgE and non-IgE mediated (no SPT or SIgE)	1 (nr) n=202	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	2 years	wheat	Both IgE and non-IgE mediated (no SPT or SIgE)	2† (1.2-3.3) n=852	-	2.4 † (1.5-3.7) n=852	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	3 years	wheat	Both IgE and non-IgE mediated (no SPT or SIgE)	0.9 (0.4-1.9) n=784	-	3.1 † (2.0-4.6) n=784	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	4 years	wheat	Both IgE and non-IgE mediated (no SPT or SIgE)	1.1 † (0.5 - 2.2) n=819	-	3.4 (2.3-5) n=819	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	wheat	Both IgE and non-IgE mediated (no SPT or SIgE)	1.5 † (0.9-2.4) n=1086	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	barley	Both IgE and non-IgE mediated	-	-	-	-	-	2.2 (1.7-2.8) n=3156	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	flour	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.5 (0.3-0.8) n=3156	0.1 (0.0-0.3) n=3156
Schafer (2001)	Germany	1997-1998	25-74 years	flour	Both IgE and non-IgE mediated	0.7 † (nr) n=nr	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	oatmeal	Both IgE and non-IgE mediated	-	-	-	-	-	1.2 (0.9-1.7) n=3156	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	rye flour	Both IgE and non-IgE mediated	-	-	-	-	-	1.2 (0.8-1.6) n=3156	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Zuberbier (2004)	Germany	1999-2000	0-80+ years	wheat	Both IgE and non-IgE mediated	-	-	-	-	-	1.2 (0.9-1.7) n=3156	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	wheat	Both IgE and non-IgE mediated	-	-	-	2.8† (nr) n=nr	-	-	-	-	-	-
Zannikos (2008)	Greece	2007	7-13 years	wheat	Both IgE and non-IgE mediated (no SPT and SIgE)	0.2 † (0.0-0.5) n=1988	-	-	-	-	-	-	-	-	-
Krause (2002)	Greenland	1998	5-18 years	wheat	IgE mediated only	-	-	-	-	2.4 † (1.6-3.6) n=1031	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	rye	IgE mediated only	-	-	-	11.1 † (3.6-27.0) n=36	0 † (0-12.0) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	rye flour	IgE mediated only	-	-	-	7.3 † (3.5-14.4) n=109	2.8 † (0.7-8.4) n=109	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	wheat	IgE mediated only	-	-	-	13.9 † (5.2-30.3) n=36	2.8 † (0.2-16.2) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	wheat	IgE mediated only	-	-	-	9.2 † (4.7-16.6) n=109	5.5 † (2.3-12.1) n=109	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	cereals	Both IgE and non-IgE mediated	0.6 † (0.1-2.5) n=324	-	-	-	-	0 † (0-1.5) n=324	-	-	-	-
Ronchetti (2008)	Italy	2005 - 2006	9 years	wheat	Both IgE and non-IgE mediated	-	-	-	0.5 † (0.0-3.5) n=184	-	-	-	-	-	6 † (3.2-10.7) n=184
Ronchetti (2008)	Italy	2005 - 2006	13 years	wheat	Both IgE and non-IgE mediated	-	-	-	1.5 † (0.4-4.8) n=196	-	-	-	-	-	5.6 † (3.0-10.1) n=196
Eggesbo (1999)	Norway	1993-1995	1 year	cereals	Both IgE and non-IgE mediated	0.8 (0.6-1.2) n=3366	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	18 months	cereals	Both IgE and non-IgE mediated	0.2 (0.1-0.5) n=3278	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	2 years	cereals	Both IgE and non-IgE mediated	0.5 (0.3-0.8) n=2979	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	cereals	Both IgE and non-IgE mediated	1.2 † (0.4-3.3) n=328	-	-	-	-	0 † (0-1.4) n=328	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Ostblom (2008 b)	Sweden	1995-2004	1 year	wheat	Both IgE and non-IgE mediated	0.8 † (0.5-1.2) n=3104	-	0.3 † (0.1-0.6) n=3104	-	-	-	-	-	-	-
Ostblom (2008 b)	Sweden	1996-1998	2 years	wheat	Both IgE and non-IgE mediated	0.8 † (0.5-1.2) n=3104	-	0.4 † (0.2-0.7) n=3104	-	-	-	-	-	-	-
Ostblom (2008 b)	Sweden	1998-2000	4 years	wheat	Both IgE and non-IgE mediated	0.5 † (0.3-0.9) n=3104	-	0.4 † (0.2-0.7) n=3104	-	-	-	-	-	-	-
Ostblom (2008 a)	Sweden	1999-2000	4 years	wheat	Both IgE and non-IgE mediated	0.7 † (0.5-1.2) n=2563	-	-	-	4 † (3.3-4.9) n=2563	-	1.3 (1.0-1.9) n=2563	-	-	-
Ostblom (2008 b)	Sweden	2002-2004	8 years	wheat	Both IgE and non-IgE mediated	0.4 † (0.2-0.7) n=3104	-	0.3 † (0.1-0.6) n=3104	-	-	-	-	-	-	-
Bjornsson (1996)	Sweden	1991-1992	20-44 years	wheat	IgE mediated only	-	-	-	-	3.1 † (2.3-4.2) n=1397	-	-	-	-	-
Orhan (2009)	Turkey	2006	6-9 years	corn	IgE mediated only	0.1 † (0.0 - 0.4) n=2739	-	-	-	-	0.1 † (0 - 0.3) n=2739	-	-	0 † (0 - 0.2) n=2739	-
Venter (2008)	United Kingdom	2001-2005	3 years	corn	Both IgE and non-IgE mediated	-	-	-	0.2 † (0-1.0) n=642	-	-	-	-	-	0.1 † (0.0-0.2) n=891
Venter (2008)	United Kingdom	2001-2005	1 year	corn flour	Both IgE and non-IgE mediated	-	-	-	0.1 † (0.0-0.8) n=763	-	-	-	-	-	0.1 † (0.0-0.7) n=900
Venter (2008)	United Kingdom	2001-2005	2 years	corn flour	Both IgE and non-IgE mediated	-	-	-	0.2 † (0.0-1.0) n=658	-	-	-	-	-	0.1 † (0.0-0.8) n=858
Venter (2008)	United Kingdom	2001-2005	1 year	gluten	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0 † (0-0.5) n=900
Venter (2008)	United Kingdom	2001-2005	2 years	gluten	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0.1 † (0.0-0.8) n=858
Venter (2008)	United Kingdom	2001-2005	3 years	gluten	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0.1 † (0.0-0.2) n=891
Venter (2008)	United Kingdom	2001-2005	1 year	wheat	Both IgE and non-IgE mediated	-	-	-	0 † (0-0.6) n=763	-	-	-	-	-	0.4 † (0.1-1.2) n=900
Venter (2008)	United Kingdom	2001-2005	2 years	wheat	Both IgE and non-IgE mediated	-	-	-	0.2 † (0.0-1.0) n=658	-	-	-	-	-	0.3 † (0.1-1.1) n=858

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Venter (2008)	United Kingdom	2001-2005	3 years	wheat	Both IgE and non-IgE mediated	-	-	-	0 † (0-0.1) n=642	-	-	-	-	-	0.2 † (0.0-0.9) n=891
Arshad (2001)	United Kingdom	1993-1994	4 years	wheat	IgE mediated only	-	-	-	0.3 † (0.1- 1) n=981	-	-	-	-	-	-
Venter (2006)	United Kingdom	2003-2004	6 years	wheat	Both IgE and non-IgE mediated	1.3 † (0.6-2.4) n=798	-	-	0.4 † (0.1-1.4) n=700	-	-	-	-	-	0.3 † (0-1.0) n=798
Pereira (2005)	United Kingdom	2002-2003	11 years	wheat	Both IgE and non-IgE mediated	1.3 † (0.7-2.4) n=775	-	-	0.6 † (0.2-1.6) n=699	-	-	-	-	-	-
Pereira (2005)	United Kingdom	2002-2003	15 years	wheat	Both IgE and non-IgE mediated	1.2 † (0.6-2.3) n=757	-	-	1.2 † (0.6-2.5) n=649	-	-	-	-	-	-
Young (1994)	United Kingdom	nr	All ages	wheat	Both IgE and non-IgE mediated	0.9 † (0.8-1.1) n=18880	-	-	-	-	-	-	-	-	-
Emmett (1999)	United Kingdom	1995-1996	15 + years	wheat/gluten	Both IgE and non-IgE mediated	0.4 † (0.3-0.5) n=16420	-	-	-	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.13: Cereals allergy prevalence in non-European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Woods (2002)	Australia	1992-1998	26-50 years	wheat	IgE mediated only	1.3 † (0.5-3.0) n=457	-	-	2.2 † (1.1-4.1) n=457	-	0 † (0-1.0) n=457	-	-	-	-
Woods (1998)	Australia	1998	20-44years	wheat products	Both I gE and non IgE mediated	0.4 † (0.1-1.4) n=669	-	-	-	-	-	-	-	-	-
Soller (2012)	Canada	2008-2009	<18 years	wheat	"likely" IgE mediated (no SPT or SIgE)	0.45 (0.08-0.83) n= nr	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Soller (2012)	Canada	2008-2009	>18 years	wheat	"likely" IgE mediated (no SPT or SIgE)	0.86 (0.63-1.08) n= nr	-	-	-	-	-	-	-	-	-
Hu (2010)	China	1999	0-24 months	wheat	IgE mediated only	-	-	-	0.3 † (0.0-2.1) n=304	-	-	-	-	-	-
Hu (2010)	China	2009	0-24 months	wheat	IgE mediated only	-	-	-	0.5 † (0.1-2.1) n=382	-	-	-	-	-	-
Sai (2011)	China	2008-2009	adults	wheat	IgG mediated only	-	-	-	-	-	-	-	-	-	1.2 † (1.0-1.4) n=12765
Obeng (2011)	Ghana	2006-2008	5-16 years	cereals (millet)	IgE mediated only	0.1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	corn	IgE mediated only	0.2 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	wheat	IgE mediated only	0.3 (nr) n= 1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	wheat flour	IgE mediated only	0 (nr) n= 1407	-	-	-	-	-	-	-	-	-
Morita (2012)	Japan	2009-2010	24-93 years	wheat	IgE mediated only	1.2 † (0.6-2.2) n=935	-	-	-	1.4 † (nr) n= 935	0.2 † (0.0-0.9) n=935	0.2 (0.0-0.9) n=935	-	-	-
Kim (2011)	Korea	2006-2007	0-12 months	wheat	IgE mediated only (no SPT or SIgE)	-	-	0.1 † (0-0.5) n=1177	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	wheat	IgE mediated only (no SPT or SIgE)	0 † (0.0-0.1) n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	wheat	IgE mediated only (no SPT or SIgE)	0.1 † (0.0-0.1) n=14777	-	-	-	-	-	-	-	-	-
Lao-araya (2012)	Thailand	2010	3-7years	wheat	IgE mediated only	0.2 † (0.0-1.4) n=452	-	-	-	-	-	-	-	-	-
Al-Hammadi (2010)	United Arab Emirates (Emirate of Abu Dhabi)	2006	6-9 years	wheat	IgE mediated only (no SPT or SIgE)	-	-	0.5 † (0.1-2.0) n=397	-	-	-	-	-	-	-
Bock (1987)	United States	1980-1984	0-3 years	corn	Both IgE and non-IgE mediated	1.2 † (0.4-2.6) n=408	-	-	-	-	-	-	-	-	0.2 † (0-1.3) n=480

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Gupta (2011)	United States	2009-2010	0-2 years	wheat	IgE mediated (no SPT or SIgE)	-	0.3 (0.1-0.5) n=5429	-	-	-	-	-	-	-	-
Bock (1987)	United States	1980-1984	0-3 years	wheat	Both IgE and non-IgE mediated	0.9 † (0.3-2.3) n=408	-	-	-	-	-	-	-	-	0.2 † (0-1.3) n=480
Gupta (2011)	United States	2009-2010	3-5 years	wheat	IgE mediated (no SPT or SIgE)	-	0.5 (0.3-0.7) n=5910	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	6-10 years	wheat	IgE mediated (no SPT or SIgE)	-	0.4 (0.3-0.5) n=9911	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	11-13 years	wheat	IgE mediated (no SPT or SIgE)	-	0.7 (0.5-0.9) n=6716	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	14-17 years	wheat	IgE mediated (no SPT or SIgE)	-	0.3 (0.2-0.4) n=10514	-	-	-	-	-	-	-	-
Greenhawt (2009)	United States	nr	18 years+	wheat	IgE mediated (no SPT or SIgE)	2.3 † (1.3-4.2) n=513	-	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	All ages	wheat	IgE mediated (no SPT or SIgE)	-	0.4 (0.3-0.5) n=3339	-	-	-	-	-	-	-	-
Vierk (2007)	United States	2001	18 years +	wheat/gluten	IgE mediated only (no SPT or SIgE)	0.6 † (0.4-0.9) n=4482	-	-	-	-	-	-	-	-	0.5 † (0.3-0.8) n=4482

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.14: Egg allergy prevalence in European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Eller (2009)	Denmark	1999-2000	3 months	egg	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	0 (nr) n=nr	-	-
Eller (2009)	Denmark	1999-2000	6 months	egg	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	0.2 (nr) n=nr	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Eller (2009)	Denmark	1999-2000	9 months	egg	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	0.2 (nr) n=nr	-	-
Eller (2009)	Denmark	1999-2000	1 year	egg	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	0.6 (nr) n=nr	-	-
Eller (2009)	Denmark	2000-2001	18 months	egg	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	2.6 (nr) n=nr	-	-
Osterballe (2005)	Denmark	2000-2001	< 3 years	egg	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	0 (nr) n=111	-	1.8 † (0.3 - 7) n=111
Osterballe (2005)	Denmark	2000-2001	3 years	egg	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	1.6 † (0.1 - 3.4) n=486	2.9 (1.7 - 4.9) n=486
Eller (2009)	Denmark	2001-2002	3 years	egg	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	2.3 (nr) n=nr	-	-
Eller (2009)	Denmark	2004-2005	6 years	egg	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	0.7 (nr) n=nr	-	-
Osterballe (2005)	Denmark	2000-2001	3-22 years	egg	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0 - 2) n=301	0 † (0 - 2) n=301
Osterballe (2009)	Denmark	2001-2002	22 years	egg	Both Ige and non-IgE mediated	0.9 † (0.4 - 1.9) n=843	-	-	-	-	-	-	-	-	0 (0) n=843
Osterballe (2005)	Denmark	2000-2001	>22 years	egg	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	0.1 † (0 - 0.7) n=936	0.2 † (0 - 1) n=936
Julge (2001)	Estonia	1993-1999	6 months	egg white	IgE mediated only	-	-	-	5.2 (nr) n=172	4.2 (nr) n=118	-	-	-	-	-
Julge (2001)	Estonia	1993-1999	1 year	egg white	IgE mediated only	-	-	-	4.1 (nr) n=220	5.6 (nr) n=126	-	-	-	-	-
Julge (2001)	Estonia	1993-1999	2 years	egg white	IgE mediated only	-	-	-	1.8 (nr) n=222	20.6 (nr) n=141	-	-	-	-	-
Julge (2001)	Estonia	1993-1999	5 years	egg white	IgE mediated only	-	-	-	0 (nr) n=208	22.7 (nr) n=208	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	1 year	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	6 (nr) n=261	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	1 year	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	2.7 † (1.8-4.1) n=853	-	1.9 † (1.1 - 3.1) n=853	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Kajosaari (1982)	Finland	1980-1981	2 years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	7 (nr) n=202	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	2 years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	4 † (2.8-5.6) n=852	-	2.2 † (1.4-3.5) n=852	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	3 years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	9 (nr) n=200	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	3 years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	3.6 † (2.4-5.2) n=784	-	3.4 † (2.3-5.0) n=784	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	4 years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	3.4 † (2.3-5.0) n=819	-	3.9 † (2.7 - 5.5) n=819	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	6 years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	1 (nr) n=203	-	-	-	-	-	-	1 (nr) n=203	-	-
Rance (2005)	France	2002	2-14 years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	0.8 † (0.6 - 1.3) n=2716	-	-	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	3 † (2.1-4.3) n=1086	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	egg	Both IgE and non IgE mediated	-	-	-	-	-	0.2 (0.1-0.5) n=3156	-	-	0.1 (0-0.3) n=3156	-
Schafer (2001)	Germany	1997-1998	25-74 years	egg	Both IgE and non IgE mediated	0.4 † (nr) n=nr	-	-	1.9 † (nr) n=nr	-	-	-	-	-	-
Schafer (1999)	Germany	1994	5-6 years	egg	Both IgE and non IgE mediated	-	-	-	2.8 † (1.9-3.9) n=1235	-	-	-	-	-	-
Sakellariou (2008)	Greece	2007	20-54 years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	1.4 † (nr) n=2003	-	-	-	-	-	-	-	-	-
Zannikos (2008)	Greece	2007	7-13 years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	2.1 † (1.5-2.9) n=1988	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Krause (2002)	Greenland	1998	5-18 years	egg	IgE mediated only	-	-	-	-	0.4 † (0.1-1.1) n=1031	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	egg white	IgE mediated only	-	-	-	8.3 † (2.2-23.6) n=36	2.8 † (0.2-16.2) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	egg white	IgE mediated only	-	-	-	10.1 † (5.4-17.7) n=109	2.8 † (0.7-8.4) n=109	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	egg yolk	IgE mediated only	-	-	-	11.1 † (3.6-27.0) n=36	0 † (0-12.0) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	egg yolk	IgE mediated only	-	-	-	7.3 † (3.5-14.4) n=109	0 † (0-4.2) n=109	-	-	-	-	-
Kristjanson (1999)	Iceland	1994	18 months	egg	Both IgE and non IgE mediated	3.1 † (1.6-5.8) n=324	-	-	-	-	1.2 † (0.4-3.3) n=324	-	-	-	-
Kilgallen (1996)	Ireland	nr	0-6 months	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	0 † (0-6.1) n=75	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	12-24 months	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	2 † (0.5-6.2) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	24-36 months	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	1.3 † (0.2-5.2) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	36-48 months	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	2 † (0.5-6.2) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	6-12 months	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	0 † (0-6.1) n=75	-	-	-	-	-	-	-	-	-
Frongia (2005)	Italy	2003	12-24 months	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	-	-	1.9 † (1.5-2.3) n=4602	-	-	-	-	-	-	-
Ronchetti (2008)	Italy	2005 - 2006	9 years	egg	Both IgE and non-IgE mediated	-	-	-	0 † (0-2.6) n=184	-	-	-	-	-	8.2 † (4.8-13.3) n=184
Ronchetti (2008)	Italy	2005 - 2006	13 years	egg	Both IgE and non-IgE mediated	-	-	-	1 † (0.2-4.0) n=196	-	-	-	-	-	10.2 † (6.5-15.5) n=196
Eggesbo (1999)	Norway	1993-1995	1 year	egg	Both IgE and non-IgE mediated	1.5 (1.1-2.0) n=3366	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Eggesbo (1999)	Norway	1993-1995	18 months	egg	Both IgE and non-IgE mediated	2.9 (2.3-3.5) n=3278	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	2 years	egg	Both IgE and non-IgE mediated	3 (2.4-3.7) n=2979	-	-	-	-	-	-	-	-	-
Ro (2012)	Norway	2002-2006	2 years	egg	IgE mediated only	-	-	-	2.8 † (1.5 - 5.3) n=352	11.4 † (8.3 - 15.3) n=352	-	-	-	-	-
Falcao (2004)	Portugal	nr	>39 years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	0.6 † (0.2-1.7) n=659	-	-	-	-	-	-	-	-	-
Martinez-Gimeno (2000)	Spain	nr	6-13 years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	13 † (12.1-14) n=5163	-	-	-	-	-	-	-	-	-
Ostblom (2008 b)	Sweden	1995-2004	1 year	egg	Both IgE and non-IgE mediated	2.5 † (2.0-3.1) n=3104	-	2.6 † (2.1-3.2) n=3104	-	-	-	-	-	-	-
Kristjanrson (1999)	Sweden	1994	18 months	egg	Both IgE and non-IgE mediated	4 † (2.2-6.9) n=324	-	-	-	-	1.5 † (0.6-3.7) n=328	-	-	-	-
Ostblom (2008 b)	Sweden	1996-1998	2 years	egg	Both IgE and non-IgE mediated	3 † (2.4-3.7) n=3104	-	1.8 † (1.4-2.4) n=3104	-	-	-	-	-	-	-
Ostblom (2008 b)	Sweden	1998-2000	4 years	egg	Both IgE and non-IgE mediated	2.6 † (2.1-3.3) n=3104	-	2.0 † (1.6-2.6) n=3104	-	-	-	-	-	-	-
Ostblom (2008 a)	Sweden	1999-2000	4 years	egg	Both IgE and non-IgE mediated	3.7 † (3.0-4.5) n=2563	-	-	-	5 † (4.2-5.9) n=2563	-	0.6 (0.3-1.0) n=2563	-	-	-
Ostblom (2008 b)	Sweden	2002-2004	8 years	egg	Both IgE and non-IgE mediated	1.6 † (1.2-2.1) n=3104	-	1.6 † (1.2-2.1) n=3104	-	-	-	-	-	-	-
Bjornnson (1996)	Sweden	1991-1992	20-44 years	egg	IgE mediated only	-	-	-	-	0.8 † (0.4-1.5) n=1397	-	-	-	-	-
Kucukosmanoglu (2008 a)	Turkey	2002-2004	8-18 months	egg	IgE mediated only	-	-	-	1.9 † (1.2-3.0) n=1015	-	-	-	-	-	-
Orhan (2009)	Turkey	2006	6-9 years	egg	IgE mediated only	1.9 † (1.4 - 2.5) n=2739	-	-	-	-	0.9 † (0.6 - 1.4) n=2739	-	-	0.1 † (0 - 0.4) n=2739	-
Mustafayev (2012)	Turkey	2010	10-11 years	egg	IgE mediated only	5.6 † (5.1-6.2) n=6963	-	-	-	-	-	-	0.1 † (0.0-0.8) n=813	-	-
Gelincik (2008)	Turkey	nr	18 years +	egg	Both IgE and non-IgE mediated	2 ‡ (1.7-2.3) n=11816	-	-	-	-	0.1 † (0.0-0.1) n=11816	0.1 (0.0-0.1) n=11816	-	0.1 † (0.0-0.1) n=11816	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Mustafayev (2012)	Turkey	2010	10-11 years	egg white	IgE mediated only	-	-	-	0.3 † (0.2-0.5) n=6134	-	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	1 year	egg	Both IgE and non-IgE mediated	-	-	-	1.8 † (1.0-3.1) n=763	-	-	-	-	-	1.8 † (1.1-2.9) n=900
Venter (2008)	United Kingdom	2001-2005	2 years	egg	Both IgE and non-IgE mediated	-	-	-	2.1 † (1.2-3.6) n=658	-	-	-	-	-	1.3 † (0.7-2.3) n=858
Venter (2008)	United Kingdom	2001-2005	3 years	egg	Both IgE and non-IgE mediated	-	-	-	1.4 † (0.7-2.7) n=642	-	-	-	-	-	1 † (0.5-2.0) n=891
Arshad (2001)	United Kingdom	1993-1994	4 years	egg	IgE mediated only	-	-	-	0.8 † (0.4 - 2) n=980	-	-	-	-	-	-
Venter (2006)	United Kingdom	2003-2004	6 years	egg	Both IgE and non-IgE mediated	1.9 † (1.1-3.2) n=798	-	-	0.9 † (0.4 - 2) n=700	-	-	-	-	-	0.3 † (0-1.0) n=798
Roberts (2005)	United Kingdom	1998-2000	7 years	egg	IgE mediated only	-	-	-	0.4 † (0.3 - 0.6) n=5066	-	-	-	-	-	-
Pereira (2005)	United Kingdom	2002-2003	11 year olds	egg	Both IgE and non-IgE mediated	1.5 † (0.8-2.8) n=775	-	-	0.3 † (0.1-1.2) n=699	-	-	-	-	-	-
Emmett (1999)	United Kingdom	1995-1996	15 + years	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	0.7 † (0.6-0.8) n=16420	-	-	-	-	-	-	-	-	-
Pereira (2005)	United Kingdom	2002-2003	15 year olds	egg	Both IgE and non-IgE mediated	3 † (2.0-4.6) n=757	-	-	0.2 † (0.0-1.0) n=649	-	-	-	-	-	-
Young (1994)	United Kingdom	nr	All ages	egg	Both IgE and non-IgE mediated	2.3 † (2.1-2.5) n=18880	-	-	-	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.15: Egg allergy prevalence in non-European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Osborne	Australia	2007-2010	12-15 months	egg (raw)	IgE mediated	-	-	-	11.8	-	-	-	9#	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
(2011)					only				(10.6-13.0) n=2768				(7.9-10.0) n=2768		
Woods (1998)	Australia	1998	20-44years	egg	Both IgE and non-IgE mediated	0.7 † (0.3-1.8) n=669	-	-	-	-	-	-	-	-	-
Woods (2002)	Australia	1992-1998	26-50 years	egg white	IgE mediated only	1.3 † (0.5-3.0) n=457	-	-	1.8 † (0.8-3.6) n=457	-	0.2 † (0-1.4) n=457	-	-	-	-
Soller (2012)	Canada	2008-2009	<18 years	egg	"Likely" IgE mediated (no SPT or SIgE)	1.23 (0.69-1.77) n=nr	-	-	-	-	-	-	-	-	-
Soller (2012)	Canada	2008-2009	>18 years	egg	"Likely" IgE mediated (no SPT or SIgE)	0.67 (0.48-0.86) n=nr	-	-	-	-	-	-	-	-	-
Chen (2011)	China	2009	0-12 months	egg	IgE mediated only	-	-	-	9.4 † (7-12.5) n=477	-	-	-	2.5 † (1.4-4.5) n=477	-	-
Hu (2010)	China	1999	0-24 months	egg	IgE mediated only	-	-	-	7.6 † (5.0-11.3) n=304	-	-	-	2.9 † (1.4-5.6) n=314	-	-
Hu (2010)	China	2009	0-24 months	egg	IgE mediated only	-	-	-	16.2 † (12.8-20.4) n=382	-	-	-	5 † (3.2-7.7) n=401	-	-
Sai (2011)	China	2008-2009	adults	egg	IgG mediated only	-	-	-	-	-	-	-	-	-	28.5 † (27.7-29.2) n=12766
Chen (2012)	China (Chongqing)	2009-2010	0-2 years	egg	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	12.0 † (9.5-15.1) n=550
Chen (2012)	China (Hangzhou)	2009-2010	0-2 years	egg	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	4.2 † (2.6-6.5) n=481
Chen (2012)	China (Zhuhai)	2009-2010	0-2 years	egg	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	3 † (1.8-4.8) n=573
Marrugo (2008)	Colombia	nr	All ages	egg	Both IgE and non-IgE mediated (no SPT or SIgE)	0.4 † (0.3-0.8) n=3099	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	egg	IgE mediated only	0.1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Leung (2009)	Hong Kong	2006-2007	2-7 years	egg	IgE mediated only (no SPT or SIgE)	0.7 † (0.5-1.1) n=3677	-	-	-	-	-	-	-	-	0.4 † (0.2-0.7) n=3677

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Dalal (2002)	Israel	nr	0-2years	egg	IgE mediated only	-	0.7 † (0.6 - 1.0) n=9070	-	-	-	0.5 † (0.3-0.6) n=9070	-	-	-	-
Kim (2011)	Korea	2006-2007	0-12 months	egg	IgE mediated only (no SPT or SIgE)	-	-	2.8 † (2.0-4.0) n=1177	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	egg	IgE mediated only (no SPT or SIgE)	1 † (0.9-1.1) n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	egg	IgE mediated only (no SPT or SIgE)	0.6 † (0.5-0.8) n=14777	-	-	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	<3 years	egg	IgE mediated only (no SPT or SIgE)	-	-	0.4 † (0.1-1.2) n=813	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	4-18 years	egg	IgE mediated only (no SPT or SIgE)	-	-	0.5 † (0.4-0.6) n=15169	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	>19 years	egg	IgE mediated only (no SPT or SIgE)	-	-	0.3 † (0.2-0.4) n=14036	-	-	-	-	-	-	-
Lao-araya (2012)	Thailand	2010	3-7 years	egg	IgE mediated only	0.9 † (0.3-2.4) n=452	-	-	-	-	-	-	-	-	-
Santadusit (2005)	Thailand	nr	6 months – 6 years	egg white	IgE mediated only	0.6 † (0.2 - 1.7) n=656	-	-	-	-	-	-	-	-	-
Santadusit (2005)	Thailand	Nr	6 months – 6 years	egg yolk	IgE mediated only	0.9 † (0.4 - 2.1) n=656	-	-	-	-	-	-	-	-	-
Al-Hammadi (2010)	United Arab Emirates (Emirate of Abu Dhabi)	2006	6-9 years	egg	IgE mediated only (no SPT or SIgE)	-	-	3.3 † (1.8-5.7) n=397	-	-	-	-	-	-	-
Branum (2009)	United States	2005-2006	< 18 years	egg	IgE mediated only (not clearly defined)	-	-	-	-	6.7 (nr) n=nr	-	-	-	-	-
Gupta (2011)	United States	2009-2010	0-2 years	egg	IgE mediated (no SPT or SIgE)	-	1 (0.7-1.3) n=5429	-	-	-	-	-	-	-	-
Bock (1987)	United States	1980-1984	0-3 years	egg	Both IgE and non-IgE mediated	2.7 † (1.2-4.2) n=408	-	-	-	-	-	-	-	-	0.6 † (0.2-2) n=480

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Kumar (2011)	United States	2011	6 months - 6 years	egg	IgE mediated only	-	-	-	-	21 † (18.7-23.6) n=1104	-	-	-	-	-
Liu (2010)	United States	2005-2006	1-5 years	egg	IgE mediated only	-	-	-	-	13.9 (nr) n=909	-	-	-	-	1.8 (nr) n=nr
Keet (2012)	United States	2005-2006	1-21 years	egg	IgE mediated only	-	-	-	-	6 (nr) n=3550	-	-	-	-	-
Gupta (2011)	United States	2009-2010	3-5 years	egg	IgE mediated (no SPT or SIgE)	-	1.3 (0.9-1.7) n=5910	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	6-10 years	egg	IgE mediated (no SPT or SIgE)	-	0.8 (0.6-1.1) n=9911	-	-	-	-	-	-	-	-
Liu (2010)	United States	2005-2006	6-19 years	egg	IgE mediated only	-	-	-	-	4.1 (nr) n=2869	-	-	-	-	0.1 (nr) n=nr
Gupta (2011)	United States	2009-2010	11-13 years	egg	IgE mediated (no SPT or SIgE)	-	0.5 (0.4-0.8) n=6716	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	14-17 years	egg	IgE mediated (no SPT or SIgE)	-	0.4 (0.3-0.5) n=10514	-	-	-	-	-	-	-	-
Vierk (2007)	United States	2001	18 years +	egg	IgE mediated only (no SPT or SIgE)	0.7 † (0.5-1.1) n=4482	-	-	-	-	-	-	-	-	0.5 † (0.3-0.8) n=4482
Greenhawt (2009)	United States	nr	18 years+	egg	IgE mediated only (no SPT or SIgE)	1.6 † (0.7-3.2) n=513	-	-	-	-	-	-	-	-	-
Liu (2010)	United States	2005-2006	20-39 years	egg	IgE mediated only	-	-	-	-	2.1 (nr) n=1672	-	-	-	-	0.1 (nr) n=nr
Liu (2010)	United States	2005-2006	40-59 years	egg	IgE mediated only	-	-	-	-	3.8 (nr) n=1361	-	-	-	-	0.2 (nr) n=nr
Liu (2010)	United States	2005-2006	60+ years	egg	IgE mediated only	-	-	-	-	3.9 (nr) n=1392	-	-	-	-	0.6 (nr) n=nr
Liu (2010)	United States	2005-2006	All ages	egg	IgE mediated only	-	-	-	-	3.9 (nr) n=8203	-	-	-	-	0.2 (nr) n=nr
Gupta (2011)	United States	2009-2010	All ages	egg	IgE mediated (no SPT or SIgE)	-	0.8 (0.7-0.9) n=3339	-	-	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper

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‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.16: Fish and Shellfish allergy prevalence in European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Osterballe (2005)	Denmark	2000-2001	< 3 years	crustaceans (shrimp)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0.0 (0.0-4.2) n=111	-	0 † (0.0-4.2) n=111
Osterballe (2005)	Denmark	2000-2001	3 years	crustaceans (shrimp)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0 - 1) n=486	0 † (0 - 1) n=486
Osterballe (2005)	Denmark	2000-2001	3-22 years	crustaceans (shrimp)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0 - 2) n=301	0.3 † (0 - 2.1) n=301
Osterballe (2009)	Denmark	2001-2002	22 years	crustaceans (shrimp)	Both IgE and non-IgE mediated	2 † (1.2 - 3.3) n=843	-	-	-	-	-	-	-	-	0.2 (0.01-0.9) n=843
Osterballe (2005)	Denmark	2000-2001	>22 years	crustaceans (shrimp)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.3 † (0.1 - 1.0) n=936	1.1 † (1 - 2.0) n=936
Osterballe (2005)	Denmark	2000-2001	< 3 years	Fish (cod)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0.0 (0.0-4.2) n=111	-	0 † (0 - 1) n=111
Osterballe (2005)	Denmark	2000-2001	3 years	Fish (cod)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0 - 1) n=486	0.8 † (0.3 - 2.2) n=486
Osterballe (2005)	Denmark	2000-2001	3-22 years	Fish (cod)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0 - 2) n=301	0.3 † (0.1 - 2.6) n=301
Osterballe (2009)	Denmark	2001-2002	22 years	Fish (cod)	Both IgE and non-IgE mediated	0.2 † (0 - 1) n=843	-	-	-	-	-	-	-	-	0.1 (0.0-0.8) n=843
Osterballe (2005)	Denmark	2000-2001	>22 years	Fish (cod)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.2 † (0 - 0.9) n=936	0.6 † (0.3 - 1.5) n=936
Osterballe (2009)	Denmark	2001-2002	22 years	mollusc (octopus)	Both IgE and non-IgE mediated	0.4 † (0.1 - 1.1) n=843	-	-	-	-	-	-	-	-	0.1 (0.0-0.8) n=843
Kajosaari (1982)	Finland	1980-1981	1 year	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	7 (nr) n=261	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	1 year	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	3.5 † (2.4-5.1) n=853	-	0.2 † (0-0.9) n=853	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Kajosaari (1982)	Finland	1980-1981	2 years	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	6 (nr) n=202	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	2 years	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	5 † (3.4-6.4) n=852	-	0.4 † (0.1-1.1) n=852	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	3 years	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	5 (nr) n=200	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	3 years	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	3.6 † (2.4-5.2) n=784	-	1 † (0.4-1.9) n=784	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	4 years	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	4.2 † (2.9 - 5.8) n=819	-	1 † (0.5-2.0) n=819	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	6 years	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	1 (nr) n=203	-	-	-	-	-	-	1 (nr) n=203	-	-
Haahtela (1980)	Finland	nr	15-17 years	fish	IgE mediated only	-	-	-	2.7 † (1.7-4.2) n=708	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	crustaceans	Both IgE and non-IgE mediated (no SPT or SIgE)	5.5 † (4.3-7.1) n=1086	-	-	-	-	-	-	-	-	-
Rance (2005)	France	2002	2-14 years	crustaceans (shrimp)	Both IgE and non-IgE mediated (no SPT or SIgE)	0.5 † (0.3 - 0.9) n=2716	-	-	-	-	-	-	-	-	-
Rance (2005)	France	2002	2-14 years	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	0.7 † (0.4 - 1.1) n=2716	-	-	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	4 † (2.9-5.3) n=1086	-	-	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	mollusc (oyster)	Both IgE and non-IgE mediated (no SPT or SIgE)	1.5 † (0.9-2.4) n=1086	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	crustaceans (crab)	Both IgE and non-IgE mediated	-	-	-	-	-	0.3 (0.2-0.6) n=3156	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Schafer (2001)	Germany	1997-1998	25-74 years	crustaceans (crab)	Both IgE and non-IgE mediated	-	-	-	1.9 † (nr) n=nr	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	fish (herring)	Both IgE and non-IgE mediated	-	-	-	-	-	0.1 (0.0-0.4) n=3156	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	fish (mackerel)	Both IgE and non-IgE mediated	-	-	-	-	-	0.1 (0.0-0.3) n=3156	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	fish (mackerel)	Both IgE and non-IgE mediated	-	-	-	1.8 † (nr) n=nr	-	-	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	fish/seafood	Both IgE and non-IgE mediated	1† (nr) n=nr	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	mollusc (mussels)	Both IgE and non-IgE mediated	-	-	-	-	-	0.0 (0.0-0.2) n=3156	-	-	-	-
Zannikos (2008)	Greece	2007	7-13 years	fish	Both IgE and non IgE mediated (no SPT or SIgE)	1.9 † (1.3-2.6) n=1988	-	-	-	-	-	-	-	-	-
Sakellariou (2008)	Greece	2007	20-54 years	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	1.5 (nr) † n=2003	-	-	-	-	-	-	-	-	-
Zannikos (2008)	Greece	2007	7-13 years	shellfish	Both IgE and non IgE mediated (no SPT or SIgE)	0.1 † (0.0-0.2) n=3821	-	-	-	-	-	-	-	-	-
Krause (2002)	Greenland	1998	5-18 years	fish	IgE mediated only	-	-	-	-	0.7 † (0.3-1.5) n=1031	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	crustaceans (crab)	IgE mediated only	-	-	-	-	-	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	crustaceans (crab)	IgE mediated only	-	-	-	-	1.8 † (0.3-7.1) n=109	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	fish (cod)	IgE mediated only	-	-	-	-	-	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	fish (cod)	IgE mediated only	-	-	-	-	0 † (0-4.2) n=109	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	fish	Both IgE and non-IgE mediated	2.2 † (1.0-4.6) n=324	-	-	-	-	0.6 † (0.1-2.5) n=324	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	shellfish	Both IgE and non-IgE mediated	1.5 † (0.6-3.8) n=324	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Eggesbo (1999)	Norway	1993-1995	1 year	fish	Both IgE and non-IgE mediated	1.2 (0.9-1.7) n=3366	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	18 months	fish	Both IgE and non-IgE mediated	1.5 (1.1-2.0) n=3278	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	2 years	fish	Both IgE and non-IgE mediated	1.5 (1.1-2.1) n=2979	-	-	-	-	-	-	-	-	-
Ro (2012)	Norway	2002-2006	2 years	fish	IgE mediated only	-	-	-	0.3 † (0 - 1.8) n=352	1.1 † (0.4 - 3.1) n=352	-	-	-	-	-
Falcao (2004)	Portugal	nr	>39 years	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	0.9 † (0.4-2.1) n=659	-	-	-	-	-	-	-	-	-
Falcao (2004)	Portugal	nr	>39 years	Molluscs	Both IgE and non-IgE mediated (no SPT or SIgE)	0.5 † (0.1-1.4) n=659	-	-	-	-	-	-	-	-	-
Martinez-Gimeno (2000)	Spain	nr	6-13 years	fish	Both IgE and non-IgE mediated (no SPT or SIgE)	6.9 † (6.2-7.6) n=5163	-	-	-	-	-	-	-	-	-
Ostblom (2008 b)	Sweden	1995-2004	1 year	fish	Both IgE and non-IgE mediated	1.5 † (1.1-2.0) n=3104	-	0.2 † (0.1-0.4) n=3104	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	fish	Both IgE and non-IgE mediated	3.1 † (1.6-5.7) n=328	-	-	-	-	0.3 † (0.0-2.0) n=328	-	-	-	-
Ostblom (2008 b)	Sweden	1996-1998	2 years	fish	Both IgE and non-IgE mediated	1.8 † (1.4-2.4) n=3104	-	0.6 † (0.4-1.0) n=3104	-	-	-	-	-	-	-
Ostblom (2008 b)	Sweden	1998-2000	4 years	fish	Both IgE and non-IgE mediated	1.2 † (0.9-1.7) n=3104	-	0.8 † (0.5-1.2) n=3104	-	-	-	-	-	-	-
Ostblom (2008 b)	Sweden	2002-2004	8 years	fish	Both IgE and non-IgE mediated	0.8 † (0.5-1.2) n=3104	-	0.6 † (0.4-1.0) n=3104	-	-	-	-	-	-	-
Bjornsson (1996)	Sweden	1991-1992	20-44 years	fish	IgE mediated only	-	-	-	-	0.3 † (0.1-0.8) n=1397	-	-	-	-	-
Ostblom (2008 a)	Sweden	1999-2000	4 years	Fish (cod)	Both IgE and non-IgE mediated	1.6 † (1.2-2.2) n=2563	-	-	-	1 † (0.7-1.5) n=2563	-	0.4 (0.2-0.8) n=2563	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	shellfish	Both IgE and non-IgE mediated	1.2 † (0.4-3.3) n=328	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	fish/crustacean	Both IgE and non-IgE (no SPT or SIgE)	0.7 † (0.5-1.0) n=4400	-	-	-	-	-	-	-	-	-
Orhan (2009)	Turkey	2006	6-9 years	fish	IgE mediated only	0.3 † (0.2 - 1) n=2739	-	-	-	-	0.2 † (0.1 - 0.5) n=2739	-	-	0 † (0 - 0.2) n=2739	-
Mustafayev (2012)	Turkey	2010	10-11 years	fish	IgE mediated only	2.3 † (2.0-2.7) n=6963	-	-	-	-	-	-	-	-	-
Gelincik (2008)	Turkey	nr	18 years +	fish	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0-0.0) n=11816	-
Gelincik (2008)	Turkey	nr	18 years +	seafood	Both Ige and non-IgE mediated	0.4 ‡ (0.3-0.5) n=11816	-	-	-	-	-	-	-	0 † (0-0.0) n=11816	-
Pereira (2005)	United Kingdom	2002-2003	11 year olds	crustaceans (prawn)	Both IgE and non-IgE mediated	0.3 † (0.1-1.0) n=775	-	-	-	-	-	-	-	-	-
Pereira (2005)	United Kingdom	2002-2003	15 year olds	crustaceans (prawn)	Both IgE and non-IgE mediated	0.7 † (0.2-1.6) n=757	-	-	-	-	-	-	-	-	-
Pereira (2005)	United Kingdom	2002-2003	11 year olds	fish	Both IgE and non-IgE mediated	0.9 † (0.4-1.9) n=775	-	-	1.3 † (0.6-2.5) n=699	-	-	-	-	-	-
Pereira (2005)	United Kingdom	2002-2003	15 year olds	fish	Both IgE and non-IgE mediated	1.8 † (1.1-3.2) n=757	-	-	1.4 † (0.7-2.7) n=649	-	-	-	-	-	-
Emmett (1999)	United Kingdom	1995-1996	15 + years	fish	Both Ige and non-IgE mediated	0.5 † (0.4-0.6) n=16420	-	-	-	-	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	1 year	fish (cod)	Both IgE and non-IgE mediated	-	-	-	0.3 † (0.0-1.0) n=763	-	-	-	-	-	0.1 † (0.0-0.7) n=900
Venter (2008)	United Kingdom	2001-2005	2 years	fish (cod)	Both IgE and non-IgE mediated	-	-	-	0.5 † (0.1-1.4) n=658	-	-	-	-	-	0 † (0-0.6) n=858
Venter (2008)	United Kingdom	2001-2005	3 years	fish (cod)	Both IgE and non-IgE mediated	-	-	-	0.5 † (0.1-1.5) n=642	-	-	-	-	-	0 † (0-0.5) n=891
Arshad (2001)	United Kingdom	1993-1994	4 years	fish (cod)	IgE mediated only	-	-	-	0.7 † (0.3 - 2) n=981	-	-	-	-	-	-
Venter (2006)	United Kingdom	2003-2004	6 years	fish (cod)	Both IgE and non-IgE mediated	0.3 † (0-1.0) n=798	-	-	1 † (0.4-2.1) n=700	-	-	-	-	-	0 † (0-0.6) n=798
Roberts (2005)	United Kingdom	1998-2000	7 years	fish (cod)	IgE mediated only	-	-	-	0 † (0 - 0.3) n=2061	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Young (1994)	United Kingdom	nr	All ages	fish/crustacean	Both IgE and non-IgE mediated	2.9 † (2.7-3.1) n=18880	-	-	-	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.17: Fish and Shellfish allergy prevalence in non-European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Tyoe of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Woods (2002)	Australia	1992-1998	26-50 years	crustaceans (shrimp)	IgE mediated only	3.3 † (1.9-5.5) n=457	-	-	3.7 † (2.3-6.0) n=457	-	0.9 † (0.3-2.4) n=457	-	-	-	-
Woods (1998)	Australia	1998	20-44years	fish/shellfish	Both IgE and non IgE mediated	2.1 † (1.2-3.6) n=669	-	-	-	-	-	-	-	-	-
Osborne (2011)	Australia	2007-2010	12-15 months	shellfish		-	-	-	0.4 (0.2-0.7) n=2375	-	-	-	-	-	-
Ben-Shoshan (2010)	Canada	2008-2009	< 18 years	fish	Both IgE and non-IgE mediated only	0.18 (0.00-0.36) n=nr	0.18 # (0.00-0.36) n=nr	0 # (nr) n=nr	-	-	-	-	-	-	-
Ben-Shoshan (2010)	Canada	2008-2009	> 18 years	fish	Both IgE and non-IgE mediated only	0.6 (0.43-0.78) n=nr	0.56 # (0.39-0.73) n=nr	0.12 # (0.08-0.16) n=nr	-	-	-	-	-	-	-
Ben-Shoshan (2010)	Canada	2008-2009	< 18 years	shellfish	Both IgE and non-IgE mediated only	0.55 (0.21-0.88) n=nr	0.5 # (0.18-0.82) n=nr	0.06 # (0.01-0.10) n=nr	-	-	-	-	-	-	-
Ben-Shoshan (2010)	Canada	2008-2009	> 18 years	shellfish	Both IgE and non-IgE mediated only	1.91 (1.60-2.23) n=nr	1.69 # (1.39-1.98) n=nr	0.71 # (0.58-0.84) n=nr	-	-	-	-	-	-	-
Chen (2011)	China	2009	0-12 months	crustaceans (shrimp)	IgE mediated only	-	-	-	0.2 † (0-1.3) n=477	-	-	-	-	-	-
Hu (2010)	China	1999	0-24 months	crustaceans (shrimp)	IgE mediated only	-	-	-	0 † (0-1.6) n=304	-	-	-	-	-	-
Hu (2010)	China	2009	0-24 months	crustaceans (shrimp)	IgE mediated only	-	-	-	0.3 † (0.0-1.7) n=382	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Tyoe of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Sai (2011)	China	2008-2009	adults	crustaceans (shrimp)	IgGmediated only	-	-	-	-	-	-	-	-	-	10 † (9.5-10.6) n=12766
Sai (2011)	China	2008-2009	adults	crustaceans (crab)	IgG mediated only	24.5 (nr) n=nr	-	-	-	-	-	-	-	-	24.5 † (23.8-25.3) n=12765
Chen (2011)	China	2009	0-12 months	fish	IgE mediated only	-	-	-	0.2 † (0-1.3) n=477	-	-	-	-	-	-
Hu (2010)	China	1999	0-24 months	fish	IgE mediated only	-	-	-	0.3 † (0.0-2.1) n=304	-	-	-	-	-	-
Hu (2010)	China	2009	0-24 months	fish	IgE mediated only	-	-	-	0.8 † (0.2-2.5) n=382	-	-	-	-	-	-
Sai (2011)	China	2008-2009	adults	fish	IgG mediated only	-	-	-	-	-	-	-	-	-	11.2 † (10.7-11.8) n=12766
Marrugo (2008)	Colombia	nr	All ages	seafood	Both IgE and non-IgE mediated (no SPT or SIgE)	4 † (3.3-4.7) n=3099	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	crustaceans (shrimp)	IgE mediated only	0.1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	fish	IgE mediated only	0.3 (nr) n=1407	-	-	-	-	-	-	-	-	-
Leung (2009)	Hong Kong	2006-2007	2-7 years	crustaceans	IgE mediated only (no SPT or SIgE)	1.3 † (1.0-1.7) n=3677	-	-	-	-	-	-	-	-	0.9 † (0.6-1.3) n=3677
Leung (2009)	Hong Kong	2006-2007	2-7 years	fish	IgE mediated only (no SPT or SIgE)	0.3 † (0.2-0.6) n=3677	-	-	-	-	-	-	-	-	0.2 † (0.1-0.5) n=3677
Dalal (2002)	Israel	nr	0-2years	fish	IgE mediated only	-	0 † (0 - 0.1) n=9070	-	-	-	0 † (0 - 0.1) n=9070	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	fish	IgE mediated only (no SPT or SIgE)	0.7 † (0.6 - 0.8) n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	fish	IgE mediated only (no SPT or SIgE)	0.6 † (0.5-0.8) n=14777	-	-	-	-	-	-	-	-	-
Kim (2011)	Korea	2006-2007	0-12 months	seafood	IgE mediated only (no SPT or SIgE)	-	-	0.5 † (0.2-1.2) n=1177	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Tyoe of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Oh (2004)	Korea	2000	6-12 years	seafood	IgE mediated only (no SPT or SIgE)	0.4 † (0.3-0.4) n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	seafood	IgE mediated only (no SPT or SIgE)	0.8 † (0.7-1.0) n=14777	-	-	-	-	-	-	-	-	-
Connett (2012)	Philippines	2007 - 2008	14 - 16 years	fish	Both IgE and non-IgE mediated only	4.3 † (3.9-4.7) n=11434	2.3 † (2.0-2.6) n=11434	-	-	-	-	-	-	-	-
Shek (2010)	Philippines	2007-2008	14-16 years	shellfish	Both IgE and non IgE mediated	8.7 † (8.2-9.2) n=11158	5.1 † (4.3-6.1) n=11158	-	-	-	-	-	-	-	-
Connett (2012)	Singapore	2007- 2008	14 - 16 years	fish	Both IgE and non-IgE mediated only	0.6 † (0.4-0.8) n=6498	0.3 † (0.2-0.4) n=6498	-	-	-	-	-	-	-	-
Shek (2010)	Singapore	2007-2008	4-6 years	shellfish	Both IgE and non IgE mediated	7.2 † (6.5-8.1) n=4115	1.2 † (0.9-1.6) n=4115	-	-	-	-	-	-	-	-
Shek (2010)	Singapore	2007-2008	14-16 years	shellfish	Both IgE and non IgE mediated	11.6 † (10.8-12.4) n=6342	5.2 † (4.5-6.1) n=6342	-	-	-	-	-	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	crustacean (lobster)	IgE mediated only	-	-	-	-	-	-	17.3 (15.1-19.8) n=1010	-	-	-
Wu (2012)	Taiwan	2004	<3 years	crustaceans (shrimp)	IgE mediated only (no SPT or SIgE)	-	-	0.6 † (0.2-1.5) n=813	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	4-18 years	crustaceans (shrimp)	IgE mediated only (no SPT or SIgE)	-	-	4 † (3.7-4.4) n=15169	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	>19 years	crustaceans (shrimp)	IgE mediated only (no SPT or SIgE)	-	-	3.3 † (3.0-3.6) n=14036	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	<3 years	crustceans (crab)	IgE mediated only (no SPT or SIgE)	-	-	0.4 † (0.1-1.2) n=813	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	4-18 years	crustceans (crab)	IgE mediated only (no SPT or SIgE)	-	-	2.6 † (2.3-2.8) n=15169	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	>19 years	crustceans (crab)	IgE mediated only (no SPT or SIgE)	-	-	2.3 † (2.0-2.5) n=14036	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	<3 years	fish	IgE mediated only (no SPT or SIgE)	-	-	0.5 † (0.2-1.3) n=813	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Wu (2012)	Taiwan	2004	4-18 years	fish	IgE mediated only (no SPT or SIgE)	-	-	1.5 † (1.3-1.7) n=15169	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	>19 years	fish	IgE mediated only (no SPT or SIgE)	-	-	1.2 † (1.0-1.4) n=14036	-	-	-	-	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	mollusc (abalone)	IgE mediated only	-	-	-	-	-	-	25.1 (22.4-27.9) n=1010	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	mollusc (clam)	IgE mediated only	-	-	-	-	-	-	4.8 (3.6-6.3) n=1010	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	mollusc (octopus)	IgE mediated only	-	-	-	-	-	-	7.5 (6.0-9.4) n=1010	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	mollusc (oyster)	IgE mediated only	-	-	-	-	-	-	9.9 (8.2-12) n=1010	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	mollusc (scallop)	IgE mediated only	-	-	-	-	-	-	24.9 (22.2-27.7) n=1010	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	mollusc (squid)	IgE mediated only	-	-	-	-	-	-	2.3 (1.5-3.5) n=1010	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	mollusc (squid)	IgE mediated only	-	-	-	-	-	-	6.8 (5.4-8.6) n=1010	-	-	-
Wu (2012)	Taiwan	2004	<3 years	Molluscs	IgE mediated only (no SPT or SIgE)	-	-	0.1 † (0.0-0.8) n=813	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	4-18 years	Molluscs	IgE mediated only (no SPT or SIgE)	-	-	1.1 † (1.0-1.3) n=15169	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	>19 years	Molluscs	IgE mediated only (no SPT or SIgE)	-	-	1.5 † (1.3-1.7) n=14036	-	-	-	-	-	-	-
Lao-araya (2012)	Thailand	2010	3-7years	crustaceans (shrimp)	IgE mediated only	3.1 † (1.8-5.3) n=452	-	-	-	-	-	-	0.9 † (0.3-2.4) n=452	-	-
Santadusit (2005)	Thailand	nr	6 months - 6years	crustaceans (shrimp)	IgE mediated only	1.2 † (0.6 - 2.5) n=656	-	-	-	-	-	-	-	-	-
Lao-araya (2012)	Thailand	2010	3-7years	crustaceans (crab)	IgE mediated only	0.7 † (0.2-2.1) n=452	-	-	-	-	-	-	0.2 † (0.0-1.4) n=452	-	-
Connett (2012)	Thailand	2007- 2008	14 - 16 years	fish	Both IgE and non-IgE mediated only	0.4 † (0.2-0.8) n=2034	0.3 † (0.1-0.7) n=2034	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Lao-araya (2012)	Thailand	2010	3-7years	fish	IgE mediated only	1.1 † (0.4-2.7) n=452	-	-	-	-	-	-	0.2 † (0.0-1.4) n=452	-	-
Santadusit (2005)	Thailand	nr	6 months - 6years	fish	IgE mediated only	0.3 † (0.1 - 1.2) n=656	-	-	-	-	-	-	-	-	-
Lao-araya (2012)	Thailand	2010	3-7years	mollusc (squid)	IgE mediated only	0.2 † (0.0-1.4) n=452	-	-	-	-	-	-	-	-	-
Lao-araya (2012)	Thailand	2010	3-7years	molluscs	IgE mediated only	0.2 † (0.0-1.4) n=452	-	-	-	-	-	-	-	-	-
Santadusit (2005)	Thailand	Nr	6 months - 6years	seafood (crab. mollusc, squid)	IgE mediated only	0.5 † (0.1 - 1.5) n=656	-	-	-	-	-	-	-	-	-
Al-Hammadi (2010)	United Arab Emirates (Emirate of Abu Dhabi)	2006	6-9 years	fish	IgE mediated only (no SPT or SIgE)	-	-	2.8 † (1.5-5.1) n=397	-	-	-	-	-	-	-
Vierk (2007)	United States	2001	18 years +	crustaceans	IgE mediated only (no SPT or SIgE)	0.7 † (0.5-1.0) n=4482	-	-	-	-	-	-	-	-	0.4 † (0.2-0.7) n=4482
Branum (2009)	United States	2005-2006	< 18 years	crustaceans (shrimp)	IgE mediated only (not clearly defined)	-	-	-	-	5.2 (nr) n=nr	-	-	-	-	-
Liu (2010)	United States	2005-2006	6-19 years	crustaceans (shrimp)	IgE mediated only	-	-	-	-	6.1 (nr) n=2869	-	-	-	-	1.1 (nr) n=nr
Liu (2010)	United States	2005-2006	20-39 years	crustaceans (shrimp)	IgE mediated only	-	-	-	-	6.7 (nr) n=1672	-	-	-	-	1.2 (nr) n=nr
Liu (2010)	United States	2005-2006	40-59 years	crustaceans (shrimp)	IgE mediated only	-	-	-	-	5.9 (nr) n=1361	-	-	-	-	0.9 (nr) n=nr
Liu (2010)	United States	2005-2006	60+ years	crustaceans (shrimp)	IgE mediated only	-	-	-	-	4.6 (nr) n=1392	-	-	-	-	0.7 (nr) n=nr
Liu (2010)	United States	2005-2006	All ages	crustaceans (shrimp)	IgE mediated only	-	-	-	-	5.9 (nr) n=8203	-	-	-	-	1 (nr) n=nr
Sicherer (2004)	United States	2002	0-5 years	fish	Both IgE and non IgE mediated	-	0 † (0-0.5) n=997	-	-	-	-	-	-	-	-
Sicherer (2004)	United States	2002	6-17 years	fish	Both IgE and non IgE mediated	-	0.2 (0.1-0.5) n=2610	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Vierk (2007)	United States	2001	18 years +	fish	IgE mediated only (no SPT or SIgE)	0.7 † (0.5-1.0) n=4482	-	-	-	-	-	-	-	-	0.6 † (0.4-0.9) n=4482
Greenhawt (2009)	United States	nr	18 years+	fish	IgE mediated (no SPT or SIgE)	2.7 † (1.6-4.7) n=513	-	-	-	-	-	-	-	-	-
Sicherer (2004)	United States	2002	18-40 years	fish	Both IgE and non IgE mediated	-	0.5 † (0.3-0.8) n=4336	-	-	-	-	-	-	-	-
Sicherer (2004)	United States	2002	41-60 years	fish	Both IgE and non IgE mediated	-	0.5 † (0.3-0.8) n=3604	-	-	-	-	-	-	-	-
Sicherer (2004)	United States	2002	61 +	fish	Both IgE and non IgE mediated	-	0.3 † (0.1-0.7) n=1876	-	-	-	-	-	-	-	-
Sicherer (2004)	United States	2002	All ages	fish	Both IgE and non IgE mediated	0.8 † (0.7-1) n=14948	0.4 † (0.3-0.5) n=14948	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	0-2 years	fish (fin fish)	IgE mediated (no SPT or SIgE)	-	0.3 (0.1-0.4) n=5429	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	3-5 years	fish (fin fish)	IgE mediated (no SPT or SIgE)	-	0.5 (0.3-0.8) n=5910	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	6-10 years	fish (fin fish)	IgE mediated (no SPT or SIgE)	-	0.5 (0.3-0.7) n=9911	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	11-13 years	fish (fin fish)	IgE mediated (no SPT or SIgE)	-	0.6 (0.4-0.8) n=6716	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	14-17 years	fish (fin fish)	IgE mediated (no SPT or SIgE)	-	0.6 (0.4-0.9) n=10514	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	All ages	fish (fin fish)	IgE mediated (no SPT or SIgE)	-	0.5 (0.4-0.6) n=3339	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	0-2 years	shellfish	IgE mediated (no SPT or SIgE)	-	0.5 (0.3-0.8) n=5429	-	-	-	-	-	-	-	-
Sicherer (2004)	United States	2002	0-5 years	shellfish	Both IgE and non IgE mediated	-	0.1 † (0-0.6) n=997	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	3-5 years	shellfish	IgE mediated (no SPT or SIgE)	-	1.2 (0.8-1.6) n=5910	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Tyoe of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Gupta (2011)	United States	2009-2010	6-10 years	shellfish	IgE mediated (no SPT or SIgE)	-	1.3 (1.1-1.6) n=9911	-	-	-	-	-	-	-	-
Sicherer (2004)	United States	2002	6-17 years	shellfish	Both IgE and non IgE mediated	-	0.7 † (0.4-1.1) n=2610	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	11-13 years	shellfish	IgE mediated (no SPT or SIgE)	-	1.7 (1.3-2.1) n=6716	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	14-17 years	shellfish	IgE mediated (no SPT or SIgE)	-	2 (1.7-2.5) n=10514	-	-	-	-	-	-	-	-
Vierk (2007)	United States	2001	18 years +	shellfish	IgE mediated only (no SPT or SIgE)	1.7 † (1.3-2.1) n=4482	-	-	-	-	-	-	-	-	1.1 † (0.8-1.5) n=4482
Greenhawt (2009)	United States	nr	18 years+	shellfish	IgE mediated only (no SPT or SIgE)	9 † (6.7-11.9) n=513	-	-	-	-	-	-	-	-	-
Sicherer (2004)	United States	2002	18-40 years	shellfish	Both IgE and non IgE mediated	-	2.2 † (1.8-2.7) n=4336	-	-	-	-	-	-	-	-
Sicherer (2004)	United States	2002	41-60 years	shellfish	Both IgE and non IgE mediated	-	3.1 † (2.5-3.7) n=3604	-	-	-	-	-	-	-	-
Sicherer (2004)	United States	2002	61 +	shellfish	Both IgE and non IgE mediated	-	2.6 † (2-3.5) n=1876	-	-	-	-	-	-	-	-
Sicherer (2004)	United States	2002	All ages	shellfish	Both IgE and non IgE mediated	2.7 † (2.5-3) n=14948	2 † (1.8-2.3) n=14948	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	All ages	shellfish	IgE mediated only (no SPT or SIgE)	-	1.4 (1.2-1.5) n=3339	-	-	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

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Table 1.18: Fruits allergy prevalence in European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Osterballe (2005)	Denmark	2000-2001	< 3 years	fruit/ vegetables	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0 (nr) N=111	-	9 † (4.6 - 16.3) n=111
Osterballe (2005)	Denmark	2000-2001	3 years	fruit/ vegetables	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0 - 1) n=486	1.4 † (0.6 - 3.1) n=486
Osterballe (2005)	Denmark	2000-2001	3-22 years	fruit/ vegetables	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	1 † (0.3-3.1) n=301	5.6 † (3.4 - 9.1) n=301
Osterballe (2005)	Denmark	2000-2001	>22 years	fruit/ vegetables	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	2.7 † (1.8 - 4) n=936	8.1 † (7 - 10.1) n=936
Kajosaari (1982)	Finland	1980-1981	3 years	fruits (apple)	Both IgE and non-IgE mediated (no SPT or SIgE)	0.5 (nr) n=200	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	6 years	fruits (apple)	Both IgE and non-IgE mediated (no SPT or SIgE)	1 (nr) n=203	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	1 year	fruits (apple, pear, cherry, peach, banana)	Both IgE and non-IgE mediated (no SPT or SIgE)	6.6 † (5.0-8.5) n=853	-	0.4 † (0.1 - 1.1) n=853	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	2 years	fruits (apple, pear, cherry, peach, banana)	Both IgE and non-IgE mediated (no SPT or SIgE)	7 † (5.3-8.8) n=852	-	0.2 † (0.0 - 0.9) n=852	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	3 years	fruits (apple, pear, cherry, peach, banana)	Both IgE and non-IgE mediated (no SPT or SIgE)	6.6 † (5.0 - 8.7) n=784	-	1 † (0.3-1.8) n=784	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	4 years	fruits (apple, pear, cherry, peach, banana)	Both IgE and non-IgE mediated (no SPT or SIgE)	6 † (4.5 - 7.9) n=819	-	1.3 † (0.7 - 2.5) n=819	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	1 year	fruits (citrus)	Both IgE and non-IgE mediated (no SPT or SIgE)	8 (nr) n=261	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	1 year	fruits (citrus)	Both IgE and non-IgE mediated (no SPT or SIgE)	3.5 † (2.4-5.1) n=853	-	0.1 † (0-0.8) n=853	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	2 years	fruits (citrus)	Both IgE and non-IgE mediated (no SPT or SIgE)	9 (nr) n=202	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Pyrhonen (2009)	Finland	2001-2009	2 years	fruits (citrus)	Both IgE and non-IgE mediated (no SPT or SIgE)	7.2 † (5.6-9.2) n=852	-	0 † (0.0 - 0.6) n=852	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	3 years	fruits (citrus)	Both IgE and non-IgE mediated (no SPT or SIgE)	11 (nr) n=200	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	3 years	fruits (citrus)	Both IgE and non-IgE mediated (no SPT or SIgE)	6.5 † (4.9 - 8.5) n=784	-	0.4 † (0.1-1.2) n=784	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	4 years	fruits (citrus)	Both IgE and non-IgE mediated (no SPT or SIgE)	5.1 † (3.8 - 6.9) n=819	-	1.3 † (0.7 - 2.5) n=819	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	6 years	fruits (citrus)	Both IgE and non-IgE mediated (no SPT or SIgE)	2 (nr) n=203	-	-	-	-	-	-	2 (nr) n=203	-	-
Kajosaari (1982)	Finland	1980-1981	1 year	fruits (strawberry)	Both IgE and non-IgE mediated (no SPT or SIgE)	7 (nr) n=261	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	2 years	fruits (strawberry)	Both IgE and non-IgE mediated (no SPT or SIgE)	4 (nr) n=202	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	3 years	fruits (strawberry)	Both IgE and non-IgE mediated (no SPT or SIgE)	7 (nr) n=200	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	6 years	fruits (strawberry)	Both IgE and non-IgE mediated (no SPT or SIgE)	0.5 (nr) n=203	-	-	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	fruits	Both IgE and non-IgE mediated (no SPT or SIgE)	1.5 † (0.9-2.4) n=1086	-	-	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	fruits (apple/peach/raspberry/cherry)	Both IgE and non-IgE mediated (no SPT or SIgE)	16 † (13.9-18.4) n=1086	-	-	-	-	-	-	-	-	-
Rance (2005)	France	2002	2-14 years	fruits (kiwi)	Both IgE and non-IgE mediated (no SPT or SIgE)	0.8 † (0.5 - 1. 3) n=2716	-	-	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	fruits (pear)	Both IgE and non-IgE mediated (no	1.5 † (0.9-2.4) n=1086	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
					SPT or SIgE)										
Zuberbier (2004)	Germany	1999-2000	0-80+ years	fruits	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	1.1 (0.8-1.5) n=3156	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	Fruits	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0.3 (0.1-0.6) n=3156
Schafer (2001)	Germany	1997-1998	25-74 years	fruits	Both IgE and non-IgE mediated	3.8 † (nr) n=nr	-	-	-	-	-	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	fruits (apple etc)	Both IgE and non-IgE mediated	3.9 † (nr) n=nr	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	fruits (apple)	Both IgE and non-IgE mediated	-	-	-	-	-	4.2 (3.6-4.8) n=4093	-	-	2.2 (1.8-2.8) n=3156	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	fruits (apricot)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.2 (0.1-0.4) n=3156	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	fruits (cherry)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.9 (0.6-1.3) n=3156	-
Schafer (2001)	Germany	1997-1998	25-74 years	fruits (citrus)	Both IgE and non-IgE mediated	4.5† (nr) n=nr	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	fruits (grape)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.1 (0.0-0.3) n=3156	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	fruits (nectarine)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.4 (0.2-0.7) n=3156	-
Schafer (2001)	Germany	1997-1998	25-74 years	fruits (peach etc)	Both IgE and non-IgE mediated	3.7 † (nr) n=nr	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	fruits (peach)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.7 (0.4-1.0) n=3156	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	fruits (pear)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.4 (0.2-0.7) n=3156	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	fruits (plum)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.5 (0.3-0.8) n=3156	-
Zannikos (2008)	Greece	2007	7-13 years	Fruits	Both IgE and non IgE mediated (no SPT or SIgE)	3.1† (2.4-4.0) n=1988	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Bakos (2006)	Hungary	2002-2004	20-69 years	fruits (apple)	IgE mediated only	-	-	-	5.6 † (1.0-20.0) n=36	0 † (0-12.0) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	fruits (apple)	IgE mediated only	-	-	-	9.2 † (5.0-17.0) n=109	0 † (0-4.2) n=109	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	fruits (banana)	IgE mediated only	-	-	-	0 † (0-12.0) n=36	-	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	fruits (banana)	IgE mediated only	-	-	-	8.3 † (4.1-15.5) n=109	-	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	fruits (orange)	IgE mediated only	-	-	-	-	-	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	fruits (orange)	IgE mediated only	-	-	-	-	4.6 † (1.7-10.9) n=109	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	fruits (apple)	Both IgE and non-IgE mediated	0.9 † (0.2-2.9) n=324	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	fruits (banana)	Both IgE and non-IgE mediated	1.2 † (0.4-3.3) n=324	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	fruits (citrus)	Both IgE and non-IgE mediated	6.8 † (4.4-10.3) n=324	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	fruits (plum/cherry)	Both IgE and non-IgE mediated	0.9 † (0.2-2.9) n=324	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	1 year	fruits	Both IgE and non-IgE mediated	7.6 (6.7-8.7) n=3366	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	18 months	fruits	Both IgE and non-IgE mediated	9.3 (8.3-10.4) n=3278	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	2 years	fruits	Both IgE and non-IgE mediated	11.5 (10.4-12.7) n=2979	-	-	-	-	-	-	-	-	-
Falcao (2004)	Portugal	nr	>39 years	fruits	Both IgE and non-IgE mediated (no SPT or SIgE)	2 † (1.1-3.4) n=659	-	-	-	-	-	-	-	-	-
Martinez-Gimeno (2000)	Spain	nr	6-13 years	fruits	Both IgE and non-IgE mediated (no SPT or SIgE)	21 † (19.9 -22.1) n=5163	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Kristjansson (1999)	Sweden	1994	18 months	fruits (apple)	Both IgE and non-IgE mediated	0.9 † (0.2-2.9) n=328	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	fruits (banana)	Both IgE and non-IgE mediated	0.9 † (0.2-2.9) n=328	-	-	-	-	-	-	-	-	-
Ostblom (2008 a)	Sweden	1999-2000	4 years	fruits (banana)	Both IgE and non-IgE mediated	0.3 † (0.1-0.6) n=2563	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	fruits (citrus)	Both IgE and non-IgE mediated	6.7 † (4.4-10-1) n=328	-	-	-	-	-	-	-	-	-
Ostblom (2008 a)	Sweden	1999-2000	4 years	fruits (citrus)	Both IgE and non-IgE mediated	5 † (4.2-5.9) n=2563	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	fruits (plum/cherry)	Both IgE and non-IgE mediated	0.9 † (0.2-2.9) n=328	-	-	-	-	-	-	-	-	-
Ostblom (2008 a)	Sweden	1999-2000	4 years	fruits (stonefruit)	Both IgE and non-IgE mediated	3.4 † (2.7-4.2) n=2563	-	-	-	-	-	-	-	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	fruits (apple)	Both IgE and non-IgE (no SPT or SIgE)	0.6 † (0.4-0.9) n=4400	-	-	-	-	-	-	-	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	fruits (banana)	Both IgE and non-IgE (no SPT or SIgE)	0.4 † (0.2-0.6) n=4400	-	-	-	-	-	-	-	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	fruits (orange)	Both IgE and non-IgE (no SPT or SIgE)	0.5 † (0.4-0.8) n=4400	-	-	-	-	-	-	-	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	fruits (strawberry)	Both IgE and non-IgE (no SPT or SIgE)	0.6 † (0.4-0.9) n=4400	-	-	-	-	-	-	-	-	-
Gelincik (2008)	Turkey	nr	18 years +	fruits (banana)	Both Ige and non-IgE mediated	0.2 ‡ (0.1-0.3) n=11816	-	-	-	-	0 † (0.0-0.1) n=11816	0 (0.0-0.1) n=11816	-	0 † (0-0.1) n=11816	-
Orhan (2009)	Turkey	2006	6-9 years	fruits (banana)	IgE mediated only	0.1 † (0.0 - 0.4) n=2739	-	-	-	-	0.1 † (0 - 0.3) n=2739	-	-	0 † (0- 0.2) n=2739	-
Gelincik (2008)	Turkey	nr	18 years +	fruits (grape)	Both Ige and non-IgE mediated	0.2 ‡ (0.1-0.3) n=11816	-	-	-	-	-	-	-	0 † (0-0.0) n=11816	-
Mustafayev (2012)	Turkey	2010	10-11 years	fruits (kiwi)	IgE mediated only	-	-	-	-	-	-	-	0.1 † (0.0-0.8) n=813	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Orhan (2009)	Turkey	2006	6-9 years	fruits (kiwi)	IgE mediated only	0.3 † (0.1 - 0.6) n=2739	-	-	-	-	0.3 † (0.1 - 0.6) n=2739	-	-	0.1 † (0 - 0.4) n=2739	-
Gelincik (2008)	Turkey	nr	18 years +	fruits (orange)	Both Ige and non-IgE mediated	0.2 ‡ (0.1-0.3) n=11816	-	-	-	-	0 † (0.0-0.1) n=11816	0 (0.0-0.1) n=11816	-	0 † (0.0-0.1) n=11816	-
Mustafayev (2012)	Turkey	2010	10-11 years	fruits (peach)	IgE mediated only	-	-	-	-	-	-	-	0.1 † (0.0-0.8) n=813	-	-
Gelincik (2008)	Turkey	nr	18 years +	fruits (peach)	Both Ige and non-IgE mediated	0.3 ‡ (0.2-0.4) n=11816	-	-	-	-	-	-	-	0 † (0-0.0) n=11816	-
Gelincik (2008)	Turkey	nr	18 years +	fruits (pear)	Both Ige and non-IgE mediated	-	-	-	-	-	0 † (0-0.1) n=11816	0 (0-0.1) n=11816	-	0 † (0-0.1) n=11816	-
Gelincik (2008)	Turkey	nr	18 years +	fruits (strawberry)	Both Ige and non-IgE mediated	0.7 ‡ (0.5-0.8) n=11816	-	-	-	-	0 † (0-0.1) n=11816	0 (0-0.1) n=11816	-	0 † (0-0.1) n=11816	-
Orhan (2009)	Turkey	2006	6-9 years	fruits (strawberry)	IgE mediated only	0.1 † (0 - 0.3) n=2739	-	-	-	-	0 † (0 - 0.2) n=2739	-	-	0 † (0 - 0.2) n=2739	-
Emmett (1999)	United Kingdom	1995-1996	15 + years	fruits	Both Ige and non-IgE mediated	0.5 † (0.4-0.6) n=16420	-	-	-	-	-	-	-	-	-
Venter (2006)	United Kingdom	2003-2004	6 years	fruits (banana)	Both IgE and non-IgE mediated	-	-	-	0.1 † (0-0.9) n=700	-	-	-	-	-	0.1 † (0-0.8) n=798
Young (1994)	United Kingdom	nr	nr	fruits (citrus)	Both IgE and non IgE mediated (no SPT or SIgE)	3.2 † (3.2-3.8) n=18880	-	-	-	-	-	-	-	-	-
Young (1994)	United Kingdom	nr	nr	fruits (non citrus)	Both IgE and non IgE mediated (no SPT or SIgE)	1 † (0.9-1.2) n=18880	-	-	-	-	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	1 year	fruits (pineapple)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0 † (0-0.5) n=900
Venter (2008)	United Kingdom	2001-2005	2 years	fruits (pineapple)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0 † (0-0.6) n=858
Venter (2008)	United Kingdom	2001-2005	3 years	fruits (pineapple)	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0.1 † (0.0-0.2) n=891
Venter (2006)	United Kingdom	2003-2004	6 years	fruits (strawberry)	Both IgE and non-IgE mediated	0.8 † (0.3-1.7) n=798	-	-	-	-	-	-	-	-	-

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† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
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Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.19: Fruits allergy prevalence in non-European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Woods (1998)	Australia	1998	20-44years	fruits	Both IgE and non IgE mediated	2.8 † (1.8-4.5) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	fruits (dried)	IgE mediated only	0.3 † (0.1-1.2) n=669	-	-	-	-	-	-	-	-	-
Soller (2012)	Canada	2008-2009	<18 years	fruits	"likely" IgE mediated (no SPT or SIgE)	1.14 (0.68-1.60) n=nr	-	-	-	-	-	-	-	-	-
Soller (2012)	Canada	2008-2009	>18 years	fruits	"likely" IgE mediated (no SPT or SIgE)	1.61 (1.32-1.89) n=nr	-	-	-	-	-	-	-	-	-
Chen (2011)	China	2009	0-12 months	fruits (orange)	IgE mediated only	-	-	-	0.2 † (0-1.3) n=477	-	-	-	-	-	-
Hu (2010)	China	1999	0-24 months	fruits (orange)	IgE mediated only	-	-	-	1 † (0.3-3.1) n=304	-	-	-	-	-	-
Hu (2010)	China	2009	0-24 months	fruits (orange)	IgE mediated only	-	-	-	0 † (0-1.2) n=382	-	-	-	-	-	-
Marrugo (2008)	Colombia	nr	All ages	fruit/ vegetables	Both IgE and non-IgE mediated (no SPT or SIgE)	6.2 † (5.4-7.2) n=3099	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	fruits (apple)	IgE mediated only	0.1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	fruits (banana)	IgE mediated only	0.1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	fruits (mango)	IgE mediated only	0.4 (nr) n=1407	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Obeng (2011)	Ghana	2006-2008	5-16 years	fruits (melon)	IgE mediated only	0.3 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	fruits (orange)	IgE mediated only	0 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	fruits (pawpaw)	IgE mediated only	0.3 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	fruits (pineapple)	IgE mediated only	1.1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Leung (2009)	Hong Kong	2006-2007	2-7 years	fruits (orange/ banana)	IgE mediated only (no SPT or SIgE)	0.1 † (0.1-.0.3) n=3677	-	-	-	-	-	-	-	-	0.1 † (0.1-0.3) n=3677
Dalal (2002)	Israel	nr	0-2years	fruits (strawberry)	IgE mediated only	-	0 † (0.0 - 0.2) n=9070	-	-	-	0 † (0 - 0.1) n=9070	-	-	-	-
Kim (2011)	Korea	2006-2007	0-12 months	fruits	IgE mediated only (no SPT or SIgE)	-	-	0.6 † (0.3-1.3) n=1177	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	fruits	IgE mediated only (no SPT or SIgE)	0.1 † (0.1-0.2) n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	fruits	IgE mediated only (no SPT or SIgE)	0.3 † (0.2-0.4) n=14777	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	fruits (apple)	IgE mediated only (no SPT or SIgE)	0 † (0.0-0.1) n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	fruits (apple)	IgE mediated only (no SPT or SIgE)	0.1 † (0.1-0.2) n=14777	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	fruits (banana)	IgE mediated only (no SPT or SIgE)	0 † (0.0-0.0) n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	fruits (banana)	IgE mediated only (no SPT or SIgE)	0 † (0.0-0.1) n=14777	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	fruits (peach)	IgE mediated only (no SPT or SIgE)	0.2 † (0.2-0.3) n=27425	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Oh (2004)	Korea	2000	12-15 years	fruits (peach)	IgE mediated only (no SPT or SIgE)	0.7 † (0.5-0.8) n=14777	-	-	-	-	-	-	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	fruits (grape)	IgE mediated only	-	-	-	-	-	-	0.7 (0.3-1.5) n=1010	-	-	-
Wu (2012)	Taiwan	2004	<3 years	fruits (kiwi)	IgE mediated only (no SPT or SIgE)	-	-	0.1 † (0.0-0.8) n=813	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	>19 years	fruits (kiwi)	IgE mediated only (no SPT or SIgE)	-	-	0.1 † (0.1-0.2) n=14036	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	4-18 years	fruits (kiwi)	IgE mediated only (no SPT or SIgE)	-	-	0.3 † (0.2-0.4) n=15169	-	-	-	-	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	fruits (litchi)	IgE mediated only	-	-	-	-	-	-	3.4 (2.4-4.7) n=1010	-	-	-
Wu (2012)	Taiwan	2004	<3 years	fruits (mango)	IgE mediated only (no SPT or SIgE)	-	-	0.1 † (0.0-0.8) n=813	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	>19 years	fruits (mango)	IgE mediated only (no SPT or SIgE)	-	-	1.2 † (1.0-1.4) n=14036	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	4-18 years	fruits (mango)	IgE mediated only (no SPT or SIgE)	-	-	1.4 † (1.2-1.6) n=15169	-	-	-	-	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	fruits (melon)	IgE mediated only	-	-	-	-	-	-	2.4 (2.4-4.7) n=1010	-	-	-
Al-Hammadi (2010)	United Arab Emirates (Emirate of Abu Dhabi)	2006	6-9 years	fruits	IgE mediated only (no SPT or SIgE)	-	-	3.3 † (1.8-5.7) n=397	-	-	-	-	-	-	-
Bock (1987)	United States	1980-1984	1 year	fruit juice	Both IgE and non-IgE mediated	10.8 † (8.3-14) n=480	-	-	-	-	-	-	-	-	7.9 † (5.7-10.8) n=480
Bock (1987)	United States	1980-1984	2 years	fruit juice	Both IgE and non-IgE mediated	5 † (3.3-7.4) n=480	-	-	-	-	-	-	-	-	4.4 † (3-6.7) n=480
Bock (1987)	United States	1980-1984	3 years	fruit juice	Both IgE and non-IgE mediated	1.7 † (0.8-3.4) n=480	-	-	-	-	-	-	-	-	1.3 † (0.5-2.8) n=480

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Gupta (2011)	United States	2009-2010	0-2 years	fruits (strawberry)	IgE mediated (no SPT or SIgE)	-	0.5 (0.3-0.7) n=5429	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	3-5 years	fruits (strawberry)	IgE mediated (no SPT or SIgE)	-	0.5 (0.3-0.8) n=5910	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	6-10 years	fruits (strawberry)	IgE mediated (no SPT or SIgE)	-	0.4 (0.3-0.5) n=9911	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	11-13 years	fruits (strawberry)	IgE mediated (no SPT or SIgE)	-	0.4 (0.3-0.6) n=6716	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	14-17 years	fruits (strawberry)	IgE mediated (no SPT or SIgE)	-	0.4 (0.3-0.6) n=10514	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	All ages	fruits (strawberry)	IgE mediated (no SPT or SIgE)	-	0.4 (0.4-0.5) n=3339	-	-	-	-	-	-	-	-
Vierk (2007)	United States	2001	18 years +	fruit/ vegetables	IgE mediated only (no SPT or SIgE)	3.3 † (2.9-3.9) n=4482	-	-	-	-	-	-	-	-	2† (1.7-2.5) n=4482

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.20: Milk/Dairy allergy prevalence in European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Host (2002	Denmark	1985-2000	0-1 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	2.2 (1.6-3.1) n=1749	-	-
Eller (2009)	Denmark	1999-2000	6 months	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0.4 (nr) n= nr	-	-
Eller (2009)	Denmark	1999-2000	1 year	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0.8 (nr) n= nr	-	-
Host (2002	Denmark	1985-2000	1 year	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	1.0 0.6-1.6 n=1749	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Eller (2009)	Denmark	2000-2001	18 months	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	1.1 (nr) n= nr	-	-
Host (2002)	Denmark	1985-2000	2 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0.5 (0.3-1.0) n=1749	-	-
Osterballe (2005)	Denmark	2000-2001	< 3 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0.0 (0.0-4.2) n= 111	-	0.9 † (0.1 - 5.6) n=111
Eller (2009)	Denmark	2001-2002	3 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0.7 (nr) n= nr	-	-
Host (2002)	Denmark	1985-2000	3 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0.3 (0.1-0.7) n=1749	-	-
Osterballe (2005)	Denmark	2000-2001	3 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.6 † (0.2 - 2) n=486	1.6 † (0.1 - 3.4) n=486
Osterballe (2005)	Denmark	2000-2001	3-22 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.3 † (0.1 - 2.6) n=301	1.1 † (0 - 2.1) n=301
Host (2002)	Denmark	1985-2000	5 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0.2 (0.0-0.5) n=1749	-	-
Eller (2009)	Denmark	2004-2005	6 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0 (nr) n= nr	-	-
Host (2002)	Denmark	1985-2000	10 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0.2 (0.0-0.5) n=1749	-	-
Host (2002)	Denmark	1985-2000	15 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0.1 (0.0-0.4) n=1749	-	-
Osterballe (2009)	Denmark	2001-2002	22 years	cow's milk	Both IgE and non-IgE mediated	3.3 † (2.3 - 4.8) n=843	-	-	-	-	-	-	-	-	0.1 (0.0-0.8) n=843
Osterballe (2005)	Denmark	2000-2001	>22 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0.3 † (0.1 - 1.0) n=936	0.8 † (0.4 - 1.7) n=936
Julge (2001)	Estonia	1993-1999	6 months	cow's milk	IgE mediated only	-	-	-	1.7 (nr) n=172	12 (nr) n=92	-	-	-	-	-
Julge (2001)	Estonia	1993-1999	1 year	cow's milk	IgE mediated only	-	-	-	0.9 (nr) n=220	20.7 (nr) n=116	-	-	-	-	-
Julge (2001)	Estonia	1993-1999	2 years	cow's milk	IgE mediated only	-	-	-	0 (nr) n=222	25.8 (nr) n=120	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Julge (2001)	Estonia	1993-1999	5 years	cow's milk	IgE mediated only	-	-	-	-	23.2 (nr) n=207	-	-	-	-	-
Saarinen (1999)	Finland	1994-1996	0-34 months	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	10.0 (9.3-10.8) n=6209	-	-	-	-	-	-	1.9 (1.6-2.3) n=555	-	-
Kajosaari (1982)	Finland	1980-1981	1 year	cow's milk	Both IgE and non-IgE mediated ((no SPT or SIgE))	2 (nr) n=261	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	1 year	cow's milk	Both IgE and non-IgE mediated ((no SPT or SIgE))	5.4 † (4.0-7.2) n=853	-	5.6 † (4.2 - 7.5) n=853	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	2 years	cow's milk	Both – not clearly specified	5 (nr) n=202	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	2 years	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	6.8 † (5.3-8.8) n=852	-	6.7 † (5.2-8.6) n=852	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	3 years	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	2 (nr) n=200	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	3 years	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	5.9 † (4.4 -7.8) n=784	-	7.5 † (5.8 - 9.7) n=784	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	4 years	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	7.6 † (5.9 - 9.7) n=819	-	6 † (4.5 - 7.9) n=819	-	-	-	-	-	-	-
Isolauri (2004)	Finland	nr	7 years (born 1990)	cow's milk	Both IgE and non IgE mediated	-	14 (7.9-22.4) n=100	-	-	9 (4.2-16.4) n=100	-		-	-	-
Isolauri (2004)	Finland	nr	27 years (born 1963-1966	cow's milk	Both IgE and non IgE mediated	-	10 (4.9-17.6) n=100	-	-	4.4 (1.2-10.8) n=100	-		-	-	-
Isolauri (2004)	Finland	nr	47 years (born 1943-1946	cow's milk	Both IgE and non IgE mediated	-	14 (8.0-22.6) n=100	-	-	1.0 (0.03-5.5) n=100	-		-	-	-
Isolauri (2004)	Finland	nr	67 years (born 1923-1926)	cow's milk	Both IgE and non IgE mediated	-	13 (7.1-21.2) n=100	-	-	7.1 (2.9-14.0) n=100	-		-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Rance (2005)	France	2002	2-14 years	cow's milk	Both IgE and non-IgE mediated ((no SPT or SIgE))	1.1 † (0.7 - 1.6) n=2716	-	-	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	5.5 † (1.3-7.1) n=1086	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	cow's milk	Both IgE and non_IgE mediated	-	-	-	-	-	0.2 (0.1-0.4) n=3156	-	-	0.2 (0.1-0.4) n=3156	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	cow's milk	Both IgE and non_IgE mediated	-	-	-	-	-	-	-	-	-	0.2 (0.1-0.5) n=3156
Schafer (1999)	Germany	1994	5-6 years	cow's milk	Both IgE and non_IgE mediated	-	-	-	3.9 † (2.9-5.2) n=1235	-	-	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	cow's milk	Both IgE and non_IgE mediated	1.8 † (nr) n= nr	-	-	2.3 † (nr) n= nr	-	-	-	-	-	-
Krause (2002)	Greenland	1998	5-18 years	cow's milk	IgE mediated only	-	-	-	-	0.5 † (0.2-1.2) n=1031	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	cow's milk	IgE mediated only	-	-	-	13.9 † (5.2-30.3) n=36	8.3 † (2.2-23.6) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	cow's milk	IgE mediated only	-	-	-	12.8 † (7.5-20.9) n=109	4.6 † (1.7-10.9) n=109	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	milk (casein)	IgE mediated only	-	-	-	5.6 † (1.0-20.0) n=36	13.9 † (2.2-23.6) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	milk (casein)	IgE mediated only	-	-	-	14.7 † (8.9-23.0) n=109	8.3 † (4.1-15.5) n=109	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	cow's milk	Both IgE and non-IgE mediated	10.8 † (7.7-14.8) n=324	-	-	-	-	0.3 † (0.0-2.0) n=324	-	-	-	-
Kilgallen (1996)	Ireland	nr	0-6 months	cow's milk	Both IgE and Non-IgE mediated(no SPT or SIgE)	0 † (0-6.1) n=75	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	6-12 months	cow's milk	Both IgE and Non-IgE mediated(no SPT or SIgE)	5.3 † (1.7-13.8) n=75	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Kilgallen (1996)	Ireland	nr	12-24 months	cow's milk	Both IgE and Non-IgE mediated(no SPT or SIgE)	5.3 † (2.5-10.6) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	24-36 months	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	1.3 † (0.2-5.2) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	36-48 months	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	2.7 † (0.9-7.1) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	0-6 months	dairy products	Both IgE and non-IgE mediated (no SPT or SIgE)	0 † (0-6.1) n=75	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	6-12 months	dairy products	Both IgE and non-IgE mediated (no SPT or SIgE)	4 † (1.0-12.0) n=75	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	12-24 months	dairy products	Both IgE and non-IgE mediated (no SPT or SIgE)	4.7 † (2.1-9.8) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	24-36 months	dairy products	Both IgE and non-IgE mediated (no SPT or SIgE)	0.7 † (0.0-4.2) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	36-48 months	dairy products	Both IgE and non-IgE mediated (no SPT or SIgE)	2 † (0.5-6.2) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	0-6 months	dairy products (yoghurt)	Both IgE and non-IgE mediated (no SPT or SIgE)	0 † (0-6.1) n=75	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	6-12 months	dairy products (yoghurt)	Both IgE and non-IgE mediated (no SPT or SIgE)	0 † (0-6.1) n=75	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	12-24 months	dairy products (yoghurt)	Both IgE and non-IgE mediated (no SPT or SIgE)	1.3 † (0.2-5.2) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	24-36 months	dairy products (yoghurt)	Both IgE and non-IgE mediated (no SPT or SIgE)	0 † (0-3.1) n=150	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Kilgallen (1996)	Ireland	nr	36-48 months	dairy products (yoghurt)	Both IgE and non-IgE mediated (no SPT or SIgE)	1.3 † (0.2-5.2) n=150	-	-	-	-	-	-	-	-	-
Frongia (2005)	Italy	2003	12-24 months	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	-	-	5.4 † (4.7-6.1) n=4602	-	-	-	-	-	-	-
Ronchetti (2008)	Italy	2005 - 2006	9 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	0.5 † (0.0-3.5) n=184	-	-	-	-	-	11.4 † (7.4-17.1) n=184
Ronchetti (2008)	Italy	2005 - 2006	13 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	2 † (0.7-5.5) n=196	-	-	-	-	-	4.1 † (1.9-8.2) n=196
Eggesbo (1999)	Norway	1993-1995	1 year	cow's milk	Both IgE and non-IgE mediated	7.5 (6.6-8.6) n=3366	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	18 months	cow's milk	Both IgE and non-IgE mediated	5.5 (4.7-6.4) n=3278	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	2 years	cow's milk	Both IgE and non-IgE mediated	5 (4.3-5.9) n=2979	-	-	-	-	-	-	-	-	-
Ro (2012)	Norway	2002-2006	2 years	cow's milk	IgE mediated only	-	-	-	0.9 † (0.2 - 2.7) n=352	4.8 † (2.9 - 7.8) n=352	-	-	-	-	-
Falcao (2004)	Portugal	2000	>39 years	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	0.3 † (0-1.2) n=659	-	-	-	-	-	-	-	-	-
Martinez-Gimeno (2000)	Spain	nr	6-13 years	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	21 † (19.9 - 22.1) n=5163	-	-	-	-	-	-	-	-	-
Ostblom (2008 b)	Sweden	1995-2004	1 year	cow's milk	Both IgE and non-IgE mediated	4.5 † (3.8-5.3) n=3104	-	2.2 † (1.7-2.8) n=3104	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	cow's milk	Both IgE and non-IgE mediated	5.2 † (3.1-8.3) n=328	-	-	-	-	0.6 † (0.1-2.4) n=328	-	-	-	-
Ostblom (2008 b)	Sweden	1996-1998	2 years	cow's milk	Both IgE and non-IgE mediated	4 † (3.3-4.8) n=3104	-	2.2 † (1.7-2.8) n=3104	-	-	-	-	-	-	-
Ostblom (2008 b)	Sweden	1998-2000	4 years	cow's milk	Both IgE and non-IgE mediated	3.6 † (3.0-4.3) n=3104	-	2.0 † (1.6-2.6) n=3104	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Ostblom (2008 a)	Sweden	1999-2000	4 years	cow's milk	Both IgE and non-IgE mediated	5 † (4.2-6.0) n=2563	-	-	-	8 † (7.0-9.1) n=2563	-	1.8 (1.3-2.4) n=2563	-	-	-
Ostblom (2008 b)	Sweden	2002-2004	8 years	cow's milk	Both IgE and non-IgE mediated	2.8 † (2.3-3.5) n=3104	-	1.8 † (1.4-2.4) n=3104	-	-	-	-	-	-	-
Bjornsson (1996)	Sweden	1991-1992	20-44 years	cow's milk	IgE mediated only	-	-	-	-	1.1 † (0.6-1.8) n=1397	-	-	-	-	-
Schrander	The Netherlands	Unclear	0-1 years	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	18.2† (16.2-20.6) 1158	-	-	-	-	-	-	2.3† (1.5-3.3) n=1158	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	1.5 † (1.2-1.9) n=4400	-	-	-	-	-	-	-	-	-
Mustafayev (2012)	Turkey	2010	10-11 years	cheese	IgE mediated only	-	-	-	-	-	-	-	0.1 † (0.0-0.8) n=813	-	-
Altintas (1995)	Turkey	1992-1993	0-1 years	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	-	-	1.4 † (0.9-2.2) n=1348	-	-	-	-	-	-	-
Kucukosmanoglu (2008 b)	Turkey	2002-2003	8-18months	cow's milk	IgE mediated only	-	-	-	0.6 † (0.2 - 1.4) n=1015	-	-	0.2 (0.0-0.8) n=1015	0.3 † (0.1-0.9) n=1015	-	-
Altintas (1995)	Turkey	1992-1993	1-2 years	cow's milk	Both IgE and non-IgE mediated	-	-	1.2 † (0.7-2.0) n=1348	-	-	-	-	-	-	-
Orhan (2009)	Turkey	2006	6-9 years	cow's milk	IgE mediated only	0.9 † (0.6 - 1.4) n=2739	-	-	-	-	0.4 † (0.2 - 0.7) n=2739	-	-	0.1 † (0.1 - 0.4) n=2739	-
Mustafayev (2012)	Turkey	2010	10-11 years	cow's milk	IgE mediated only	1.5 † (1.2-1.8) n=6963	-	-	1.1 † (0.9-1.4) n=6134	-	-	-	-	-	-
Gelincik (2008)	Turkey	nr	18 years +	cow's milk	Both IgE and non-IgE mediated	0.2 ‡ (0.2-0.4) n=11816	-	-	-	-	-	-	-	0.1† (0.0-0.1) n=11816	-
Emmett (1999)	United Kingdom	1995-1996	15 + years	cheese	Both IgE and non-IgE mediated (no SPT or Spes IgE)	0.2 † (0.1-0.3) n=16420	-	-	-	-	-	-	-	-	-
Young (1994)	United Kingdom	nr	All ages	cheese	Both IgE and non-IgE mediated (no	2.5 † (2.3-2.7) n=18880	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
					SPT or Spes IgE)										
Venter (2008)	United Kingdom	2001-2005	1 year	cow's milk	Both IgE and non-IgE mediated	-	-	-	0.3 † (0.0-1.0) n=763	-	-	-	-	-	2.4 † (1.6-3.7) n=900
Venter (2008)	United Kingdom	2001-2005	2 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	0.5 † (0.1-1.4) n=658	-	-	-	-	-	1.2 † (0.1-2.2) n=858
Venter (2008)	United Kingdom	2001-2005	3 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	0.5 † (0.1-1.5) n=642	-	-	-	-	-	0.4 † (0.1-1.2) n=891
Arshad (2001)	United Kingdom	1993-1994	4 years	cow's milk	IgE only	-	-	-	1.3 † (0.7 - 2.3) n=981	-	-	-	-	-	-
Venter (2006)	United Kingdom	2003-2004	6 years	cow's milk	Both IgE and non-IgE mediated	3.6 † (2.5-5.2) n=798	-	-	0.4 † (0.1 -1.4) n=700	-	-	-	-	-	0.8 † (0.3-1.7) n=798
Roberts (2005)	United Kingdom	1998-2000	7 years	cow's milk	IgE mediated	-	-	-	0.2 † (0.1 - 0.6) n=2007	-	-	-	-	-	-
Emmett (1999)	United Kingdom	1995-1996	15 + years	cow's milk	Both IgE and non-IgE mediated	0.7 † (0.6-0.8) n=16420	-	-	-	-	-	-	-	-	-
Young (1994)	United Kingdom	nr	All ages	cow's milk	Both IgE and non-IgE mediated	2.7 † (2.5-3.0) n=18880	-	-	-	-	-	-	-	-	-
Pereira (2005)	United Kingdom	2002-2003	11 year olds	milk/dairy	Both IgE and non-IgE mediated	2.8 † (1.8-4.3) n=775	-	-	0.3 † (0.1-1.2) n=699	-	-	-	-	-	-
Pereira (2005)	United Kingdom	2002-2003	15 year olds	milk/dairy	Both IgE and non-IgE mediated	3.4 † (2.3-5.1) n=757	-	-	0.3 † (0.1-1.2) n=649	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

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Table 1.21: Milk/Dairy allergy prevalence in non-European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Types of food allergy		Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC		
							95% Prevalence (CI)									
Osborne (2011)	Australia	2007-2010	12-15 months	cow's milk	IgE mediated only	6.1 (5.1-7.0) n= nr	-	2.7 (2.1-3.4) n= nr	-	5.6 (3.2-8.0) n=355	-	-	-	-	-	-
Woods (2002)	Australia	1992-1998	26-50 years	cow's milk	IgE mediated only	4.8 † (3.1-7.3) n=457	-	-	-	0.7 † (0.2-2.1) n=457	-	0 † (0-1.0) n=457	-	-	-	-
Woods (1998)	Australia	1998	20-44years	dairy products	Both IgE and non-IgE mediated	1.9 † (1.1-3.4) n=669	-	-	-	-	-	-	-	-	-	-
Soller (2012)	Canada	2008-2009	<18 years	cow's milk	"likely" IgE mediated (no SPT or SIgE)	2.23 (1.51-2.95) n= nr	-	-	-	-	-	-	-	-	-	-
Soller (2012)	Canada	2008-2009	>18 years	cow's milk	"likely" IgE mediated (no SPT or SIgE	1.89 (1.56-2.21) n= nr	-	-	-	-	-	-	-	-	-	-
Gerrard (1973)	Canada	nr	6-36 months	cow's milk	Both IgE and non-IgE mediated (no SPT or SIgE)	-	-	7.5 † (5.8-9.6) n=787	-	-	-	-	-	-	-	-
Chen (2011)	China	2009	0-12 months	cow's milk	IgE mediated only	-		-	-	2.7 † (1.5-4.7) n=477	-	-	-	1.3 † (0.5-2.9) n=477	-	-
Hu (2010)	China	1999	0-24 months	cow's milk	IgE mediated only	-	-	-	-	3.3 † (1.7-6.2) n=304	-	-	-	1.6 † (0.6-3.9) n=314	-	-
Hu (2010)	China	2009	0-24 months	cow's milk	IgE mediated only	-	-	-	-	6.5 † (4.4-9.6) n=382	-	-	-	3.5 † (2-5.9) n=401	-	-
Sai (2011)	China	2008-2009	adults	cow's milk	IgG mediated only	-	-	-	-	-	-	-	-	-	-	24.5 † (23.8-25.3) n=12765
Chen (2012)	China (Chongqing)	2009-2010	0-2 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	-	3.5 † (2.2-5.4) n=550
Chen (2012)	China (Hangzhou)	2009-2010	0-2 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	-	0.8 † (0.3-2.3) n=481
Chen (2012)	China (Zhuhai)	2009-2010	0-2 years	cow's milk	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	-	2.8 † (1.7-4.6) n=573
Marrugo (2008)	Colombia	nr	All ages	cow's milk	IgE and non-IgE mediated (no SPT or SIgE)	1.4 † (1.1-1.9) n=3099	-	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Types of food allergy		Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC		
						95% Prevalence (CI)										
Obeng (2011)	Ghana	2006-2008	5-16 years	cow's milk	IgE mediated only	0.2 (nr) n=1407	-	-	-	-	-	-	-	-	-	-
Leung (2009)	Hong Kong	2006-2007	2-7 years	cheese	IgE mediated only (no SPT or SIgE)	0.2 † (0.1-0.5) n=3677	-	-	-	-	-	-	-	-	-	0.2 † (0.1-0.5) n=3677
Leung (2009)	Hong Kong	2006-2007	2-7 years	cow's milk	IgE mediated only (no SPT or SIgE)	0.5 † (0.3-0.8) n=3677	-	-	-	-	-	-	-	-	-	0.3 † (0.2-0.6) n=3677
Dalal (2002)	Israel	nr	0-2years	cow's milk	IgE mediated only	-	-	0.4 † (0.3 -0.6) n=9070	-	-	-	0.3 † (0.2-0.5) n=9070	-	-	-	-
Katz (2010)	Israel	2004-2006	0-2 years	cow's milk	Both IgE and non-IgE mediated	2.9 (2.6-3.2) n=13019	-	-	-	-	-	-	-		-	-71 + 66 out of 13019 – (1.1%) based on hx or SPT and pos challenge
Kim (2011)	Korea	2006-2007	0-12 months	cow's milk	IgE mediated only((no SPT or SIgE))	-	-	-	1.7 † (1.1-2.7) n=1177	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	cow's milk	IgE mediated only (no SPT or SIgE)	0.7 † (0.6-0.8) n=27425	-	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	cow's milk	IgE mediated only (no SPT or SIgE)	0.4 † (0.3-0.5) n=14777	-	-	-	-	-	-	-	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	alpha lactalbumin	IgE mediated only	-	-	-	-	-	-	-	14.5 (12.4-16.8) n=1010	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	BLG	IgE mediated only	-	-	-	-	-	-	-	6.7 (5.3-8.5) n=1010	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	cheese	IgE mediated only	-	-	-	-	-	-	-	6.2 (4.9-8.0) n=1010	-	-	-
Wu (2012)	Taiwan	2004	<3 years	cow's milk	IgE mediated only (no SPT or SIgE)	-	-	-	1.1 † (0.5-2.2) n=813	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	>19 years	cow's milk	IgE mediated only (no SPT or SIgE)	-	-	-	0.5 † (0.4-0.6) n=14036	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	4-18 years	cow's milk	IgE mediated only (no SPT or SIgE)	-	-	-	0.9 † (0.8-1.1) n=15169	-	-	-	-	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	milk	IgE mediated	-	-	-	-	-	-	-	13.3	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC		
							95% Prevalence (CI)									
				(casein)	only								(11.3-15.5) n=1010			
Wan (2012)	Taiwan	nr	6-8 years	milk (goat)	IgE mediated only	-	-	-	-	-	-	-	10.7 (8.9-12.8) n=1010	-	-	-
Santadusit (2005)	Thailand	nr	6 months - 6yrs	cow's milk	IgE mediated only	1.7 † (0.9 -3.1) n=656	-	-	-	-	-	-	-	-	-	-
Lao-araya (2012)	Thailand	2010	3-7years	cow's milk	IgE mediated only	2 † (1.0-3.9) n=452	-	-	-	-	-	-	-	0 † (0-1.1) n=452	-	-
Al-Hammadi (2010)	United Arab Emirates (Emirate of Abu Dhabi)	2006	6-9 years	cow's milk	IgE mediated only (no SPT or SIgE)	-	-	-	1 † (0.3-2.7) n=397	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	0-2 years	cow's milk	IgE mediated only (no SPT or SIgE)	-	-	2 (1.6-2.4) n=5429	-	-	-	-	-	-	-	-
Branum (2009)	United States	2005-2006	< 18 years	cow's milk	IgE mediated only (not clearly defined)	-	-	-	-	-	12.2 (nr) n=nr	-	-	-	-	-
Kumar (2011)	United States	2011 (yr pub)	6 months - 6 yrs	cow's milk	IgE mediated only	-	-	-	-	-	21.6 † (19.3-24.2) n=1104	-	-	-	-	-
Bock (1987)	United States	1980-1984	1 year	cow's milk	Both IgE and non-IgE mediated	13.1 † (10.3-16.6) n=480	-	-	-	-	-	-	-	-	-	5 † (3.3-7.4) n=480
Liu (2010)	United States	2005-2006	1-5 years	cow's milk	IgE mediated only	-	-	-	-	-	22 (nr) n=909	-	-	-	-	1.8 (nr) n=nr
Keet (2012)	United States	2005-2006	1-21 years	cow's milk	IgE mediated only	-	-	-	-	-	11 (nr) n=3550	-	-	-	-	-
Bock (1987)	United States	1980-1984	2 years	cow's milk	Both IgE and non-IgE mediated	1.3 † (0.5-3.0) n=480	-	-	-	-	-	-	-	-	-	0.2 † (0-1.3) n=480
Bock (1987)	United States	1980-1984	3 years	cow's milk	Both IgE and non-IgE mediated	0.6 † (0.2-2) n=480	-	-	-	-	-	-	-	-	-	0 † (0-1) n=480
Gupta (2011)	United States	2009-2010	3-5 years	cow's milk	IgE mediated (no SPT or SIgE)	-	-	2 (1.7-2.5) n=5910	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	6-10 years	cow's milk	IgE mediated (no SPT or SIgE)	-	-	1.5 (1.2-1.8) n=9911	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Types of food allergy		Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC		
							95% Prevalence (CI)									
Liu (2010)	United States	2005-2006	6-19 years	cow's milk	IgE mediated only	-	-	-	-	-	8.1 (nr) n=2869	-	-	-	-	0.3 (nr) n=nr
Gupta (2011)	United States	2009-2010	11-13 years	cow's milk	IgE mediated (no SPT or SIgE)	-	-	1.4 (1.1-1.8) n=6716	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	14-17 years	cow's milk	IgE mediated (no SPT or SIgE)	-	-	1.6 (1.3-1.9) n=10514	-	-	-	-	-	-	-	-
Greenhawt (2009)	United States	nr	18 years+	cow's milk	IgE mediated (no SPT or SIgE)	10.5 † (8.1-13.6) n=513	-	-	-	-	-	-	-	-	-	-
Liu (2010)	United States	2005-2006	20-39 years	cow's milk	IgE mediated only	-	-	-	-	-	3.2 (nr) n=1672	-	-	-	-	0.2 (nr) n=nr
Liu (2010)	United States	2005-2006	40-59 years	cow's milk	IgE mediated only	-	-	-	-	-	4.9 (nr) n=1361	-	-	-	-	0.5 (nr) n=nr
Liu (2010)	United States	2005-2006	60+ years	cow's milk	IgE mediated only	-	-	-	-	-	3.8 (nr) n=1392	-	-	-	-	0.3 (nr) n=nr
Liu (2010)	United States	2005-2006	All ages	cow's milk	IgE mediated only	-	-	-	-	-	5.7 (nr) n=8203	-	-	-	-	0.4 (nr) n=nr
Gupta (2011)	United States	2009-2010	All ages	cow's milk	IgE mediated (no SPT or SIgE)	-	-	1.7 (1.5-1.8) n=3339	-	-	-	-	-	-	-	-
Vierk (2007)	United States	2001	18 years +	milk/dairy	IgE mediated only (no SPT or SIgE)	2.4 † (2.0-2.9) n=4482	-	-	-	-	-	-	-	-	-	1.4 † (1.1-1.8) n=4482

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

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Table 1.22: Mustard allergy prevalence in European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Touraine (2002)	France	2000-2001	5-17 years	mustard	Both IgE and non-IgE mediated (no SPT or SIgE)	3 † (2.1-4.3) n=1086	-	-	-	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

No Non-European studies looking at mustard where included within this review

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Table 1.23: Peanut allergy prevalence in European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen)	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Osterballe (2005)	Denmark	2000-2001	< 3 years	peanut	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0 † (0.0-4.2) n=111	-	0 † (0.0-4.2) n=111
Osterballe (2005)	Denmark	2000-2001	>22 years	peanut	Both IgE and non IgE mediated	-	-	-	-	-	-	-	-	0.4 † (0.1-1.2) n=936	1.2 † (0.6-2.2) n=936
Eller (2009)	Denmark	1998-1999	3 months	peanut	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	0 (nr) n=nr	-	-
Eller (2009)	Denmark	1999-2000	6 months	peanut	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	0 (nr) n=nr	-	-
Eller (2009)	Denmark	1999-2000	9 months	peanut	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	0 (nr) n=nr	-	-
Eller (2009)	Denmark	1999-2000	1 year	peanut	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	0 (nr) n=nr	-	-
Osterballe (2005)	Denmark	2000-2001	3 years	peanut	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	0.2 † (0.0-1.3) n=486	1.6 † (0.8-3.4) n=486
Eller (2009)	Denmark	2001-2002	3 years	peanut	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	0.4 (nr) n=nr	-	-
Osterballe (2005)	Denmark	2000-2001	3-22 years	peanut	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0.0-1.6) n=301	1 † (0.3-3.1) n=301
Eller (2009)	Denmark	2004-2005	6 years	peanut	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	0.4 (nr) n=nr	-	-
Mortz (2005)	Denmark	1995-1996	14 years	peanut	IgE mediated only	-	-	-	3.4 † (2.1-5.4) n=558	5.8 † (4.4-7.6) n=862	-	-	0.5 † (0.2-1.3) n=979	-	-
Osterballe (2009)	Denmark	2001-2002	22 years	peanut	Both Ige and non-IgE mediated	5.3 † (4.0-7.1) n=843	-	-	-	-	-	-	-	-	0.6 (0.2-1.4) n=843
Rance (2005)	France	2002	2-14 years	peanut	Both IgE and non-IgE mediated (no SPT or SIgE)	0.7 † (0.5 - 1.2) n=2716	-	-	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	peanut	Both IgE and non-IgE mediated (no SPT or SIgE)	15 † (13-17.3) n=1086	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen)	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Zuberbier (2004)	Germany	1999-2000	0-80+ years	peanut	Both IgE and non-IgE mediated	-	-	-	-	-	0.6 (0.4-1.0) n=3156	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	peanut	Both IgE and non-IgE mediated	1.3† (nr) n=nr	-	-	6.8† (nr) n=nr	-	-	-	-	-	-
Krause (2002)	Greenland	1998	5-18 years	peanut	IgE mediated only	-	-	-	-	2.6 † (1.8-3.8) n=1031	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	peanut	IgE mediated only	-	-	-	5.6 † (1.0-20.0) n=36	0 † (0-12.0) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	peanut	IgE mediated only	-	-	-	6.4 † (2.8-13.2) n=109	1.8 † (0.3-7.1) n=109	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	peanut	Both IgE and non-IgE mediated	0 † (0-1.5) n=324	-	-	-	-	0 † (0-1.5) n=324	-	-	-	-
Ro (2012)	Norway	2002-2006	2 years	peanut	IgE mediated only	-	-	-	2.8 † (1.5-5.3) n=352	3.4 † (1.9-6.0) n=352	-	-	-	-	-
Ostblom (2008 b)	Sweden	1995-1997	1 year	peanut	Both IgE and non-IgE mediated	0.4 † (0.2-0.7) n=3104	-	0.2 † (0.1-0.4) n=3104	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	peanut	Both IgE and non-IgE mediated	0.3 † (0.0-2.0) n=328	-	-	-	-	0 † (0-1.4) n=328	-	-	-	-
Ostblom (2008 b)	Sweden	1996-1998	2 years	peanut	Both IgE and non-IgE mediated	1.2 † (0.9-1.7) n=3104	-	0.8 † (0.5-1.2) n=3104	-	-	-	-	-	-	-
Ostblom (2008 b)	Sweden	1998-2000	4 years	peanut	Both IgE and non-IgE mediated	2.8 † (2.3-3.5) n=3104	-	2.2 † (1.7-2.8) n=3104	-	-	-	-	-	-	-
Ostblom (2008 a)	Sweden	1999-2000	4 years	peanut	Both IgE and non-IgE mediated	4 † (3.3-4.8) n=2563	-	-	-	5 † (4.2-5.9) n=2563	-	2.4 (1.9-3.1) n=2563	-	-	-
Ostblom (2008 b)	Sweden	2002-2004	8 years	peanut	Both IgE and non-IgE mediated	5.2 † (4.5-6.0) n=3104	-	4 † (3.4-4.8) n=3104	-	-	-	-	-	-	-
Bjornsson (1996)	Sweden	1991-1992	20-44 years	peanut	IgE mediated only	-	-	-	-	3.1 † (2.3-4.2) n=1397	-	-	-	-	-
Mustafayev (2012)	Turkey	2010	10-11 years	peanut	IgE mediated only	1.4 † (1.1-1.7) n=6963	-	-	0.7 † (0.5-1.0) n=6134	-	-	-	0.1 † (0.0-0.8) n=813	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Gelincik (2008)	Turkey	nr	18 years +	peanut	Both Ige and non-IgE mediated	-	-	-	-	-	0 † (0.0-0.1) n=11816	0 (0-0.1) n=11816	-	0 † (0-0.0) n=11816	-
Orhan (2009)	Turkey	2006	6-9 years	peanut	IgE mediated only	0.1 † (0.0-0.4) n=2739	-	-	-	-	0.1 † (0 - 0.3) n=2739	-	-	0 † (0 - 0.2) n=2739	-
Emmett (1999)	United Kingdom	1995-1996	0-14 years	peanut	Both Ige and non-IgE mediated	-	0.2 † (0.1-0.3) n=16420	-	-	-	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	1 year	peanut	Both Ige and non-IgE mediated	-	-	-	0.4 † (0.0-1.2) n=763	-	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	2 years	peanut	Both Ige and non-IgE mediated	-	-	-	2 † (1.1-3.4) n=658	-	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	3 years	peanut	Both Ige and non-IgE mediated	-	-	-	2 † (1.1-3.5) n=642	-	-	-	-	-	1.2 † (0.6-2.3) n=891
Grundy (2002)	United Kingdom	1999-2000	3-4 years	peanut	IgE mediated only	1 † (0.6-1.8) n=1273	-	-	3.3 † (2.4-4.5) n=1246	-	-	-	1.4 † (0.9-2.3) n=1246	-	-
Hourihane (2007)	United Kingdom	2003-2005	3-6 years	peanut	IgE mediated only	-	-	-	2.8 (1.8-3.8) n=1072	-	-	-	-	1.8 # (1.1-2.7) n=1072	-
Tariq (1996)	United Kingdom	1993-1994	4 years	peanut	IgE mediated only	0.5 † (0.2-1.1) n=1218	-	-	1.3 † (0.7-2.3) n=981	-	0.5 † (0.2-1.1) n=1218	-	-	-	-
Lack (2003)	United Kingdom	1997-1998	4-6 years	peanut	IgE mediated only	-	0.4 † (0.3-0.6) n=12090	-	-	-	0.2 † (0.2-0.4) n=12090	-	-	0.2 † (0.1-0.3) n=12090	-
Venter (2006)	United Kingdom	2003-2004	6 years	peanut	Both Ige and non-IgE mediated	1.9 † (1.1-3.2) n=798	-	-	2.6 † (1.6-4.1) n=700	-	-	-	-	-	0.9 † (0.4-1.9) n=798
Roberts (2005)	United Kingdom	1998-2000	7 years	peanut	IgE mediated only	-	-	-	1.4 † (1.2-1.8) n=6213	-	-	-	-	-	-
Nicolaou (2010)	United Kingdom	2003	8 years	peanut	IgE mediated only	-	-	-	5.1 † (3.8-6.8) n=919	12.2 † (9.7-15.2) n=582	-	-	-	-	1.9 † (1.2-2.9) n=1029
Pereira (2005)	United Kingdom	2002-2003	11 year olds	peanut	Both IgE and non-IgE mediated	1.8 † (1.0-3.1) n=775	-	-	3.7 † (2.5-5.5) n=699	-	-	-	-	-	-
Pereira (2005)	United Kingdom	2002-2003	15 year olds	peanut	Both IgE and non-IgE mediated	2.5 † (1.6-4.0) n=757	-	-	2.6 † (1.6-4.3) n=649	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen)	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Emmett (1999)	United Kingdom	1995-1996	15 + years	peanut	Both Ige and non-IgE mediated	0.4 † (0.3-0.5) n=16420	0.5 † (0.4-0.7) n=16420	-	-	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.24: Peanut allergy prevalence in non-European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Osborne (2011)	Australia	2007-2010	12-15 months	peanut	IgE mediated only	-	-	-	6.4 (5.5-7.3) n=2757	-	-	-	2.9# (2.2-3.5) N=2757	-	-
Woods (2002)	Australia	1992-1998	26-50 years	peanut	IgE mediated only	1.1 † (0.4-2.7) n=457	-	-	5.7 † (3.8-8.3) n=457	-	0.4 † (0.1-1.8) n=457	-	-	-	-
Ben-Shoshan (2010)	Canada	2008-2009	< 18 years	peanut	Both IgE and non-IgE mediated only	1.77 # (1.21-2.33) n=nr	1.68 # (1.14-2.23) n=nr	1.03 # (0.67-1.39) n=nr	-	-	-	-	-	-	-
Ben-Shoshan (2010)	Canada	2008-2009	> 18 years	peanut	Both IgE and non-IgE mediated only	0.78 # (0.58-0.97) n=nr	0.71 # (0.52-0.90) n=nr	0.26 # (0.18-0.34) n=nr	-	-	-	-	-	-	-
Kagan (2003)	Canada	2000-2002	5-9 years	peanut	IgE mediated only	-	-	-	-	-	-	-	-	-	1.5 † (1.2-1.9) n=4254
Ben-Shoshan (2009)	Canada	2000-2002	7 year	peanut	IgE mediated only	-	-	-	-	-	-	-	-	-	1.34 (1.08-1.64) n=nr
Ben-Shoshan (2009)	Canada	2005-2007	7 year	peanut	IgE mediated only	-	-	-	-	-	-	-	-	-	1.62 (1.31-1.98) n=nr
Chen (2011)	China	2009	0-12 months	peanut	IgE mediated only	-	-	-	0.4 † (0.1-1.7) n=477	-	-	-	-	-	-
Hu (2010)	China	1999	0-24 months	peanut	IgE mediated only	-	-	-	0.3 † (0.0-2.1) n=304	-	-	-	-	-	-
Hu (2010)	China	2009	0-24 months	peanut	IgE mediated only	-	-	-	1.6 † (0.6-3.6) n=382	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Obeng (2011)	Ghana	2006-2008	5-16 years	peanut	IgE mediated only	1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Leung (2009)	Hong Kong	2006-2007	2-7 years	peanut	IgE mediated only (no SPT or SIgE)	0.7 † (0.4-1.0) n=3677	-	-	-	-	-	-	-	-	0.5 † (0.3-0.8) n=3677
Dalal (2002)	Israel	Nr	0-2years	peanut	IgE mediated only	-	0.1 † (0.0 - 0.2) n=9070	-	-	-	0 † (0.0 - 0.1) n=9070	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	peanut	IgE mediated only (no SPT or SIgE)	0.1 † (0.1-0.2) n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	peanut	IgE mediated only (no SPT or SIgE)	0.1 † (0.1-0.2) n=14777	-	-	-	-	-	-	-	-	-
Shek (2010)	Philippines	2007-2008	14-16 years	peanut	Both IgE and non IgE mediated	1.3 † (1.1-1.5) n=11322	0.4 † (0.3-0.6) n=11322	-	-	-	-	-	-	-	-
Shek (2010)	Singapore	2007-2008	14-16 years	peanut	Both IgE and non IgE mediated	1.2 † (0.9-1.5) n=6450	0.5 † (0.4-0.6) n=6450	-	-	-	-	-	-	-	-
Shek (2010)	Singapore	2007-2008	4-6 years	peanut	Both IgE and non IgE mediated	3.6 † (3.1-4.2) n=4390	0.6 † (0.4-1.0) n=4390	-	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	<3 years	peanut	IgE mediated only (no SPT or SIgE)	-	-	0.4 † (0.1-1.2) n=813	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	>19 years	peanut	IgE mediated only (no SPT or SIgE)	-	-	0.5 † (0.4-0.6) n=14036	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	4-18 years	peanut	IgE mediated only (no SPT or SIgE)	-	-	0.9 † (0.8-1.1) n=15169	-	-	-	-	-	-	-
Al-Hammadi (2010)	United Arab Emirates (Emirate of Abu Dhabi)	2006	6-9 years	peanut	IgE mediated only (no SPT or SIgE)	-	-	2.3 † (1.1-4.4) n=397	-	-	-	-	-	-	-
Branum (2009)	United States	2005-2006	< 18 years	peanut	IgE mediated only (not clearly defined)	-	-	-	-	9.3 (nr) n=nr	-	-	-	-	-
Sicherer (1999)	United States	1997	<18 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.4 † (0.2-0.7) n=2998	-	-	-	-	-	-	-	-
Sicherer (1999)	United States	1997	≥18 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.7 † (0.6-1.0) n=8049	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Sicherer (2003)	United States	2002	≥65 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.5 † (0.2-1.0) n=1345	-	-	-	-	-	-	-	-
Sicherer (2010)	United States	2008	≥65 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.7 (0.4-1.2) n=2481	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	0-2 years	peanut	IgE mediated (no SPT or SIgE)	-	1.4 (1.1-1.8) n=5429	-	-	-	-	-	-	-	-
Bock (1987)	United States	1980-1984	0-3 years	peanut	Both IgE and non-IgE mediated	1.4 † (0.5-2.8) n=408	-	-	-	-	-	-	-	-	0.8 † (0.3-2.3) n=480
Sicherer (2003)	United States	2002	0-5 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.8 † (0.4-1.7) n=869	-	-	-	-	-	-	-	-
Sicherer (2010)	United States	2008	0-5 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.9 † (0.4-1.9) n=860	-	-	-	-	-	-	-	-
Kumar (2011)	United States	2011	6 months - 6 years	peanut	IgE mediated only	-	-	-	-	13.5 (11.6-15.7) n=1104	-	-	-	-	-
Keet (2012)	United States	2005-2006	1-21 years	peanut	IgE mediated only	-	-	-	-	10 (nr) n=3550	-	-	-	-	-
Liu (2010)	United States	2005-2006	1-5 years	peanut	IgE mediated only	-	-	-	-	7.1 (nr) n=909	-	-	-	-	1.8 (nr) n=nr
Gupta (2011)	United States	2009-2010	3-5 years	peanut	IgE mediated (no SPT or SIgE)	-	2.8 (2.3-3.4) n=5910	-	-	-	-	-	-	-	-
Sicherer (2003)	United States	2002	6-10 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.6 † (0.2-1.4) n=851	-	-	-	-	-	-	-	-
Sicherer (2010)	United States	2008	6-10 years	peanut	IgE mediated only (no SPT or SIgE)	-	1.3 † (0.7-2.3) n=861	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	6-10 years	peanut	IgE mediated (no SPT or SIgE)	-	1.9 (1.6-2.3) n=9911	-	-	-	-	-	-	-	-
Liu (2010)	United States	2005-2006	6-19 years	peanut	IgE mediated only	-	-	-	-	10.7 (nr) n=2869	-	-	-	-	2.7 (nr) n=nr
Gupta (2011)	United States	2009-2010	11-13 years	peanut	IgE mediated (no SPT or SIgE)	-	2.3 (1.9-2.8) n=6716	-	-	-	-	-	-	-	-
Sicherer (2003)	United States	2002	11-17 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.2 † (0-0.6) n=1228	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Sicherer (2010)	United States	2008	11-17 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.7 † (0.3-1.4) n=1151	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	14-17 years	peanut	IgE mediated (no SPT or SIgE)	-	1.7 (1.4-2.1) n=10514	-	-	-	-	-	-	-	-
Vierk (2007)	United States	2001	18 years +	peanut	IgE mediated only (no SPT or SIgE)	0.7 † (0.5-1.0) n=4482	-	-	-	-	-	-	-	-	0.5 † (0.3-0.8) n=4482
Greenhawt (2009)	United States	nr	18 years+	peanut	IgE mediated (no SPT or SIgE)	8.4 (6.2-11.2) n=513	-	-	-	-	-	-	-	-	-
Sicherer (2003)	United States	2002	18-20 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.5 † (0.1-1.6) n=579	-	-	-	-	-	-	-	-
Sicherer (2010)	United States	2008	18-20 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.4 † (0.1-1.7) n=456	-	-	-	-	-	-	-	-
Liu (2010)	United States	2005-2006	20-39 years	peanut	IgE mediated only	-	-	-	-	8.7 (nr) n=1672	-	-	-	-	1 (nr) n=nr
Sicherer (2003)	United States	2002	21-30 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.4 † (0.1-0.9) n=1491	-	-	-	-	-	-	-	-
Sicherer (2010)	United States	2008	21-30 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.2 † (0-0.8) n=1019	-	-	-	-	-	-	-	-
Sicherer (2003)	United States	2002	31-40 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.5 † (0.2-1) n=1556	-	-	-	-	-	-	-	-
Sicherer (2010)	United States	2008	31-40 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.6 † (0.3-1.2) n=1311	-	-	-	-	-	-	-	-
Liu (2010)	United States	2005-2006	40-59 years	peanut	IgE mediated only	-	-	-	-	6.5 (nr) n=1361	-	-	-	-	1.1 (nr) n=nr
Sicherer (2003)	United States	2002	41-50 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.2 † (0.1-0.6) n=1809	-	-	-	-	-	-	-	-
Sicherer (2010)	United States	2008	41-50 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.6 † (0.3-1.1) n=1754	-	-	-	-	-	-	-	-
Sicherer (2003)	United States	2002	51-60 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.4 † (0.2-1.0) n=1352	-	-	-	-	-	-	-	-
Sicherer (2010)	United States	2008	51-60 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.4 † (0.2-0.8) n=1894	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Liu (2010)	United States	2005-2006	60+ years	peanut	IgE mediated only	-	-	-	-	4.5 (nr) n=1392	-	-	-	-	0.3 (nr) n=nr
Sicherer (2003)	United States	2002	61-64 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.3 † (0-1.8) n=355	-	-	-	-	-	-	-	-
Sicherer (2010)	United States	2008	61-64 years	peanut	IgE mediated only (no SPT or SIgE)	-	0.3 † (0.1-1.3) n=610	-	-	-	-	-	-	-	-
Arbes (2005)	United States	1988-1994	All ages	peanut	IgE mediated	-	-	-	8.6 † (8.1-9.2) n=10508	-	-	-	-	-	-
Liu (2010)	United States	2005-2006	All ages	peanut	IgE mediated only	-	-	-	-	7.6 (nr) n=8203	-	-	-	-	1.3 (nr) n=nr
Gupta (2011)	United States	2009-2010	All ages	peanut	IgE mediated (no SPT or SIgE)	-	2 (1.8-2.2) n=3339	-	-	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.25: Sesame allergy prevalence in European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Touraine (2002)	France	2000-2001	5-17 years	sesame	Both IgE and non-IgE mediated (no SPT or SIgE)	1.5 † (0.9-2.4) n=1086	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	sesame	Both IgE and non-IgE mediated	-	-	-	-	-	2.2 (1.7-2.7) n=4093	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	sesame	IgE mediated only	-	-	-	-	0 † (0-4.2) n=109	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	1 years	sesame	Both IgE and non IgE mediated	-	-	-	0.3 † (0.0-1.0) n=763	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Venter (2008)	United Kingdom	2001-2005	2 years	sesame	Both IgE and non IgE mediated	-	-	-	0.8 † (0.3-1.9) n=658	-	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	3 years	sesame	Both IgE and non IgE mediated	-	-	-	1.4 † (0.7-2.7) n=642	-	-	-	-	-	0.6 † (0.2-1.4) n=891
Venter (2006)	United Kingdom	2003-2004	6 years	sesame	Both IgE and non IgE mediated	0.6 † (0.2-1.6) n=798	-	-	0.4 † (0.1 -1.4) n=700	-	-	-	-	-	0.1 † (0-0.8) n=798
Roberts (2005)	United Kingdom	1998-2000	7 years	sesame	IgE mediated only	-	-	-	0.1 † (0 - 0.5) n=2003	-	-	-	-	-	-
Pereira (2005)	United Kingdom	2002-2003	11 years	sesame	Both IgE and non-IgE mediated	-	-	-	0.6 † (0.2-1.6) n=699	-	-	-	-	-	-
Pereira (2005)	United Kingdom	2002-2003	15 years	sesame	Both IgE and non-IgE mediated	-	-	-	0.9 † (0.4-2.1) n=649	-	-	-	-	-	-
Emmett (1999)	United Kingdom	1995-1996	15 + years	sesame	Both Ige and non-IgE mediated	0 † (0-0.1) n=16420	-	-	-	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
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Table 1.26: Sesame allergy prevalence in non-European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Osborne (2011)	Australia	2007-2010	12-15 months	sesame	IgE mediated only	-	-	-	1.6 (1.2-2.1) n=2695	-	-	-	0.7# (0.4-1.0) N=2695	-	-
Ben-Shoshan (2010)	Canada	2008-2009	< 18 years	sesame	Both IgE and non-IgE mediated	0.2 (0.03-0.43) n=nr	0.23 # (0.03-0.43) n=nr	0.03 # (0.00-0.06) n=nr	-	-	-	-	-	-	-
Ben-Shoshan (2010)	Canada	2008-2009	> 18 years	sesame	Both IgE and non-IgE mediated	0.1 (0.01-0.13) n=nr	0.05 # (0.00-0.11) n=nr	0.01 # (0.00-0.02) n=nr	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Dalal (2002)	Israel	nr	0-2years	sesame	IgE mediated only	-	0.2 † (0.1 - 0.3) n=9070	-	-	-	0.2 † (0.1-0.3) n=9070	-	-	-	-
Sicherer (2010)	United States	2008	<18 years	sesame	IgE- only (no SPT or SIgE)	-	0 † (0-0.1) n=13534	-	-	-	-	-	-	-	-
Sicherer (2010)	United States	2008	>18 years	sesame	IgE- only (no SPT or SIgE)	-	0.1 † (0-0.1) n=13534	-	-	-	-	-	-	-	-
Sicherer (2010)	United States	2008	All ages	sesame	IgE- only (no SPT or SIgE)	-	0.1 † (0.1-0.2) n=13534	-	-	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.27: Soya allergy prevalence in European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Osterballe (2005)	Denmark	2000-2001	< 3 years	soya	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	0 (nr) n=111	-	0 † (0.0-4.2) n=111
Osterballe (2005)	Denmark	2000-2001	3 years	soya	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0 - 1) n=486	0.4 † (0.1 - 1.6) n=486
Osterballe (2005)	Denmark	2000-2001	3-22 years	soya	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0 (0 - 2) n=301	0.3 (0 - 2.1) n=301
Osterballe (2009)	Denmark	2001-2002	22 years	soya	Both IgE and non-IgE mediated	0.6 † (0.2 - 1.5) n=843	-	-	-	-	-	-	-	-	0.1 (0.0-0.8) n=843
Osterballe (2005)	Denmark	2000-2001	>22 years	soya	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0 - 0.5) n=936	0.3 † (0.1 - 1.0) n=936
Zuberbier (2004)	Germany	1999-2000	0-80+ years	soya	Both IgE and non-IgE mediated	-	-	-	-	-	0.9 (0.6-1.3) n=3156	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Schafer (2001)	Germany	1997-1998	25-74 years	soya	Both IgE and non-IgE mediated	0.3† (nr) n=nr	-	-	1.7 † (nr) n=nr	-	-	-	-	-	-
Krause (2002)	Greenland	1998	5-18 years	soya	IgE mediated only	-	-	-	-	2.1 † (1.4-3.3) n=1031	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	soya	IgE mediated only	-	-	-	8.3 † (2.2-23.6) n=36	2.8 † (0.2-16.2) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	soya	IgE mediated only	-	-	-	7.3 † (3.5-14.4) n=109	3.7 (1.2-9.7) n=109	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	Soya	Both IgE and non-IgE mediated	0.3 † (0.0-2.0) n=324	-	-	-	-	0 † (0-1.5) n=324	-	-	-	-
Ostblom (2008 b)	Sweden	1995-1997	1 year	soya	Both IgE and non-IgE mediated	0.6 † (0.4-1.0) n=3104	-	0.2 † (0.1-0.4) n=3104	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	Soya	Both IgE and non-IgE mediated	0 † (0-1.4) n=328	-	-	-	-	0 † (0-1.4) n=328	-	-	-	-
Ostblom (2008 b)	Sweden	1996-1998	2 years	soya	Both IgE and non-IgE mediated	0.6 † (0.4-1.0) n=3104	-	0.6 † (0.4-1.0) n=3104	-	-	-	-	-	-	-
Ostblom (2008 b)	Sweden	1998-2000	4 years	soya	Both IgE and non-IgE mediated	1 † (0.7-1.4) n=3104	-	0.8 † (0.5-1.2) n=3104	-	-	-	-	-	-	-
Ostblom (2008 a)	Sweden	1999-2000	4 years	soya	Both IgE and non-IgE mediated	1.2 † (0.8-1.7) n=2563	-	-	-	3 † (2.4-3.8) n=2563	-	1.6 (1.1-2.1) n=2563	-	-	-
Ostblom (2008 b)	Sweden	2002-2004	8 years	soya	Both IgE and non-IgE mediated	0.8 † (0.5-1.2) n=3104	-	0.8 † (0.5-1.2) n=3104	-	-	-	-	-	-	-
Bjornsson (1996)	Sweden	1991-1992	20-44 years	soya	IgE mediated only	-	-	-	-	2.1 † (1.4-3.0) n=1397	-	-	-	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	soya	Both IgE and non-IgE (no SPT or SIgE)	0.6 † (0.4-0.9) n=4400	-	-	-	-	-	-	-	-	-
Arshad (2001)	United Kingdom	1993-1994	4 years	soya	IgE mediated only	-	-	-	0.3 † (0.1 - 1) n=981	-	-	-	-	-	-
Roberts (2005)	United Kingdom	1998-2000	7 years	soya	IgE mediated only	-	-	-	0.2 † (0 - 0.7) n=1173	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Emmett (1999)	United Kingdom	1995-1996	15 + years	soya	Both Ige and non-IgE mediated	0 † (0-0.1) n=16420	-	-	-	-	-	-	-	-	-
Young (1994)	United Kingdom	nr	All ages	soya	Both IgE and non IgE mediated (no SPT or SIgE)	0.3 † (0.3-0.4) n=18880	-	-	-	-	-	-	-	-	-

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‡ Percentage prevalence inferred from graph provided (no raw data reported).
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Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.28: Soya allergy prevalence in non-European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Soller (2012)	Canada	2008-2009	<18 years	soya	"likely" IgE mediated (no SPT or SIgE)	0.32 (0.08-0.55) n=nr	-	-	-	-	-	-	-	-	-
Soller (2012)	Canada	2008-2009	>18 years	soya	"likely" IgE mediated (no SPT or SIgE)	0.16 (0.07-0.25) n=nr	-	-	-	-	-	-	-	-	-
Hu (2010)	China	1999	0-24 months	soya	IgE mediated only	-	-	-	1 † (0.3-3.1) n=304	-	-	-	-	-	-
Hu (2010)	China	2009	0-24 months	soya	IgE mediated only	-	-	-	0.5 † (0.1-2.1) n=382	-	-	-	-	-	-
Sai (2011)	China	2008-2009	adults	soya	IgG mediated only	-	-	-	-	-	-	-	-	-	7.2 † (6.6-7.7) n=12766
Obeng (2011)	Ghana	2006-2008	5-16 years	soya	IgE mediated only	0.2 (nr) n=1407	-	-	-	-	-	-	-	-	-
Dalal (2002)	Israel	nr	0-2years	soya	IgE mediated only	-	0 † (0.0 - 0.2) n=9070	-	-	-	0 † (0.0 - 0.1) n=9070	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Kim (2011)	Korea	2006-2007	0-12 months	soya	IgE mediated only (no SPT or SIgE)	-	-	0.3 † (0.1-0.9) n=1177	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	soya	IgE mediated only (no SPT or SIgE)	0.2 † (0.1-0.2) n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	soya	IgE mediated only (no SPT or SIgE)	0.1 † (0.1-0.2) n=14777	-	-	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	<3 years	soya	IgE mediated only (no SPT or SIgE)	-	-	0 † (0.0-0.6) n=813	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	>19 years	soya	IgE mediated only (no SPT or SIgE)	-	-	0.2 † (0.1-0.3) n=14036	-	-	-	-	-	-	-
Wu (2012)	Taiwan	2004	4-18 years	soya	IgE mediated only (no SPT or SIgE)	-	-	0.2 (0.2-0.3†) n=15169	-	-	-	-	-	-	-
Santadusit (2005)	Thailand	nr	6 months - 6years	soya	IgE mediated only	0.2 † (0.0 - 1.0) n=656	-	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	0-2 years	soya	IgE mediated (no SPT or SIgE)	-	0.3 (0.2-0.4) n=5429	-	-	-	-	-	-	-	-
Bock (1987)	United States	1980-1984	0-3 years	soya	Both IgE and non-IgE mediated	2.7 † (1.2-4.2) n=408	-	-	-	-	-	-	-	-	0.8 † (0.3-2.3) n=480
Gupta (2011)	United States	2009-2010	3-5 years	soya	IgE mediated (no SPT or SIgE)	-	0.5 (0.3-0.7) n=5910	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	6-10 years	soya	IgE mediated (no SPT or SIgE)	-	0.3 (0.2-0.5) n=9911	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	11-13 years	soya	IgE mediated (no SPT or SIgE)	-	0.6 (0.4-0.8) n=6716	-	-	-	-	-	-	-	-
Gupta (2011)	United States	2009-2010	14-17 years	soya	IgE mediated (no SPT or SIgE)	-	0.3 (0.2-0.4) n=10514	-	-	-	-	-	-	-	-
Vierk (2007)	United States	2001	18 years +	soya	IgE mediated only (no SPT or SIgE)	0.1 † (0.0-0.3) n=4482	-	-	-	-	-	-	-	-	0.1 † (0.0-0.2) n=4482
Greenhawt (2009)	United States	nr	18 years+	soya	IgE mediated only (no SPT or SIgE)	1.8 † (0.9-3.4) n=513	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Gupta (2011)	United States	2009-2010	All ages	soya	IgE mediated only (no SPT or SIgE)	-	0.4 (0.3-0.4) n=3339	-	-	-	-	-	-	-	-

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Table 1.29: Tree nuts allergy prevalence in European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Kajosaari (1982)	Finland	1980-1981	1 year	unspecified nuts	Both IgE and non-IgE mediated (no SPT or SIgE)	2 (nr) n=261	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	1 year	unspecified nuts	Both IgE and non-IgE mediated (no SPT or SIgE)	0.8 † (0.4-1.8) n=853	-	0.1 † (0 - 0.6) n=853	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	2 years	unspecified nuts	Both IgE and non-IgE mediated (no SPT or SIgE)	1 (nr) n=202	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	2 years	unspecified nuts	Both IgE and non-IgE mediated (no SPT or SIgE)	2 † (1.2-3.3) n=852	-	0 † (0-0.6) n=852	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	3 years	unspecified nuts	Both IgE and non-IgE mediated (no SPT or SIgE)	2 (nr) n=200	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	3 years	unspecified nuts	Both IgE and non-IgE mediated (no SPT or SIgE)	1.4 † (0.7-2.6) n=784	-	0.5 † (0.2-1.4) n=784	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	4 years	unspecified nuts	Both IgE and non-IgE mediated (no SPT or SIgE)	1.7 † (1.0 -2.9) n=819	-	0.4 † (0.1-1.2) n=819	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Zuberbier (2004)	Germany	1999-2000	0-80+ years	tree nuts (hazelnut)	Both IgE and non-IgE mediated	-	-	-	-	-	5.9 (5.1-6.8) n=3156	-	-	2.2 (1.8-2.8) n=3156	-
Schafer (2001)	Germany	1997-1998	25-74 years	tree nuts (hazelnut)	Both IgE and non-IgE mediated	-	-	-	11.3† (nr) n=nr	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	tree nuts (walnut)	Both IgE and non-IgE mediated	-	-	-	-	-	1.8 (1.4-2.4) n=3156	-	-	1.0 (0.7-1.4) n=3156	-
Schafer (2001)	Germany	1997-1998	25-74 years	unspecified nuts	Both IgE and non-IgE mediated	5.3† (nr) n=nr	-	-	-	-	-	-	-	-	-
Sakellariou (2008)	Greece	2007	20-54 years	unspecified nuts	Both IgE and non-IgE mediated (no SPT or SIgE)	1.3† (nr) n=2003	-	-	-	-	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	tree nuts (almond)	IgE mediated only	-	-	-	-	-	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	tree nuts (almond)	IgE mediated only	-	-	-	-	0 † (0-4.2) n=109	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	tree nuts (hazelnut)	IgE mediated only	-	-	-	2.8 † (0.2-16.2) n=36	0 † (0-12.0) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	tree nuts (hazelnut)	IgE mediated only	-	-	-	3.7 † (1.2-9.7) n=109	9.2 † (4.7-16.6) n=109	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	tree nuts (walnut)	IgE mediated only	-	-	-	-	-	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	tree nuts (walnut)	IgE mediated only	-	-	-	-	3.7 † (1.2-9.7) n=109	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	tree nuts (almond)	Both IgE and non-IgE mediated	0 † (0-1.5) n=324	-	-	-	-	0 † (0-1.5) n=324	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	unspecified nuts	Both IgE and non-IgE mediated	0 † (0-1.5) n=324	-	-	-	-	0 † (0-1.5) n=324	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	1 year	unspecified nuts	Both IgE and non-IgE mediated	0.4 (0.3-0.8) n=3366	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	18 months	unspecified nuts	Both IgE and non-IgE mediated	1.2 (0.9-1.7) n=3278	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	2 years	unspecified nuts	Both IgE and non-IgE mediated	1.2 (0.9-1.7) n=2979	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Martinez-Gimeno (2000)	Spain	nr	6-13 years	unspecified nuts	Both IgE and non-IgE mediated (no SPT or SIgE)	6.9 † (6.2-7.6) n=5163	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	tree nuts (almond)	Both IgE and non-IgE mediated	0 † (0-1.4) n=328	-	-	-	-	0 † (0-1.4) n=328	-	-	-	-
Ostblom (2008 a)	Sweden	1999-2000	4 years	tree nuts (almond)	Both IgE and non-IgE mediated	3.8 † (3.1-4.7) n=2563	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	unspecified nuts	Both IgE and non-IgE mediated	0.3 † (0.0-2.0) n=328	-	-	-	-	0 † (0-1.4) n=328	-	-	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	unspecified nuts	Both IgE and non-IgE (no SPT or SIgE)	1.3 † (1.0-1.7) n=4400	-	-	-	-	-	-	-	-	-
Mustafayev (2012)	Turkey	2010	10-11 years	tree nuts (hazelnut)	IgE mediated only	1.5 † (1.2-1.8) n=6963	-	-	0.4 † (0.3-0.6) n=6134	-	-	-	0.1 † (0.0-0.8) n=813	-	-
Gelincik (2008)	Turkey	nr	18 years +	tree nuts (hazelnut)	Both Ige and non-IgE mediated	-	-	-	-	-	0 † (0-0.1) n=11816	0 (0-0.0) n=11816	-	0 † (0-0.1) n=11816	-
Orhan (2009)	Turkey	2006	6-9 years	tree nuts (hazelnut)	IgE mediated only	0.3 † (0.1 - 0.6) n=2739	-	-	-	-	0.1 † (0 - 0.3) n=2739	-	-	0 † (0 - 0.2) n=2739	-
Mustafayev (2012)	Turkey	2010	10-11 years	tree nuts (pistachio)	IgE mediated only	0.8 † (0.6-1.1) n=6963	-	-	-	-	-	-	-	-	-
Mustafayev (2012)	Turkey	2010	10-11 years	tree nuts (walnut)	IgE mediated only	1.2 † (1.0-1.5) n=6963	-	-	4.5 † (4.0-5.1) n=6134	-	-	-	0.4 † (0.1-1.2) n=813	-	-
Gelincik (2008)	Turkey	nr	18 years +	tree nuts (walnut)	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0-0.1) n=11816	-
Orhan (2009)	Turkey	2006	6-9 years	tree nuts (walnut)	IgE mediated only	0.1 † (0.0 - 0.4) n=2739	-	-	-	-	0.1 † (0 - 0.3) n=2739	-	-	0 † (0- 0.2) n=2739	-
Gelincik (2008)	Turkey	nr	18 years +	unspecified nuts	Both Ige and non-IgE mediated	0.1 ‡ (0-0.2) n=11816	-	-	-	-	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	1 year	tree nuts (almond)	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	-	0 † (0-0.5) n=900
Venter (2008)	United Kingdom	2001-2005	2 years	tree nuts (almond)	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	-	0 † (0-0.6) n=858

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Venter (2008)	United Kingdom	2001-2005	3 years	tree nuts (almond)	Both Ige and non-IgE mediated	-	-	-	0.3 † (0.0-1.2) n=642	-	-	-	-	-	0.2 † (0.0-0.9) n=891
Roberts (2005)	United Kingdom	1998-2000	7 years	tree nuts (almond)	IgE mediated only	-	-	-	0.5 † (0.2 - 0.9) n=1935	-	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	3 years	tree nuts (brazil)	Both Ige and non-IgE mediated	-	-	-	0.3 † (0.0-1.2) n=642	-	-	-	-	-	0.2 † (0.0-0.9) n=891
Roberts (2005)	United Kingdom	1998-2000	7 years	tree nuts (brazil)	IgE mediated only	-	-	-	0.5 † (0.3 - 1) n=1977	-	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	1 year	tree nuts (cashew)	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	-	0 † (0-0.5) n=900
Venter (2008)	United Kingdom	2001-2005	3 years	tree nuts (cashew)	Both Ige and non-IgE mediated	-	-	-	0.2 † (0.0-1.0) n=642	-	-	-	-	-	0.1 † (0.0-0.2) n=891
Tariq (1996)	United Kingdom	1993-1994	4 years	tree nuts (cashew)	IgE mediated only	-	-	-	-	-	0.1 † (0-0.5) n=1218	-	-	-	-
Roberts (2005)	United Kingdom	1998-2000	7 years	tree nuts (cashew)	IgE mediated only	-	-	-	0.4 † (0.2 - 0.8) n=1998	-	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	3 years	tree nuts (hazelnut)	Both Ige and non-IgE mediated	-	-	-	0.2 † (0.0-1.0) n=642	-	-	-	-	-	0.1 † (0.0-0.2) n=891
Tariq (1996)	United Kingdom	1993-1994	4 years	tree nuts (hazelnut)	IgE mediated only	-	-	-	-	-	0.1 † (0-0.5) n=1218	-	-	-	-
Roberts (2005)	United Kingdom	1998-2000	7 years	tree nuts (hazelnut)	IgE mediated only	-	-	-	0.1 † (0 - 0.5) n=2076	-	-	-	-	-	-
Roberts (2005)	United Kingdom	1998-2000	7 years	tree nuts (pecan)	IgE mediated only	-	-	-	0.2 † (0 - 0.5) n=1989	-	-	-	-	-	-
Roberts (2005)	United Kingdom	1998-2000	7 years	tree nuts (walnut)	IgE mediated only	-	-	-	0.5 † (0.3 - 1) n=1997	-	-	-	-	-	-
Young (1994)	United Kingdom	nr	All ages	unspecified nuts	Both IgE and non IgE mediated (no SPT or SIgE)	1.7 † (1.5-1.9) n=18880	-	-	-	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

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Table 1.30: Tree nuts allergy prevalence in non-European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Woods (1998)	Australia	1998	20-44years	unspecified nuts	Both IgE and non IgE mediated	0.6 † (0.2-1.6) n=669	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	tree nuts (palm)	IgE mediated only	0.2 (nr) n=1407	-	-	-	-	-	-	-	-	-
Kim (2011)	Korea	2006-2007	0-12 months	unspecified nuts	IgE mediated only (no SPT or SIgE)	-	-	0.7 † (0.3-1.4) n=1177	-	-	-	-	-	-	-
Shek (2010)	Philippines	2007-2008	14-16 years	unspecified nuts	Both IgE and non IgE mediated	1.7 † (1.5-2) n=11390	0.7 † (0.5-0.8) n=11390	-	-	-	-	-	-	-	-
Shek (2010)	Singapore	2007-2008	14-16 years	unspecified nuts	Both IgE and non IgE mediated	1.5 † (1.2-1.8) n=6465	0.5 † (0.4-0.8) n=6465	-	-	-	-	-	-	-	-
Shek (2010)	Singapore	2007-2008	4-6 years	unspecified nuts	Both IgE and non IgE mediated	4.7 † (4.1-5.4) n=4416	0.7 † (0.5-1.0) n=4416	-	-	-	-	-	-	-	-
Wan (2012)	Taiwan	Not Reported	6-8 years	tree nuts (pistachio)	IgE mediated only	-	-	-	-	-	-	2.2 (1.4-3.3) n=1010	-	-	-
Sicherer (2010)	United States	2008	< 18 years	unspecified nuts	IgE mediated only (unclear)	-	0.4 † (0.3-0.6) n=13534	-	-	-	-	-	-	-	-
Sicherer (1999)	United States	1997	<18 years	unspecified nuts	IgE- only (no SPT or SIgE)	-	0.2 † (0.1-0.4) n=8049	-	-	-	-	-	-	-	-
Sicherer (2003)	United States	2002	<18 years	unspecified nuts	IgE- only (no SPT or SIgE)	-	0.2 † (0.1-0.3) n=13493	-	-	-	-	-	-	-	-
Sicherer (2010)	United States	2008	> 18 years	unspecified nuts	IgE- only (no SPT or SIgE)	-	1 † (0.8-1.1) n=13534	-	-	-	-	-	-	-	-
Sicherer (2003)	United States	2002	>18 years	unspecified nuts	IgE- only (no SPT or SIgE)	-	0.9 † (0.7-1.1) n=13493	-	-	-	-	-	-	-	-
Sicherer (1999)	United States	1997	≥18 years	unspecified nuts	IgE- only (no SPT or SIgE)	-	1.6 † (1.4-1.9) n=8049	-	-	-	-	-	-	-	-

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

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Table 1.31: All Other Foods allergy prevalence in European countries by age group

Study ID	Country	Year(s) study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Osterballe (2005)	Denmark	2000-2001	< 3 years	additives	Both IgE and non IgE mediated	-	-	-	-	-	-	-	0 (nr) n=111	-	0 † (0 - 1) n=111
Osterballe (2005)	Denmark	2000-2001	>22 years	additives	Both IgE and non IgE mediated	-	-	-	-	-	-	-	-	0.1 † (0 - 0.7) n=936	0.6 † (0.3 - 1.5) n=936
Osterballe (2005)	Denmark	2000-2001	3 years	additives	Both IgE and non IgE mediated	-	-	-	-	-	-	-	-	0 † (0 - 1) n=486	2.3 † (1.2 - 4.1) n=486
Osterballe (2005)	Denmark	2000-2001	3-22 years	additives	Both IgE and non IgE mediated	-	-	-	-	-	-	-	-	0 † (0 - 2) n=301	0.7 † (0.1 - 2.6) n=301
Osterballe (2009)	Denmark	2001-2002	22 years	additives	Both IgE and non IgE mediated	6.6 † (5.1 - 8.6) n=843	-	-	-	-	-	-	-	-	0.5 (0.1-1.3) n=843
Kajosaari (1982)	Finland	1980-1981	1 year	chocolate	Both IgE and non-IgE mediated (no SPT or SIgE)	2 (nr) n=261	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	2 years	chocolate	Both IgE and non-IgE mediated (no SPT or SIgE)	4 (nr) n=202	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	3 years	chocolate	Both IgE and non-IgE mediated (no SPT or SIgE)	4 (nr) n=200	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	6 years	chocolate	Both IgE and non-IgE mediated (no SPT or SIgE)	1 (nr) n=203	-	-	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	1 year	legumes	Both IgE and non-IgE mediated (no SPT or SIgE)	1.6 † (0.9-2.8) n=853	-	0.7 † (0.3-1.6) n=853	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	2 years	legumes	Both IgE and non-IgE mediated (no SPT or SIgE)	3.2 † (2.1-4.6) n=852	-	0.8 † (0.4-1.8) n=852	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	3 years	legumes	Both IgE and non-IgE mediated (no SPT or SIgE)	2.8 † (1.8 - 4.3) n=784	-	1.4 † (0.7-2.6) n=784	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	4 years	legumes	Both IgE and non-IgE mediated (no SPT or SIgE)	2.9 † (1.9 - 4.4) n=819	-	1.5 † (0.8 - 2.6) n=819	-	-	-	-	-	-	-

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Study ID	Country	Year(s) study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Pyrhonen (2009)	Finland	2001-2009	1 year	strawberry/chocolate/tomato	Both IgE and non-IgE mediated (no SPT or SIgE)	6.6 † (5.0-8.5) n=853	-	0.1 † (0-0.8) n=853	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	2 years	strawberry/chocolate/tomato	Both IgE and non-IgE mediated (no SPT or SIgE)	13.8 † (11.6-16.4) n=852	-	0.4 † (0.1 - 1.1) n=852	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	3 years	strawberry/chocolate/tomato	Both IgE and non-IgE mediated (no SPT or SIgE)	13 † (10.4-15.2) n=784	-	1 † (0.4-1.9) n=784	-	-	-	-	-	-	-
Pyrhonen (2009)	Finland	2001-2009	4 years	strawberry/chocolate/tomato	Both IgE and non-IgE mediated (no SPT or SIgE)	13.4 † (11.2-16.0) n=819	-	2.1 † (1.3-3.4) n=819	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	1 year	tomato	Both IgE and non-IgE mediated (no SPT or SIgE)	7 (nr) n=261	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	2 years	tomato	Both IgE and non-IgE mediated (no SPT or SIgE)	7 (nr) n=202	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	3 years	tomato	Both IgE and non-IgE mediated (no SPT or SIgE)	11 (nr) n=200	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	6 years	tomato	Both IgE and non-IgE mediated (no SPT or SIgE)	2 (nr) n=203	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	1 year	vegetables (peas)	Both IgE and non-IgE mediated (no SPT or SIgE)	3 (nr) n=261	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	2 years	vegetables (peas)	Both IgE and non-IgE mediated (no SPT or SIgE)	2 (nr) n=202	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	3 years	vegetables (peas)	Both IgE and non-IgE mediated (no SPT or SIgE)	3 (nr) n=200	-	-	-	-	-	-	-	-	-
Kajosaari (1982)	Finland	1980-1981	6 years	vegetables (peas)	Both IgE and non-IgE mediated (no SPT or SIgE)	0.5 (nr) n=203	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Touraine (2002)	France	2000-2001	5-17 years	chocolate	Both IgE and non-IgE mediated (no SPT or SIgE)	1.5 † (0.9-2.4) n=1086	-	-	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	garlic	Both IgE and non-IgE mediated (no SPT or SIgE)	1.5 † (0.9-2.4) n=1086	-	-	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	honey	Both IgE and non-IgE mediated (no SPT or SIgE)	1.5 † (0.9-2.4) n=1086	-	-	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	Latex-kiwi/melon/banana/chestnut	Both IgE and non-IgE mediated (no SPT or SIgE)	14 † (12.0-16.2) n=1086	-	-	-	-	-	-	-	-	-
Touraine (2002)	France	2000-2001	5-17 years	pork	Both IgE and non-IgE mediated (no SPT or SIgE)	1.5 † (0.9-2.4) n=1086	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	additives	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0.3 (0.1-0.5) n=3156
Schafer (2001)	Germany	1997-1998	25-74 years	additives	Both IgE and non-IgE mediated	0.7† (nr) n=nr	-	-	-	-	-	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	alcohol (sparkling wine)	Both IgE and non-IgE mediated	1.9† (nr) n=nr	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	cacao	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0.1 (0.0-0.3) n=3156
Zuberbier (2004)	Germany	1999-2000	0-80+ years	carob	Both IgE and non-IgE mediated	-	-	-	-	-	0.9 (0.6-1.3) n=3156	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	carrageen	Both IgE and non-IgE mediated	-	-	-	-	-	0.2 (0.1-0.4) n=3156	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	guargum	Both IgE and non-IgE mediated	-	-	-	-	-	0.2 (0.1-0.5) n=3156	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	herbs/spices	Both IgE and non-IgE mediated	1.1† (nr) n=nr	-	-	-	-	-	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	meat	Both IgE and non-IgE mediated	0.5† (nr) n=nr	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Zuberbier (2004)	Germany	1999-2000	0-80+ years	poppy seeds	Both IgE and non-IgE mediated	-	-	-	-	-	0.7 (0.5-1.1) n=3156	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	pork	Both IgE and non-IgE mediated	-	-	-	-	-	0.2 (0.1-0.4) n=3156	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	pork	Both IgE and non-IgE mediated	-	-	-	2† (nr) n=nr	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	potato	Both IgE and non-IgE mediated	-	-	-	-	-	4.9 (4.2-5.7) n=3156	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	sugar	Both IgE and non-IgE mediated	0.5† (nr) n=nr	-	-	-	-	-	-	-	-	-
Schafer (2001)	Germany	1997-1998	25-74 years	tomato	Both IgE and non-IgE mediated	1.3† (nr) n=nr	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	vegetables	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	1.8 (1.4-2.4) n=3156	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	vegetables	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0.3 (0.1-0.6) n=3156
Schafer (2001)	Germany	1997-1998	25-74 years	vegetables	Both IgE and non-IgE mediated	1.7† (nr) n=nr	-	-	-	-	-	-	-	-	-
Zuberbier (2004)	Germany	1999-2000	0-80+ years	vegetables (carrot)	Both IgE and non-IgE mediated	-	-	-	-	-	3.6 (2.9-4.3) n=3156	-	-	-	-
Zannikos (2008)	Greece	2007	7-13 years	chocolate	Both IgE and non IgE mediated (no SPT or SIgE)	1.9 † (1.3-2.6) n=1988	-	-	-	-	-	-	-	-	-
Sakellariou (2008)	Greece	2007	20-54 years	chocolate	Both IgE and non-IgE mediated (no SPT or SIgE)	0.9 † (nr) n=2003	-	-	-	-	-	-	-	-	-
Sakellariou (2008)	Greece	2007	20-54 years	meat	Both IgE and non-IgE mediated (no SPT or SIgE)	0.8† (nr) n=2003	-	-	-	-	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	potato	IgE mediated only	-	-	-	2.8 † (0.2-16.2) n=36	0 † (0-12.0) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	Potato	IgE mediated only	-	-	-	2.8 † (0.7-8.4) n=109	3.7 † (1.2-9.7) n=109	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Bakos (2006)	Hungary	2002-2004	20-69 years	tomato	IgE mediated only	-	-	-	-	-	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	tomato	IgE mediated only	-	-	-	-	2.8 † (0.7-8.4) n=109	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	20-69 years	vegetables (carrot)	IgE mediated only	-	-	-	8.3 † (2.2-23.6) n=36	2.8 † (0.2-16.2) n=36	-	-	-	-	-
Bakos (2006)	Hungary	2002-2004	60-97 years	vegetables (carrot)	IgE mediated only	-	-	-	3.7 † (1.2-9.7) n=109	7.3 † (3.5-14.4) n=109	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	Chicken	Both IgE and non-IgE mediated	0.6 † (0.1-2.5) n=324	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	Chocolate	Both IgE and non-IgE mediated	1.5 † (0.6-3.8) n=324	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	Tomato	Both IgE and non-IgE mediated	3.1 † (1.6-5.8) n=324	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	vegetables (carrot)	Both IgE and non-IgE mediated	0.9 † (0.2-2.9) n=324	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Iceland	1994	18 months	vegetables (peas)	Both IgE and non-IgE mediated	1.5 † (0.6-3.8) n=324	-	-	-	-	0 † (0-1.5) n=324	-	-	-	-
Kilgallen (1996)	Ireland	nr	0-6 months	additives	Both IgE and non-IgE mediated (no SPT or SIgE)	0 † (0-6.1) n=75	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	12-24 months	additives	Both IgE and non-IgE mediated (no SPT or SIgE)	1.3 † (0.2-5.2) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	24-36 months	additives	Both IgE and non-IgE mediated (no SPT or SIgE)	0.7 † (0.0-4.2) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	36-48 months	additives	Both IgE and non-IgE mediated (no SPT or SIgE)	0.7 † (0.0-4.2) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	6-12 months	additives	Both IgE and non-IgE mediated (no SPT or SIgE)	0 † (0-6.1) n=75	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Kilgallen (1996)	Ireland	nr	0-6 months	colourings	Both IgE and non-IgE mediated (no SPT or SIgE)	0 † (0-6.1) n=75	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	12-24 months	colourings	Both IgE and non-IgE mediated (no SPT or SIgE)	2.7 † (0.9-7.1) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	24-36 months	colourings	Both IgE and non-IgE mediated (no SPT or SIgE)	2 † (0.5-6.2) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	36-48 months	colourings	Both IgE and non-IgE mediated (no SPT or SIgE)	4.7 † (2.1-9.8) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	6-12 months	colourings	Both IgE and non-IgE mediated (no SPT or SIgE)	0 † (0-6.1) n=75	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	0-6 months	soft drinks	Both IgE and non-IgE mediated (no SPT or SIgE)	0 † (0-6.1) n=75	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	6-12 months	soft drinks	Both IgE and non-IgE mediated (no SPT or SIgE)	1.3 † (0.1-8.2) n=75	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	12-24 months	soft drinks	Both IgE and non-IgE mediated (no SPT or SIgE)	6 † (3.0-11.4) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	24-36 months	soft drinks	Both IgE and non-IgE mediated (no SPT or SIgE)	2.7 † (0.9-7.1) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	36-48 months	soft drinks	Both IgE and non-IgE mediated (no SPT or SIgE)	6 † (3.0-11.4) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	0-6 months	sweets	Both IgE and non-IgE mediated (no SPT or SIgE)	0 † (0-6.1) n=75	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	6-12 months	sweets	Both IgE and non-IgE mediated (no SPT or SIgE)	2.7 † (0.5-10.2) n=75	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Kilgallen (1996)	Ireland	nr	12-24 months	sweets	Both IgE and non-IgE mediated (no SPT or SIgE)	4.7 † (2.1-9.8) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	24-36 months	sweets	Both IgE and non-IgE mediated (no SPT or SIgE)	7.3 † (3.9-13.1) n=150	-	-	-	-	-	-	-	-	-
Kilgallen (1996)	Ireland	nr	36-48 months	sweets	Both IgE and non-IgE mediated (no SPT or SIgE)	3.3 † (1.2-8.0) n=150	-	-	-	-	-	-	-	-	-
Frongia (2005)	Italy	2003	12-24 months	tomato	Both IgE and non-IgE mediated (no SPT or SIgE)	-	-	0.6 † (0.4-0.8) n=4602	-	-	-	-	-	-	-
Ronchetti (2008)	Italy	2005 - 2006	9 years	tomato	Both IgE and non-IgE mediated	-	-	-	1.1 † (0.2-4.3) n=184	-	-	-	-	-	4.3 † (2.0-8.7) n=184
Ronchetti (2008)	Italy	2005 - 2006	13 years	tomato	Both IgE and non-IgE mediated	-	-	-	3.1 † (1.3-6.9) n=196	-	-	-	-	-	3.8 † (1.6-8.6) n=156
Eggesbo (1999)	Norway	1993-1995	1 year	chocolate	Both IgE and non-IgE mediated	0.8 (0.6-1.2) n=3366	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	18 months	chocolate	Both IgE and non-IgE mediated	1.3 (0.9-1.8) n=3278	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	2 years	chocolate	Both IgE and non-IgE mediated	1.9 (1.4-2.4) n=2979	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	1 year	vegetables	Both IgE and non-IgE mediated	3.3 (2.7-4.1) n=3366	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	18 months	vegetables	Both IgE and non-IgE mediated	2.9 (2.3-3.6) n=3278	-	-	-	-	-	-	-	-	-
Eggesbo (1999)	Norway	1993-1995	2 years	vegetables	Both IgE and non-IgE mediated	3.3 (2.7-4.0) n=2979	-	-	-	-	-	-	-	-	-
Falcao (2004)	Portugal	nr	>39 years	Chocolate	Both IgE and non-IgE mediated (no SPT or SIgE)	0.3 † (0-1.2) n=659	-	-	-	-	-	-	-	-	-
Falcao (2004)	Portugal	nr	>39 years	Legumes	Both IgE and non-IgE mediated (no SPT or SIgE)	0.3 † (0-1.2) n=659	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Falcao (2004)	Portugal	nr	>39 years	Meat	Both IgE and non-IgE mediated (no SPT or SIgE)	1.8 † (1-3.2) n=659	-	-	-	-	-	-	-	-	-
Falcao (2004)	Portugal	nr	>39 years	Spices	Both IgE and non-IgE mediated (no SPT or SIgE)	0.3 † (0-1.2) n=659	-	-	-	-	-	-	-	-	-
Martinez-Gimeno (2000)	Spain	nr	6-13 years	legumes	Both IgE and non-IgE mediated (no SPT or SIgE)	12.6 † (11.7-13.6) n=5163	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	Chicken	Both IgE and non-IgE mediated	0 † (0-1.4) n=328	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	Chocolate	Both IgE and non-IgE mediated	3.4 † (1.8-6.1) n=328	-	-	-	-	-	-	-	-	-
Ostblom (2008 a)	Sweden	1999-2000	4 years	Chocolate	Both IgE and non-IgE mediated	2.6 † (2.0-3.3) n=2563	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	Tomato	Both IgE and non-IgE mediated	13.7 † (10.3-18.0) n=328	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	vegetables (carrot)	Both IgE and non-IgE mediated	1.5 † (0.6-3.7) n=328	-	-	-	-	-	-	-	-	-
Kristjansson (1999)	Sweden	1994	18 months	vegetables (peas)	Both IgE and non-IgE mediated	0 † (0-1.4) n=328	-	-	-	-	0 † (0-1.4) n=328	-	-	-	-
Ostblom (2008 a)	Sweden	1999-2000	4 years	vegetables (peas)	Both IgE and non-IgE mediated	1.2 † (0.9-1.8) n=2563	-	-	-	-	-	-	-	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	additives	Both IgE and non-IgE (no SPT or SIgE)	3.1 † (2.6-3.6) n=4400	-	-	-	-	-	-	-	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	chocolate	Both IgE and non-IgE (no SPT or SIgE)	2.7 † (2.2-3.2) n=4400	-	-	-	-	-	-	-	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	mayonnaise	Both IgE and non-IgE (no SPT or SIgE)	1 † (0.7-1.3) n=4400	-	-	-	-	-	-	-	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	pork	Both IgE and non-IgE (no SPT or SIgE)	1.5 † (1.2-1.9) n=4400	-	-	-	-	-	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	soft drinks	Both IgE and non-IgE (no SPT or SIgE)	1.2 † (0.9-1.6) n=4400	-	-	-	-	-	-	-	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	sugar	Both IgE and non-IgE (no SPT or SIgE)	1.4 † (1.1-1.8) n=4400	-	-	-	-	-	-	-	-	-
Brugman (1998)	The Netherlands	1993- 1994	4-15 years	tomato	Both IgE and non-IgE (no SPT or SIgE)	0.7 † (0.5-1.0) n=4400	-	-	-	-	-	-	-	-	-
Gelincik (2008)	Turkey	nr	18 years +	additives	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	0.0† (0.0-0.1) n=11816	-
Orhan (2009)	Turkey	2006	6-9 years	beef	IgE mediated only	1.4 † (1 - 1.9) n=2739	-	-	-	-	0.3 † (0.2 - 0.7) n=2739	-	-	0.3 † (0.1 - 0.6) n=2739	-
Mustafayev (2012)	Turkey	2010	10-11 years	beef	IgE mediated only	-	-	-	-	-	-	-	0.2 † (0.0-1.0) n=813	-	-
Orhan (2009)	Turkey	2006	6-9 years	black pepper	IgE mediated only	0.2 † (0.1 - 0.5) n=2739	-	-	-	-	0.1 † (0.1 - 0.4) n=2739	-	-	0 † (0 - 0.2) n=2739	-
Gelincik (2008)	Turkey	nr	18 years +	black pepper	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	0† (0-0.1) n=11816	-
Gelincik (2008)	Turkey	nr	18 years +	cacao	Both Ige and non-IgE mediated	1 † (0.9-1.2) n=11816	-	-	-	-	-	-	-	-	-
Orhan (2009)	Turkey	2006	6-9 years	Chickpea	IgE mediated only	0.2 † (0.1 - 0.5) n=2739	-	-	-	-	0.1 † (0 - 0.3) n=2739	-	-	0 † (0 - 0.2) n=2739	-
Gelincik (2008)	Turkey	nr	18 years +	Chocolate	Both Ige and non-IgE mediated	1 ‡ (0.9-1.2) n=11816	-	-	-	-	-	(0-0.0) n=11816	-	0† (0-0.1) n=11816	-
Orhan (2009)	Turkey	2006	6-9 years	Cocoa	IgE mediated only	3 † (2.4 - 3.7) n=2739	-	-	-	-	0.5 † (0.3 - 0.8) n=2739	-	-	0.1 † (0.1 - 0.4) n=2739	-
Gelincik (2008)	Turkey	nr	18 years +	Eggplant	Both Ige and non-IgE mediated	0.4 ‡ (0.3-0.6) n=11816	-	-	-	-	0 † (0.0-0.1) n=11816	0 (0-0.1) n=11816	-	0† (0-0.1) n=11816	-
Gelincik (2008)	Turkey	nr	18 years +	garlic	Both Ige and non-IgE mediated	0.1 ‡ (0-0.2) n=11816	-	-	-	-	-	-	-	-	-
Gelincik (2008)	Turkey	nr	18 years +	Meat	Both Ige and non-IgE mediated	0.3 ‡ (0.2-0.4) n=11816	-	-	-	-	-	-	-	0 † (0-0.0) n=11816	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Gelincik (2008)	Turkey	nr	18 years +	Mushroom	Both Ige and non-IgE mediated	0.2 ‡ (0.1-0.3) n=11816	-	-	-	-	-	-	-	-	-
Gelincik (2008)	Turkey	nr	18 years +	Pickle	Both Ige and non-IgE mediated	0.3 ‡ (0.2-0.4) n=11816	-	-	-	-	-	-	-	0 † (0-0.0) n=11816	-
Orhan (2009)	Turkey	2006	6-9 years	Potato	IgE mediated only	0.1† (0 - 0.3) n=2739	-	-	-	-	0 † (0 - 0.2) n=2739	-	-	0 † (0 - 0.2) n=2739	-
Gelincik (2008)	Turkey	nr	18 years +	Potato	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	0† (0-0.1) n=11816	-
Gelincik (2008)	Turkey	nr	18 years +	Red chilli	Both Ige and non-IgE mediated	-	-	-	-	-	-	-	-	0 † (0-0.1) n=11816	-
Gelincik (2008)	Turkey	nr	18 years +	Spices	Both Ige and non-IgE mediated	0.5 ‡ (0.4-0.6) n=11816	-	-	-	-	-	-	-	0 † (0-0.0) n=11816	-
Mustafayev (2012)	Turkey	2010	10-11 years	spinach	IgE mediated only	-	-	-	-	-	-	-	0.1 † (0.0-0.8) n=813	-	-
Gelincik (2008)	Turkey	nr	18 years +	Spinach	Both Ige and non-IgE mediated	-	-	-	-	-	0 † (0-0.1) n=11816	0 (0-0.0) n=11816	-	0 † (0-0.1) n=11816	-
Orhan (2009)	Turkey	2006	6-9 years	Tomato	IgE mediated only	0.3 † (0.1 - 0.6) n=2739	-	-	-	-	0.1 † (0 - 0.3) n=2739	-	-	0.0 † (0 - 0.2) n=2739	-
Gelincik (2008)	Turkey	nr	18 years +	Tomato	Both Ige and non-IgE mediated	2.3 ‡ (2.0-2.5) n=11816	-	-	-	-	0.1 † (0.0-0.1) n=11816	0 (0.0-0.1) n=11816	-	0.1† (0-0.1) n=11816	-
Gelincik (2008)	Turkey	nr	18 years +	vegetables (carrot)	Both Ige and non-IgE mediated	-	-	-	-	-	0 † (0-0.1) n=11816	0 (0-0.1) n=11816	-	0 † (0-0.1) n=11816	-
Pereira (2005)	United Kingdom	2002-2003	11 year olds	additives	Both IgE and non-IgE mediated	3.4 † (2.2-4.9) n=775	-	-	-	-	-	-	-	-	-
Pereira (2005)	United Kingdom	2002-2003	15 year olds	additives	Both IgE and non-IgE mediated	1.8 † (1.1-3.2) n=757	-	-	-	-	-	-	-	-	-
Venter (2006)	United Kingdom	2003-2004	6 years	additives	Both IgE and non-IgE mediated	1.6 † (0.9-2.9) n=798	-	-	-	-	-	-	0 † (0-0.6) n=798	0 † (0-0.6) n=798	-
Young (1994)	United Kingdom	nr	All ages	additives	Both IgE and non IgE mediated (no SPT or SIgE)	5.3 † (5.0-5.6) n=18880	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Young (1994)	United Kingdom	nr	All ages	alcohol	Both IgE and non IgE mediated (no SPT or SIgE)	1.4 † (1.2-1.6) n=18880	-	-	-	-	-	-	-	-	-
Young (1994)	United Kingdom	nr	All ages	caffeine	Both IgE and non IgE mediated (no SPT or SIgE)	1.3 † (1.1-1.5) n=18880	-	-	-	-	-	-	-	-	-
Emmett (1999)	United Kingdom	1995-1996	15 + years	Chocolate	Both Ige and non-IgE mediated	0.2 † (0.1-0.3) n=16420	-	-	-	-	-	-	-	-	-
Young (1994)	United Kingdom	nr	All ages	chocolate	Both IgE and non IgE mediated (no SPT or SIgE)	6.7 † (6.4-7.1) n=18880	-	-	-	-	-	-	-	-	-
Young (1994)	United Kingdom	nr	All ages	meat	Both IgE and non IgE mediated (no SPT or SIgE)	1.9 † (1.7-2.1) n=18880	-	-	-	-	-	-	-	-	-
Emmett (1999)	United Kingdom	1995-1996	15 + years	Pulses	Both Ige and non-IgE mediated	0 † (0-0.1) n=16420	-	-	-	-	-	-	-	-	-
Venter (2008)	United Kingdom	2001-2005	1 year	salicylate	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0.1 † (0.0-0.7) n=900
Venter (2008)	United Kingdom	2001-2005	2 years	salicylate	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0.1 † (0.0-0.8) n=858
Venter (2008)	United Kingdom	2001-2005	3 years	salicylate	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0.1 † (0.0-0.2) n=891
Venter (2008)	United Kingdom	2001-2005	1 year	tomato	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0.1 † (0.0-0.7) n=900
Venter (2008)	United Kingdom	2001-2005	2 years	tomato	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0 † (0-0.6) n=858
Venter (2008)	United Kingdom	2001-2005	3 years	tomato	Both IgE and non-IgE mediated	-	-	-	-	-	-	-	-	-	0 † (0-0.5) n=891
Young (1994)	United Kingdom	nr	All ages	tomato	Both IgE and non IgE mediated (no SPT or SIgE)	1.2 † (1.1-1.4) n=18880	-	-	-	-	-	-	-	-	-
Young (1994)	United Kingdom	nr	All ages	vegetables	Both IgE and non IgE mediated (no SPT or SIgE)	0.5 † (0.4-0.6) n=18880	-	-	-	-	-	-	-	-	-

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† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

Table 1.32: All Other Foods allergy prevalence in non-European countries by age group

Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Woods (1998)	Australia	1998	20-44years	alcohol	Both IgE and non IgE mediated	0.9 † (0.4-2.1) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	chocolate	Both IgE and non IgE mediated	0.7 † (0.3-1.8) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	Fats/oils, butter, margarine/ cream/ salad dressing	Both IgE and non IgE mediated	0.7 † (0.3-1.8) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	herbs/spices/condiments/ garlic, chilli	Both IgE and non IgE mediated	1 † (0.5-2.3) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	High fat foods	Both IgE and non IgE mediated	0.6 † (0.2-1.6) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	meat	Both IgE and non IgE mediated	0.3 † (0.1-1.2) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	meat (red)	Both IgE and non IgE mediated	0.7 † (0.3-1.8) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	Monosodium glutamate	Both IgE and non IgE mediated	0.9 † (0.4-2.1) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	Poultry	Both IgE and non IgE mediated	0.3 † (0.1-1.2) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	Restaurant meals/take away meals	Both IgE and non IgE mediated	0.3 † (0.1-1.2) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	Sauces	Both IgE and non IgE mediated	0.3 † (0.1-1.2) n=669	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Woods (1998)	Australia	1998	20-44years	Spicy foods	Both IgE and non IgE mediated	0.3 † (0.1-1.2) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	sugar	Both IgE and non IgE mediated	0.3 † (0.1-1.2) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	Tea/coffee	Both IgE and non IgE mediated	0.3 † (0.1-1.2) n=669	-	-	-	-	-	-	-	-	-
Woods (1998)	Australia	1998	20-44years	vegetables	Both IgE and non IgE mediated	0.7 † (0.3-1.8) n=669	-	-	-	-	-	-	-	-	-
Soller (2012)	Canada	2008-2009	<18 years	vegetables	"likely" IgE mediated (no SPT or SIgE)	0.45 (0.17-0.74) n=nr	-	-	-	-	-	-	-	-	-
Soller (2012)	Canada	2008-2009	>18 years	vegetables	"likely" IgE mediated (no SPT or SIgE)	1.29 (1.02-1.55) n=nr	-	-	-	-	-	-	-	-	-
Sai (2011)	China	2008-2009	adults	Beef	IgG mediated only	-	-	-	-	-	-	-	-	-	2.1 † (1.9-2.4) n=12766
Sai (2011)	China	2008-2009	adults	Chicken	IgG mediated only	-	-	-	-	-	-	-	-	-	1.6 † (1.4-1.9) n=12766
Sai (2011)	China	2008-2009	adults	Mushroom	IgG mediated only	-	-	-	-	-	-	-	-	-	1.2 † (1.0-1.4) n=12766
Sai (2011)	China	2008-2009	adults	Pork	IgG mediated only	-	-	-	-	-	-	-	-	-	0.4 † (0.3-0.6) n=12766
Sai (2011)	China	2008-2009	adults	Rice	IgG mediated only	-	-	-	-	-	-	-	-	-	2.3 † (2.1-2.6) n=12766
Sai (2011)	China	2008-2009	adults	Sweetcorn	IgG mediated only	-	-	-	-	-	-	-	-	-	4.2 † (3.9-4.6) n=12764
Sai (2011)	China	2008-2009	adults	Tomato	IgG mediated only	-	-	-	-	-	-	-	-	-	4.3 † (3.9-4.7) n=12766
Marrugo (2008)	Colombia	Nr	All ages	additives	Both IgE and non-IgE mediated (no SPT or SIgE)	0.4† (0.2-0.7) n=3099	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Marrugo (2008)	Colombia	Nr	All ages	alcohol	Both IgE and non-IgE mediated (no SPT or SIgE)	2 † (1.5-2.5) n=3099	-	-	-	-	-	-	-	-	-
Marrugo (2008)	Colombia	Nr	All ages	meat	Both IgE and non-IgE mediated (no SPT or SIgE)	3.1 † (2.5-3.8) n=3099	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Avocado	IgE mediated only	0.3 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Beans	IgE mediated only	1.3 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Cassava	IgE mediated only	0.6 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Coconut	IgE mediated only	0.1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Cocoyam	IgE mediated only	0.1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Kontomire	IgE mediated only	0.4 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Nutmeg	IgE mediated only	0.3 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Okro	IgE mediated only	0.9 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Potato	IgE mediated only	0.1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Rice	IgE mediated only	0.1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Sorghum	IgE mediated only	0.4 (nr) n=1407	-	-	-	-	-	-	-	-	-

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Obeng (2011)	Ghana	2006-2008	5-16 years	Sweet potato	IgE mediated only	0.3 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Tomato	IgE mediated only	0 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	vegetables (carrot)	IgE mediated only	0.1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Obeng (2011)	Ghana	2006-2008	5-16 years	Water yam	IgE mediated only	0.1 (nr) n=1407	-	-	-	-	-	-	-	-	-
Leung (2009)	Hong Kong	2006-2007	2-7 years	Beef	IgE mediated only (no SPT or SIgE)	0.5 † (0.3-0.8) n=3677	-	-	-	-	-	-	-	-	0.3† (0.2-0.6) n=3677
Leung (2009)	Hong Kong	2006-2007	2-7 years	Chocolate	IgE mediated only (no SPT or SIgE)	0.3 † (0.2-0.6) n=3677	-	-	-	-	-	-	-	-	0.3 † (0.2-0.6) n=3677
Leung (2009)	Hong Kong	2006-2007	2-7 years	Lamb	IgE mediated only (no SPT or SIgE)	0.2 † (0.1-0.5) n=3677	-	-	-	-	-	-	-	-	0.1 † (0.1-0.5) n=3677
Leung (2009)	Hong Kong	2006-2007	2-7 years	Tomato	IgE mediated only (no SPT or SIgE)	0.2 † (0.1-0.5) n=3677	-	-	-	-	-	-	-	-	0.2 † (0.1-0.5) n=3677
Babu (2008)	India	nr	5-60 years	eggplant	IgE mediated only	9.2 † (7.3-11.6) n=741	-	-	6.5 † (4.9-8.6) n=741	-	-	0.8 (0.3-1.9) n=741	-	-	-
Dalal (2002)	Israel	nr	0-2years	beef	IgE mediated only	-	0 † (0 - 0.1) n=9070	-	-	-	0 † (0 - 0.1) n=9070	-	-	-	-
Dalal (2002)	Israel	nr	0-2years	chicken	IgE mediated only	-	0 † (0 - 0.1) n=9070	-	-	-	0 † (0 - 0.1) n=9070	-	-	-	-
Dalal (2002)	Israel	nr	0-2years	chocolate	IgE mediated only	-	0 † (0 - 0.1) n=9070	-	-	-	0 † (0 - 0.1) n=9070	-	-	-	-
Dalal (2002)	Israel	nr	0-2years	garlic	IgE mediated only	-	0 † (0 - 0.1) n=9070	-	-	-	0 † (0 - 0.1) n=9070	-	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Dalal (2002)	Israel	nr	0-2years	tomato	IgE mediated only	-	0 † (0 - 0.1) n=9070	-	-	-	0 † (0 - 0.1) n=9070	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	Beef	IgE mediated only (no SPT or SIgE)	0.4 † (0.3-0.6) n=14777	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	Beef	IgE mediated only (no SPT or SIgE)	0.2 † (0.2-0.3) n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	Beef	IgE mediated only (no SPT or SIgE)	0.2 † (0.2-0.3 n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	Buckwheat	IgE mediated only (no SPT or SIgE)	0.1 † (0.1-0.2) n=14777	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	Buckwheat	IgE mediated only (no SPT or SIgE)	0.1 † (0.1-0.1) n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	chicken	IgE mediated only (no SPT or SIgE)	0.2 † (0.2-0.3) n=14777	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	chicken	IgE mediated only (no SPT or SIgE)	0.3 † (0.3-0.4) n=27425	-	-	-	-	-	-	-	-	-
Kim (2011)	Korea	2006-2007	0-12 months	perilla seeds	IgE mediated only (no SPT or SIgE)	-	-	0.1 † (0-0.5) n=1177	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	Pork	IgE mediated only (no SPT or SIgE)	0.3 † (0.3-0.5) n=14777	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	Pork	IgE mediated only (no SPT or SIgE)	0.5 † (0.4-0.6) n=27425	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	12-15 years	Tomato	IgE mediated only (no SPT or SIgE)	0 † (0.0-0.1) n=14777	-	-	-	-	-	-	-	-	-
Oh (2004)	Korea	2000	6-12 years	Tomato	IgE mediated only (no SPT or SIgE)	0 † (0.0-0.1) n=27425	-	-	-	-	-	-	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	bamboo shoot	IgE mediated only	-	-	-	-	-	-	1.2 (0.7-2.1) n=1010	-	-	-

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						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Wan (2012)	Taiwan	nr	6-8 years	cacao	IgE mediated only	-	-	-	-	-	-	0.3 (0.1-1.0) n=1010	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	garlic	IgE mediated only	-	-	-	-	-	-	11.6 (9.7-13.8) n=1010	-	-	-
Wan (2012)	Taiwan	nr	6-8 years	onion	IgE mediated only	-	-	-	-	-	-	1.6 (0.9-2.6) n=1010	-	-	-
Lao-araya (2012)	Thailand	2010	3-7years	ant eggs	IgE mediated only	0.9 † (0.3-2.4) n=452	-	-	-	-	-	-	-	-	-
Lao-araya (2012)	Thailand	2010	3-7years	beef	IgE mediated only	0.4 † (0.0-1.8) n=452	-	-	-	-	-	-	0 † (0-1.1) n=452	-	-
Lao-araya (2012)	Thailand	2010	3-7years	chocolate	IgE mediated only	0.4 † (0.0-1.8) n=452	-	-	-	-	-	-	0 † (0-1.1) n=452	-	-
Lao-araya (2012)	Thailand	2010	3-7years	coconut	IgE mediated only	0.2 † (0.0-1.4) n=452	-	-	-	-	-	-	-	-	-
Santadusit (2005)	Thailand	nr	6 months - 6years	Duck	IgE mediated only	0.2 † (0.0 - 1.0) n=656	-	-	-	-	-	-	-	-	-
Lao-araya (2012)	Thailand	2010	3-7years	insect	IgE mediated only	0.4 † (0.0-1.8) n=452	-	-	-	-	-	-	-	-	-
Santadusit (2005)	Thailand	nr	6 months - 6years	Junk food	IgE mediated only	0.3 † (0.1 - 1.2) n=656	-	-	-	-	-	-	-	-	-
Al-Hammadi (2010)	United Arab Emirates (Emirate of Abu Dhabi)	2006	6-9 years	vegetables	IgE mediated only (no SPT or SIgE)	-	-	0.5 † (0.1-2.0) n=397	-	-	-	-	-	-	-
Vierk (2007)	United States	2001	18 years +	additives	IgE mediated only (no SPT or SIgE)	0.6 † (0.4-0.9) n=4482	-	-	-	-	-	-	-	-	0.4 † (0.2-0.7) n=4482
Bock (1987)	United States	1980-1984	0-3 years	chocolate	Both IgE and non-IgE mediated	1.9 † (0.8-3.4) n=408	-	-	-	-	-	-	-	-	0 † (0-1) n=480
Vierk (2007)	United States	2001	18 years +	Chocolate	IgE mediated only (no SPT or SIgE)	0.6 † (0.4-0.9) n=4482	-	-	-	-	-	-	-	-	0.4 † (0.2-0.6) n=4482

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Study ID	Country	Year(s) of study	Age group	Allergen	Type of food allergy	Questionnaire-based methods			Sensitisation		Sensitisation with clinical history		Food challenge with clinical history		Other
						Self-reported	Clinical history	Clinician-diagnosed	Skin prick test	Serum-specific IgE	History and SPT	History and SIgE	History and OFC	History and DBPCFC	
						95% Prevalence (CI)									
Bock (1987)	United States	1980-1984	0-3 years	rice	Both IgE and non-IgE mediated	0.9 † (0.3-2.3) n=408	-	-	-	-	-	-	-	-	0.2 † (0-1.3) n=480

† Percentage prevalence and/or confidence intervals calculated from raw data provided in the paper
‡ Percentage prevalence inferred from graph provided (no raw data reported).
# Data has been subject to correction or estimation by the authors (presented as reported in the paper).
Note: Where confidence intervals are missing the data has either been inferred from a graph or they have not been provided by the paper and, in the absence of raw data, could not be calculated.

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1.2.7. Time Trends

There are only a few cases in which it is appropriate to compare prevalence rates across decades. In these instances, studies have adopted similar methodologies in similar age groups in the same country. It would not make sense to compare across countries, where diet changes significantly, and across methodologies as each one carries its own level of risk/bias.

1.2.7.1. Celery

There are no studies available which are appropriate to compare to show any time trends in sesame allergy.

1.2.7.2. Cereals

Two studies were conducted on the prevalence of cereal allergy in Finland, one in 1980 and the other 20 years later in 2001. As similar methodologies were used comparisons can be made to reveal time trends in wheat allergy in 1 and 2 year old children. At 1 year of age, self-reported allergy to wheat in 1980 was estimated at 1% (95% CI: not reported) (Kajosaari 1982), this rose to 2.1 % (95% CI:1.3-3.4%) (Pyrhonen 2009) when studied in 2001. At 2 years of age, self-reported allergy to wheat was 1% (95% CI: not reported) (Kajosaari 1982), again doubling to 2% (95% CI: 1.2-3.2%) in 2001 (Pyrhonen 2009).

Two further studies in the UK, both measuring sensitisation to wheat using skin prick tests, found 0.3% (95% CI:0.1-1.0) sensitisation in 4 year olds in the 1993 cohort (Arshad 2001) and 0% (95% CI: 0.0-0.1) in 3 year olds in the 2001 cohort (Venter 2008). Skin prick tests were conducted using the same allergens and the same research nurses. A study conducted in China, looked at sensitisation rates to wheat, as determined by a positive skin prick test in 1999 and 10 years later in 2009 in children aged 0-24 months. They found a 0.2% increase, from 0.3% (95% CI: 0.0-2.1%) (Hu 2010) in 1999 to 0.5 % (95% CI: 0.1-2.1%) in 2009 (Hu 2010).

1.2.7.3. Egg

Two studies looking at egg allergy in Finland were carried out in 1980 and 2001, and as similar methods were utilised, we are able to compare the prevalence rates, At 1 year of age, 6% (95% CI: not reported) of parents reported an adverse reaction to egg in 1980, whereas in 2001 only 2.7% (95% CI: 1.8-4.1%) (Kajosaari 1982) parents reported a problem with egg. At 2 years of age, there was a 7% (95% CI: not reported) (Kajosaari 1982) self-reported prevalence of egg allergy in 1980 compared to 4% (95% CI: 2.8-5.6%) (Pyrhonen 2009) prevalence found at the same age in 2001. At 3 years of age, 9% (95% CI: not reported) of parents reported an egg allergy in their children (Kajosaari 1982), this dropped to only 3.6% (95% CI:2.4-5.2%) reporting a problem in the same age group in 2001 (Pyrhonen 2009).

In the UK, a study was conducted in 1995 which reported self-reported egg allergy at 15 years of age, this showed a 0.7% (95% CI: 0.6-0.8%) prevalence (Emmett 1999). When compared to a later study also in the UK with 15 year olds, self-reported egg allergy had risen to 3%. (95% CI: 2.0-4.6%) (Pereira 2005). Two further studies in the UK, both measuring sensitisation to egg using skin prick tests, found 0.8% (95% CI: 0.4-2.0) sensitisation in 4 year olds in a 1993 cohort (Arshad 2001) and 1.4% (95% CI:0.7-2.7) in 3 year olds in 2001 cohort (Venter 2008). In China, sensitisation to egg increased from 7.6% (95% CI: 5.0-11.3%) in 0-24 month olds in 1999, to 16.2% (95% CI: 12.8-20.4%) in the same age group in 2009 (Hu 2010). Of note, Osborne 2011 reports the highest challenge proven rate of egg allergy in young children worldwide (9%; 95% CI: 7.9-10.0) in a study conducted in Australia, however the challenges were performed using raw egg.

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1.2.7.4. Fish and Shellfish

The prevalence of self-reported allergy to fish in Finland in 1980 was 6% (95% CI: not reported) (Kajosaari 1982) which declined slightly when assessed in 2001 when it was reported to be 5% (95% CI: 3.4-6.4%) (Pyrhonen 2009). In 1980 5% (95% CI: not reported) of parents reported that their child experienced an adverse reaction after consumption of fish (Kajosaari 1982), this declined to 3.6% (95% CI: 2.4-5.2%) in 2001 (Pyrhonen, 2009). Two further studies in the UK, both measuring sensitisation to cod using skin prick tests, found 0.7% (95% CI:0.3-2.0) sensitisation in 4 year olds in a 1993 cohort (Arshad 2001) and 0.5% (95% CI: 0.1-1.5) in 3 year olds in a 2001 cohort (Venter 2008). In China, 0-24 month olds were skin prick tested, which resulted in 0% (95% CI: 0.0-1.6%) prevalence to shrimp in 1999 and 0.3% (95% CI: 0.0-1.7%) in 2009 (Hu, 2010). Prevalence of sensitisation to fish was 0.3% (95% CI: 0.0-2.1%) in 1999 and 0.8 % (95% CI: 0.2-2.5%) in 2009 (Hu, 2010).

1.2.7.5. Fruits

At 1 year of age, self-reported allergy to citrus fruits was reported to be 8% (95% CI: not reported) in 1980 (Kajosaari 1982) and 3.5% (95% CI: 2.4-5.1%) in 2001 (Pyrhonen 2009). At 2 years of age the prevalence rates were 9% (95% CI: not reported) in 1980 (Kajosaari 1982) and 7.2% (95% CI: 5.6-9.2%) in 2001 (Pyrhonen 2009). At 3 years of age the self-reported prevalence was 11% (95% CI: not reported) in 1980 (Kajosaari 1982) compared to 6.5% (95% CI: 4.9-8.5%) in 2001 (Pyrhonen 2009). In China, sensitisation to orange fell from 1% (95% CI: 0.3-3.1%) in 1999 to 0% (95% CI:0-1.2%) in 2009 (Hu 2010).

1.2.7.6. Milk/dairy

Comparing cow's milk allergy in Finland in 1980 to 2001, self-reported rates were 2% (95% CI: not reported) at age 1, 5% (95% CI: not reported) at age 2 and 2% (95% CI: not reported) at age 3 in 1980 (Kajosaari 1982). Rates in 2001 were somewhat higher; 5.4% (95%CI: 4.0-7.2%) at age 1, 6.8% (95% CI:5.2-8.6%) at age 2 and 5.9% (95% CI: 4.4-7.8%) at age 3 (Pyrhonen 2009). Two further studies in the UK, both measuring sensitisation to milk using skin prick tests, found 1.3% (95% CI:0.7-2.3) sensitisation in 4 year olds in a 1993 cohort (Arshad 2001) and 0.5% (95% CI:0.1-1.5) in 3 year olds in a 2001 cohort (Venter 2008). In China, cow's milk sensitivity diagnosed using skin prick tests in 0-24 month olds almost doubled from 3.3 % (95% CI: 1.7-6.2%) in 1999 to 6.5% (95% CI: 4.4-9.6%) in 2009 (Hu 2010).

1.2.7.7. Mustard

There was only one study found on mustard allergy and so no time trends can be assessed.

1.2.7.8. Peanut

Two cohorts of children (age 3–4 years) born on the Isle of Wight, were assessed for peanut allergy and the outcomes compared: Cohort A: Born in 1989; (Tarik) reviewed at 4 years of age (n = 2181). Cohort B: Born between 1994 and 1996; reviewed between 3 and 4 years of age (n = 1273). Peanut sensitization increased significantly from 1.3% in Cohort A to 3.3% (P = 0.003) in Cohort B (Grundy) before falling back to 2.0% in Cohort C (P = 0.145) (Venter 2008). Similarly, clinical peanut allergy increased significantly from 0.5% in Cohort A to 1.4% (P = 0.023) in Cohort B, with a subsequent fall to 1.2% in Cohort C (P = 0.850).

1.2.7.9. Sesame

There were limited studies on sesame allergy, with only two studies worldwide utilising food challenges, and both studies were done within the same decade so no time trends can be reported.

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1.2.7.10. Soya

One study in China looked at sensitisation to soya, reporting a 1% (95% CI: 0.3-3.1) prevalence in 0-24 month olds in 1999, compared to 0.5% (95% CI: 0.1-2.1) in the same age group in 2009 (Hu 2010). In addition, in the United States Bock 1987 reported a rate of 2.7% (95% CI: 1.2-4.2) for self-reported soya allergy in 0-3 year olds in the 1980s, compared to Gupta 2011 who found a prevalence of 0.3% (95% CI: 0.2-0.4) for 0-2 year olds in 2009 when assessing a convincing clinical history.

1.2.7.11. Tree Nuts

A study conducted in Finland in 1980 reported the prevalence of self-reported allergy to nuts (unspecified). It reported a 2% (95% CI: not reported) prevalence at age 1, 0% (95% CI: not reported) prevalence at age 2 and 2% (95% CI: not reported) prevalence at 3 years of age (Kajosaari 1982). A similar study also looking at self-reported allergy to nuts in Finland in 2001 found 0.8% (95% CI: 0.4-1.8%) prevalence at 1 year of age, 2% (95% CI: 1.2-3.3%) at 2 years of age and 1.4% (95% CI: 0.7-2.6%) prevalence at 3 years of age (Pyrhonen 2009). In the US, Sicherer 1999 and Sicherer 2010 reported allergy to nuts based on a convincing clinical history in 1997 and 2008 for children under the age of 18 years and adults. In the children the prevalence of allergy to nuts was 0.2% (95% CI: 0.1-0.4%) in 1997 (Sicherer 1999), which doubled to 0.4% (95% CI: 0.3-0.6%) almost 10 years later in 2008 (Sicherer 2010). For adults, the same studies reported a prevalence rate of 1.6% (95% CI: 1.4-1.9%) prevalence in 1997 (Sicherer 1999) which dropped to 1.0% (95% CI: 0.8-1.1%) prevalence in 2008 (Sicherer 2010).

1.2.7.12. Other Foods

A vast array of allergens was included in this group and so comparing across decades is challenging. However, prevalence rates of self-reported allergy in studies published before 2000 varied between 0% in allergens such as additives and colourings, sweets, chicken, soft drinks, pulses and vegetables (Killgallen 1996; Kristjansson 1999; Emmett 1999). The highest self-reported prevalence was seen in tomato allergy at 11% (95% CI: not reported) (Kajosaari 1982). Studies published after 2000 report self-reported rates of allergy between 0.0% in tomato allergy (Oh 2004; Obeng 2011) and 14% in strawberry, chocolate, tomato, latex associated foods (kiwi, melon, banana, chestnut) (Pyrhonen 2009; Touraine 2002).

1.2.8. Discussion

In this systematic review we have focused on the 14 major allergens as identified by the EU including: milk, egg, wheat, fish, shellfish, molluscs, soya, peanut, tree nuts, sesame, mustard, lupin and celery. We have excluded sulphites from the systematic review as agreed with EFSA. Additionally, we have also looked at fruit, vegetable and other reported allergens.

Celery

Celery allergy is considered to be a big problem in mainland Europe. The main problem with studying the prevalence of celery allergy is that celery salt is considered much more allergenic than celery itself; none of the identified studies utilized celery salt in their food challenges. In fact, despite being considered as one of the major 14 food allergens, there appear to be only six studies reported on celery allergy. Two studies presented rates of self-reported allergy, three studies focused on SPT results and three reported on specific IgE levels. The best information we have on possible celery allergy is based on the data from Zuberbier 2004 indicating that 3.5% of the German population suffer from celery allergy based on SPT and a good clinical history and the data from Wan 2012 indicating that 1.8% of 6-8 year olds in Taiwan suffer from celery allergy based on serum IgE levels and a good clinical history.

Cereals (Wheat)

Wheat allergy prevalence based on food challenge is reported in three studies only. Osterballe 2004 reported no wheat allergy in all ages in Denmark, Orhan 2009 found no wheat allergy in 6-9 year old

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children in Turkey and Venter 2008 reported that 0.4% of 1 year olds, 0.3% of two year olds, 0.2% of three year olds, and 0.3% of six year olds suffer from wheat allergy in the UK.

Egg

Egg allergy is probably one of the most common allergies seen in early childhood and provides a clinical dilemma in terms of diagnosis and management due to the effect of heating on allergenicity as discussed in Objective 4a. A number of studies have looked at challenge-proven egg allergy in Europe. As with peanut, the two main studies using a food challenge outcome are Osterballe 2005 (Denmark) and Venter 2008 (United Kingdom). In young children (age 0-3 years) challenge-proven egg allergy prevalence rates have been found to be 1.8% (Osterballe 2005) in under 3's and at 2.9% at 3 years. Slightly lower rates are reported in the UK: 1.8% at 1 year, 1.3% at 2 year and 1% at 3 years (Venter 2008).

In older children (>3 years) rates based on food challenges were 0.7% (Eller 2009; Demark), 1% (Kajosaari 1982; Finland), 0.1% (Mustafayev 2012; Turkey), 1.9% (Orhan 2009; Turkey) and 0.3% (Venter 2006). In adults, challenge proven egg allergy data is from two studies only, showing no egg allergy in 22 year olds in Denmark (Osterballe 2005) and 0.1% of a whole population in Germany (Zuberbier 2004).

In studies from the rest of the world, the prevalence of food-challenge proven egg allergy has been reported for Australia and China and only in young children. Osborne identified egg allergy rates of 9% in 12 – 15 month old children in Melbourne, Australia. This is much higher than rates reported in Europe and other countries in the rest of the world such as China. Egg allergy rates in China were reported to be 2.5% in 0-12 month olds (Chen 2011), 2.9% in 0-24 month olds seen in 1999, and a much higher rate of 5% in 0-24 month olds in 2009 (Hu 2010). Chen 2012 also reported rates of 4.4%, 4.2% and 3% in 0-2 year olds in 2009-2010 in 3 different areas in China.

Fish and Shellfish

Challenge-proven data on the prevalence of fish allergies is surprisingly weak. In terms of fish (cod) allergy, the majority of data is derived from the UK cohort (Venter 2008), showing that 0.1% of one, two, three and six year olds suffered from a codfish allergy despite rates of sensitisation of between 0.3 – 1%. This information was echoed in Osterballe 2005 who found that none of the children under 3 years in their study had a fish allergy and only 0.6% of the adults studied had a challenge-proven allergy. Sensitisation rates for fish/cod were not available from this study. The only other adult study available, found that 0% of adults have a fish allergy in Turkey (Gelinicik 2008). In 6 year olds, 1% of a Finnish group studied showed the 6 year olds had a positive food challenge to fish (Kajosaari 1999). No fish allergy was found in the same age group in Turkey (Orhan 2009).

In terms of shellfish allergy, only one study (Osterballe 2005) showed any challenge proven data finding a prevalence rate of 0% shellfish (prawn) allergy in young children and 1.1% in adults (Osterballe 2005). Mollusc allergy has only been investigated in four studies across Europe, three of which presented self-reported allergy only. The rates of self-reported allergy were 0.5% to octopus for a group of 22 year olds in Denmark (Osterballe 2005), 0.4% to oyster in 5-7 year olds in France (Touraine 2002) and 0.5% to all molluscs in >39year olds in Spain (Falcáo 2004). Zuberbier 2004 reported a 0% prevalence of sensitisation to mollusc in all ages in Germany.

Looking at the rest of the world, despite a large number of questionnaire-based studies indicating reported rates of 0.2% (Ben-Shoshan 2010) to 4.3% (Connett 2012) and sensitization rates of 0.3% to 0.8%, only one study performed food challenges to fish reporting that 0.2% of 3-7 year olds in Thailand have a confirmed fish allergy (Lao-Araya 2012). Self-reported rates of shellfish allergy varied between 0.1%-11.7%. Sensitization rates measured by SPT were between 0-3.7% and 4.6- 6.7% as measured by

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SIgE testing. However only one study reported on shellfish allergy prevalence based on SPT plus a good clinical history and found that 17.3% of 6-8 year olds in Taiwan have a shellfish allergy (Wan 2012).

Data on mollusc allergy is even more sparse, with Lao-Araya showing that 0.2% of 3 -7 years olds in Thailand self-report problems to eating molluscs, Wu 2012 report clinician diagnosed mollusc allergy ranging from 0.1% in under 3 year olds to 1.5% in adults in Taiwan. Wan 2012 indicates very high sensitisation rates in the same country, based on SPT and a good clinical history ranging from 2.3% (squid) to 25.1% (abalone) in children. Importantly, there were no studies conducted worldwide that used food challenges to confirm the prevalence of mollusc allergy in children or adults. The majority of studies identified on fish and shellfish allergy reported prevalence rates of IgE-mediated and non-IgE mediated allergy collectively. However 22 studies reported IgE-mediated allergy only, using sensitisation rates and a convincing history of IgE- associated symptoms following ingestion of fish or shellfish to confirm this type of allergic reaction.

Fruit

A large variety of fruits have been studied including: a mixture of fruit and vegetables, apple, citrus/orange fruits, strawberry, kiwi, pear, apricot, cherry, grape, nectarine, peach, plum, banana, and pineapple. Those studied in the rest of the world but not in Europe included: pawpaw, mango and melon, litchi, “fruit juice”, “dried fruit”. Adverse reactions to cherry, plum and apricot were reported in Europe but not in the rest of the world. Considering the debates surrounding the use DBPCFC in diagnosing fruit allergies a surprisingly large number were conducted, questioning the allergenicity of the challenge food. The potential for adverse reactions to be linked to oral allergy syndrome or latex allergy and possible cross-reactions were not mentioned either.

Milk

One of the main problems with reporting the prevalence of milk allergy is that many studies have failed to distinguish between IgE and non-IgE mediated cow’s milk allergy. The latter has also been incorrectly referred to as milk intolerance prior to 2004. Finally, due to the time to onset of symptoms, non-IgE mediated cow’s milk allergy may be missed in many cases if food challenges were only performed over one day rather than at least 3-4 days.

Studies indicated the prevalence of food-challenge proven milk allergy in the EU as 0.9% in under 3 year olds (Osterballe 2005; Denmark) and 1.6% in 3 year olds (Osterballe 2005; Denmark). Also in Denmark, prevalence rates of 0.4% in 6 month olds, 0.6% in 9 month olds, 1.1% in 18 month olds, 0.8% in 1 year olds, 0.7% in 3 year olds and 0% in 6 year olds have been reported (Eller 2009). Similar prevalence rates have been found in the UK: 2.4% in 1 year olds, 1.2% in two year olds and 0.4% in 3 year olds (Venter 2008). Only two studies looked at milk allergies in older children which found 0.1% challenge proven milk allergy in 6 -9 year olds in Turkey (Orhan 2009) and 0.8% in 6 year olds in the UK (Venter 2008).

In adults only three studies looked at challenge-proven milk allergy reporting prevalence rates of 0.8% in over 22 year olds in Denmark (Osterballe 2005), 0% in adults in Turkey (Gelincik 2008) and 0.2% in a whole population in Germany (Zuberbier 2004). Looking at the rest of the world, milk allergy prevalence in children younger than 3 years ranged from 1.3% (Chen 2009) to 5% (Bock 1987). In older children only one study, conducted in Taiwan, reported challenge-proven milk allergy and found none of the children to be milk allergic (Loa-araya 2012). There are no studies looking at the prevalence of milk allergy in the rest of the world.

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Mustard

The prevalence of mustard allergy has been examined by a single study, which presented self-report data only, finding that 3% of teenagers in France self report problems with mustard (Touraine 2002). No other studies on the prevalence of mustard allergy could be found in the literature. Hence, there are huge gaps in our knowledge of the prevalence of mustard allergy, notably the prevalence of mustard allergy confirmed by food challenge.

Peanut

Peanut allergy is probably the most discussed allergen in the world and, due to the severity of the reactions, most of the studies investigating immunotherapy to foods have focused on peanut allergy. The prevalence of peanut allergy has been studied widely in the EU and the rest of world with using varied methodologies. In Europe, the landmark studies have included those conducted by Osterballe et al. (Denmark), Venter et al. (UK), Hourihane et al. (UK) and Nicolau et al. (UK). These studies provide valuable data on the prevalence of challenge-proven peanut allergy in young children with the highest rate reported as 1.8% in a group of 3-6 year olds in the UK (Hourihane 2007). In older children, Nicolau et al. (UK) reported a prevalence of 1.9% challenge-proven peanut allergy in 8 year olds (Nicolau 2009). The only data on challenge proven peanut allergy in adults in Europe is from Osterballe et al. showing that 1.2% of adults in Denmark suffer from peanut allergy (Osterballe 2005).

Studies investigating peanut allergy in the rest of the world has been dominated to some extent by questionnaire based studies in the US and Canada. In terms of challenge-proven peanut allergy, Osborne et al. (2011; Australia) found the highest prevalence (2.9%) in 12-15 month olds. Using a complex definition of peanut allergy, which included food challenges in some participants, Ben-Shoshan 2010 (Canada) reported a prevalence of 1.6% in 7 year olds in 2005; a slight increase from that reported for 7 year olds in 2000-2002 (1.3%). In the same country, Kagan 2003 reported prevalence for peanut allergy of 1.5% in 5-9 year olds using similar definitions to that of Ben-Shoshan 2010. The geographical disparities in allergies to individual foods is highlighted by the findings of Dalal 2002 who did not diagnose any peanut allergy in a group of 0-2 year olds in Israel.

Sesame

The prevalence of self-reported sesame allergy ranged between 0 - 1.5% (Touraine 2002; Emmett 1999), with only one study in Europe reporting challenge-proven sesame allergy. This study found the prevalence to be 0.6% in 3 year olds and 0.1% in 6 year olds (Venter 2006; Venter 2008). Sensitisation rates varied between 0.1 – 1.4%. In the rest of the world, the prevalence of self-reported sesame allergy ranged between 0.1 – 0.2%, although data was only available for Canada (Ben-Shoshan 2010). Sensitisation rates determined by SPT and the prevalence of challenge-proven sesame allergy was reported by only one study, which found that 1.6% of 12-15 month olds in Australia are sensitized to sesame and 0.7% (95% CI: 0.4-1.0) had challenge-proven sesame allergy (Osborne 2011). Despite the finding of a study conducted in Israel that there was no challenge-proven peanut allergy in a study group of 0-2 year olds, 0.2% of the study group did have challenge-proven sesame allergy (Dalal 2002).

Soya

Soya allergy is often mentioned in relation to cow's milk allergy IN infants and it is estimated that up to 60% of children with gastro-intestinal milk allergy may suffer from co-existing soya allergy. This figure is much lower in children with IgE-mediated allergy. Soya milk as an alternative for children with cow's milk allergy is also often debated. However, despite all the hype surrounding soya and possible soya allergy only one study conducted in Europe and another conducted in the US report challenge-proven allergy to soya. Osterballe 2005 found no soya allergy in a group of under 3 year olds from Denmark although Bock 1987 found that 0.8% of children in the same age group in the United States had soya allergies (Bock 1987). In addition, Osterballe 2005 also reported that 0.4% of 3 year olds and 0.3% of adults in Denmark suffer from a soya allergy.

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Tree nuts

Many studies looking into tree nut allergy disappointingly did not specify the type of nuts being studied. Self-reported tree nut allergy to unspecified nuts however ranged from between 0.2 – 4.7% (Obeng 2011; Shek 2010). The studies that reported prevalence based on a challenge-proven diagnosis to specific tree nuts focused on hazelnut, walnut, almond, cashew nut and pistachio. These studies indicate that 2.2% of the German population suffer from a hazelnut allergy (Zuberbier 2004), as do 0.1% of the adult Turkish population and 0-1% of older children in Turkey (Orhan 2009; Mustayev 2012; Gelincik 2008), and 0.1% of 3 year old children in the UK (Venter 2008). Walnut allergy based on food challenges is reported in 1% of the German population (Zuberbier 2004) as well as 0.1% of Turkish adults and 0-0.4% of older children in Turkey (Orhan 2009; Mustayev 2012; Gelincik 2008). Food challenge proven almond and cashew nut allergy was reported by only one study (Venter 2008) in 0.2% and 0.1% of 3 year olds in the United Kingdom respectively. None of the studies in the rest of the world report challenge-proven tree nut allergies, apart from a 2.2% prevalence rate for pistachio allergy in 6-8 year olds in Taiwan (Wan 2012).

Other foods

Numerous foods have been reported in the literature to cause adverse reactions in individuals in both Europe and throughout the world. The majority of studies utilised self-reported methods for calculating prevalence. Before the year 2000 studies reported prevalence rates ranging from 0% to 11%, which increased to 14% in studies published after 2000. This suggests a minimum of a 3% increase in reported levels of prevalence. This could be due to the increased knowledge and awareness of allergy worldwide and also the expanding availability of different foods. However, compared to other allergens, there was a lack of studies adopting the gold standard of diagnosis which incorporates both open and double-blind placebo-controlled food challenges. This is the most effective way to determine prevalences of food allergy.

Therefore in summary, it is surprising to find such paucity of information on the prevalence of food allergy, although the published literature does give us a good indicating of the scale of the problem. The lack of information may be explained by the cost incurred of performing large scale epidemiological studies and the difficulties in performing food challenges, particularly DBPCFC. It is hoped that the evidence base will be enriched once the Europrevall studies funded by the EU are published.

Emerging allergens

This section presents a summary and analysis of the data gathered on allergens other than: milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame that have either increased in prevalence or have been highlighted as there was a significant reported prevalence in at least one country in Europe.

The prevalence of allergy to citrus fruits was found in this review to be relatively high for self-reported allergy with values between 3.2 to 11% being reported from a range of countries including Finland, Germany, Iceland, Sweden and the United Kingdom. Fewer studies reported challenge results and these tended to give a lower prevalence of 2% and under. Allergy to citrus is often reported as resulting in mild symptoms however there are reports of severe reactions including anaphylaxis. Those at risk of these more severe reactions are possible those with allergy to the lipid transfer proteins (Ebo, Ahrazem, Lopez-Torrejon, Bridts, Salcedo and Stevens; 2007).

Kiwi allergy has been reported in the wider literature as being one of the more common causes of allergy to fruit. We found that this food was not reported as a separate item by the majority of included studies. The data we could extract indicated self-report in France of 0.8% and open food challenge in Turkey at 0.1 %. Although it is thought that many people with allergy to kiwi fruit experience mild symptoms, there are reports of more severe reactions including anaphylaxis recently reviewed by Lucas (2003).

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The prevalence of positive skin prick tests to tomato was relatively high in Italy Ronchette (2008) 3.1%. Those that used more robust methods such as food challenge gave much lower values, the highest being in Turkey, Gelincik (2008) with 0.1% for those over 18, and other studies indicating less than 01%, Orhan (2009) and Venter (2006).

Additives as a group were highlighted in a number of studies countries. Self-reported allergy could be quite high at 6.6 % Osterballe (2005), and 3.4 % in the United Kingdom Pereira (2005), however on challenge the figures were lower at less than 0.1%.

Cocoa allergy was reported in a number of countries, Orhan (2009) supplied the self-reported, skin prick test with history and challenge findings with the later indicating a 0.1% prevalence. That this could cause symptoms in 0.1% was concurred by Zurberbier (2004) and Gelincik (2008) . There are very few reports of allergic reactions to cocoa in the wider literature and no reports on anaphylaxis.

Wan (2012) carried out skin prick tests on those who reported a positive history of garlic allergy and relatively high prevalence at 11.6% for children in Taiwan for children. The same study showed relatively high for onion and bamboo shoot. In European studies self-reported allergy to garlic was lower at only 1.5% in France, Touraine (2002), 0.1% in Turkey Gelincik (2008). For all other allergens the self-reported rates are higher than challenge findings and so we would expect very garlic allergy rates Europe according to our data. The wider literature does include reports of allergy to garlic including anaphylaxis, (Pérez-pimiento, Santaolalla, De Paz, Fernández-parra, Domínguez-lázaro, and Moneo; 1999).

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1.3. List of Included Studies

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1.4. List of Excluded Studies

Below is a table of studies excluded from the review and their reasons for exclusion. These have been selected on the basis that they might be expected to have been included in the review (e.g. they have been included in previous systematic reviews), but did not meet the eligibility criteria (Table 1.12).

Table 1.33: Studies excluded from the systematic review

Short Title	Title	Reason for exclusion
Aardoom (1997)	Food intolerance (food hypersensitivity) and chronic complaints in children: the parents' perception	Food intolerance rather than food allergy
Aba-Alkhail (2000)	Prevalence of food allergy in asthmatic patients	Sample, all had asthma
Altman (1996)	Public perception of food allergy	Duplicate
Avila (2002)	Hypersensitivity detected by skin tests to food in allergic patients in the Hospital Infantil de Mexico Federico Gomez	Inappropriate sample (allergic patients only)
Bernardini (1998)	Prevalence and risk factors of latex sensitization in an unselected pediatric population	No separated data for each allergen
Biagini (2004)	Evaluation of the prevalence of antiwheat-, anti-flour dust, and anti-alpha-amylase specific IgE antibodies in US blood donors	Sample, not a representative population
Bival'kevich (1990)	Allergic diathesis in infants in the first year of life	Topic
Garcia (2003)	Incidence of allergy to cow's milk protein in the first year of life and its effect on consumption of hydrolyzed formulae	Sample, all had suspected cow's milk allergy
Gislason (2000)	Allergy and intolerance to food in an Icelandic urban population 20-44 years of age	Sample, enriched with participants with asthma
Hill (1997)	The frequency of food allergy in Australia and Asia	Sample inappropriate (Limited to children of atopic parents only)
Hossny (2011)	Peanut sensitization in a group of allergic Egyptian children	Inappropriate sample (allergic patients only)
Host (1990)	A prospective study of cow milk allergy in Danish infants during the first 3 years of life. Clinical course in relation to clinical and immunological type of hypersensitivity reaction	Linked to Host 2002

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Short Title	Title	Reason for exclusion
Isolauri (2004)	The allergy epidemic extends beyond the past few decades	Excluded in error for extraction
Jansen (1994)	Prevalence of food allergy and intolerance in the adult Dutch population	Excluded in error for extraction
Kaczmarski (1999)	The prevalence of food allergies in infants in North-Eastern Poland	Review article
Kanny (2001)	Population study of food allergy in France	Data not provided by individual allergen
Keiding, (1997)	Asthma, allergy and other types of hypersensitivity in Denmark: and the development	Unable to locate article
Levin (2011)	Associations between asthma and bronchial hyper-responsiveness with allergy and atopy phenotypes in urban black South African teenagers	Topic, mainly concerning allergy to inhalant allergens
Lunet (2005)	Self-reported food and drug allergy in Maputo, Mozambique	Reports allergy to foods collectively i.e not per allergen
Marklund (2004)	Health-related quality of life among adolescents with allergy-like conditions - with emphasis on food hypersensitivity	Sample, not cross section of community
Marklund (2006)	Health-related quality of life in food hypersensitive schoolchildren and their families: parents' perceptions	Sample, not cross section of community
Ouahidi (2010)	The effect of thermic and acid treatment on the allergenicity of peanut proteins among the population of the region of Fes-Meknes in Morocco	Topic not suitable
Penard-Morand (2005)	Prevalence of food allergy and its relationship to asthma and allergic rhinitis in schoolchildren	Topic not suitable
Ramos (1993)	Hypersensitivity to common allergens in the central region of Coahuila	Review article
Rodriguez-Ortiz (2009)	Epidemiological characteristics of patients with food allergy assisted at Regional Center of Allergies and Clinical Immunology of Monterrey	Sample, all had allergy
Roehr (2004)	Food allergy and non-allergic food hypersensitivity in children and adolescents	Excluded in error
Schrander (1993)	Cow's milk protein intolerance in infants under 1 year of age: a prospective epidemiological study	Included for extraction

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Short Title	Title	Reason for exclusion
Takahashi (1998)	Buckwheat allergy in 90,000 school children in Yokohama	Incidence rather than prevalence
Tariq (2000)	Egg allergy in infancy predicts respiratory allergic disease by 4 years of age	Data reported elsewhere
van Bockel-Geelkerken (1992)	Prevalence of putative food hypersensitivity in young children	Excluded
Venter (2006)	Incidence of parentally reported and clinically diagnosed food hypersensitivity in the first year of life	Prevalence data reported elsewhere
Westritschnig (2003)	Analysis of the sensitization profile towards allergens in central Africa	Sample all participants had allergy
Woods (2001)	International prevalences of reported food allergies and intolerances. Comparisons arising from the European Community Respiratory Health Survey (ECRHS) 1991-1994	Australian data presented in Woods 2002 and no separated data for individual countries for specific allergens.
Woods (2002)	Prevalence of food allergies in young adults and their relationship to asthma, nasal allergies, and eczema	Inappropriate sample (the sample has been enriched from a group reporting asthma-like symptoms)

1.5. Additional references

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2. THE EFFECT OF FOOD PROCESSING ON THE ALLERGENICITY IN RELATION TO EACH OF THE FOLLOWING FOOD ALLERGENS: MILK/DAIRY, EGGS, CEREALS, BUCKWHEAT, PEANUTS, NUTS, CELERY, CRUSTACEANS, FISH, MOLLUSCS, SOY, LUPIN, MUSTARD AND SESAME? (OBJECTIVE 4A)

2.1. Introduction

2.1.1. Assessing allergenicity of the processed food

Guidelines indicate that double blind placebo controlled food challenges are the method of choice assessing allergenicity of foods and diagnosis of allergy for those with immediate and delayed type reactions in Europe (Bindslev-Jensen et al., 2004; Fiocchi et al., 2010) and the United States of America (Boyce et al., 2010). However open challenges can be used for specific situations (Bindslev-Jensen, et al., 2004) and have been shown to have reasonable negative predictive values (Venter, 2007). In food challenges participants are challenged with increasing doses of the food and once symptoms are experienced the challenge halted. Allergenicity of that food for an individual may be expressed as the dose eliciting a reaction, or the dose combined with the type of symptoms experienced (Hourihane et al., 2005) (Nowak-Wegrzyn et al., 2009) but there are no agreed standards for doing this.

Skin prick tests with food allergens and measurement of specific IgE in serum without clinical history or challenge results have been shown to have poor accuracy for diagnosis of food allergy (Fiocchi et al., 2002; Järvinen & Sicherer, 2012), and these tests do not indicate accurately enough the intensity of reaction on food challenge or the threshold dose that could elicit symptoms (Hourihane, et al., 2005) (Osterballe & Bindslev-Jensen, 2003). In addition these tests are not appropriate for non IgE mediated allergy (Fiocchi et al., 2010).

Therefore changes in the allergenicity of the processed foods have been assessed using evidence from studies comparing open or blind challenge data, studies comparing ability to bind specific IgE or ability to provoke a positive skin prick test will not be included in this review. This review presents details of the challenge procedure for quality assessment and comparability.

2.1.2. Participants

The participants involved with challenge studies are key to the quality of the research; hence one of the quality criteria for assessing the studies was whether the participants are representative of those with food allergy. A random sample would reduce the risk of bias. As person specific factors affect symptoms experienced by individuals on exposure to a particular food it is of paramount importance that the population studies were described in detail so that the generalisability of the findings to specific populations could be assessed.

2.1.3. Food processing methods

A wide range of methods were assessed. We have distinguished between studies of laboratory prepared foods and those using commercially available or kitchen prepared foods that could be less reliable but more relevant to real world situations.

2.2. Materials and Methods

2.2.1. Literature search strategy

The following databases were searched from Web of Science (1970-November 2012), BIOSIS Citation Index (1969-November 2012), BIOSIS reviews (1969-2008), Medline (1950-November 2012), Pubmed (-November 2012), using the search terms shown (Table 2.1). No limits were used.

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Table 2.1: Search strategy in Web of Knowledge. Each group was combined with the terms within a group were linked with 'or' and the groups were linked with 'and'

Topics	Search terms Web of Knowledge	Search terms (Including appropriate MeSH terms) PubMed
Group 1. Food		
Milk and dairy	milk OR butter or cream or dairy or cheese or yoghurt or petit filous or casein or whey or lacto Infant NEAR/2 formula	milk[Tiab] OR milk[MeSH Terms] OR lactose[MeSH Terms] OR lactose[Tiab] OR dairy[Tiab] OR butter[Tiab] OR cream[Tiab] OR "infant formula"[Tiab] OR cheese[Tiab] OR yoghurt[Tiab] OR "petit filous"[Tiab] OR casein[Tiab] OR whey[Tiab]
Egg	Egg	egg[Tiab] OR eggs[Tiab]
Cereals	Cereal or gluten or wheat or rye or barley or oats or spelt or kamut	cereals[Tiab] OR glutens[MeSH Terms] OR glutens[Tiab] OR gluten[Tiab] OR wheat[Tiab] OR rye[Tiab] OR barley[Tiab] OR oats [Tiab] OR oat[Tiab] OR spelt[Tiab] OR kamut[Tiab]
Peanuts	peanut or arachis	peanut[Tiab] OR arachis[Tiab]
Nuts	nut or arachis or cashew or brasil or almond or hazel or walnut or pecan or macadamia or pistachio or filbert	nuts[MeSH Terms] OR nuts[Tiab] OR nut[Tiab] OR almond[Tiab] OR almonds[Tiab] OR hazelnut[Tiab] OR hazelnuts[Tiab] OR walnut[Tiab] OR walnuts[Tiab] OR cashew[Tiab] OR cashews[Tiab] OR pecan[Tiab] OR pecans[Tiab] OR macadamia[Tiab] OR macadamias[Tiab] OR pistachio[Tiab] OR pistachios[Tiab] OR beechnut[Tiab] OR beechnuts[Tiab] OR filbert[Tiab] OR filberts[Tiab]
Celery	Celery	celery[tiab]
Crustaceans	crustacea OR crustacean OR crustaceans OR crab OR crabs OR lobster OR lobsters OR shrimp OR shrimps OR prawn OR prawns OR crayfish OR shellfish OR langoustine OR langoustines	crustacea[MeSH Terms] OR crustacea[Tiab] OR crustacean[Tiab] OR crustaceans[Tiab] OR crab[Tiab] OR crabs[Tiab] OR lobster[Tiab] OR lobsters[Tiab] OR shrimp[Tiab] OR shrimps[Tiab] OR prawn[Tiab] OR prawns[Tiab] OR crayfish[Tiab] OR shellfish[MeSH Terms] OR shellfish[Tiab] OR langoustine[Tiab] OR langoustines[Tiab]

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Topics	Search terms Web of Knowledge	Search terms (Including appropriate MeSH terms) PubMed
Fish	fish OR pollock OR carp OR cod OR mackerel OR salmon OR tuna OR shark OR “sea bass” OR swordfish OR hake OR sole OR megrim OR sardine OR sardines OR halibut OR anchovy OR anchovies OR catfish OR trout	fishes[MeSH Terms] OR fish[Tiab] OR pollock[Tiab] OR carp[Tiab] OR cod[Tiab] OR mackerel[Tiab] OR salmon[Tiab] OR tuna[Tiab] OR shark[tiab] OR “sea bass”[tiab] OR swordfish[tiab] OR hake[tiab] OR sole[tiab] OR megrim[tiab] OR sardine[tiab] OR sardines[tiab] OR halibut[tiab] OR anchovy[tiab] OR anchovies[tiab] OR catfish[tiab] OR trout[tiab] mollusca[MeSH Terms] OR mollusc[Tiab] OR molluscs[Tiab] OR oyster[Tiab] OR oysters[Tiab] OR snail [Tiab] OR snails[Tiab] OR squid[Tiab] OR mussel[Tiab] OR mussels[Tiab] OR clam[Tiab] OR clams[Tiab] OR abalone[tiab] OR octopus[tiab] OR scallop[tiab] OR scallops[tiab]
Soy	Soy*	soy[Tiab] OR soybeans[MeSH Terms] OR soybean[Tiab] OR soybeans[Tiab] OR soya[Tiab]
Lupin	LUPINUS-ALBUS, Lupin*	lupinus[MeSH Terms] OR lupin[Tiab]
Mustard	Mustard	"mustard plant"[MeSH Terms] OR mustard[Tiab]
Sesame	Sesame*	sesamum[MeSH Terms] OR "sesame"[Tiab]
Group 2. Food Challenge		
Open food challenge	(Food or oral or open or mucosal or ingestion) near/2 Challenge	Challenge*[tiab]
Double blind placebo controlled food challenge	((food or oral or mucosal or ingestion) near/2 challenge*) OR (DBPC)	DBPC*[tiab] “double blind placebo controlled”

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Topics	Search terms Web of Knowledge	Search terms (Including appropriate MeSH terms) PubMed
Group 3. Food processing		
Heat and chemical Cooking, heavy salting,, microwaving, filtration, fermenting, smoking, drying, UV treatment for sterilisation, acid, alkaline (lyme treatment) treatment in powder production e.g. coffee, other heating treatments (ohmic) and chemical peeling of fruit (lipid transfer protein in skin).	(heat* or cook* or roast* or fry* or pasteuri* or boil) or (heavy near/2 salting) or dying or microwav* OR ferment* or smoking or drying or (UV NEAR/2 treatment) or lyme or ohmic OR (chemical near/4 peeling)) or Hydrostatic pressure or (food near/1 process*) or (food near/1 process*) or (digest*) or (hydrol*) or filtration	Heat*[tiab] OR cook*[tiab] OR roast* [tiab] OR fry*[tiab] OR pasteuri*[tiab] OR boil[tiab] OR Hydrolysis [tiab] OR digestion [tiab] OR enzymatic treatment [tiab] OR fermented [tiab] OR Hydrostatic pressure[tiab] OR food process* [tiab] OR "heavy salting" [tiab] OR dying [tiab] OR microwav* [tiab] OR ferment* [tiab] OR smoking [tiab] OR drying [tiab] OR UV [tiab] OR lyme [tiab] OR ohmic [tiab] OR "chemical peeling"[tiab]
Filtration/specific product related	(wine OR beer OR clarif*)	wine [tiab] OR beer [tiab] Or clarify* [tiab]

2.2.1.1. Selection procedure

All titles and abstracts were imported to Endnote and duplicates removed. One reviewer, SK, screened the titles and abstracts to remove studies not relevant to the objective. The full texts were obtained for the remaining studies; a second screen by SK then removed studies that were not relevant to the research question and the reasons identified.

2.2.1.2. Types of studies

We included any study that reported on the effect of food processing on the allergenicity of the named foods, a wide range of sampling designs were acceptable including those involving people from:

- a random sample from a cross-section of a community or clinic population.
- a non-random sample from a cross-section of a community or clinic population.
- convenience or self or clinician selected volunteers with food allergy.

Studies that used the following designs were included:

- cross-over with challenge with a comparison form of the food against another test form of the food or a test from that has a different intensity of treatment, for example oven treatment at 200°C at 30 minutes versus 300 ^{o}C for 1 hour. Studies with random or non-random order of cross over were included.

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- Random or non random between group comparisons in which a group with proven allergy (with positive challenge) to the food are allocated to exposure to a comparison form of the food or a test form of the food using random or non random methods. Each participant being exposed to either the comparison or test form of the food only. For example infants with allergy to cow milk being allocated to partially hydrolysed or fully hydrolysed cow milk formula.

- Non-comparison studies were included only if those being challenged had a recent diagnosis of allergy to that food using a valid method that included food challenge. We did not include non comparison studies where the participants did not have a recent positive food challenge result to the comparison food.

2.2.1.3. Type of participants

We included studies whose participants were either adults or children (residing in any country) with gastro intestinal food allergies such as eosinophilic esophagitis, eosinophilic gastroenteritis, food protein induced allergic proctocolitis, food protein induced enterocolitis syndrome, oral allergy syndrome, those with cutaneous reactions to foods such as acute urticaria, angiodema, atopic dermatitis, allergic contact dermatitis, contact urticaria, and respiratory symptoms (Boyce et al, 2010) with a positive diagnosis using recognized procedures such as of a history of symptoms and a positive serum specific IgE or skin prick test to the relevant food (any foods containing milk/dairy, eggs, cereals, peanuts, nuts, celery, crustaceans, fish, molluscs, soy, lupin, mustard and sesame), or a positive food challenge (Boyce et al 2010).

2.2.1.4. Methods of food processing

We included studies comparing different types of processing (e.g. frying, boiling, dry oven, sterilisation, pasteurisation, enzyme degradation or heating and/or pressure; mechanical concentration or fractionation; chemical treatment including action of enzymes) or different intensities (e.g. duration or temperature) of processing methods or processing methods compared to the raw or native product. The full list is included in the group 3 terms of the search strategy (Table 2.1).

2.2.1.5. Types of outcome measure

The review included studies that assessed allergenicity of the food determined by observation of:

- Type and intensity of symptoms (self reported or clinician assessed)

- Dose to elicit a reaction

- Combination of the above

on contact with or ingestion of the food product within a clinic or office setting, using open or double blind placebo controlled challenge by participants with the relevant food allergy (see definition above).

2.2.2. Extraction of data

Data on the methods of recruiting participants, description of participants, diagnosis, food processing methods, challenge procedures and allergenicity of the foods was collected, by SK. A second reviewer checked that the data extraction and interpretation was accurate, and any differences resolved by discussion. Data was collected and stored in EPPI-Reviewer 4 software (Social Science Research Unit at the Institute of Education, University of London, UK).

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2.2.3. Assessing the quality of studies

We assessed the quality of the studies using the following categories:

1. Quality of diagnosis of food allergy for the study participants
2. Sampling procedure
3. How representative the sample is likely to be if people with severe allergy
4. Challenge procedure, i.e. is it accurate for the individual food
5. Comparison of challenge findings between processed foods

The methods for doing this quality assessment are outlined in Table 2.2.

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Table 2.2: The method for assessing the quality of the included studies

Criteria	Very low risk of bias	Low risk of bias	High risk of bias	Unclear
Quality of diagnosis	Double blind placebo controlled food challenge	Open challenge or convincing history with sensitization (serum specific IgE &/or SPT) (if appropriate)	Sensitisation (skin prick test and/or serum specific IgE) without clinical history Clinical history alone Self-report	Not stated
Sampling procedure	NA	Random sampling, clearly defined population	Non random sampling	Not stated
How representative the sample is likely to be if people with severe allergy	NA	random sampling from a group with severe allergy	Non random sampling	Not stated
Challenge procedure, i.e. is it accurate for each individual form of food	Double blind placebo controlled food challenge with random sequence of placebo/active, and taste tested masking recipe	Stated as double blind placebo controlled food challenge	Open challenge	Not stated
Comparison of challenge findings between different forms of processed foods or raw foods	NA	Individual challenges had low risk of bias and all participants underwent challenges with both forms of the food	Individual challenges had high risk of bias, or not all participants underwent challenge with both forms of the food.	Not stated

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2.2.4. Analysis of Data

We classified the two or more forms of the food used in the challenge as comparison or test, if there was more than one test food these were labeled sequentially. Normally the form of the food that was used to select participants was considered to be the comparison food e.g. 'we recruited people with allergy to raw peanut', comparison food= raw peanut. In studies that were not specific about whether the selection criteria was allergy was to processed or unprocessed forms of the food we selected the least processed food form as the comparison e.g. 'we recruited people with egg allergy and then challenged to extensively heated and raw egg (comparison =raw egg, test =extensively heated egg). For dichotomous data such as percentage demonstrating a positive challenge if possible we presented the findings as Odds Ratio or Risk Ratio. For continuous data such as the minimum dose to elicit symptoms we presented the findings as mean difference.

2.3. Results

2.3.1. Results of Search

After removal of duplicates 1040 references were retrieved of these 86 were potentially eligible after full text screening (Figure 2.1). The types of studies excluded at this stage were listed as those without human participants, not involving food allergy, did not involve one of the listed foods, and studies where there was no comparison of different processing methods on the allergenicity of the foods (Figure 2.1). Within the 'other' category studies were excluded because data was not available for individual foods or the study involved only one or two cases.

The studies excluded after full text screening are show (Section 2.5 List of excluded studies), common reasons for exclusion at this stage were that participants were only challenged to one form of the food and that the majority of challenges were negative, therefore current food allergy could not be confirmed. Other groups of excluded studies were those investigated immune reactivity and not challenge findings, being represented by studies that investigated how IgE binding to food proteins within ELISA, or western blotting are altered by different processing methods. Studies investigating primary prevention of allergy by introduction of special infant milk formulas were also excluded at this stage as they were beyond the remit of this review.

We attempted to find studies investigating the effect of using egg or milk as fining agents within the wine making industry. However we were unable to find studies that included egg and milk allergic participants who were confirmed with oral food challenges and who also underwent challenges with the wine products.

Although a large number of studies are carried out on peanut allergy we could not find studies that challenged participants with two forms of peanut, for example raw and roasted, however we did find one study that investigated the allergenicity of crude or refined peanut oil.

We included 25 studies and they are detailed in the following section.

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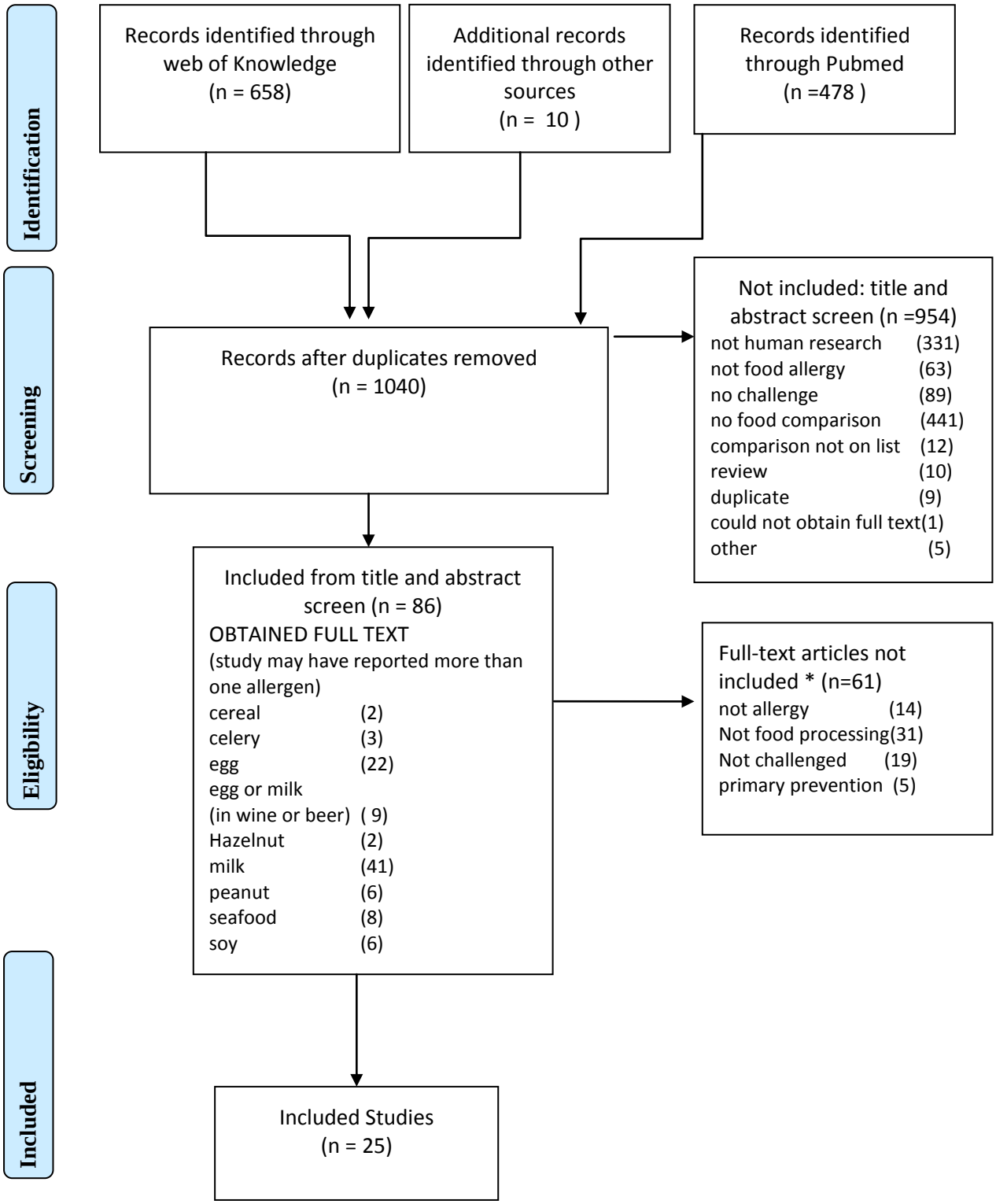
Figure 2.2.: PRISMA flow diagram, ending with included studies, and number included for each food, Full text screening excluded and reasons



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2.3.2. Description of Studies

The studies included investigated the allergenicity of the following reported allergens celery (1), wheat (1), egg (6), hazelnut (2), milk and dairy (14), peanut oil (1) these studies are listed in food order (Table 2.3). Studies tended to focus on the effect of heat, commonly, boiling, roasting or baking. The exceptions were the studies investigating hydrolysis and fractioning of milk for infant milk formulas, Ammar (1999), Burks (2008), Caffarelli (2002), Giampietro (2001), Kaczmarski (2005), Niggemann (2008), Ragno (1993), Rugo (1992), Sampson (1991) and one study investigating the effect of maturation time for cheese production for those with allergy to the additive lysozyme (from egg), Marseglia (2012) or milk allergens Alessandri (2012).

The majority of studies utilized a cross-over design with each participant underwent challenge to two forms of the food. The order in which the participants were allocated to the challenge with each type of food was determined randomly for only a small proportion of studies and these investigated milk, Giampietro (2001), Host (1988), Ragno (1993), Sampson (1991) and one peanut oil, Hourihane (1997). A random order of challenge reduces the risk of bias for the study, as those that are challenge positive to one form of food could refuse challenge with the second form of food, or one type of food is perceived as being more allergenic than another.

The remaining cross-over studies used a non-random order, usually because the participants were challenged to the food considered least allergenic first as exemplified by Nowak-Wegrzyn (2008). Within this study participants were challenged with heated milk, only those that were challenge negative then went on to have the challenge with un-heated milk. A similar study was carried out with egg, Lemon-Mule (2008), where challenges were first carried out to baked eggs and again only those that tolerated this challenge went on to be challenged with regular egg (scrambled or cooked in French toast). The authors designed these studies to investigate whether a diet including extensively heated egg or milk could lead to increased tolerance rather than the effect of processing on allergenicity.

In one study, a large group of participants was challenged to raw hazelnut first and then a subset challenged to the roasted product. This study was considered as a between group comparison as the paired cross-over data was not available Worm (2009). The selection procedure for this subset was not clear and so the group receiving challenge with the roasted product could have been naturally more or less sensitive to hazelnut protein than the main group.

Table 2.3: Summary of description of studies (alphabetical order by food)

Study ID	Food	Type of allergy	Source	Comparison processing method	Test processing method	Design
Ballmer-Weber (2002)	Celery	Both IgE and non IgE mediated	Clinic Allergy Unit of University Hospital Zurich	Raw	Baked 110 °C for 15 min	Cross over non random
Scibilia (2006)	Cereals Wheat	IgE mediated allergy	Clinic Allergy Units in Europe, Niguarda Ca Granda Hospital, Milan, Italy; Milan University Hospital, Milan, Italy; or Odense University	Raw	Boiled	Cross over non random

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Study ID	Food	Type of allergy	Source	Comparison processing method	Test processing method	Design
			Hospital, Odense, Denmark			
Alessandri (2012)	Egg	IgE mediated allergy	Clinic Centre for Molecular Allergology, IDI-IRCSS, Rome, Italy	Raw	Boiled 10 min	Cross over non random
Ando (2008)	Egg	IgE mediated allergy	Clinic	Raw egg white	Extensively heated 90 °C for 60 min	Cross over non random
Lemon-Mule (2008)	Egg	IgE mediated allergy	Clinic Mount Sinai Medical Centre New York	Heated (regular) Scrambled/French toast	Extensively heated	Cross over non random
Boyano Martinez (2001)	Egg	Both IgE and non IgE mediated	Not reported	Raw	Boiled 10 min	Cross over non random
Urisu (1997)	Egg white	IgE mediated allergy	Not reported	Raw	Extensively heated 90 °C for 60 min	Cross over non random
Marseglia (2012)	Egg, Lysozyme	IgE mediated allergy	Clinic Pediatric Unit, University Hospital Pavia, Italy	Unclear if raw or heated egg	Cheese; Granda Padano 12 month matured; Granda Padano 24 month matured	Cross over Non random
Hansen (2003)	Hazelnut	IgE mediated allergy	Clinic Allergy units, University Hospitals in Copenhagen and Zurich	Raw	Roasted 140 °C 40 min	Cross over non random
Worm (2009)	Hazelnut	IgE mediated allergy	Clinic Dermatology outpatient clinic	Raw	Roasted 144 °C	Between group comparison non random
Alessandri (2012)	Milk/ Dairy	IgE mediated allergy	Clinic Centre for Molecular Allergology, IDI-IRCSS, Rome, Italy	Pasteurized	Cheese; Parmigiano-Reggiano	Cross over non random
Ammar (1999)	Milk/ Dairy	Both IgE and non IgE mediated	Clinic	Hydrolysed A range of products	Amino acid–based formulas; Neocate	Cross over non random
Burks (2008)	Milk/ Dairy	IgE mediated allergy	Clinic Fourteen clinical sites in the USA	Pasteurized	Amino acid–based formulas;	Cross over non random
Caffarelli (2002)	Milk/ Dairy	IgE mediated allergy	Not reported	Pasteurized	Whey partially hydrolysed; Whey extensively	Cross over non random

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Study ID	Food	Type of allergy	Source	Comparison processing method	Test processing method	Design
					hydrolysed; Casein extensively hydrolysed;	
Giampietro (2001)	Milk/ Dairy	IgE mediated allergy	Clinic Tertiary referral centres in Italy and Sweden	Pasteurized	Whey extensively hydrolysed; Nutrilon Pepti	Cross over random
Host (1988)	Milk/ Dairy	Both IgE and non IgE mediated	Not reported	Raw	Pasteurised Cow's milk	Cross over random
Kaczmarski (2005)	Milk/ Dairy	IgE mediated allergy	Clinic Hospitalised children	Low lactose Cow's milk	Casein extensively hydrolysed; Nutramigen	Cross over non random
Komata (2009)	Milk/ Dairy	IgE mediated allergy	Not reported	Pasteurized	Extensively heated	Cross over non random
Niggeman (2008)	Milk/ Dairy	IgE mediated allergy	Not reported	Pasteurized	Extensively hydrolysed	Cross over non random
Nowak-Wegrzyn (2008)	Milk/ Dairy	IgE mediated allergy	Clinic Mount Sinai Pediatric Allergy Clinic	Pasteurized	Extensively heated	Cross over non random
Ragno (1993)	Milk/ Dairy	IgE mediated allergy	Clinic Allergy and Immunology Division, Depart. Paediatrics, University of Rome "La Sapienza" Italy	Pasteurized	Casein hydrolysate; Nutramigen	Cross over random
Rugo (1992)	Milk/ Dairy	IgE mediated allergy	Clinic	Pasteurized	Whey hydrolysate;; Profylac Whey hydrolysate; Nidina Casein hydrolysate Nutramigen Casein hydrolysate Pregestimil Whey hydrolysate; Alfare Whey hydrolysate; Beba HA Whey hydrolysate; Ultrafiltered	Cross over non random
Sampson (1991)	Milk/ Dairy	IgE mediated allergy	Clinic John Hopkins Paediatric Clinical Research Unit	Pasteurized	Casein Hydrolysate; Alimentum	Cross over random

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Study ID	Food	Type of allergy	Source	Comparison processing method	Test processing method	Design
Kim (2011)	Milk/ Dairy Cow's milk and cheese	IgE mediated allergy	Clinic Mount Sinai Pediatric Allergy Clinic	Pasteurized	Baked 350 °C for 30 min	Cross over non random
Hourihane (1997)	Peanut	IgE mediated allergy	Community Those responding to a survey and who volunteered	Roasted, salted peanuts	Crude peanut oil Refines peanut oil	Cross over random

2.3.3. Participants

The details of the study participants are presented in study author order for easy reference (Table 2.4). All studies of egg and milk were carried out with children (Table 2.4), perhaps reflecting the higher prevalence within these age groups. Studies involving adults were carried out for celery Ballmer-Weber (2002), for wheat Scibilia (2006), and a mixed population for milk, Hansen (2003), Nowak-Wegrzyn (2008) and, Ragno (1993), and for peanuts, Hourihane (1997).

We made the decision to include only those individuals who were challenge positive to one or more of the forms of the food. Therefore, we excluded the data from those who were challenge negative to both forms of the food. So for example using this rule we excluded from our analysis in the study by Nowak-Wegrzyn (2008) on milk 9 of the 100 participants, for the by Boyano Martinez on reactivity to egg we excluded 17 of 56 participants and for the study on peanut oil , by Hourihane (1997) we excluded two of the 62 potential participants.

Of the 25 studies, 20 included participants with either skin prick test or specific IgE sensitivity to the allergen, in addition to the food challenge findings. Studies did not tend to include a high proportion of participants with severe allergy, although many did enroll at least one person with a history of anaphylaxis and within one study, Komata (2009), 49% of participants had a history of anaphylaxis.

Table 2.4: Participants included in the studies (alphabetical order by study author)

Study ID	Food	Type of Allergy	Sensitisation	History of Symptoms	Challenge	Country	Sex	Age Range
Alessandri (2012 a)	Milk/ Dairy	IgE mediated allergy	Either SPT or specific IgE	Not reported	DBPCFC	Italy	Female 23%	Children
Alessandri (2012 b)	Egg Whole egg	IgE mediated allergy	IgE and SPT	Anaphylaxis, Gastrointestinal, Oral allergy syndrome, Rhinitis, Respiratory Urticaria, Other: Worsening of eczema, conjunctivitis	DBPCFC	Italy Rome	Female 36%	Children 1-11y
Ammar	Milk/ Dairy	Both IgE and non	Either SPT or specific IgE	Gastrointestinal Other	Open challenge	France	Female 73%	Children 15 days –3

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Study ID	Food	Type of Allergy	Sensitisation	History of Symptoms	Challenge	Country	Sex	Age Range
(1999)		IgE mediated	13 positive SPT, 6 specific IgE Not reported	Failure to thrive				months
Ando (2008)	Egg	IgE mediated allergy	Specific IgE	Anaphylaxis Gastrointestinal Respiratory Atopic dermatitis/ eczema	DBPCFC	Japan	Female 41%	Children median 30, range 14-72 months
Ballmer-Weber (2002)	Celery	Both IgE and non IgE mediated	Not reported	Not reported	DBPCFC (12)	Switzerland	Female 58%	Adults Mean 27.9, SD ±7.3y (range 21-42 y)
Boyano Martinez (2001)	Egg	Both IgE and non IgE mediated	Specific IgE SPT	Gastrointestinal Oral allergy syndrome: Sneezing, nasal itching and or congestion. Respiratory Urticaria, Angioedema	Open challenge	Spain Madrid	Female 39.5%	Children 11– 24 months
Burks (2008)	Milk/ Dairy	IgE mediated allergy	Specific IgE SPT	Atopic dermatitis/ eczema	DBPCFC (Five Cow's milk +ve by DBPCFC; 24 +ve specific IgE not included in this review)	USA	Not reported	Children
Caffarelli (2002)	Milk/ Dairy	IgE mediated allergy	Specific IgE SPT	Respiratory Urticaria Atopic dermatitis/ eczema Angioedema	Open DBPCFC	Italy	Not reported	Children
Giampietro (2001)	Milk/ Dairy	IgE mediated allergy	Not reported	Urticaria Atopic dermatitis/ eczema	Open Elimination	Italy Sweden	Not reported	Children Mean 37 months
Hansen (2003)	Hazelnuts	IgE mediated allergy	Specific IgE CAP- raw or roasted. SPT Prick to prick- raw or roasted	Oral allergy syndrome 17/17 Systemic reaction 3/17	DBPCFC All except for one patient with a convincing clinical history	Denmark Switzerland	Female 65%	Adults and children median 24.5; range 14–65 years

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Study ID	Food	Type of Allergy	Sensitisation	History of Symptoms	Challenge	Country	Sex	Age Range
Host (1988)	Milk/ Dairy	Both IgE and non IgE mediated	Not reported	Gastrointestinal: Vomiting, Oral allergy syndrome: Allergic rhinitis, Respiratory: Asthma. Atopic dermatitis/ eczema, Urticaria	Open Elimination diet	Denmark Odense	Female 80%	Children 12-40 months
Hourihane (1997)	Peanut	IgE mediated allergy	Specific IgE SPT	Pruritus, urticarial, swollen lips, erythema, facial swelling, oedema, wheeze and anaphylaxis	Open and DBPCFC	UK	Female 78%	Adults and children Mean 26, range 14-48 years
Kaczmarski (2005)	Milk/ Dairy	IgE mediated allergy	Either SPT or specific IgE	Atopic dermatitis/ eczema	Open	Poland	Female 36%	Children mean 11.34 months, range 1 - 28 months
Kim (2011)	Milk/ Dairy Cows' milk	IgE mediated allergy	Either SPT or specific IgE	Not reported (Exclusion criteria: recent reaction to baked milk)	Open challenge	USA	Not reported	Children mean 6.6 y, range 2.1-17.3 y
Komata (2009)	Milk/ Dairy	IgE mediated allergy	Either SPT or specific IgE	Anaphylaxis 48.6 % Atopic dermatitis/ eczema 91.9 % Angioedema 91.9 %	Open challenge	Japan	Female 13.5 %	Children mean 63.2 years, SD (±6.3)
Lemon-Mule (2008)	Egg	IgE mediated allergy	either SPT or specific IgE	Not reported (exclusion criteria: recent reaction to extensively heated egg)	Open challenge	**US**A New York	Not reported	Children mean 6.9 y, range 1.6-18.6 y
Marseglia (2012)	Egg Lysozyme	IgE mediated allergy	Specific IgE		Open challenge			unclear
Niggeman (2008)	Milk/ Dairy	IgE mediated allergy	SPT	Yes, but not described	DBPCFC	Germany	Female 60%	Infants approx. 36 weeks old
Nowak-Wegrzyn (2008)	Milk/ Dairy	IgE mediated allergy	Specific IgE SPT	Not reported (exclusion criteria: recent	Open	USA New York	Not reported	Mean 7.5 y range 0.5 - 21 y

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Study ID	Food	Type of Allergy	Sensitisation	History of Symptoms	Challenge	Country	Sex	Age Range
				reaction to heated milk)				
Ragno (1993)	Milk/ Dairy	IgE mediated allergy	Either SPT or specific IgE	Gastrointestinal Respiratory: Asthma. Urticaria Angioedema	Open challenge	Italy	Not reported	Children 15 -76 months
Rugo (1992)	Milk/ Dairy	IgE mediated allergy	Specific IgE Fluorescence Immunoassay SPT	Gastrointestinal Respiratory Asthma Urticaria Atopic dermatitis/ eczema	Open challenge	Germany	Female 24%	Children Median 16 months, range 5 months - 9.5 y
Sampson (1991)	Milk/ Dairy	IgE mediated allergy	Specific IgE SPT	Gastrointestinal Oral allergy syndrome Respiratory Atopic dermatitis/ eczema Urticaria Other Skin rash	DBPCFC	USA	Not reported	Children 8 months - 9.5 y
Scibilia (2006)	Cereals Wheat	IgE mediated allergy	Not reported	Gastrointestinal Oral allergy syndrome Rhinitis Respiratory Atopic dermatitis/ eczema Urticaria. Exercise-induced Angioedema	DBPCFC	Denmark Italy	Female 67%	Adults 19-60y Children 14-16y
Urisu (1997)	Egg	IgE mediated allergy	Specific IgE	Anaphylaxis Erythema Gastrointestinal Respiratory Urticaria. Atopic dermatitis/ eczema	DBPCFC	Japan	Female 39%	Children 1–10 y
Worm (2009)	Hazelnut and mostly birch pollen sensitive	IgE mediated allergy	Specific IgE Phadia CAP SPT Prick to prick and ALK extract- raw and roasted	Oral allergy syndrome Systemic group no data Urticaria	DBPCFC	Germany (presumed)	Not reported	Not reported

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2.3.4. Processing methods

The summary of the processing methods are shown (Table 2.5), in study author order. Many of the methods used are relevant to commercial and home cooking such as baking milk within a muffin. For the comparison food we listed the form of the food that we considered being least processed. In most cases, this was listed by the authors as being the more allergenic form as exemplified by raw egg, compared to egg baked within a cake. The study authors reported wet weight or volume rather than standardizing challenges by dry weight or protein concentration.

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Table 2.5: Method of processing comparison and test food. Wet weight indicates the weight of the processed or unprocessed food was used without adjusting for the moisture or protein content (alphabetical order by author).

Study ID	Comparison processing method	Comparison supplier	Comparison variety	Comparison quantified by	Test processing method	Test brand and supplier	Test variety	Test quantified by
Alessandri (2012 b)	Raw **Prepared by** Laboratory prepared	Not reported	Unclear	Other 1 whole egg	**Test 1** Boiled 10 min **Prepared by** Laboratory prepared	Not reported	Unclear	Dry weight 1 whole egg
Alessandri (2012a)	Pasteurised **Prepared by** Unclear	Not reported	Unclear	Wet weight	Cheese Parmigiano-Reggiano (PR) **Prepared by** Unclear	Not reported	Parmigiano-Reggiano	Wet weight or volume 200 ml Cow's milk = 13.3 g PR
Ammar (1999)	Hydrolysed A range of products **Prepared by** Shop bought	Not reported	Not reported	Not stated	Amino acid–based formulas (AAFs) Neocate **Prepared by** Shop bought	**Test 1** Neocate	Not reported	Not reported
Ando (2008)	Raw **Prepared by** Laboratory prepared Raw liquid egg white freeze dried, homogenised	Not reported	Unclear	Wet weight Highest possible dose Equivalent to one egg	Extensively heated Liquid egg white, 90 ˚C for 60 min, freeze dried milled	Not reported	Unclear	Wet weight or volume One egg
Ballmer-Weber (2002)	Raw **Prepared by** Laboratory prepared	Unclear	Unclear	Wet weight 20 g raw added to the drink (1 ml of drink contains 0.144 g raw celery)	**Test 1** Baked, 110 ˚C for 15 min. Small additional open challenge of samples cooked for 7.45, 13.12, 23.64, 76.07 min at 100 ˚C **Prepared by** Laboratory prepared	**Test 1** Dr N Sauerwald (Nestle, Frankfurt, Germany)	Unclear	Wet weight or volume

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Study ID	Comparison processing method	Comparison supplier	Comparison variety	Comparison quantified by	Test processing method	Test brand and supplier	Test variety	Test quantified by
					Test 2 Dried and pulverized Celery spice	**Test 2** Dr N Sauerwald (Nestle, Frankfurt, Germany)		
Boyano Martinez (2001)	Raw egg whites **Prepared by** Not reported	Not reported	Not reported	Wet weight	**Test 1** Boiled 10 min **Prepared by** Not reported	Not reported	Unclear	Wet weight or volume
Burks (2008)	Pasteurised **Prepared by** Unclear	Not reported	Unclear	Wet weight	**Test 1** Amino acid–based formulas (AAFs) **Prepared by** Unclear **Test 2** Extensively hydrolysed formula (EHF) **Prepared by** Laboratory prepared	**Test 1** Neocate **Test 2** Mead Johnson Nutritionals, Evansville, Indiana	Not applicable Not applicable	Wet weight
Caffarelli (2002)	Pasteurised **Prepared by** Laboratory prepared	Not reported	Not reported	Wet weight	**Test 1** Whey partially hydrolysed Humana **Test 2** Extensively hydrolysed whey formula (EHF) Hypolac **Test 3** Extensively hydrolysed casein.	**Test 1** Humana, Milano, Italy. **Test 2** ALK, Lainate, Milano, Italy **Test 3** Nutramigen, Mead Johnson, Roma, Italy.		Wet weight or volume

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Study ID	Comparison processing method	Comparison supplier	Comparison variety	Comparison quantified by	Test processing method	Test brand and supplier	Test variety	Test quantified by
					Test 4 Amino acid derived formula Nutri-junior,	**Test 4** Nutricia, Milano, Italy		
Giampietro (2001)	Pasteurised **Prepared by** Laboratory prepared	Not reported	Unclear	Wet weight	**Test 1** Nutrilon Pepti extensively hydrolysed whey **Test 2** Profylac Extensively hydrolysed whey **Test 3** Nan HA Partial whey hydrolysate	Nutricia, Zoetermeer, Netherlands ALK, Horsholm, Denmark Nestlé, Vevey, Switzerland	Unclear	Wet weight or volume
Hansen (2003)	Raw **Prepared by** Shop bought	Sorematec, Arlon-Schoppach, Belgium	Piemonte	Wet weight	**Test 1** Roasted, (140 ˚C, 40 min) **Prepared by** Shop bought	**Test 1** Sorematec, Arlon-Schoppach, Belgium.	Piemonte	Wet weight or volume
Host (1988)	Raw **Prepared by** Laboratory prepared	J.Kollerup, Enigheden Dairy Copenhagen, Denmark	Unclear Not reported	Wet weight Volume	**Test 1** Pasteurised Cow's Milk **Prepared by** Laboratory prepared **Test 2** Homogenised and pasteurised cow's milk	**Test 1** J.Kollerup, Enigheden Dairy Copenhagen, Denmark **Test 2** J. Kollerup, Enigheden Diary. Copenhagen, Denmark	Unclear Not reported	Wet weight or volume
Hourihane (1997)	Roasted, salted peanuts	KP Foods, Leicester	Not reported	Nuts	**Test 1** Refined peanut oil	Random batches of oil supplied by the Seed Crushers' and	Unclear	Wet weight

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Study ID	Comparison processing method	Comparison supplier	Comparison variety	Comparison quantified by	Test processing method	Test brand and supplier	Test variety	Test quantified by
					Test 2 Crude peanut oil	Oil Processors' Association.		
Kaczmarski (2005)	Low lactose cow's milk **Prepared by** Not reported	Bebilon Nutricia	Not reported	Wet weight	**Test 1** Casein extensively hydrolysed **Test 2** Whey extensively hydrolysed	**Test 1** Nutramigen, Mead Johnson **Test 2** Bebilon Pepti 1 or 2, Nutricia		Wet weight or volume
Kim (2011)	Pasteurised **Prepared by** Shop bought	Unclear	Unclear	Wet weight	**Test 1** Baked Muffin with 1.3 g of milk protein. Baked at 350 ˚C for 30 min. Also Baked cheese within a Pizza **Prepared by** prepared in house	Unclear	Unclear	Wet weight
Komata (2009)	Pasteurised	Unclear	Unclear	Volume	**Test 1** Extensively heated	Unclear	Unclear	Volume
Lemon-Mule (2008)	Heated Scrambled/French toast **Prepared by** Unclear	Not reported	Not reported	Volume	**Test 1** Extensively heated **Prepared by** Unclear	Not reported	Not reported	Volume
Marseglia (2012)	Unclear Egg not clear if raw or cooked	Not reported	Not reported	Volume	**Test 1** Cheese Granda Padano 12 month matured **Test 2** Cheese Granda Padano 24 month matured			Wet weight
Niggeman (2008)	Pasteurised **Prepared by** Shop bought	Not reported	Not reported	Volume	**Test 1** extensively hydrolysed ultra	**Test 1** Althera, Nestle´, Switzerland	Not reported	Volume

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Study ID	Comparison processing method	Comparison supplier	Comparison variety	Comparison quantified by	Test processing method	Test brand and supplier	Test variety	Test quantified by
					filtered whey **Test 2** Amino acid based	**Test 2** Neocate, SHS, UK		
Nowak-Wegrzyn (2008)	Pasteurised **Prepared by** Shop bought	Nestle Carnation, Glendale, California	Non-fat	Wet weight	**Test 1** Extensively heated **Prepared by** In house	Nestle Carnation, Glendale, California	Non-fat	Wet weight
Ragno (1993)	Pasteurised **Prepared by** Unclear			Wet weight	**Test 1** Casein hydrolysed **Test 2** Whey extensively hydrolysed **Test 3** Whey partially hydrolysed	**Test 1** Alimentum,Ross **Test 2** Profylac, ALK **Test 3** Nidina HA, Nestle		Wet weight or volume
Rugo (1992)	Pasteurised **Prepared by** Not reported	Not reported	Not reported	Wet weight ml Highest possible dose 40ml	**Test 1** Casein hydrolysate **Test 2** Casein hydrolysate **Test 3** Whey hydrolysate **Test 4** Whey hydrolysate **Test 5** Whey hydrolysate Ultrafiltered	**Test 1** Nutramigen **Test 2** Pregestimil **Test 3** Alfaré **Test 4** Beba HA **Test 5** Ultrafiltered		Dry weight g/ml dissolved hydrolysate
Sampson (1991)	Pasteurised Prepared by Unclear	Not reported		Wet weight	**Test 1** Casein hydrolysed **Placebo** Casein extensively hydrolysed	**Test 1** Alimentum, Ross Laboratories, Columbus, Ohio **Placebo**		Wet weight or volume

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Study ID	Comparison processing method	Comparison supplier	Comparison variety	Comparison quantified by	Test processing method	Test brand and supplier	Test variety	Test quantified by
						Nutramigen, Mead Johnson,		
Scibilia (2006)	Raw Prepared by Laboratory prepared	Not reported	Variety Unclear	Dry weight 25g	**Test 1** **Boiled** No details on how the wheat was cooked **Prepared by** Laboratory prepared	Not reported	Unclear	Dry weight 25g
Urisu (1997)	Raw Freeze-dried egg white Prepared by Laboratory prepared	Not reported	Unclear	Dry weight	**Test 1** Extensively heated 90 ˚C for 60 m **Prepared by** Laboratory prepared **Test 2** Heated ovomucoid depleted **Prepared by** Laboratory prepared	Not reported	Unclear	Dry weight
Worm (2009)	Raw finely ground Prepared by Unclear	Unclear	Unclear	Wet weight	**Test 1** Roasted, 144 ˚C **Prepared by** laboratory **Test 2** HN flour Capsule		Unclear	Wet weight or volume

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2.3.5. Challenge procedure

The challenge procedures are summarised (Table 2.6). In those studies that carried out a DBPCFC the method of masking and the procedure for randomisation was not clearly reported in many of the studies. Although many studies reported the method of masking and the recipe, they did not report whether this was taste tested. The method of generating the random sequence, the ratio of active to placebo challenge and the way in which the sequence was concealed from the participants and the study personnel during the challenge and while the symptoms were assessed was not described in the majority of studies. We attempted to record how the study authors dealt with positive reactions to placebo; however in the majority of instances this was not reported. If the test food challenge procedure differed significantly for the comparison food then this was shown (Table 2.7).

Studies that reported their challenge methods in detail include Alessandri (2012 b), Hansen (2003) and Sampson (1991).

Table 2.6: Challenge procedure for comparison food (study author order)

Study ID	Avoid foods prior to challenge	Masking	Was the sequence placebo : active random	Random sequence concealment	Doses	Outcome description
Alessandri (2012 b)	Yes	Taste tested: unclear	Random	Concealed	0.1, 0.5, 2, 10, 50 ml up to 1 egg equivalent to 6 g egg protein Time delay: 20 min **Method** Ingestion	Positive response local non-objective restricted to area in contact with allergen, oral allergy syndrome or isolated digestive complaints, systemic objective, e.g. urticaria, asthma, or anaphylaxis Dose response within 6 hours of the first dose **Handling of positive placebo** Not reported
Alessandri (2012a)	Yes	Taste tested: unclear	Random	Unclear	0.05, 0.15, 0.3, 1, 3, 10, 30, 50, 100, 195 ml Time delay: 20 min	Positive response **Handling of positive placebo** Not reported
Ammar (1999)	Not reported	Taste tested: unclear Open challenge			1, 5, 80 ml Time delay: unclear **Method** Ingestion	Positive response Any reaction also any delayed reaction **Handling of positive placebo** Not reported
Ando (2008)	Not reported	Taste tested: Unclear Placebo: glucose in the	Unclear	Unclear	0.1 ml, 1 ml, 10 ml and then the remainder of the egg white Time delay: 30 min	Positive response Continued until objective symptoms developed or entire challenge dose ingested. **Handling of positive placebo**

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Study ID	Avoid foods prior to challenge	Masking	Was the sequence placebo : active random	Random sequence concealment	Doses	Outcome description
		same juice as the active			**Method** Ingestion	Not reported
Ballmer-Weber (2002)	Not reported	Taste tested: Unclear	Unclear	Unclear	0.7 g, 28.5 g Time delay: Unclear **Method** Ingestion	Positive response Oral allergy syndrome, dyspnoea, rhinitis, conjunctivitis, flush, vertigo, angioedema. Dose response **Handling of positive placebo** All placebo negative
Boyano Martinez (2001)	Not reported	Taste tested: unclear Open challenge	Not applicable	Not concealed	1/8 , 1/4 and 1/2 of the egg white Time delay 90 min **Method** Ingestion	Positive response Objective symptoms
Burks (2008)	Not reported	Taste tested: unclear	Unclear	Unclear	Time delay: unclear **Method** Ingestion	Positive response **Handling of positive placebo** Not reported
Caffarelli (2002)	Yes, 3 months before	Taste tested: unclear	Random Ingestion	Unclear	Time delay: 20 min **Method** Ingestion	A positive response **Handling of positive placebo** Not reported
Giampietro (2001)	Not reported	Not taste tested	Random 'the order was randomised'	Concealed blind	0.2, 2, 20, 50 and 150 ml Time delay: 20 min **Method** Ingestion	Positive response Urticaria, asthma, gastrointestinal, rhinitis and erythema and itch. **Handling of positive placebo** Not reported
Hansen (2003)	Not reported	Taste tested: unclear	Random 'the challenge order was randomized'	Concealed A dietician prepared the foods, code not broken until complete	Max. Dose Copenhagen 10 g, Zurich 18.2 g **Method** Ingestion	Positive response **Handling of positive placebo** Not reported
Hourihane (1997)	Yes	Open challenge	na	na	labial challenge, followed by ¼ ½ 1 peanut up to 32 peanuts Time interval 10-15 min	Positive response Objective symptoms

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Study ID	Avoid foods prior to challenge	Masking	Was the sequence placebo : active random	Random sequence concealment	Doses	Outcome description
					Method Ingestion	
Host (1988)	Yes a milk free diet 4 weeks prior to chall-enge	Not taste tested	Random	Concealed	5, 10, 20, 40, 80, up to 160 ml Time delay: 2h **Method** Ingestion	Positive response a) the child displayed definitive allergic reactions in-keeping with the child's history of CMA. b) The provoked symptoms disappeared after withdrawal of the milk preparation in question or c) coincidental infection could be excluded
Kaczmarski (2005)	Not reported	Not taste tested open challenge	na	na	0.1, 1, 3, 10, 30, 50, 100 ml according to age Time delay: unclear **Method** Ingestion	Positive response. If infants did not show a positive response they were fed the milk for up to 2 weeks.
Kim (2011)	Yes	Open challenge	na	na	**Method** Ingestion	Positive response
Komata (2009)	Unclear	Open challenge	Unclear	Unclear	**Method** Ingestion	Positive response
Lemon-Mule (2008)	Not reported	Open challenge	na	na	Not reported Time delay: unclear **Method** Ingestion Objective symptoms	Positive response Objective symptoms
Marseglia (2012)	Unclear	Comparison-open challenge	NA	na	**Method** Ingestion description Oral provocation test (University of Pavia, number 2-2009)	Positive response
Niggeman (2008)	Yes	DBPCFC	Yes	Unclear	**0.1,0.3, 1.0, 3.0, 10.0, 30.0, and 100.0 ml** **Method** Ingestion	Positive response Growth rates **Handling of positive placebo** Not described

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Study ID	Avoid foods prior to challenge	Masking	Was the sequence placebo : active random	Random sequence concealment	Doses	Outcome description
Nowak-Wegrzyn (2008)	Not reported	Comparison-open challenge	na	na	**Method** Ingestion	Positive response Objective symptoms
Ragno (1993)	Not reported	Not taste tested Comparison- open challenge	Random	Concealed	A drop of the inner lower lip then 5 ml then 100ml. Time delay 30 min - 1 week gap between challenges **Method** Ingestion Rubbing on lips	Positive response Objective symptoms **Handling of positive placebo** Not described
Rugo (1992)	Yes Milk/dairy elimination diet for at least one week prior	Comparison- open challenge	Objective symptoms		0.2ml - 1ml - 2ml - 5ml - 10ml - 20ml Time delay 20-30 min **Method** Objective symptoms	Positive response Challenges were stopped once symptoms occurred or at highest dose. Immediate and late (up to one hour recorded) Dose response
Sampson (1991)	Not reported	Not taste tested Comparison- open challenge	Random	Concealed	Up to 10 g in 100ml of formula in a period of 60 - 90 minutes. Each challenge was initiated with 5ml formula. Time delay 15 minutes	Positive response **Handling of positive placebo** No positive reactions to placebo
Scibilia (2006)	Yes 1 week prior	Taste tested: unclear Comparison- open challenge	Unclear	Unclear	Cumulative dose schedule: 100 mg, 600 mg, 1.6 g, 3.1 g, 6.1 g, 12.1 g, 25 g Time delay 20 min **Method** Ingestion	Positive response Dose response **Handling of positive placebo** No positive reactions to placebo
Urisu (1997)	Not reported	Taste tested: unclear	Unclear	Unclear	Increasing to 8 g Time delay 30 minutes	Positive response
Worm (2009)	Yes 1 week	Taste tested: unclear	Unclear	Unclear	0.01–0.02–0.03–0.05–0.1–0.2–0.4–1.0–2.5–5.0– 10.0 g. Time delay 15 min	Positive response % Dose response Single amount of HN eliciting symptoms

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Table 2.7: Challenge procedures for test food (alphabetical order by author) if different to comparison food

Study ID	Avoid foods prior to challenge	Masking	Was the sequence placebo : active random	Random sequence concealment	Doses	Outcome description
Alessandri (2012a)	Yes	Open challenge carried out	na	na	0.003, 0.01, 0.02, 0.07, 0.2, 0.7, 2.0, 3.03, 6.07, 13.0 g time delay 20 min **Method** Ingestion	Not defined
Ammar (1999)	Unclear	Open challenge carried out	na	na	**Method** Ingestion Used as infant formula	Not define
Burks (2008)	Avoided food prior to challenge	Unclear if taste tested Open challenge carried out	Unclear	Unclear	Unclear DBPCFC followed by open feeding for up to 7d **Method** Ingestion Extended over 7d	Convincing reaction **Positive reactions to placebo:** Not stated
Hourihane (1997)	Yes	DBPCFC Taste tested	Sequence devised by another member of the team	Dietitian made up the foods for challenge	labial challenge, followed by ¼ ½ 1 peanut up to 32 peanuts Time interval 10-15 min **Method** Ingestion	Positive response Objective symptoms
Marseglia (2012)	Not stated				0.5, 1, 2, 4, 8, 14.5 g time delay between doses: 20 min	
Niggeman (2008)	Yes	DBPCFC Unclear if taste tested	Yes	Unclear	Allocated to either amino acid or hydrolysed milk diet	Any reaction
Nowak-Wegrzyn (2008)	Not reported	Open food challenge	Na	na	**Method** Ingestion ¼ portions muffin (1.3 g milk protein) over 1h. If no symptoms within 2 hr a waffle given **Method** Ingestion	Positive response Objective symptoms

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Study ID	Avoid foods prior to challenge	Masking	Was the sequence placebo : active random	Random sequence concealment	Doses	Outcome description
Sampson (1991)	Not reported	Not taste tested (Comparison- open challenge)	Random	Concealed	Up to 10 g in 100ml of Nutramigen in a period of 60 - 90 min. Each challenge was initiated with 5ml formula. Time delay 15 minutes	Positive response
Scibilia (2006)	Yes 1 week prior	Taste tested: unclear DBPCFC	Unclear	Unclear	Cumulative dose schedule: 100 mg, 600 mg, 1.6 g, 3.1 g, 6.1 g, 12.1 g, 25 g Time delay 20 min **Method** Ingestion	Positive response Dose response **Handling of positive placebo** No positive reactions to placebo
Worm (2009)	Avoided food prior to challenge	Not taste tested	Unclear	Unclear	0.01–0.02–0.03–0.05–0.1–0.2–0.4–1.0–2.5–5.0– 10.0 g doses initiation dose dependent on response to raw Time delay between doses:15 min	**Challenge halted when:** Not stated **Positive reactions to placebo** Not stated

2.3.6. Study design

The study designs are shown (Table 2.8). Within a number of studies that used a cross over design the order of receiving the different types of foods was fixed, studies with this fixed order can be grouped into two categories:

- participants received the form of the food thought to the least allergenic first (either open or by DBPCFC). If a participant demonstrated symptoms they were not challenged to the second food. This was because a positive response was assumed and further challenge thought unethical;

- participants were challenged with the form thought to be more allergenic first to confirm food allergy, only those that were challenge positive had the second challenge with the processed food e.g. Niggemann (2008), first challenged the infants to pasteurized milk (using DBPCFC) and subsequently carried out a randomised cross over study comparing two processed formulas to test allergenicity and the effect on growth.

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Table 2.8: Study design and outcome assessment (alphabetical order by author)

Study ID	Overarching design	Order of challenge comparison/test	Outcome results
Alessandri (2012 b)	Cross over non random	Not random Time delay between different foods, given on separate days Challenge carried out first with boiled egg those that were positive were not challenged to raw egg as assumed positive.	Any reaction
Alessandri (2012a)	Cross over non random	Not random, milk given first	Any reaction
Ammar (1999)	Cross over non random	Not random, all challenged with hydrolysate after a diet of Neocate	Any reaction
Ando (2008)	Cross over non random	Not random, heated egg challenge carried out first, those that were negative were challenged to raw egg, those positive to heated egg were not challenged to raw egg as they were assumed to be positive.	Any reaction
Ballmer-Weber (2002)	Cross over non random	Unclear	Any reaction **Dose reaction**
Boyano Martinez (2001)	Cross over non random	Not random Cooked egg white given first for 45 participants, only those negative to cooked egg were challenge with raw. Another 10 were challenged with raw only, as they stated they tolerated cooked egg.	Any reaction
Burks (2008)	Cross over non random (study 2 only)	Not random	Any reaction
Caffarelli (2002)	Cross over non random	Not random	Any reaction
Giampietro (2001)	Cross over random	Random	Any reaction
Hansen (2003)	Cross over non random	Not random Zurich: raw challenge first, time delay between different foods was one year Copenhagen: random, time delay between different foods was different days	Any reaction
Host (1988)	Cross over random	Random	Any reaction Dose of reaction
Hourihane (1997)	Cross over non-random Cross over random	Non random Crude and refines oil first then roasted peanut Random 'random order determined by a member of staff not involved in the evaluation of the subject'	Any reaction Dose of reaction
Kaczmarski (2005)	Cross over non random	Not random Low lactose cow's milk first, however not clear on order of EHC or EHW	Any reaction

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Study ID	Overarching design	Order of challenge comparison/test	Outcome results
Kim (2011)	Cross over non random	Not random Time delay between different foods 6 months	Any reaction
Komata (2009)	Cross over non random	Not random, Unheated milk given first then given heated milk	Any reaction
Lemon-Mule (2008)	Cross over non random	Not random, extensively heated egg was given first, however time delay between different foods was not reported	Any reaction Dose of reaction
Marseglia (2012)	Cross over non random	Time delay between different foods Egg challenge was done previous year. Cheese challenges carried out at least 48h apart	Any reaction
Niggemann (2008)	Cross over non random	All challenged with pasteurised milk first then given extensively hydrolysed or amino acid based challenge in random order	Any reaction
Nowak-Wegrzyn (2008)	Cross over non random	None random, assumed same day.	Any reaction.
Ragno (1993)	Cross over random	Random	Any reaction
Rugo (1992)	Cross over non random	Not random, comparison food given first	Any reaction.
Sampson (1991)	Cross over random	Random	Any reaction
Scibilia (2006)	Cross over non random	Unclear	Any reaction
Urisu (1997)	Cross over non random	Not random. Ovomucoid depleted, heated then raw	Any reaction
Worm (2009)	Between group comparison non random	Not random. All (90) were challenged to raw given first, a non-random subset (20) were challenged to roasted.	Any reaction Dose of reaction

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2.3.7. The Quality of studies

The quality of diagnosis for most studies

Table 2.9: Quality of Studies (alphabetical order by author)

Study ID	Diagnosis*	Challenge order comp/test*	Sampling*	Severe allergy represented*
Alessandri (2012 b)	Very low risk of bias	High risk of bias	High risk of bias Specially selected for being negative to the test food	High risk of bias
Alessandri (2012a)	Very low risk of bias	High risk of bias	High risk of bias	High risk of bias
Ammar (1999)	Low risk of bias	High risk of bias	High risk of bias Specially selected for being positive to comparison food	High risk of bias
Ando (2008)	Low risk of bias	High risk of bias	High risk of bias Specially selected for being negative to the test food	High risk of bias
Ballmer-Weber (2002)	Very low risk of bias	Unclear	High risk of bias	High risk of bias
Boyano Martinez (2001)	Low risk of bias	High risk of bias	High risk of bias	High risk of bias
Burks (2008)	Very low risk of bias	High risk of bias	High risk of bias	Unclear risk of bias
Caffarelli (20020	Low risk of bias	High risk of bias	High risk of bias	High risk of bias
Giampietro (2001)	Low risk of bias	Low risk of bias	High risk of bias	High risk of bias
Hansen (2003)	Very low risk of bias	High risk of bias	Unclear	High risk of bias
Host (1988)	Low risk of bias	Low risk of bias	High risk of bias	High risk of bias
Hourihane (1997)	Low risk of bias	Low risk of bias (between oils)	High risk of bias	High risk of bias
Kaczmarski (2005)	Low risk of bias	High risk of bias	High risk of bias	High risk of bias
Kim (2011)	Low risk of bias	High risk of bias	High risk of bias Specially selected for being negative to the test food	High risk of bias
Komata (2009)	Low risk of bias	High risk of bias	Low risk of bias	Low risk of bias
Lemon-Mule (2008)	Low risk of bias	High risk of bias	High risk of bias	High risk of bias
Marseglia (2012)	Low risk of bias	High risk of bias	High risk of bias	High risk of bias
Niggeman (2008)	Very low risk of bias	High risk of bias	High risk of bias	Unclear
Nowak-Wegrzyn (2008)	Low risk of bias	High risk of bias	High risk of bias	High risk of bias

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Study ID	Diagnosis*	Challenge order comp/test*	Sampling*	Severe allergy represented*
Ragno (1993)	Low risk of bias	High risk of bias	High risk of bias	High risk of bias
Rugo (1992)	Low risk of bias	High risk of bias	High risk of bias	High risk of bias
Sampson (1991)	Very low risk of bias	Low risk of bias	High risk of bias	High risk of bias
Scibilia (2006)	Low risk of bias	Unclear	High risk of bias	High risk of bias
Urisu (1997)	Low risk of bias	High risk of bias	High risk of bias	High risk of bias
Worm (2009)	Low risk of bias	High risk of bias	Unclear	High risk of bias

For the method of assessing the quality of studies see Table 2.2

2.3.8. Findings on effect of processing on allergenicity

The table below (Table 2.10) shows the findings for all studies in food order. The percentage of participants tested showing a positive response are shown together with the threshold dose that elicited a reaction if provided. We excluded data from participants that were challenge negative to both comparison and test food. In the majority of cases, the least processed food provided the greater response rate i.e. was the more allergenic.

2.3.8.1. Celery

The one study that investigated celery, Ballmer-Weber (2002), demonstrated that for approximately half of those who reacted to raw celery challenges were negative for celery cooked at 100 ^{o}C for 15 minutes (Table 2.10). In the four cases that reacted to both raw and cooked celery the threshold dose was increased by heating in all cases. The trend seems to be decrease but not elimination in allergenicity with heat, however the small sample size and the non-representative sample make it difficult to generalise the findings to the wider population. Of the five tested, all reacted to the celery spice, three of these had negative responses to the cooked celery.

2.3.8.2. Cows' milk

We included four studies, a total of 121 participants, in which challenges were performed to amino acid based formulas with infants who were challenge positive to cow's milk, Alessandri (2012 a), Ammar (1999), Burks (2008), Caffarelli (2002), Niggemann (2008), (Table 2.10). All participants were negative except for two, in Caffarelli (2002), who were skin prick test and specific IgE negative to the formula and developed eczema more than 12 hours after the challenge.

Of the included studies, five, Caffarelli (2002), Kaczmarski (2005), Ragno (1993), Rugo (1992), and Sampson (1991) investigated hydrolysed casein formulas in a total of 119 participants, who were challenge positive to cows' milk. Studies showed that between zero to 35 % of the sample populations, a total of 20 participants were challenge positive to the hydrolysed casein formulas. In the study showing the highest reactivity (17/48), the inclusion criteria was atopic eczema or dermatitis and the challenge positive showed symptoms such as dermatitis, gastrointestinal or irritability and the challenge was carried out over a prolonged period.

Of the included studies, six investigated hydrolysed whey based formulas Caffarelli (2002), Giampietro (2001), Kaczmarski (2005), Niggemann (2008), Ragno (1993), Rugo (1992) in a total of 156 children who were proven cow's milk allergic by challenge. The proportion positive to whey derived formulas ranged from zero to nearly 35%. The formula providing the greatest reduction in allergenicity was the extensively hydrolysed, ultra filtered formula, tested by Niggemann (2008), and formulas giving the higher proportion of reactive infants were for the partially hydrolysed formulas, 36% Giampietro (2001)

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and 25% Caffarelli (2002). In Caffarelli (2002), the same participants were exposed to hydrolysed whey and extensively hydrolysed casein giving percentage responders of 25 and 5% respectively.

Only one study Host (1988) investigated pasteurization, and there was limited evidence (5 participants) that there was no effect on allergenicity. Kim (2011), Komata (2009) and Nowak-Wegrzyn (2008) studied the effect of heating on milk allergy. They showed that a proportion of those allergic to pasteurized milk are tolerant to heated or baked milk. However, Kim (2011) and Nowak-Wegrzyn (2008) selected for those who thought they had developed tolerance to baked milk and they still found positive challenge result (26% and 36% respectively). These participants did not go on to be challenged with uncooked milk, as a positive response was assumed. The selection criteria of Kim (2011) and Nowak-Wegrzyn (2008) make it impossible for us to generalize the findings a wider population. A much higher percentage (94.7%) of participants in the study by Komata (2009) reacted to heated milk. These participants were selected as they were hospitalized and a relatively high proportion (48.6%) had reported anaphylaxis to milk.

One study Alessandri (2012) looked at the effect of cheese making (Parmigiano-Reggiano) on the allergenicity of cow's milk. Of the 50 participants that had positive challenges to cows' milk, only 42% reacted to the matured hard cheese. The study authors analysis of specific IgE binding *in vitro* indicated that the partial breakdown of casein in the cheese making process could account for the decrease in reactivity by participants in the challenge. Beta-lacto globulin was unaffected by the cheese making process. Those found to be tolerant in challenge were advised not to avoid Parmigiano-Reggiano cheese, and after a two year follow up no adverse events were recorded.

2.3.8.3. Egg

The effect of heating on egg allergy was studies by Alessandri (2012 b), Ando (2008), Boyano Martinez (2001), Lemon-mule (2008) and Urisu (1997) in a total of 146 participants (Table 2.10). Between 10.6 % and 57.6 % of egg challenge positive participants reacted to extensively heated egg. In the lowest percentage study, Lemon-Mule (2008), study participants who had reported recent reactions to heated egg were excluded from the study, so this is a biased estimate. One study, Urisu (1997) additionally investigated ovomucoid-depleted egg and found that of the 36 participants that were egg challenge positive, only 2.6% reacted to the heated ovomucoid depleted egg compared to 44.7% that reacted to the heated egg.

The effect of cheese processing on the allergenicity of egg lysozyme was tested by one study, Marseglia (2012) in 21 participants, in which cheese matured for 24 months was found to be less allergenic than 12 month matured cheese.

2.3.8.4. Tree nuts

There were only two studies that used challenge to investigate heat on tree nuts and they both studies hazelnut Hansen (2003) and Worm (2009). Both found that roasting reduced allergenicity in terms of the percentage responding (29.4 and 85% respectively) and in addition roasting seemed to increase the threshold dose to elicit a reaction (Table 2.10).

2.3.8.5. Wheat

One study, Scibilia (2006), looked at boiling wheat in 10 participants and found no reduction in allergenicity (Table 2.10)

2.3.8.6. Peanut

No studies were found that compared the challenge responses to peanut processed using different methods. The studies on allergenicity of different peanut preparations investigated IgE binding or other *in vitro* methods. We also searched for studies that looked at challenges with peanut oil in those with

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proven peanut allergy. One study, Hourihane (1993) investigated whether people with allergy to peanut would react to crude peanut oil. In this study 10% of those tested had positive challenge results to the crude peanut oil (Table 2.10) and non to the refined peanut oil.

Table 2.10: Allergenicity of processed foods (alphabetical order by food)

Paper ID	Food	Number showing positive response		Threshold dose Mean (±SD)	
		Comparison food	Test food challenge	Comparison food	Test food

Ballmer-Weber 2002	Celery	Raw
		9/9 (100 %)
		
		Raw
		10/10 (100%) §
			Heated
			4/9 (44.4%)
			
			Heated
			6/11 (54.5%) §
				Raw (all)
				n =10;
				9.0 (±13.4) g
				Raw (paired)
				n =4;
				0.7 (±0.0) g
					Heated (all)
					n=6;
					6.8 (±13.6) g
					Heated (paired)
					n=4;
					9.5 (±14.4) g

		Raw 5/5 (100 %) Raw 10/10 (100%) §	Spice 5/5 (100 %) Spice 5/5 (100 %) §		
Alessandri (2012 a) milk	Cows' milk	Pasteurised 50/50 (100 %)	Cheese (Parmigiano-Reggiano) 21/50 (42 %)		
Ammar 1999	Cows' milk-AA	Hydrolysate 30/30 (100 %)	Amino acid based (Neocate) 0/30 (0 %)		
Burks 2008	Cows' milk-AA	Pasteurised 5/5 (100 %)	Amino acid based (Neocate) 0/5 (0 %)		
Caffarelli (2002)	Cows' milk-AA	Pasteurised 20/20 (100 %)	Amino acid based (Nutri-junior) 2/20 (10 %)		
Niggemann 2008	Cows' milk-AA	Pasteurised 66/66 (100%)	Amino acid based formula (Neocate) 0/66 (0%)		
Caffarelli (2002)	Cows' milk-casein	Pasteurised 20/20 (100 %)	Casein extensively hydrolysed (Nutramigen) 1/20 (5 %)		
Kaczmarski 2005	Cows' milk-casein	Low lactose (Bebilon) 48/48 (100 %)	Casein extensively hydrolysed (Nutramigen) 17/48 (35.4 %)		
Ragno (1993)	Cows' milk-casein	Pasteurised 20/20 (100 %)	Casein extensively hydrolysate (Alimentum) 2/20 (10 %)		
Rugo (1992)	Cows' milk-casein	Pasteurised 8/8 (100 %)	Casein extensively hydrolysed (Nutramigen) 0/8 (0 %)		

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Paper ID	Food	Number showing positive response		Threshold dose Mean (±SD)	
		Comparison food	Test food challenge	Comparison food	Test food
Rugo (1992)	Cows' milk-casein	Pasteurised 8/8 (100 %)	Casein extensively hydrolysed (Pregestimil) 0/8 (0 %)		
Sampson (1991)	Cows' milk-casein	Pasteurised 23/23 (100 %)	Casein extensively hydrolysed (Alimentum) 0/23 (0 %)		
Caffarelli (2002)	Cows' milk -whey	Pasteurised 20/20 (100 %)	Whey partially hydrolysed (Humana) 5/20 (25 %)		
Caffarelli (2002)	Cows' milk-whey	Pasteurised 20/20 (100 %)	Whey extensively hydrolysed (Hypolac) 3/20 (15 %)		
Giampietro (2001)	Cows' milk-whey	Pasteurised 31/31 (100 %)	Whey extensively hydrolysed (Nutrilon Pepti) 6/31 (19.4 %)		
Giampietro (2001)	Cows' milk-whey	Pasteurised 26/26 (100 %)	Whey extensively hydrolysed (Profylac) 2/26 (7.7 %)		
Giampietro (2001)	Cows' milk-whey	Pasteurised 26/26 (100 %)	Whey partially hydrolysed (Nan HA/Nidina) 9/26 (34.6 %)		
Kaczmarski 2005	Cows' milk-whey	Low Lactose (Bebilon) 19/19 (100 %)	Whey extensively hydrolysed (Bebilon pepti) 4/19 (21.1 %)		
Niggemann 2008	Cows' milk-whey	Pasteurised 66/66 (100%)	Whey extensively hydrolysed ultra filtered (Althera) 0/66 (0%)		
Ragno (1993)	Cows' milk-whey	Pasteurised 20/20 (100 %)	Whey extensively hydrolysate (Profylac) 2/15 (13.3 %)		
Ragno (1993)	Cows' milk-whey	Pasteurised 20/20 (100 %)	Whey partially hydrolysate (Nidina) 9/20 (45 %)		
Rugo (1992)	Cows' milk-whey	Pasteurised 8/8 (100 %)	Whey hydrolysed (Alfare) 2/8 (25 %)		
Rugo (1992)	Cows' milk-whey	Pasteurised 8/8 (100 %)	Whey hydrolysate (Beba HA) 5/8 (62.5 %)		

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Paper ID	Food	Number showing positive response		Threshold dose Mean (±SD)	
		Comparison food	Test food challenge	Comparison food	Test food
Rugo (1992)	Cows' milk-whey	Pasteurised 8/8 (100 %)	Whey hydrolysate (Ultrafiltered) 4/8 (50 %)		
Host 1988	Cows' milk-heat	Raw 5/5 (100 %)	Pasteurised 5/5 (100 %)	Raw n =5; 41g (±38.9g)	Pasteurised n=5; 23g (±29.4g)
Host 1988	Cows' milk-heat	Raw 5/5 (100 %)	Homogenised & pasteurised 5/5 (100 %)	Raw n =5; 41g (±38.9g)	Homogenised & pasteurised n=5; 27g (±29.4g)
Kim (2011)	Cows' milk-heat	Pasteurised 65/65 (100 %) Pasteurised # 88/88 (100 %)	Baked 0/65 (0 %) Baked # 23/88 (26.1 %)		
Komata (2009)	Cows' milk-heat	Pasteurised 19/19 (100 %)	Heated 18/19 (94.7 %)		
Nowak-Wegrzyn (2008)	Cows' milk-heat	Pasteurised 41/41 (100 %) Pasteurised # 64/64 (100 %)	Heated 0/41 (0 %) Heated # 23/64 (35.9 %)		
Alessandri (2012 b) egg	Egg	Raw 14/14 (100 %) Raw # 33/33 (100 %)	Boiled 0/14 (0 %) Boiled # 19/33 (57.6 %)		
Ando (2008)	Egg	Raw 29/29 (100 %) Raw # 67/67 (100 %)	Heated 0/29 (0 %) Heated # 38/67 (56.7 %)		
Boyano Martinez (2001)	Egg	Raw 20/20 (100 %)	Heated 0/20 (0 %)		
		Raw # 38/38 (100 %)	Heated # 18/38 (47.36 %)		
Lemon-mule (2008)	Egg	Regular 27/27 (100 %) Regular 66/66 (100 %)	Ext. heated 0/27 (0 %) Ext.heated 2 7/66 (10.6 %)		
Urisu (1997)	Egg	Raw 38/38 (100 %)# Raw 38/38 (100 %)#	Heated 17/38 (44.7%)# Heated ovomucoid depleted 1/38 (2.6 %)#		
Marseglia (2012)	Egg (lysozyme)	Regular 21/21 (100 %)	Cheese (12 month matured) 5/21 (23.8 %)		
		Regular 21/21 (100 %)	Cheese (24 month matured) 1/21 (4.8 %)		

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Paper ID	Food	Number showing positive response		Threshold dose Mean (±SD)	
		Comparison food	Test food challenge	Comparison food	Test food
Hansen 2003	Hazelnut	Raw 17/17 (100 %)	Roasted 5/17 (29.4 %)		
Worm (2009)	Hazelnut	Raw 82/90 (91.1 %)	Roasted 17/20 (85 %)	Raw n =82; median 0.1g range 0.01-2.0 g	Roasted n=17; median 0.23 g range 0.01-10 g
Scibilia (2006)	Wheat	Raw 11/11 (100 %)	Boiled 11/11 (100 %)	Raw n =10; 12.1 g (±11.6g)	Boiled n=10; 10.6 g (±10.5g)
Hourihane (1993)	Peanut	Roasted 60/60 (100%)	Crude oil 6/60 (10%)		

for this row of data the positive challenge response to the 'raw or comparison' food was assumed to be positive if participants had a positive response to the cooked form of the food.

§ in this study challenge data for both foods was available for only 9 participants, for the remaining participants only one challenge was carried out.

2.4. Discussion and Conclusions

The included studies were of high quality for the criterion of methods used to diagnose allergy with most studies considered at least low risk of bias for diagnosis as they carried out double blind challenges or open challenges with positive specific IgE. In contrast nearly all studies were considered high risk of bias for sampling. This was because the study reports did not provide a sampling strategy that would ensure that the samples were an accurate reflection of the allergic population as a whole, or accurately represented those with severe allergies. However random sampling from the allergic population would be costly and difficult. The health risks of taking part in such research, and undergoing repeated challenges makes random sampling from the wider food allergic population contentious. For participants the health risks may not out way the potential benefits of being more informed about their own allergies and those who had experienced severe reactions in the past may have been reluctant to take part. However the lack of robust evidence with large study populations for foods sold specifically as being allergen reduced, such as the hydrolysed infant milk formulas, does not support evidence-based decision making.

Those studies that included participants with positive specific IgE and a clinical history found that some individuals did not react to any form of the food. This is not surprising as the positive predictive value for these tests or a combination of tests is not 100%. Both skin tests and specific IgE tests have low positive predictive values (Cianferoni, Garrett, Naimi, Khullar and Spergel; 2012). If possible we excluded cases that were not proven food allergic by challenge for this review.

There was evidence that heat reduced the allergenicity for egg, milk, celery and hazelnut. However, the reduction varies for individual people and for the different foods.

The research studies included highlight that a number of people allergic to uncooked or lightly cooked milk or egg develop tolerance to the baked product, and that this is maintained after long term consumption. The proportion of the allergic population that this applies to is not clear as the sampling strategies selected for those who suspected that they were tolerant to baked products. However, the research does confirm that there is a subpopulation in whom challenge with cooked milk or egg could reduce unnecessary dietary restrictions. One study, Urisu (1997), additionally investigated ovomucoid depleted cooked egg and found there was a further reduction in the number of people having a positive challenge. This could be a potentially useful innovation however further testing would be required.

One small single study investigated the reactivity to cooked celery, Ballmer-Weber (2002). Although allergenicity was reduced a large proportion remained reactive to cooked celery, even if heated for over 70 minutes at 100 oC. However it is not clear from this study if there would be any long term effect of introducing cooked celery into the diet of these tolerant individuals. The positive responses to celery spice

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by even those who were challenge negative to raw celery make it essential that food labelling is clear. More research should be conducted on the effect of the cooking process on celery spice, to establish of the allergenicity in this form can also be reduced by the cooking process. Specific IgE to crude extract or purified proteins of celery did not show promise for predicting tolerance to heated celery, Ballmer-Weber (2002).

Roasting reduced the allergenicity to hazelnut for some people, Hanson (2003) Worm (2009), however within the later study 85% remained reactive. Those with a history of anaphylaxis to hazelnut have been shown to have specific IgE for a 9 kd lipid transfer protein that is heat stable (Pastorello, Vieths, Pravettoni, Farioli, Trambaioli et al, 2002). The presence of this very heat stable allergen/s could explain the persistence of reactivity even after roasting. Although there were no studies included comparing challenge with roasted compared to raw peanut there is strong evidence in the wider literature that roasted peanut remains allergenic, and that the major allergens remain stable and may even have enhanced allergenicity in vitro after extensive heating due to the Maillard reaction (Paschke, 2009; Maleki, Chung, Champagne and Raufman, 2000). Refined peanut oil contains only trace quantities of protein and was found not to cause reactions in those tested, Hourihane (1993), however there were a small proportion (10%) who showed mild symptoms to the crude oil preparation.

The one study that investigated wheat showed that boiling did not reduce allergenicity and this finding is perhaps due to cereals also containing lipid transfer proteins (Pastorello, Pompei, Pravettoni, Farioli, Calamari, Scibilia, and Ortolani, 2003).

Processing to reduce allergenicity of infant formulas has been investigated in a number of studies. All of the studies were relatively small. We excluded a number of studies as the infants did not have cows' milk allergy confirmed by oral challenge. Overall there was a reduction in the number of infants showing a positive response to hydrolysed formulas compared to standard cows' milk formula. There is a need for studies to follow guidelines on testing these formulas (Muraro, 2011).

In conclusion the evidence suggests allergenicity of foods can be altered by food processing. However, although there are trends for certain foods such as extensive heat for egg, milk, celery and to some extent hazelnut reducing allergenicity this reduction will not be experienced by all people with that allergy. The studies we reviewed were small and were not representative of the wider allergic population. More high quality research is required to determine if certain types of processing increase allergenicity, especially for foods where this is suggested by the in vitro research evidence, for example peanut. It would be useful to identify groups of people more likely to tolerate certain types of processed foods, so that more specific diagnostic challenges can be accessed and lead to individualised management strategies.

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2.5. List of included studies

Alessandri C, Zennaro D, Scala E, Ferrara R, Bernardi ML, Santoro M, Palazzo P and Mari A, 2012. Ovomucoid (Gal d 1) specific IgE detected by microarray system predict tolerability to boiled hen's egg and an increased risk to progress to multiple environmental allergen sensitisation. Clinical and Experimental Allergy, 42, 441-450.

Alessandri C, Sforza S, Palazzo P, Lambertini F, Paolella S, Zennaro D, Rafaiani C, Ferrara R, Bernardi Maria L, Santoro M, Zuzzi S, Giangrieco I, Dossena A and Mari A, 2012. Tolerability of a fully maturated cheese in cow's milk allergic children: biochemical, immunochemical, and clinical aspects. PloS one, 7, e40945-e40945.

Ammar F, de B and Dupont C, 1999. Allergy to protein hydrolysates in 30 infants. Archives De Pediatrie, 6, 837-843.

Ando H, Moverare R, Kondo Y, Tsuge I, Tanaka A, Borres Magnus P and Urisu A, 2008. Utility of ovomucoid-specific IgE concentrations in predicting symptomatic egg allergy. Journal of Allergy and Clinical Immunology, 122, 583-588.

Ballmer-Weber BK, Hoffmann A, Wuthrich B, Luttkopf D, Pompei C, Wangorsch A, Kastner M and Vieths S, 2002. Influence of food processing on the allergenicity of celery: DBPCFC with celery spice and cooked celery in patients with celery allergy. Allergy, 57, 228-235.

Boyano Martinez TB, Garcia-Ara C, Diaz-Pena JM, Munoz FM, Sanchez GG and Esteban MM, 2001. Validity of specific IgE antibodies in children with egg allergy. Clinical and Experimental Allergy, 31, 1464-1469.

Burks W, Jones Stacie M, Berseth Carol L, Harris C, Sampson Hugh A and Scalabrin Deolinda MF, 2008. Hypoallergenicity and effects on growth and tolerance of a new amino acid-based formula with docosahexaenoic acid and arachidonic acid. Journal of Pediatrics, 153, 266-271.

Caffarelli C, Plebani A, Poiesi C, Petroccione T, Spattini A and Cavagni G, 2002. Determination of allergenicity to three cow's milk hydrolysates and an amino acid-derived formula in children with cow's milk allergy. Clinical and Experimental Allergy, 32, 74-79.

Giampietro PG, Kjellman NIM, Oldaeus G, Wouters-Wesseling W and Businco L, 2001. Hypoallergenicity of an extensively hydrolyzed whey formula. Pediatric Allergy and Immunology, 12, 83-86.

Hansen KS, Ballmer-Weber BK, Luttkopf D, Skov PS, Wuthrich B, Bindslev-Jensen C, Vieths S and Poulsen LK, 2003. Roasted hazelnuts - allergenic activity evaluated by double-blind, placebo-controlled food challenge. Allergy, 58, 132-138.

Host A and Samuelsson EG, 1988. Allergic reactions to raw, pasteurized, and homogenized/pasteurized cow milk: a comparison. A double-blind placebo-controlled study in milk allergic children. Allergy, 43, 113-118.

Hourihane JOO, Bedwani SJJ, Dean TPP and Warner JOO, 1997. Randomised, double blind, crossover challenge study of allergenicity of peanut oils in subjects allergic to peanuts. BMJ (Clinical research ed.), 314, 1084-1088.

Kaczmarski M, Wasilewska J and Lasota M, 2005. Hypersensitivity to hydrolyzed cow's milk protein formula in infants and young children with atopic eczema/dermatitis syndrome with cow's milk protein allergy. Roczniki Akademii Medycznej w Bialymstoku (1995), 50, 274-278.

Kim Jennifer S, Nowak-Wegrzyn A, Sicherer Scott H, Noone S, Moshier Erin L and Sampson Hugh A, 2011. Dietary baked milk accelerates the resolution of cow's milk allergy in children. Journal of Allergy and Clinical Immunology, 128, 125-U205.

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Komata T, Shukuya A, Imai T, Tachimoto H and Ebisawa M, 2009. Single blind food challenge using dried food powder --2nd Report. milk. Arerugi = [Allergy], 58, 779-789.

Lemon-Mule H, Sampson Hugh A, Sicherer Scott H, Shreffler Wayne G, Noone S and Nowak-Wegrzyn A, 2008. Immunologic changes in children with egg allergy ingesting extensively heated egg. Journal of Allergy and Clinical Immunology, 122, 977-983.

Marseglia A, Castellazzi AM, Valsecchi C, Licari A, Piva G, Rossi F, Fiorentini L and Marseglia GL, 2012. Outcome of oral provocation test in egg-sensitive children receiving semi-fat hard cheese Grana Padano PDO (protected designation of origin) containing, or not, lysozyme. Eur J Nutr.

Niggemann B, von B, Bollrath C, Berdel D, Schauer U, Rieger C, Haschke-Becher E and Wahn U, 2008. Safety and efficacy of a new extensively hydrolyzed formula for infants with cow's milk protein allergy. Pediatric Allergy and Immunology, 19, 348-354.

Nowak-Wegrzyn A, Bloom Katherine A, Sicherer Scott H, Shreffler Wayne G, Noone S, Wanich N and Sampson Hugh A, 2008. Tolerance to extensively heated milk in children with cow's milk allergy. Journal of Allergy and Clinical Immunology, 122, 342-347.

Ragno V, Giampietro PG, Bruno G and Businco L, 1993. Allerginicity of milk protein hydrolysate formulas in children with cows milk allergy. European Journal of Pediatrics, 152, 760-762.

Rugo E and Wahn U, 1992. Invivo studies on residual allergenic activity of hydrolyzed formula. Monatsschrift Kinderheilkunde, 140, 472-475.

Sampson HA, Bernhiselbroadbent J, Yang E and Scanlon SM, 1991. Safety of casein hydrolysate formula in children with cow milk allergy. Journal of Pediatrics, 118, 520-525.

Scibilia J, Pastorello EA, Zisa G, Ottolenghi A, Bindslev-Jensen C, Pravettoni V, Scovena E, Robino A and Ortolani C, 2006. Wheat allergy: A double-blind, placebo-controlled study in adults. Journal of Allergy and Clinical Immunology, 117, 433-439.

Urisu A, Ando H, Morita Y, Wada E, Yasaki T, Yamada K, Komada K, Torii S, Goto M and Wakamatsu T, 1997. Allergenic activity of heated and ovomucoid-depleted egg white. Journal of Allergy and Clinical Immunology, 100, 171-176.

Worm M, Hompes S, Fiedler EM, Illner AK, Zuberbier T and Vieths S, 2009. Impact of native, heat-processed and encapsulated hazelnuts on the allergic response in hazelnut-allergic patients. Clinical and Experimental Allergy, 39, 159-166.

2.6. List of Excluded Studies

Amonette MS, Schwartz RH, Mattson L, Peers LB and Eldredge DM, 1991. Double-blind placebo-controlled food challenges DBPCFC demonstrating acute ige-mediated allergic reactions to good start ultrafiltered good start alfare nutramigen and alimentum in a seven-year-old. Pediatric Asthma Allergy and Immunology, 5, 245-252.

Anton M, Dieguez M, Garcia A, Rubio G and Diez M, 2004. A case of beer and wheat allergy. Journal of Allergy and Clinical Immunology, 113, S316-S316.

Armentia A, Duenas-Laita A, Pineda F, Herrero M and Martin B, 2010. Vinegar decreases allergenic response in lentil and egg food allergy. Allergologia Et Immunopathologia, 38, 74-77.

Belloni B, Kirschner S, Kugler C, Ring J and Brockow K, 2008. Tolerance of wines fined with allergenic proteins in allergic individuals-double-blind, placebo controlled food challenge study. Allergy, 63, 119-119.

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3. WHAT NEW ANALYTICAL METHODS ARE AVAILABLE TO ANALYSE/DETECT THE FOLLOWING FOOD ALLERGENS IN PROCESSED FOODS: MILK/DAIRY, EGGS, CEREALS, BUCKWHEAT, PEANUTS, NUTS, CELERY, CRUSTACEANS, FISH, MOLLUSCS, SOY, LUPINE, MUSTARD AND SESAME? (OBJECTIVE 4B)

3.1. Search Strategy

We searched the databases presented in section1.1.1, using the search terms outlined in Table 3.1, words within groups linked by OR and between groups liked with AND. In addition we asked experts within the field for published studies (to be completed). The following databases were searched from Web of Science (1970-November 2012), BIOSIS Citation Index (1969-November 2012), BIOSIS reviews (1969-2008), Medline (1950-November 2012), Pubmed (- November 2012), using the search terms shown (Table 3.1). No limits were used. The included studies were limited to those published after 2004.

Table 3.1: Search terms for identifying assays that detect allergenic foods

Topics	Search terms Web of Knowledge	Search terms Pubmed
Group 1. Food web of Knowledge		
Milk and dairy	milk OR butter or cream or dairy or cheese or yoghurt or petit filous or casein or whey or lacto Infant NEAR/2 formula	milk[Tiab] OR milk[MeSH Terms] OR lactose[MeSH Terms] OR lactose[Tiab] OR dairy[Tiab] OR butter[Tiab] OR cream[Tiab] OR "infant formula"[Tiab] OR cheese[Tiab] OR yoghurt[Tiab] OR "petit filous"[Tiab] OR casein[Tiab] OR whey[Tiab]
Egg	Egg	egg[Tiab] OR eggs[Tiab]
Cereals	Cereal or gluten or wheat or rye or barley or oats or spelt or kamut	cereals[MeSH Terms] OR cereal[Tiab] OR cereals[Tiab] OR glutens[MeSH Terms] OR glutens[Tiab] OR gluten[Tiab] OR wheat[Tiab] OR rye[Tiab] OR barley[Tiab] OR oats [Tiab] OR oat[Tiab] OR spelt[Tiab] OR kamut[Tiab]
Buckwheat	Buckwheat	Buckwheat,
Peanuts	nut or arachis	peanut[Tiab] OR arachis[Tiab]
Nuts	nut or arachis or cashew or brazil brasil or almond or hazel or walnut or pecan or macadamia or pistachio or filbert	nuts[MeSH Terms] OR nuts[Tiab] OR nut[Tiab] OR almond[Tiab] OR almonds[Tiab] OR hazelnut[Tiab] OR hazelnuts[Tiab] OR walnut[Tiab] OR walnuts[Tiab] OR cashew[Tiab] OR cashews[Tiab] OR pecan[Tiab] OR pecans[Tiab] OR macadamia[Tiab] OR macadamias[Tiab] OR pistachio[Tiab] OR pistachios[Tiab] OR beechnut[Tiab] OR beechnuts[Tiab] OR filbert[Tiab] OR filberts[Tiab]
Celery	Celery	celery[tiab]
Crustaceans	crustacea OR crustacean OR crustaceans OR crab OR crabs OR lobster OR lobsters OR shrimp OR	crustacea[MeSH Terms] OR crustacea[Tiab] OR crustacean[Tiab] OR crustaceans[Tiab] OR crab[Tiab] OR crabs[Tiab] OR lobster[Tiab] OR

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Topics	Search terms Web of Knowledge	Search terms Pubmed
	shrimps OR prawn OR prawns OR crayfish OR shellfish OR langoustine OR langoustines	lobsters[Tiab] OR shrimp[Tiab] OR shrimps[Tiab] OR prawn[Tiab] OR prawns[Tiab] OR crayfish[Tiab] OR shellfish[MeSH Terms] OR shellfish[Tiab] OR langoustine[Tiab] OR langoustines[Tiab]
Fish,	fish OR pollock OR carp OR cod OR mackerel OR salmon OR tuna OR shark OR "sea bass" OR swordfish OR hake OR sole OR megrim OR sardine OR sardines OR halibut OR anchovy OR anchovies OR catfish OR trout	fishes[MeSH Terms] OR fish[Tiab] OR pollock[Tiab] OR carp[Tiab] OR cod[Tiab] OR mackerel[Tiab] OR salmon[Tiab] OR tuna[Tiab] OR shark[tiab] OR "sea bass"[tiab] OR swordfish[tiab] OR hake[tiab] OR sole[tiab] OR megrim[tiab] OR sardine[tiab] OR sardines[tiab] OR halibut[tiab] OR anchovy[tiab] OR anchovies[tiab] OR catfish[tiab] OR trout[tiab] mollusca[MeSH Terms] OR mollusc[Tiab] OR molluscs[Tiab] OR oyster[Tiab] OR oysters[Tiab] OR snail [Tiab] OR snails[Tiab] OR squid[Tiab] OR mussel[Tiab] OR mussels[Tiab] OR clam[Tiab] OR clams[Tiab] OR abalone[tiab] OR octopus[tiab] OR scallop[tiab] OR scallops[tiab]
Mollusces	mollusc OR molluscs OR oyster OR oysters OR snail OR snails OR squid OR mussel OR mussels OR clam OR clams OR abalone OR octopus OR scallop OR scallops	mollusca[MeSH Terms] OR mollusc[Tiab] OR molluscs[Tiab] OR oyster[Tiab] OR oysters[Tiab] OR snail [Tiab] OR snails[Tiab] OR squid[Tiab] OR mussel[Tiab] OR mussels[Tiab] OR clam[Tiab] OR clams[Tiab] OR abalone[tiab] OR octopus[tiab] OR scallop[tiab] OR scallops[tiab]
Soy	Soy	soy[Tiab] OR soybeans[MeSH Terms] OR soybean[Tiab] OR soybeans[Tiab] OR soya[Tiab]
Lupin	LUPINUS-ALBUS, Lupine	lupinus[MeSH Terms] OR lupin*[Tiab]
Mustard	Mustard	"mustard plant"[MeSH Terms] OR mustard[Tiab]
Sesame	Sesame	sesamum[MeSH Terms] OR "sesame"[Tiab]
Group 2. Allergen/antigenicity or protein		
Allergen, antigen or protein	Allerg [OR Antigen* OR Epitope* OR IgE OR protein	Allerg [OR Antigen* OR Epitope* OR IgE OR protein
Group 3 Processing methods		
Heat and chemical Cooking, heavy	(heat* or cook* or roast* or fry* or pasteuri* or boil) or (heavy near/2 salting) or dying or microwav* OR ferment* or smoking or drying or (UV	Heat*[tiab] OR cook*[tiab] OR roast* [tiab] OR fry*[tiab] OR pasteuri*[tiab] OR boil[tiab] OR

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Topics	Search terms Web of Knowledge	Search terms Pubmed
salting,, microwaving, filtration, fermenting, smoking, drying, UV treatment for sterilisation, acid, alkaline (lyme treatment) treatment heating treatments, ohmic.	NEAR/2 treatment) or lyme or ohmic OR (chemical near/4 peeling) or Hydrostatic pressure or (food near/1 process*) or ("food proces*") or (digest*) or (hydrol*) or filtration	Hydrolysis [tiab] OR digestion [tiab] OR enzymatic treatment [tiab] OR fermented [tiab] OR Hydrostatic pressure[tiab] OR food process* [tiab] OR "heavy salting" [tiab] OR dying [tiab] OR microwav* [tiab] OR ferment* [tiab] OR smoking [tiab] OR drying [tiab] OR UV [tiab] OR lyme [tiab] OR ohmic [tiab] OR "chemical peeling"[tiab]
Product related	(wine OR beer OR clarif*)	wine [tiab] OR beer [tiab] Or clarify* [tiab]
Group 6 Assay quality		
	(Sensitivity near/10 specificity) or (detection near/2 limit) or (receiver near/1 operator) or (limit near/2 detection) or limit near/2 quantification	
Group 7 Assay/test		
	spectrometry or PCR or polymerase near/1 chain OR Immuno near/1 assay OR Competitive near/1 lateral OR Bioreceptor* OR Dog* OR canine* OR Sens* OR ELISA or RIA or biosensor*	spectrometry [tiab] OR PCR[tiab] OR polymerase chain [tiab] OR 'Immuno assay' [tiab] OR 'Competitive lateral flow' [tiab] OR Bioreceptor*[tiab] OR Dog*[tiab] OR canine*[tiab] OR Sens*[tiab] OR Detection limit*[tiab] OR 'Receiver operator curve' [tiab] OR ELISA [tiab], RIA [tiab], CAP [tiab] , biosensor* [tiab]

3.1.1. Selection criteria

All titles and abstracts were imported to Endnote and duplicates removed. One reviewer, SK, screened the titles and abstracts to remove studies not relevant to the objective. The full texts were obtained for the remaining studies; a second screen by SK then removed studies that were not relevant to the research question and the reasons identified.

3.1.1.1. Types of studies

We set out to include studies investigating extraction and detection of the food/proteins in a food matrix of relevance to the real world setting. Studies investigating food matrixes spiked with allergen were included, and those using samples taken from 'field' samples (manufactured, laboratory processed or home produced).

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3.1.1.2. Types of detection methods

All methods of detection that quantify the specific food or allergenic proteins within the food source or indicate that the allergen is present or absent. It was anticipated that mass spectrometry, polymerase chain reaction, immunoassay, Molditoff would be included in the review; however there was no exclusion criteria on the type of assay.

3.1.1.3. Types of outcome measure

The review included studies that assessed quality of studies as laid out by the International Committee for Harmonisation Topic Q 2 (R1) Validation of Analytical Procedures. For spiked samples we were interested in measures of validity and reliability of assays: specificity, linearity, range, accuracy, precision, detection limit, quantification limit, robustness, system suitability testing. We were interested in showing values for the extraction method and the assay in combination, in which case these were labelled 'sampling', or of the assay alone and these are labelled 'assay', e.g. assay limit of quantification, or sample limit of quantification. For studies of 'field samples', that is samples taken from kitchens or from commercial sources with unknown quantities the assays were compared with the best available assay either using continuous or binary data (e.g. cut off for allergen present or absent).

3.1.2. Extraction of data

The following data was collected: food allergen assessed, method of detection, test mechanism for example protein detection, antigen or epitope detection using immunoassay, detection of DNA, allergenicity using food challenge, type of study, comparison of reference test against an index test or percentage retrieval if spiked sample, the name of the test, the commercial company and address.

3.1.3. Assessment of methodological quality of included studies

The quality of the studies have been assessed for the range of food processing techniques used. We divided studies into those investigating the analytical quality of the assays and those investigating the effectiveness of the assays for 'field' samples. Assays investigating the analytical quality were assessed according to the adapted criteria from the International Committee for Harmonisation Topic Q 2 (R1) Validation of Analytical Procedures and by adapting the scoring system for assessing diagnostic tests QUADAS (Whiting et al., 2003). Criteria used are outlined in the following Table 3.2.

Table 3.2: Quality assessment of studies

Criteria	Low risk of bias	High risk of bias	Unclear
Spiking procedure	Likely to incorporate allergen into the matrix e.g. tempered chocolate	Method unlikely to incorporate allergen into matrix e.g. mixing powdered allergen with powdered matrix	Not reported
Spiking or extract used for standard curve	Standardised source, or source clearly identified	Not standardised	Not reported
Sampling/extraction	Each replicate involves separate extraction and sampling	One sample made into separate aliquots	Not reported
'Field' sampling	Random sample, or all samples from a representative source of food.	Non random sample, or all from an isolated source	Not reported
Assessment of data	Blind or methodology for measurement or calculation technique rigorous and objective	Not blind and methodology not rigorous or introduces subjectivity	Not reported

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3.1.4. Data synthesis and presentation

Tables will present for each allergen and each processing method the range of the limit of detection and quantification for each analytical technique with the corresponding extraction technique.

3.2. Results

The search strategy yielded 1475 studies after removal of duplications. The initial screening of title and abstract removed 1351 studies. Of those excluded a large proportion were about the development of laboratory diagnostic for allergy, detection of parasite eggs, and a range of other biological substances these studies were excluded under the heading, 'not detecting listed foods'. Some studies were review articles and so were listed under 'not contain primary data' and there were a group of studies that did not investigate detection within a suitable food related matrix.

Full text screening was carried out on 124 studies, yielding 84 included studies and excluding 40 studies. The reasons for exclusion were grouped under six headings. One: that there was no data for accuracy i.e. percentage recovery from spiked food samples, or comparison with the results from another assay with known accuracy, and there was no data for the limit of detection of the assay for a suitable food matrix. Two: although there may be data on assay validation, this did not include recovery or sensitivity of detecting the allergenic compound within a suitable food matrix. Three: the data was not in a suitable format and we could not calculate the percentage recovery or identify the limit of detection. Four: the study showed data that was presented within another study or the study was a duplicate. Five: the assay was developed to assess if a food was contaminated with food from another specifies for example goat's milk adulterated with cheaper bovine cow's milk and Six: the study was published prior to 2004. The studies excluded at the full text stage are shown (Section 3.5).

3.2.1. Almond

We included two studies, one evaluating ELISA and one PCR for detecting almond in cakes, confectionary and cereals (Table 3.3), Garber (2010a) used almond sources from a local shop whereas Roeder (2011) sourced their almond extract for the spiking experiments from a research institute (Table 3.4). Garber (2010) tested three commercial ELISAs, Veratox, Ridascreen and ELISA systems showing that the limit of detection ranged between 3-9 µg/g for extraction and measurement of the almond proteins in food matrices such as cake, oatmeal, chocolate and muffins. The real-time PCR tested by Roeder (2011) gave a limit of detection of 5 mg/kg^{-1} or 50 µg/g (Table 3.5).

Table 3.3: Almond: characteristics of included studies

Study ID	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Garber (2010a)	Crude extract	Breakfast cereals *oatmeal* Cake *muffins* Chocolate	Spiked **Source of spike** *purchased locally*	ELISA
Roeder (2011)	Specific protein/peptide or gene *nsLTP*	Chocolate Cookie **Field** Foods sampled *Chocolate, yogurt, cookies, muesli*	Spiked Field **Source of spike** *Whole unroasted almonds with seed coat from Institut Fur Produktqualitat (Berlin, Germany)*	PCR

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Table 3.4: Almond: description of assay

Study ID	Assay details	Additional information
Garber (2010a) almond	Test 1 *Veratox (Neogen Corporation, Lansing, MI, USA);* Test 2 *Ridascreen Fast (R-Biopharm Inc, Marshal, MI, USA),* Test 3 *Elisa systems-bioMerieux Industry (Hazelwood, MO, USA)*	**ELISA** Sandwich Commercial company
Roeder (2011)	Test 1 *Real time PCR*	**PCR** Real-time PCR In-house

Table 3.5: Almond: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type		Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Garber 2010	almond	crude	1	ELISA, Veratox	cake			9 µg/g	
Garber 2010	almond	crude	1	ELISA, Veratox	cereals			4 µg/g	
Garber 2010	almond	crude	1	ELISA, Veratox	chocolate			4.2 µg/g	
Garber 2010	almond	crude	2	ELISA, RIDASCREEN	cake			7 µg/g	
Garber 2010	almond	crude	2	ELISA, RIDASCREEN	cereals			2.5 µg/g	
Garber 2010	almond	crude	2	ELISA, RIDASCREEN	chocolate			3 µg/g	
Garber 2010	almond	crude	3	ELISA Systems	cake			7 µg/g	
Garber 2010	almond	crude	3	ELISA Systems	cereals			4.3 µg/g	
Garber 2010	almond	crude	3	ELISA Systems	chocolate			5 µg/g	
Roeder 2011	almond	nsLTP	1	Real time PCR	chocolate			50 µg/g	
Roeder 2011	almond	nsLTP	1	Real time PCR	cookie			50 µg/g	

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3.2.2. Brazil nut

The type of assay and the foods spiked with Brazil nut extract of the three included studies are shown (Table 3.6 and 3.7). The ELISA investigated by Ben Rejab (2005) provided similar sensitivity, 1 ppm (equivalent to 1 µg/g), as the PCR assay described by Roeder (2010) for brazil nut added to chocolate. Roeder (2010) tested the assay for a wider range of products including cookie, dough and cereals and in all cases the allergen could be detected at concentrations as low as 5 µg/g. Sharma (2009) investigated percentage recovery for their in house ELISA and found good recoveries down to 10 µg/g. However there was overestimation particularly for wheat flour (Table 3.8), Sharma (2009) found that the lowest recovery was from dark chocolate µg/g, perhaps due to Brazil nut chocolate complexes that were insoluble in the extraction buffer. Although blank samples of most foods gave negligible readings, apart from cinnamon, the recoveries for wheat flour and cookie were over 100%, which indicate that there was an interaction between the food matrix and the Brazil but proteins that altered the antibody protein binding giving higher than expected results. Both the ELISAs were developed to detect the crude protein mix, whereas the PCR was directed against the gene for the major Brazil nut allergen.

Table 3.6: Brazil nut: characteristics of included studies

Study ID	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Ben Rejeb (2005b)	Crude extract	Chocolate	Spiked Roasted defatted peanuts and nuts extracted, dialysed **Source of spike** *Not reported* Standardisation Made up to 1mg/ml^{-1} protein content measured using BCA test	ELISA
Roeder (2010)	Specific protein/peptide or gene *Ber e 1 gene*	Chocolate, Cookie Dough **Field** Foods sampled *Range of cereals, chocolate bars, snacks and other nut products*	Spiked Field **Source of spike** *Brazil nuts heat treated for drying but not roasted*	PCR
Sharma (2009)	Crude extract	Breakfast cereals *Oat* Chocolate Cookie, *Shortbread* Flour, *Wheat flour*	Spiked **Source of spike** *Local grocery store*	ELISA

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Table 3.7: Brazil nut: description of assay

Study ID	Allergen	Assay details	Additional information
Ben Rejeb (2005)	Brazil nut	Test 1 ELISA	**ELISA** Polyclonal detection antibody Competitive inhibition In house
Roeder (2010)	Brazil nut	Test 1 *real time PCR*	In-house
Sharma (2009)	Brazil nut	Test 1 *ELISA*	**ELISA** Polyclonal detection antibody Competitive inhibition In-house

Table 3.8: Brazil nut: accuracy and limit of detection and quantification

Study ID	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Ben Rejeb 2005		1 ELISA	chocolate dark			1 µg/g	
Roeder 2010	Ber e 1 gene	1 Real time PCR	chocolate			5 µg/g	
Roeder 2010	Ber e 1 gene	1 Real time PCR	cookie			5 µg/g	
Roeder 2010	Ber e 1 gene	1 Real time PCR	dough			5 µg/g	
Sharma 2009	Protein	1 ELISA	cereals	10-100 µg/g	105-119		
Sharma 2009	Protein	1 ELISA	chocolate dark	10-100 µg/g	90-95		
Sharma 2009	Protein	1 ELISA	cookie	10-100 µg/g	123-130		
Sharma 2009	Protein	1 ELISA	wheat flour	10-100 µg/g	150-189		

3.2.3. Buckwheat

We included three studies that investigated the validation of assays for detecting buckwheat within a variety of foods including noodles and cake. All three tests were immunoassays directed against crude extracts, two ELISA and one a dipstick test (Table 3.9 and Table 3.10). The ELISAs produced by Morinaga Institute of Biological Science and the commercial ELISA, FASTKIT, tested by Akiyama (2004a) in an experiment where cake samples were spiked with between 5 and 20 ng/ml of buckwheat, gave between 89 and 94 % recoveries. However this recovery was reduced for snacks and noodles spiked with buckwheat (Table 3.11). The in-house ELISA developed and tested by Panda (2010) provided a limit of detection of 2 ppm (2 µg/g) for spiked noodles or cake. The same authors investigated the ELISA-

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Systems kit and this was found to be not as sensitive with a limit of detection of 100 µg/g. The IC dipstick, in addition to being easy to use and not requiring specialised equipment to develop the test, gave sensitivity down to 5 µg/g in a range of products, Morishita (2006) (Table 3.11).

Table 3.9: Buckwheat: characteristics of included studies

Study ID	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Akiyama (2004a)	Crude extract	Cake Bun Noodles Udon Snack	Spiked **Source of spike** FASMAC Standardisation Protein measured using BCA protein assay kit concentration adjusted to 100-300 µg/ml	ELISA
Morishita (2006)	Crude extract	Carrot Sherbet Cookie, Jam Pickles (Soy Sauce, vinegar) Potato Salad, Sauce, Tomato Soup, Steamed and fried Chinese dumpling Hamburger	Spiked	ELISA Immuno-chromatographic test kits Dip stick
Panda (2010)	Crude extract	Cake Muffins Noodles	Spiked **Source of spike** Buckwheat flour (Hodgsons Mill, Effingham, ILL, USA)	ELISA

Table 3.10: Buckwheat: description of assay

Study ID	Allergen	Assay details	Additional information
Akiyama (2004a)	Buckwheat	Test 1 Buckwheat ELISA (MORINAGA Institute of Biological Science) Test 2 FASTKIT Buckwheat ELISA kit (Nippon Meat Packers Inc.)	Not stated
Morishita (2006)	Buckwheat	Test 1 Immunochromatographic test kits, dipstick. Test 2 ELISA: FASTKIT	Not stated
Panda (2010)	Buckwheat	Test 1	**ELISA** Polyclonal capture antibody

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Study ID	Allergen	Assay details	Additional information
		Test 2 ELISA Systems Pty. Ltd., Windsor, Queensland, Australia	Polyclonal detection antibody Sandwich. In-house Commercial assay

Table 3.11: Buckwheat: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Akiyama 2004a	buckwheat	crude	1 ELISA *MORINAGA*	buffer			1 ng/ml	1 ng/ml
Akiyama 2004a	buckwheat	crude	1 ELISA *MORINAGA*	cake	5-20 ng/ml	62-102		
Akiyama 2004a	buckwheat	crude	1 ELISA *MORINAGA*	noodles	5-20 ng/ml	43-56		
Akiyama 2004a	buckwheat	crude	1 ELISA *MORINAGA*	snack	5-20 ng/ml	50-54		
Akiyama 2004a	buckwheat	crude	2 ELISA, FASTKIT	buffer			1 ng/ml	4 ng/ml
Akiyama 2004a	buckwheat	crude	2 ELISA, FASTKIT	cake	5-20 ng/ml	89-94		
Akiyama 2004a	buckwheat	crude	2 ELISA, FASTKIT	noodles	5-20 ng/ml	76-94		
Akiyama 2004a	buckwheat	crude	2 ELISA, FASTKIT	snack	5-20 ng/ml	63-64		
Morishita 2006	buckwheat	crude	1 IC - dipstick	chicken meatball or burger			5 µg/g	
Morishita 2006	buckwheat	crude	1 IC - dipstick	cookie			5 µg/g	
Morishita 2006	buckwheat	crude	1 IC - dipstick	Dumplings fried/ steamed			5 µg/g	
Morishita 2006	buckwheat	crude	1 IC - dipstick	jelly			5 µg/g	
Morishita 2006	buckwheat	crude	1 IC - dipstick	Pickles in Vinegar/soy			5 µg/g	
Morishita 2006	buckwheat	crude	1 IC - dipstick	Potato salad			5 µg/g	
Morishita 2006	buckwheat	crude	1 IC - dipstick	sauce			5 µg/g	
Panda 2010	buckwheat	crude	1 ELISA	cake	3-1000 µg/g	58-69	2 µg/g	2 µg/g
Panda 2010	buckwheat	crude	1 ELISA	noodles	3-1000 µg/g	83-108	2 µg/g	2 µg/g
Panda 2010	buckwheat	crude	2 ELISA-Systems	noodles	3-1000 µg/g	0-95	100 µg/g	

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3.2.4. Cashew

Four studies investigated detection systems for cashew. The ELISA assays provided good sensitivity and good recovery from a wide range of spiked products such as ice-cream, pesto, and chocolate, Geskin (2011), Ben Rejeb (2005). The PCR assay tested by Brezezinski (2006) did not improve the limit of detection giving only 100 µg/g limit of detection for with a cookie food matrix.

Ehlert (2002) developed and validated a multi-target method for the simultaneous detection of a range of allergens food matrices. The Ligation-dependent probe amplification assay is based on PCR and enables several different allergens to be tested in one tube. The limit of detection of this test for cashew was estimated at 5 mg/kg^{-1} equivalent to 50 µg/g for detecting pesto sauce spiked with cashew proteins.

Table 3.12: Cashew: characteristics of included studies

Study ID	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Ben Rejeb (2005c)	Crude extract	Chocolate	Spiked Roasted defatted peanuts and nuts extracted, dialysed **Source of spike** Not reported Standardisation Made up to 1mg/ml^{-1} protein content measured using BCA test	ELISA
Brzezinski (2006)	Specific protein/peptide or gene Cashew 2S albumin gene	Cookie	Spiked **Source of spike** locally purchased	PCR
Ehlert (2009)	Crude extract Specific protein/peptide or gene DNA	Cookie Pesto cashew	Spiked **Source of spike** Nut materials, sesame seeds, ingredients of self-prepared DNA plant and animal materials used to test the specificity of the method and spike samples of chocolate, were obtained from the Bavarian Health and Food Safety Authority (Oberschleibheim, Germany)	ELISA PCR Ligation-dependent probe amplification
Gaskin (2011)	Crude extract roasted	Chocolate Cookie Ice cream	Spiked **Source of spike** Purchased locally	ELISA

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Table 3.13: Cashew: description of assay

Study ID	Allergen	Assay details	Additional information
Ben Rejeb (2005c) cashew	Cashew	Test 1 ELISA	**ELISA** Polyclonal detection antibody Competitive inhibition
Brzezinski (2006)	Cashew	Test 1 PCR	**PCR** Real-time PCR
Ehlert (2009)	Cashew	Test 1 Ligation dependent probe amplification (LPA) Test 2 Cashew real time PCR Test 3 Hazelnut and peanut: ELISA Ridascreen	Test 1 LPA for simultaneous detection of DNA from different foods Test 2 PCR, In house Test 3 ELISA, commercial, R-Biopharm AG, Darmstadt, Germany
Gaskin (2011)	Cashew	Test 1 Sheep antibody Test 2 Goat antibody	**ELISA** Polyclonal capture antibody Sandwich, In-house

Table 3.14: Cashew: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Ben Rejeb 2005	cashew	Crude	1 ELISA	chocolate dark			1 µg/g	
Brezezinski 2006	cashew	cashew 2S albumin	1 PCR	cookie			10 µg/g 0	
Ehlert 2009	cashew	DNA	1 LPA	pesto			50 µg/g	
Ehlert 2009	cashew	DNA	2 PCR real time	pesto			20 µg/g	
Geskin 2011	cashew	crude	1 ELISA sheep	chocolate milk	1-1000 µg/g	100-110		
Geskin 2011	cashew	crude	1 ELISA sheep	cookie	1-100 µg/g	75-99		
Geskin 2011	cashew	crude	1 ELISA sheep	ice cream	1-102 µg/g	111-128		
Gesking 2011	cashew	crude	1 ELISA sheep	buffer			0. µg/g 11	0.46 µg/g
Gesking 2011	cashew	crude	2 ELISA goat	buffer			0. µg/g 11	0.46 µg/g

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3.2.5. Celery

Three studies investigated detection of celery allergen in foods (Table 3.15 and Table 3.16) and one looked at ELISA. Wang (2011) and two looked at PCR Coisson (2010) and Wu (2010). Wang (2011) investigated an in house ELISA system, and recovery data was carried out at one concentration only 10µg/g. Good recovery was observed in a range of powdered foods. This recovery rate may not be maintained if more complex matrices such as dough were used. The PCR assay sensitivities varied from 0.1%, Wu (2010) to 5 % w/w, Coisson (2010) when meatball samples were spiked with the allergen. The PCR assay sensitivity was reduced by heating of the food matrix but this was still acceptable at 1 % w/w, Wu (2010) (Table 3.17).

Table 3.15: Celery: characteristics of included studies

Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Coisson (2010)	Celery	Specific protein/peptide or gene DNA Celery 2S albumin AgMTD, sesame mannitol dehydrogenase Si2S	Meat meat balls	Spiked **Source of spike** A. graveolens L (celery leaves). Samples purchased from commercial stores in Italy.	PCR
Wang (2011)	Celery	Specific protein/peptide or gene Api g 1.01	corn powder wheat powder Rice, rice powder soy powder	Spiked **Source of spike** Apium graveolens bought from local market	ELISA
Wu (2010)	Celery	Mannitol transporter protein gene	Pork powder **Field** Foods sampled Dumplings, hundun, biscuits, powdered chicken, mushroom soup, vegetable/fruit juice, sauce	Spiked Field **Source of spike** Celery powder from local markets	PCR

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Table 3.16: Celery: description of assay

Study ID	Allergen	Assay details	Additional information
Coisson (2010)	Celery	Test 1 PCR with multiplex	
Wang (2011)	Celery	Test 1 ELISA	**ELISA** Monoclonal capture antibody Monoclonal detection antibody Sandwich In-house
Wu (2010)	Celery	Test 1 Real time PCR (Mat3)	**PCR** Real-time PCR SYBR green

Table 3.17: Celery: accuracy and limit of detection and quantification

Study ID	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Coisson 2010	DNA	1 PCR	meatball or burger			5 % w/w	
Wang 2011	Api g 1.01	1 ELISA	buffer			5.6 µg/g	
Wang 2011	Api g 1.01	1 ELISA	corn powder	10 µg/g	102		
Wang 2011	Api g 1.01	1 ELISA	rice powder	10 µg/g	100		
Wang 2011	Api g 1.01	1 ELISA	soy powder	10 µg/g	83		
Wang 2011	Api g 1.01	1 ELISA	wheat flour	10 µg/g	115		
Wang 2011	Heated Api g 1.01	1 ELISA	Heated/ buffer			5.7 µg/g	
Wu 2010	Mannitol transporter protein gene	1 Real-Time PCR SYBR green	meatball or burger			0.1 % w/w	
Wu 2010	Heated Mannitol transporter protein gene	1 Real-Time PCR SYBR green	Heated meatball or burger			1 % w/w	

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3.2.6. Cereals

Five included studies investigated detection of wheat or gluten allergens and the key characteristics and the type of assay are shown (Table 3.18 and Table 3.19) ELISA, immune assay dip sticks and mass spectrometry were tested. Akiyama (2004) and Mena (2012) investigated ELISA. The FASTKIT ELISA was tested by Akiyama 2004, at relatively low concentration of allergens and although this ELISA gave recovery rates of less than 50% it provided a limit of detection down to 1 ng/ml, the Gliadin test kit gave similar or slightly better recovery rates. Mena (2012) with the ELISA R5 system, found that there was good recovery of gluten from a range of foods such as biscuit, bread, cereals, however in products containing chocolate there was very poor recovery perhaps due to interaction of tannins with the proteins. Mena (2012) developed a modified extraction procedure and this solved the problem enabling yields of nearly 100% when chocolate samples were spiked with 55 µg/g of gluten. The authors were using a commercial extraction system UPEX, such extraction systems could lead to increased reproducibility providing there is strict quality control.

The other type of immunoassay investigates were the test strips, EZ Gluten, which gave good sensitivity of 5 µg/g in rice, dog food, beer and cooked dough, and a dipstick system, Morishita 2006 that provided a good limit of detection of 5 µg/g Morishita 2006.

One study developed and validated mass spectrometry for gluten, investigating a range of sequences, Sealey-Voyksner 2010. This assay provided good recovery rates, 69-112 percent, of more than at very low concentrations of allergen (Table 3.20).

Table 3.18: Cereals: characteristics of included studies

Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Akiyama (2004)	Wheat	Specific protein/peptide or gene Gliadin and various proteins (not specified)	Cereal Fish Fish paste Sauce Sauce and pasta sauce Sausage	Spiked **Source of spike** Wheat: provided by FASMAC Equal mix of 14 different brands	ELISA
Allred (2012)	Gluten	Gliadins and glutenin fractions	Alcohol Beer Dog food Flour Rice	Spiked **Source of spike** National Institute of Standards and Technology SRM 1567a wheat flour, and a commercial bleached all-purpose wheat flour	EZ Gluten assay immunoassay
Mena (2012)	Gluten	Gliadin	Baby food Biscuit, Breakfast cereals, Bread, Cake, Chips, Cookie, Custard, Flour, Jam, Meat, Pancakes Pudding, Snack, Spice, **Field** Foods sampled	Spiked Field **Source of spike** Not reported	ELISA

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
			wide range of cereals, puddings and baby foods		
Morishita (2006)	Wheat	Crude extract	Carrot Sherbet Cookie Jam Pickles (Soy Sauce, vinegar) Potato Salad Sauce Tomato Soup Steamed and fried Chinese dumpling Hamburger	Spiked	ELISA Immuno-chromatographic test kits Dip stick
Sealey-Voyksner (2010)	Gluten Wheat	Gluten peptides	Cereal Corn flour and wheat	Spiked **Source of spike** Unclear	Mass spectrometry

Table 3.19: Cereals: description of assay

Study ID	Allergen	Assay details	Additional information
Akiyama (2004)	Wheat	Test 1 Wheat protein ELISA kit (Gliadin kit) Test 2 FASTKIT Wheat ELISA Kit	Not stated
Allred (2012)	Gluten	Test 1 EZ Gluten assay	**Immunoassay** Test strips (manufacturer states 10 ppm limit of detection)
Mena (2012)	Gluten	Test 1 ELISA R5 UPEX Test 2 ELISA R5 Modified UPEX	**ELISA** Polyclonal capture antibody Polyclonal detector antibody Competitive inhibition Sandwich
Morishita (2006)	Wheat	Test 1 Immunochromatographic test kits, dipstick. Test 2 ELISA: FASTKIT	
Sealey-Voyksner (2010)	Gluten Wheat	Test 1 Mass spectrometry	**Mass spectrometry** Liquid chromatography-mass spectrometry

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Table 3.20: Cereals: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Akiyama 2004	wheat	Gliadin	1 ELISA	cereals	5-20 ng/ml	53-65		
Akiyama 2004	wheat	Gliadin	1 ELISA	fish paste	5-20 ng/ml	56-59		
Akiyama 2004	wheat	Gliadin	1 ELISA	pasta sauce	5-20 ng/ml	62-65		
Akiyama 2004	wheat	Gliadin	1 ELISA	sauce	5-20 ng/ml	35-44		
Akiyama 2004	wheat	Gliadin	1 ELISA	sausage	5-20 ng/ml	58-68	1 ng/ml	1 ng/ml
Akiyama 2004	wheat	Various proteins	2 ELISA, FASTKIT	cereals	5-20 ng/ml	27-30		
Akiyama 2004	wheat	Various proteins	2 ELISA, FASTKIT	fish paste	5-20 ng/ml	48-52		
Akiyama 2004	wheat	Various proteins	2 ELISA, FASTKIT	pasta sauce	5-20 ng/ml	44-46		
Akiyama 2004	wheat	Various proteins	2 ELISA, FASTKIT	sauce	5-20 ng/ml	48-50		
Akiyama 2004	wheat	Various proteins	2 ELISA, FASTKIT	sausage	5-20 ng/ml	41-45	1 ng/ml	5 ng/ml
Allred 2012	gluten	Crude	1 EZ Gluten	beer			5 ppm	
Allred 2012	gluten	Crude	1 EZ Gluten	dog food			5 ppm	
Allred 2012	gluten	Crude	1 EZ Gluten	dough cooked			5 ppm	
Allred 2012	gluten	Crude	1 EZ Gluten	rice			5 ppm	
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	baby food	55µg/g (one conc.)	101		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	biscuit	55µg/g (one conc.)	105		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	bread	55µg/g (one conc.)	104		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	cake	55µg/g (one conc.)	25		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	cereals	55µg/g (one conc.)	102		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	chips	5 µg/g 5one conc.)	1.3		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	chocolate biscuit	55 ppm (one conc.)	41		

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	chocolate cookie	55µg/g (one conc.)	8		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	cold meat	55µg/g (one conc.)	94		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	cooked ham	55µg/g (one conc.)	103		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	curry powder	55µg/g (one conc.)	21		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	custard	55µg/g (one conc.)	95		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	flour	55µg/g (one conc.)	109		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	jam	55µg/g (one conc.)	32		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	maize pancakes	55µg/g (one conc.)	94		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	paprika	55µg/g (one conc.)	99		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	pepper	55µg/g (one conc.)	44		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	pizza dough	55µg/g (one conc.)	107		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	pudding	55µg/g (one conc.)	108		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	sausage	55µg/g (one conc.)	106		
Mena 2012	gluten	Gliadins	1 ELISA UPEX extraction	snack	55µg/g (one conc.)	92		
Mena 2012	gluten	Gliadins	2 ELISA modified UPEX extraction	biscuit	55µg/g (one conc.)	99		
Mena 2012	gluten	Gliadins	2 ELISA modified UPEX extraction	cake	55µg/g (one conc.)	98		
Mena 2012	gluten	Gliadins	2 ELISA modified UPEX extraction	chocolate cookie	55µg/g (one conc.)	101		

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Mena 2012	gluten	Gliadins	2 ELISA modified UPEX extraction	curry powder	55µg/g (one conc.)	102		
Mena 2012	gluten	Gliadins	2 ELISA modified UPEX extraction	jam	55µg/g (one conc.)	102		
Mena 2012	gluten	Gliadins	2 ELISA modified UPEX extraction	pepper	55µg/g (one conc.)	122		
Morishita 2006	wheat	crude	1 IC - dipstick	chicken meatball or burger			5 µg/g	
Morishita 2006	wheat	crude	1 IC - dipstick	cookie				
Morishita 2006	wheat	crude	1 IC - dipstick	Dumplings fried/ steamed				
Morishita 2006	wheat	crude	1 IC - dipstick	jelly			5 µg/g	
Morishita 2006	wheat	crude	1 IC - dipstick	Pickles in Vinegar/ soy			5 µg/g	
Morishita 2006	wheat	crude	1 IC - dipstick	Potato salad			5 µg/g	
Morishita 2006	wheat	crude	1 IC - dipstick	sauce			5 µg/g	
Sealey-Voyksner 2010	gluten	LQPQNPQQQPQEQVPL	1 Mass Spec	corn	0.06-60 pg/mg	93-99		
Sealey-Voyksner 2010	gluten	TQQPQQPFPQQPQQPFPQ	1 Mass Spec	corn	0.06-60 pg/mg	86-96		
Sealey-Voyksner 2010	gluten	VPVPQLQPQNPSQQQPQEQVPL	1 Mass Spec	corn	0.06-60 pg/mg	97-104		
Sealey-Voyksner 2010	gluten	RPQQPYPQPQPQY	1 Mass Spec	corn	0.06-60 pg/mg	90-98		
Sealey-Voyksner 2010	gluten	PQQSPF	1 Mass Spec	corn	0.06-60 pg/mg	69-108		
Sealey-Voyksner 2010	gluten	LQPQNPQQQPQEQVPL	1 Mass Spec	corn flour	10-1000 pg/mg	78-104	3.5	20
Sealey-Voyksner 2010	gluten	TQQPQQPFPQQPQQPFPQ	1 Mass Spec	corn flour	10-1000 pg/mg	91-102	25	100
Sealey-Voyksner 2010	gluten	VPVPQLQPQNPSQQQPQEQVPL	1 Mass Spec	corn flour	10-1000 pg/mg	93-103	14	50

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Sealey-Voyksner 2010	gluten	RPQQPYPQPQPQY	1 Mass Spec	corn flour	10-1000 pg/mg	85-103	3	20
Sealey-Voyksner 2010	gluten	QPQQPFPQTQQPQQPFPQ	1 Mass Spec	corn flour	10-1000 pg/mg	77-109	30	100
Sealey-Voyksner 2010	gluten	PQQSPF	1 Mass Spec	corn flour	10-1000 pg/mg	83-112	1	10
Sealey-Voyksner 2010	gluten	LQPQNPQQQPQEQVPL	1 Mass Spec	wheat	0.06-60 pg/mg	93-99		
Sealey-Voyksner 2010	gluten	TQQPQQPFPQQPQQPFPQ	1 Mass Spec	wheat	0.06-60 pg/mg	86-96		
Sealey-Voyksner 2010	gluten	VPVPQLQPQNPSQQQPQEQVPL	1 Mass Spec	wheat	0.06-60 pg/mg	97-104		
Sealey-Voyksner 2010	gluten	RPQQPYPQPQPQY	1 Mass Spec	wheat	0.06-60 pg/mg	90-98		
Sealey-Voyksner 2010	gluten	QPQQPFPQTQQPQQPFPQ	1 Mass Spec	wheat	0.06-60 pg/mg	90-99		
Sealey-Voyksner 2010	gluten	PQQSPF	1 Mass Spec	wheat	0.06-60 pg/mg	69-108		

Mass Spec= Mass spectrometry

3.2.7. Egg

Ovomucoid, the major allergen (Gal d1) is less abundant in the egg white than ovalbumin which is also a major allergen (Gal d2) and the most abundant protein in egg white. Other major allergens in egg white are ovotransferrin and lysozyme. The latter is of particular interest as hen's egg lysozyme can be used in wine production and cheese making. Alphalivetin Gal d 5 is present in the egg yolk. Within this review studies developing or validating assay to detect crude extract, ovalbumin, ovomucoid or lysozyme were found and included.

Six studies investigating assays or egg proteins were included in the review, the assays included ELISA (in-house and commercial), time-of-flight mass spectrometry and dipstick techniques (Table 3.21). Akiyama (2003) compared three ELISAs using bread, cereals and sauces as the food matrix. The two ELISA kits developed by the Morinaga Institute of Biological Sciences detected ovalbumin and ovomucoid and provided good recovery (in most cases more than 80%) for samples spiked with 5-20 ng/ml, and the limit of detection in a sausage mixture was 4 ng/ml (0.001 µg/ml). Khuda 2012b also found good recovery from dark chocolate, but very poor recovery from sugar cookie at just 15%. However, it should be noted that none of the assays tested by Khuda demonstrated good recoveries from the sugar cookie mix. The FASTKIT ELISA tested by Akiyama (2003) did not perform as well as the Morinaga system, giving recoveries of less than 50% for sausage spiked with egg. The Veratox ELISA was tested by Khuda (2012b) where recoveries were very high, more than 200% from dark chocolate but as mentioned before poor from sugar cookie at less than 10%. The ELISA-BIOKITS was less effective than the other assays for dark chocolate, and similarly poor for the sugar cookie mixture, Khuda (2012 b). Shon (2010) developed an in-house ELISA against ovomucoid and achieved acceptable recoveries from

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sausage and milk substitute when spiked with 10 µg/g. Lacorn (2011), used an in-house sandwich ELISA to detect egg powder or cooked egg powder in wine and achieved recoveries of 76-110% and a limit of detection of 0.27 µg/ml.

The IC – dipstick provided a limit of detection of 5 µg/g in a wide range of foods, Morishita (2006), and had the advantage of ease of use. Schneider (2010a) developed a method for mass spectrometry that achieved a limit of detection of 5 µg/g for lysozyme in cheese.

Table 3.21: Egg: characteristics of included studies

Study ID	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Akiyama (2003)	Specific protein/peptide or gene Ovalbumin Ovomucoid	Bread Cereal Cookie Sauce Sausage	Spiked Dose of spike 5-20ng/mL **Source of spike** Egg (Nippon Meat Packers, Inc), fresh egg from white leghorn hens	ELISA
Hefle (2001)	Specific protein/peptide or gene Ovalbumin	Pasta	Spiked **Source of spike** Spray-dried egg yolk solids (Hershey foods Co, Hershey, Pa, USA)	ELISA
Khuda (2012a) egg	Crude extract Spray dried egg powder	Dark Chocolate	**Source of spike** Spray dried egg powder-NIST RM 8445 (National Institute of Standards and Technology, Gaithersburg, MD, USA) Standardisation Unclear	ELISA
Khuda (2012b) egg	Crude extract	Cookie	**Source of spike** Spray dried whole egg powder, NIST RM 8445 (National Institute of Standards and Technology)	ELISA
Lacorn (2011)	Crude extract	Wine	**Spiked and source** Spray dried whole egg powder (National Institute of standards and Technology,) Whole egg and white: Henningsen Foods (Omaha, NE) Cooked egg white: prepared in house. Food grade liquid egg white (Eifix Eiweiss, Wiesenhof, Germany) Standardisation Total protein: whole egg 48± 1 %, Durmas method. Egg white powder: P-11 protein content 83.8 % (Kjehldahl determination), cooked egg white: protein, using BCA (2.8 mg/ml) Food grade liquid egg: 99 g protein/kg Kjehldahl	ELISA
Morishita (2006)	Crude extract	Carrot Sherbet Cookie Jam, Pickles (Soy Sauce, vinegar),	Spiked	ELISA Immuno-chromatographic test kits

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Study ID	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
		Potato Salad, Sauce Tomato, Soup Steamed and fried Chinese dumpling Hamburger		Dip stick
Schneider (2010a)	Specific protein/peptide or gene Lysozyme	Cheese **Field** Foods sampled Commercial parmesan cheese	Spiked Field **Source of spike** Cheese samples (Manchego, Grana Padano, Parmigiano Reggiano and hard cheese mixtures) were purchased in a local supermarket	Mass spectrometry
Schneider (2010b)	Specific protein/peptide or gene Lysozyme	Cheese	Spiked **Source of spike** Lysozyme from Sigma-Aldrich	ELISA
Shon (2010)	**Specific protein/peptide or gene** Ovomucoid	Milk milk substitute Sausage commercial sausage, in-house sausage **Field** Foods sampled crab meat analogue, sausage	Spiked Field **Source of spike** whole egg powder and egg white powder provided by Nonghyup (Pyeongtaek, Korea)	ELISA

Table 3.22: Egg: description of assay

Study ID	Allergen	Assay details	Additional information
Akiyama (2003)	Egg	Test 1 Egg protein ovalbumin ELISA kit (Morinaga Institute of Biological Sciences) Test 2 Egg protein ovomucoid ELISA kit (Morinaga Institute of Biological Sciences) Test 3 FASTKIT Egg ELISA Kit (Nippon Meat Packers, Inc.)	Commercial assay
Hefle (2001)	Egg	Test 1 ELISA	**ELISA** ICP-MS Sandwich In-house
Khuda (2012a) egg	Egg	Test 1 RIDASCREEN FAST peanut, egg, and casein from R-Biopharm (RB, Washington, MO, USA) Test 2	**ELISA** Commercial company

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Study ID	Allergen	Assay details	Additional information
		Veratox peanut, egg, and total milk allergen quantitative test kits from Neogen (NE) Corp. (Lansing, MI, USA) Test 3 Morinaga (MO) peanut, egg, and milk (casein and BLG) protein ELISA kits (Crystal Chem, Downers Grove, IL, USA) Test 4 Tepnel (TE) BIOKITS peanut, egg, casein, and BLG assay kits (Neogen Corp.) Test 5 ELISA Systems (ES) peanut, egg, casein, and BLG residue kits (BioMerieux, Durham, NC, USA)	
Khuda (2012b) egg	Egg	Test 1 RIDASCREEN FAST peanut, egg, and casein from R-Biopharm (RB, Washington, MO, USA) Test 2 Veratox peanut, egg, and total milk allergen quantitative test kits from Neogen (NE) Corp. (Lansing, MI, USA) Test 3 Morinaga (MO) peanut, egg, and milk (casein and BLG) protein ELISA kits (Crystal Chem, Downers Grove, IL, USA) Test 4 Tepnel (TE) BIOKITS peanut, egg, casein, and BLG assay kits (Neogen Corp.) Test 5 ELISA Systems (ES) peanut, egg, casein, and BLG residue kits (BioMerieux, Durham, NC, USA)	**ELISA** Commercial company
Lacorn (2011)	Egg	Test 1 ELISA In- house	**ELISA** Polyclonal detector Sandwich
Morishita (2006)	Egg	Test 1 Immunochromatographic test kits, dipstick. Test 2 ELISA: FASTKIT	Commercial company
Schneider (2010a)	Egg	Test 1 **Mass spectrometry**	**Mass spectrometry** Time of flight-mass spectrometry
Schneider (2010b)	Egg lysozyme	Test 1 ELISA	**ELISA** Monoclonal detection antibody Competitive inhibition In-house
Shon (2010)	Egg	Test 1 ELISA	**ELISA** Polyclonal detection antibody Competitive inhibition In-house

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Table 3.23: Egg: accuracy and limit of detection and quantification

Study ID	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Akiyama 2003	Ovalbumin	1 ELISA Morinaga	bread	5-20 ng/ml	81-86		
Akiyama 2003	Ovalbumin	1 ELISA Morinaga	cereals	5-20 ng/ml	85-87		
Akiyama 2003	Ovalbumin	1 ELISA Morinaga	cookie	5-20 ng/ml	90-101		
Akiyama 2003	Ovalbumin	1 ELISA Morinaga	sauce	5-20 ng/ml	71-82		
Akiyama 2003	Ovalbumin	1 ELISA Morinaga	sausage	5-20 ng/ml	92-105	4 ng/ml	8 ng/ml
Akiyama 2003	Ovomucoid	2 ELISA Morinaga	bread	5-20 ng/ml	88-107		
Akiyama 2003	Ovomucoid	2 ELISA Morinaga	cereals	5-20 ng/ml	89-108		
Akiyama 2003	Ovomucoid	2 ELISA Morinaga	cookie	5-20 ng/ml	104-167		
Akiyama 2003	Ovomucoid	2 ELISA Morinaga	sauce	5-20 ng/ml	57-65		
Akiyama 2003	Ovomucoid	2 ELISA Morinaga	sausage	5-20 ng/ml	91-131	5 ng/ml	10 ng/ml
Akiyama 2003	Standard egg protein	3 ELISA, FASTKIT	bread	5-20 ng/ml	45-49		
Akiyama 2003	Standard egg protein	3 ELISA, FASTKIT	Cookie	5-20 ng/ml	45-48		
Akiyama 2003	Standard egg protein	3 ELISA, FASTKIT	sauce	5-20 ng/ml	46-49		
Akiyama 2003	Standard egg protein	3 ELISA, FASTKIT	sausage	5-20 ng/ml	44-45		
Akiyama 2004	Standard egg protein	3 ELISA, FASTKIT	cereals	5-20 ng/ml	42-43		
Khuda 2012a	crude	1 ELISA, RIDASCREEN FAST	chocolate dark	linear regression	255		
Khuda 2012a	crude	2 ELISA, Veratox	chocolate dark	linear regression	283		
Khuda 2012a	crude	3 ELISA, Morinaga	chocolate dark	linear regression	76		
Khuda 2012a	crude	4 ELISA, BIOKITS	chocolate dark	linear regression	58		
Khuda 2012a	crude	5 ELISA Systems	chocolate dark	linear regression	66		
Khuda 2012b	crude	1 ELISA, RIDASCREEN FAST	sugar cookie	linear regression	10		
Khuda 2012b	crude	2 ELISA, Veratox	sugar cookie	linear regression	9		

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Study ID	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Khuda 2012b	crude	3 ELISA, Morinaga	sugar cookie	linear regression	15		
Khuda 2012b	crude	4 ELISA, BIOKITS	sugar cookie	linear regression	4		
Khuda 2012b	crude	5 ELISA Systems	sugar cookie	linear regression	3		
Lacorn 2011	egg white powder	1 ELISA	wine	1-9 µg /ml	98-110		
Lacorn 2011	cooked egg white	1 ELISA	wine	2-18 µg /ml	76-88		
Lacorn 2011	whole egg powder	1 ELISA	wine	1.5-13.5 µg /ml	87-109	0.27 µg /ml	0.5 mg/L
Morishita 2006	crude	1 IC - dipstick	chicken meatball or burger			5 µg/g	
Morishita 2006	crude	1 IC - dipstick	cookie			5 µg/g	
Morishita 2006	crude	1 IC - dipstick	Dumplings fried/steamed			5 µg/g	
Morishita 2006	crude	1 IC - dipstick	jelly			5 µg/g	
Morishita 2006	crude	1 IC - dipstick	Pickles in Vinegar/soy			5 µg/g	
Morishita 2006	crude	1 IC - dipstick	Potato salad			5 µg/g	
Morishita 2006	crude	1 IC - dipstick	sauce			5 µg/g	
Schneider 2010a	lysozyme	1 Time-of-flight mass spectrometry	cheese			5 µg/g	
Schneider 2010b	lysozyme	1 ELISA	cheese	50-400 mg/kg	87-94	2.73 ng/ml	
Shon 2010	ovomucoid	1 ELISA	egg free sausage	10-100 mg/kg	74		
Shon 2010	ovomucoid	1 ELISA	in house sausage	5-30mg/kg	66		
Shon 2010	ovomucoid	1 ELISA	milk substitute	10-100 mg/kg	129		

3.2.8. Fish and Shellfish

The majority of assays for fish and shellfish in foods were developed to detect parvalbumin the major fish allergen or tropomyosin the main allergen for a wide range of shellfish (Table 3.24). Both these allergenic proteins are relatively heat stable. The assays investigated included ELISA and PCR (Table 3.25). The majority of food matrices tested were liquids such as soups, and in the findings indicated that for most assays there was relatively good recovery (Table 3.26)

Cai (2013) developed and tested a parvalbumin ELISA over a wide range of concentrations 10-1000 ng/ml and demonstrated good recovery rates of 70-140%. Faeste (2008) developed their own in-house

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ELISA that gave a limit of detection of 0.01 µg/g, however they did not present the recovery rates. Shibahara (2013b) also developed and validated an ELISA to parvalbumin showing acceptable recoveries for matrices such as meatballs and potato products when were with 10 ppm (10 µg/g).

Shibahara (2007) developed an ELISA for detecting shrimp and crab tropomyosin and although the limit of detection for different food matrices was not presented, the study demonstrated that for foods spiked with as little as 10ppm (10 µg /g) the percentage recovery ranged from 64-82%. Fuller (2007) showed similar sensitivity for an in-house ELISA to detect tropomyosin and Wener (2007) showed good recovery rates when samples were spiked with as little as 1 µg/ml and the limit of detection was as low as 0.2 µg/ml in certain foods.

One by study by Taguchi (2011) investigated PCR to detect the DNA of crab, and this assay showed a limit of detection in a similar region of 10 µg/g. This PCR had the advantage that it can discriminate between shrimp and crab unlike the two commercial LISA kits that it was tested against (*Table 3.25)*.

Table 3.24: Fish and Shellfish: characteristics of included studies

Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Cai (2013)	Fish Silver Carp	Parvalbumin Silver Carp	Soup Tofu and mushroom	Spiked **Source of spike** Local market in Xiamen, China	ELISA
Faeste (2008)	Fish Cod	Specific protein/peptide or gene Parvalbumin	Sauce white sauce, soy sauce Soup fish soup, mushroom soup **Field** Foods sampled fish soup	Spiked Field **Source of spike** Gadus morhua	ELISA
Fuller (2006)	Fish and shellfish Crustaceans	Specific protein/peptide or gene Tropomyosin	Ocean Pie, Quiche Rice: pilau rice Sauce, Soy sauce, lemon and dill sauce, Spread: tuna and sweet corn spread Thai crackers Vegetable balti	Spiked **Source of spike** Penaeus latisulcatus, shop brought	ELISA
Shibahara (2007)	Fish and shellfish Crustaceans	Specific protein/peptide or gene Tropomyosin	cream croquette Pork dumpling Tomato sauce	Spiked **Source of spike** Extracted from freeze-dried black tiger prawns	ELISA
Shibahara (2013b)	Fish	Parvalbumin	cream croquette Meat: chicken meatball, pork meatball Rice: rice gruel	Spiked Field **Source of spike** Five species of fish: Japanese eel Anguilla	ELISA

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
			Soup: vegetable and chicken soup	japonica, horse mackerel Trachurus japonicus, crimson sea bream Evynnis japonica, pacific mackerel S.japonicus and bigeye tuna Thunnus obesus.	
Taguchi (2011)	Fish and shellfish Crustacean shrimp, crab	Crude extract	Cream croquette Rice dry condiment sprinkled on rice, rice gruel Soup freeze-dried soup, miso soup paste, soup powder **Field** Foods sampled 27 commercial food products, purchased from local stores	Spiked Field **Source of spike** Markets in Tokyo and Chiba, Japan, or provided by Maruha Nichiro Holdings, Inc.	ELISA PCR
Werner (2007)	Fish and shellfish Crustaceans	Specific protein/peptide or gene Tropomyosin	Fish breaded codfish, fish cake Sauce fish sauce, mayonnaise Surimi	Spiked **Source of spike** Pandalus borealis	ELISA

Table 3.25: Fish and Shellfish: description of assay

Study ID	Allergen	Assay details	Additional information
Cai (2012)	Fish Silver Carp Parvalbumin	Test 1 ELISA	**ELISA** Monoclonal detection antibody Competitive inhibition In-house
Faeste (2008)	Fish	Test 1 ELISA	**ELISA** Sandwich In-house
Fuller (2006)	Crustaceans	Test 1 ELISA	**ELISA** Polyclonal capture antibody Polyclonal detection antibody Sandwich In-house
Shibahara (2007)	Crustaceans	Test 1 ELISA	**ELISA** Monoclonal capture antibody Polyclonal detection antibody

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Study ID	Allergen	Assay details	Additional information
			In-house
Shibahara (2013b)	Fish	Test 1 ELISA	**ELISA** Polyclonal capture antibody Polyclonal detection antibody Sandwich In-house
Taguchi (2011)	Crustaceans shrimp, crab	Test 1 EIA crustacean 'Nissui' ELISA Test 2 crustacean kit 'Maruha' ELISA Test 3 Shrimp PCR Test 4 Crab PCR In-house	Test 1 Nissui Pharmaceutical Co., Ltd., Toshima-ku, Tokyo Test 2 Maruha Nichiro Holdings, Inc. Test 3 PCR, in-house Test 4 PCR, in-house
Werner (2007)	Crustaceans	ELISA	ELISA Polyclonal capture antibody Polyclonal detection antibody Sandwich In-house

Table 3.26: Fish and Shellfish: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Cai 2013	silver carp	parvalbumin	1 ELISA	mushroom soup	10-1000 ng/ml	87.7 - 97.8		
Cai 2013	silver carp	parvalbumin	1 ELISA In-house	tofu soup	10-1000 ng/ml	70.3 - 134.8		
Faeste 2008	fish	cod parvalbumin	1 ELISA In-house	buffer			0.01 µg/g	0.02 µg/g
Faeste 2008	fish	cod parvalbumin	1 ELISA In-house	mushroom soup			0.01 µg/g	0.02 µg/g
Faeste 2008	fish	cod parvalbumin	1 ELISA In-house	sauce			0.01 µg/g	0.02 µg/g
Faeste 2008	fish	cod parvalbumin	1 ELISA In-house	soup			0.01 µg/g	0.02 µg/g
Faeste 2008	fish	cod parvalbumin	1 ELISA In-house	soy sauce			0.01 µg/g	0.02 µg/g
Fuller 2007	crustaceans	tropomyosin	1 ELISA In-house	buffer	nr	nr	1 µg/g	
Fuller 2007	crustaceans	tropomyosin	1 ELISA In-house	casserole/ curry	7.5 ppm	67-86		
Fuller 2007	crustaceans	tropomyosin	1 ELISA In-house	crisps/ Thai cracker	7.5 ppm	99-140		
Fuller 2007	crustaceans	tropomyosin	1 ELISA In-house	pie/ quiche	7.5 ppm	41-112		

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Fuller 2007	crustaceans	tropomyosin	1 ELISA In-house	pie/ quiche	7.5 ppm	74-84		
Fuller 2007	crustaceans	tropomyosin	1 ELISA In-house	rice	7.5 ppm	76-117		
Fuller 2007	crustaceans	tropomyosin	1 ELISA In-house	sauce	7.5 ppm	85-124		
Fuller 2007	crustaceans	tropomyosin	1 ELISA In-house	soy sauce	7.5 ppm	76-87		
Fuller 2007	crustaceans	tropomyosin	1 ELISA In-house	spread	7.5 ppm	117-143		
Shibahara 2007	crustaceans	tropomyosin	1 ELISA In-house	buffer			0.4 ng/ml	1.2 ng/ml
Shibahara 2007	crustaceans	tropomyosin	1 ELISA In-house	croquette	2,10,16 ppm	88-103		
Shibahara 2007	crustaceans	tropomyosin	1 ELISA In-house	Dumplings fried/steamed	2,10,16 ppm	94-105		
Shibahara 2007	crustaceans	tropomyosin	1 ELISA In-house	sauce	2,10,16 ppm	94-104		
Shibahara 2013b	fish	parvalbumin	1 ELISA In-house	buffer			0.23 ng/ml	0.7 µg/g
Shibahara 2013b	fish	parvalbumin	1 ELISA In-house	chicken meatball or burger	10 ppm	73.5		
Shibahara 2013b	fish	parvalbumin	1 ELISA In-house	pork meatball or burger	10 ppm	81.8		
Shibahara 2013b	fish	parvalbumin	1 ELISA In-house	potato croquette or mash	10 ppm	63.6		
Shibahara 2013b	fish	parvalbumin	1 ELISA In-house	range of products				
Shibahara 2013b	fish	parvalbumin	1 ELISA In-house	rice gruel/ porridge	10 ppm	78.8		
Shibahara 2013b	fish	parvalbumin	1 ELISA In-house	soup	10 ppm	78.7		
Taguchi 2011	crustaceans	DNA -shrimp	3 shrimp PCR	miso			10 µg/g	
Taguchi 2011	crustaceans	DNA -shrimp	3 shrimp PCR	soup			10 µg/g	
Taguchi 2011	crustaceans	DNA -shrimp	3 shrimp PCR	soup			10 µg/g	
Taguchi 2011	crustaceans	DNA -crab	4 crab-PCR	miso			10 µg/g	
Taguchi 2011	crustaceans	DNA -crab	4 crab-PCR	soup			10 µg/g	
Taguchi 2011	crustaceans	DNA -crab	4 crab-PCR	soup			10 µg/g	
Taguchi 2011	crustaceans	DNA -shrimp	3 shrimp PCR	chicken meatball or burger			10 µg/g	

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Taguchi 2011	crustaceans	DNA -shrimp	3 shrimp PCR	croquette			10 µg/g	
Taguchi 2011	crustaceans	DNA -shrimp	3 shrimp PCR	range of products				
Taguchi 2011	crustaceans	DNA -shrimp	3 shrimp PCR	rice gruel/ porridge			10 µg/g	
Taguchi 2011	crustaceans	DNA -crab	4 crab-PCR	chicken meatball or burger			10 µg/g	
Taguchi 2011	crustaceans	DNA -crab	4 crab-PCR	croquette			10 µg/g	
Taguchi 2011	crustaceans	DNA -crab	4 crab-PCR	range of products				
Taguchi 2011	crustaceans	DNA -crab	4 crab-PCR	rice gruel/ porridge			10 µg/g	
Werner 2007	crustaceans	tropomyosin	1 ELISA In-house	fish	1-100 µg/ml	68-83	0.3 µg/g	
Werner 2007	crustaceans	tropomyosin	1 ELISA In-house	mayonnaise	1-100 µg/ml	102-120	0.2 µg/g	
Werner 2007	crustaceans	tropomyosin	1 ELISA In-house	sauce	1-100 µg/ml	79-94	0.3 µg/g	
Werner 2007	crustaceans	tropomyosin	1 ELISA In-house	surimi	1-100 µg/ml	66-88	0.9 µg/g	

3.2.9. Hazelnut

A range of immunoassays including dipstick tests and real-time PCR assays were validated by the studies included within this review and the food matrices used included chocolate, cereals and cookies (Table 3.27). Commercial and in-house tests were investigated (Table 3.28). Most of the assays investigated could detect to below 10 ppm (10 µg/g) in a range of samples, including milk chocolate (Table 3.29).

Akkerdaas (2004), Ben Rejeb (2003), Ben Rejeb (2005h), Blais (2001), Cucu (2012), Drs (2004), Holzhauser (1999) developed and validated in-house ELISAs. All provided a limit of detection as low as or lower than 1 µg/g with recoveries of over 50%.

Commercial assays tested included Veratox, Garber (2010), ELISA systems, Garber 2010) Ridascreen, Ehlert (2009), Garber (2010), Piknova (2008) with a limit of detection between 1 and 6 µg/g The exception was Ridascreen tested by Ehlert (2009) that gave a limit of detection of 10 mg/kg^{-1}, equivalent to 100 µg/g. All assays were directed against crude extracts rather than purified proteins.

Ehlert (2009) developed and validated a ligation-dependent probe amplification system for simultaneous detection of DNA from a number of allergenic foods. The limit of detection in food matrices such as chocolate and cookies was 10 mg/kg^{-1} that equates to 100 µg/g. Faeste (2006) developed a time-resolved fluoro-imunoassay and this had reasonable recoveries of between 5-123 % for matrices spiked with 1-150 mg/kg (1-150 µg/g).

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Table 3.27: Hazelnut: characteristics of included studies

Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Akkerdaas (2004)	Hazelnut	**Tree nut** Hazelnut Crude extract Pepsin stable	Chocolate	Spiked **Source of spike** Turkish variety, local store	ELISA
Ben Rejeb(2003)	Hazelnut	Crude extract	Breakfast cereals Chocolate Cookie Ice cream	Spiked **Source of spike** Roasted hazelnuts from a local store Standardisation Total protein content using the Bradford test	ELISA
Ben Rejeb (2005h)	Hazelnut	Crude extract	Chocolate	Spiked Roasted defatted peanuts and nuts extracted, dialysed **Source of spike** Not reported Standardisation Made up to 1mg/ml^{-1} protein content measured using BCA test	ELISA
Blais (2001)	Hazelnut	Crude extract	Breakfast cereals Cake cake mix Cereal Bar fruit and almond granola bars Chocolate Cookie Ice cream	Spiked **Source of spike** Shelled raw hazelnuts ground and defatted salted and centrifuged. Standardisation Total protein 35 mg/ml (determined using Biorad)	ELISA
Costa (2012)	Hazelnut	Specific protein/peptide or gene hsp1 gene m RNA	Pasta	Spiked Field **Source of spike** Unclear 'commercial hazelnut'	PCR
Cucu (2012)	Hazelnut	Crude extract	Cookie Both before and after cooking	Spiked **Source of spike** 9 different brands of hazelnut (8 raw, 1 roasted) were purchased in supermarkets	ELISA
Drs (2004)	Hazelnut	Crude extract roasted hazelnut	Cookie	Spiked **Source of spike** Masterfoods (Breitenbiunn, Austria)	ELISA
Ehlert (2009)	Hazelnut	Crude extract Specific protein/peptide or gene DNA	Cookie Pesto cashew	Spiked **Source of spike** Nut materials, sesame seeds, ingredients of self-prepared DNA plant and animal materials used to	ELISA PCR Ligation-dependent probe amplification

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
				test the specificity of the method and spike samples of chocolate, were obtained from the Bavarian Health and Food Safety Authority (Oberschleibheim, Germany)	
Faeste (2006)	Hazelnut	Specific protein/peptide or gene Corylin fraction	Breakfast cereals Chocolate Cookie	Spiked. **Source of spike** Raw Mina hazelnuts (Iran) Standardisation Raw nuts were homogenised, suspended in buffer, vortexed, centrifuged and filtered through glass wool. Total protein content was determined using the Lowrey method, and protein standard solution was diluted with PBS to 2 mg/ml	Time-resolved fluoroimmuno-assay
Garber (2010b)	Hazelnut	Crude extract	Breakfast cereals oatmeal Cake muffins Chocolate	Spiked **Source of spike** purchased locally	ELISA
Holzhauser (1999)	Hazelnut	Crude extract	Cereal Bar yoghurt cereal bar Chocolate almond candy cream chocolate bar Cookie	Spiked Field **Source of spike** Piemonte and Nocciole Ordu, provided by Dr G Malgarini, Sorematx, Arlon-Schoppach, Belgium. toasted. Standardisation soluble protein was quantified by the Bradford method	ELISA
Holzhauser (2002)	Hazelnut	Crude extract	Cereal bar Chocolate **Field** Foods sampled A range of products including chocolates, chocolates with nuts, nougat, milk products	**Source of spike** Hazelnuts of the variety Nocciole Ordu (Turkey) both native and toasted at 140 ^{o}C for 30 min, were provided by Dr G Malgarini, Sorematec, Arlon-Schoppach, Belguim. Commercial food products were bought at a local food store.	ELISA PCR PCR ELISA

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Kiening (2005)	Hazelnut	Crude extract	Breakfast cereals Chocolate Cookie Ice cream **Field** Foods sampled cookie, cereals and chocolate	Spiked Field **Source of spike** Roasted hazelnut samples were provided by R. Fila from Masterfoods, Breitenbrunn, Austria.	ELISA
Piknova (2008)	Hazelnut	Specific protein/peptide or gene hsp1 gene	Pastry or dough **Field** Foods sampled Chocolate, wafers, muesli and biscuits	Spiked Field **Source of spike** Five cultivars from Botanical garden, Slovak Agricultural University, Nitra Slovkia.	ELISA PCR
Stephan (2002)	Hazelnut	Specific protein/peptide or gene Corylin fraction Hazelnut	Chocolate Rausch Schokoladen Gmbh (Peine, Germany) **Field** Foods sampled Range of foods labelled as containing, not containing and may contain peanut or hazelnut	Spiked Field **Source of spike** Not reported	Dipstick

Table 3.28: Hazelnut: description of assay

Study ID	Allergen	Assay details	Additional information
Akkerdaas (2004)	Hazelnut	Test 1 ELISA	**ELISA** Polyclonal capture antibody Polyclonal detection antibody In-house
Ben Rejeb (2003)	Hazelnut	Test 1 ELISA	**ELISA** Competitive Polyclonal detector antibody In-house
Ben Rejeb (2005h)	Hazelnut	ELISA	**ELISA** Polyclonal detection antibody Competitive inhibition In-house
Blais (2001)	Hazelnut	Test 1 ELISA	**ELISA** Polyclonal capture antibody Polyclonal detection antibody Sandwich In-house

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Study ID	Allergen	Assay details	Additional information
Costa (2012)	Hazelnut	Test 1 Real-time PCR Test 2 Nested real-time PCR	PCR
Cucu (2012)	Hazelnut	Test 1 ELISA	**ELISA** Polyclonal detection antibody Competitive inhibition In-house
Drs (2004)	Hazelnut	Test 1	**ELISA** Competitive inhibition Indirect competitive ELISA In-house
Ehlert (2009)	Hazelnut	Test 1 Ligation dependent probe amplification Test 2 Hazelnut and peanut: real-time PCR Surefood allergen kit (Congen Biotechnology GmbH, Berlin, Germany) cashew real time PCR In house Test 3 Hazelnut and peanut: ELISA Ridascreen (R-Biopharm AG, Darmstadt, Germany)	Test 1 PCR-LPA Test 2 PCR Test 3 ELISA Commercial
Faeste (2006)	Hazelnut	Test 1 Fluoro-immunoassay	Time-resolved fluoro-immunoassay
Garber (2010b) hazelnut	Hazelnut	Test 1 Veratox (Neogen Corporation, Lansing, MI, USA); Test 2 Ridascreen Fast (R-Biopharm Inc, Marshal, MI, USA), Test 3 Elisa systems(bioMerieux Industry (Hazelwood, MO, USA)	**Test 1, 2 and 3** **ELISA** Sandwich Commercial company
Holzhauser (1999)	Hazelnut	Test 1 ELISA	**ELISA** Polyclonal capture antibody Polyclonal detection antibody Sandwich In-house
Holzhauser (2002)	Hazelnut	Test 1 ELISA Test 2 PCR-ELISA	**Test 1** ELISA Polyclonal capture antibody Polyclonal detection antibody Sandwich Commercial company **Test 2** PCR-ELISA Commercial company SureFood-Allergen Hazelnut test (Congen Biotechnology, No.

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Study ID	Allergen	Assay details	Additional information
			S3002)
Piknova (2008)	Hazelnut	Test 1 Real-time PCR hsp 1 Test 2 ELISA RidaScreen FAST Hazelnut (R-Biopharm, Darmstadt, Germany	**Test 1** **PCR** **Test 2** **ELISA** Commercial company
Stephan (2002)	Hazelnut	Test 1 Dipstick: in-house	Dipstick method In house. Polyclonal capture antibody Polyclonal detector antibody. In-house

Table 3.29: Hazelnut: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Akkerdaas 2004	hazelnut	Crude pepsin stable	1 ELISA	buffer			0.7 ng/ml	
Akkerdaas 2004	hazelnut	Crude pepsin stable	1 ELISA	chocolate milk	0.5 -100 µg/g	53-120	0.5 µg/g	
Akkerdaas 2004	hazelnut	Crude pepsin stable	1 ELISA	range of products				
Ben Rejeb 2003	hazelnut	Crude-roasted	1 ELISA	breakfast cereal	1-10 µg/g	80-93		
Ben Rejeb 2003	hazelnut	Crude-roasted	1 ELISA	chocolate dark	1-10 µg/g	64-83	0.5 µg/g	
Ben Rejeb 2003	hazelnut	Crude-roasted	1 ELISA	chocolate milk	1-10 µg/g			
Ben Rejeb 2003	hazelnut	Crude-roasted	1 ELISA	cookie	1-10 µg/g	89-97		
Ben Rejeb 2003	hazelnut	Crude-roasted	1 ELISA	ice cream	1-10 µg/g	78-83		
Ben Rejeb 2005	hazelnut		1 ELISA	chocolate dark			1 ppm	
Blais 2001	hazelnut	Crude	1 EIA (ELISA)	breakfast cereal			0.25 ppm	
Blais 2001	hazelnut	Crude	1 EIA (ELISA)	Cake			0.12 ppm	
Blais 2001	hazelnut	Crude	1 EIA (ELISA)	chocolate milk			0.25 ppm	
Blais 2001	hazelnut	Crude	1 EIA (ELISA)	cookie			0.5 ppm	
Blais 2001	hazelnut	Crude	1 EIA (ELISA)	ice cream			0.25 ppm	
Blais 2001	hazelnut	Crude	1 EIA (ELISA)	muesli			0.5 ppm	

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Blais 2001	hazelnut	Crude	1 EIA (ELISA)	snack cereal			1 ppm	
Blais 2001	hazelnut	Crude	1 EIA (ELISA)	snack cereal			0.5 ppm	
Costa 2012	hazelnut	hsp 1	1 Real time PCR	Pasta	Nr	nr	100 mg/kg	100 mg/kg
Costa 2012	hazelnut	hsp 1	2 Nested Real time PCR	Pasta			50 mg/kg	50 mg/kg
Costa 2012	hazelnut	hsp 1	2 Nested real-time PCR	range of products	Nr	nr	nr	nr
Cucu 2012	hazelnut	crude	1 ELISA	cookie	30-100 µg/g	10 - 20		
Cucu 2012	hazelnut	crude	1 ELISA	cookie (spiked after baking)	3-25 µg/g	73-107		
Drs 2004	hazelnut	crude	1 ELISA	cookie		128	10 µg/L^{-1}	30 µg/L-1
Ehlert 2009	hazelnut	DNA	1 LPA	chocolate	Nr	nr	5 mg/kg^{-1}	
Ehlert 2009	hazelnut	DNA	1 LPA	walnut cookies	Nr	nr	100 mg/kg^{-1}	
Ehlert 2009	hazelnut	DNA	2 PCR real time	chocolate			10 mg/kg^{-1}	
Ehlert 2009	hazelnut	DNA	2 PCR real time	walnut cookies			10 mg/kg^{-1}	
Ehlert 2009	hazelnut	crude	3 ELISA *Ridascreen*	chocolate			10 mg/kg^{-1}	
Ehlert 2009	hazelnut	crude	3 ELISA *Ridascreen*	walnut cookies			1 mg/kg^{-1}	
Faeste 2006	hazelnut	Corylin fraction	1 *Fluoro-IA*	cereals	1-150 mg/kg	77-123		
Faeste 2006	hazelnut	Corylin fraction	1 *Fluoro-IA*	cereals	1-150 mg/kg	54-77		
Faeste 2006	hazelnut	Corylin fraction	1 *Fluoro-IA*	chocolate milk	1-150 mg/kg	50-71		
Faeste 2006	hazelnut	Corylin fraction	1 *Fluoro-IA*	cookie	1-150 mg/kg	73-97		
Garber 2010 hazelnut	hazelnut	crude	1 ELISA, Veratox	Cake			5.6 µg/g	
Garber 2010 hazelnut	hazelnut	crude	1 ELISA, Veratox	cereals			1.4 µg/g	
Garber 2010 hazelnut	hazelnut	crude	1 ELISA, Veratox	chocolate			1.1 µg/g	
Garber 2010 hazelnut	hazelnut	crude	2 ELISA, RIDASCREEN	Cake			2 µg/g	
Garber 2010	hazelnut	crude	2 ELISA, RIDASCRE	cereals			2 µg/g	

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
hazelnut			EN					
Garber 2010 hazelnut	hazelnut	crude	2 ELISA, RIDASCREEN	chocolate			1 µg/g	
Garber 2010 hazelnut	hazelnut	crude	3 ELISA Systems	Cake			38 µg/g	
Garber 2010 hazelnut	hazelnut	crude	3 ELISA Systems	cereals			5.8 µg/g	
Garber 2010 hazelnut	hazelnut	crude	3 ELISA Systems	chocolate			8 µg/g	
Holzhauser 1999	hazelnut	crude	1 ELISA	almond			1.1 ppm	1.4 ppm
Holzhauser 1999	hazelnut	crude	1 ELISA	cashew			4.5 ppm	6.9 ppm
Holzhauser 1999	hazelnut	crude	1 ELISA	chocolate	0.001 - 10 %	103-132		
Holzhauser 1999	hazelnut	crude	1 ELISA	chocolate milk	0.001 - 10 %	83-118	0.07 ppm	0.13 ppm
Holzhauser 1999	hazelnut	crude	1 ELISA	chocolate milk			0.11 ppm	0.19 ppm
Holzhauser 1999	hazelnut	crude	1 ELISA	cookie	0.001 - 10 %	90-127		
Holzhauser 1999	hazelnut	crude	1 ELISA	crisps/ Thai cracker			0.09 ppm	0.14 ppm
Holzhauser 1999	hazelnut	crude	1 ELISA	ice cream			0.07 ppm	0.12 ppm
Holzhauser 1999	hazelnut	crude	1 ELISA	popcorn			0.43 ppm	0.67 ppm
Holzhauser 1999	hazelnut	crude	1 ELISA	pumpkin seed			10.3 ppm	14.1 ppm
Holzhauser 1999	hazelnut	crude	1 ELISA	range of products				
Holzhauser 1999	hazelnut	crude	1 ELISA	snack cereal	0.001 – 10%	67-127		
Holzhauser 1999	hazelnut	crude	1 ELISA	snack cereal			0.05 ppm	0.05 ppm
Holzhauser 1999	hazelnut	crude	1 ELISA	walnut			5.5 ppm	6.9 ppm
Kiening 2005	hazelnut	crude	1 ELISA	cereals	1-10 mg/kg	95-101		
Kiening 2005	hazelnut	crude	1 ELISA	chocolate dark	1-10 mg/kg	86-94		
Kiening 2005	hazelnut	crude	1 ELISA	chocolate milk	1-10 mg/kg	0-115		
Kiening 2005	hazelnut	crude	1 ELISA	cookie	1-10 mg/kg	95-127		

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Kiening 2005	hazelnut	crude	1 ELISA	ice cream	1-10 mg/kg	93-111		
Piknova 2008	hazelnut		2 ELISA (Ridascreen)	dough			0.01 % w/w	
Piknova 2008	hazelnut		1 Real time PCR	range of products				

IA- Immuno-assay

3.2.10. Lupine

We had findings from three studies that investigated ELISA and PCR in food matrixes such as bread, cakes and sausage meat. The assays all provided a limit of detection of approximately 1 ppm equivalent to 1µg/g with good recoveries when spiked with between 1 and 1000µg/g of lupine. The ELISA assays, Holden (2005), Holden (2007), and Kaw (2008) were developed in-house and were directed against crude antigens rather than specific allergens. All the studies presented limit of detection and percentage recovery and this was as low as 1 µg/g with the Holden (2005) assay detecting down to 0.1µg/g in sausage or pastry matrices.

Demmel (2011) tested a real time PCR assay in pizza, flour and dough and demonstrated a consistent limit of detection of 0.1µg/g of Lupine flour. However the percentage recovery was not presented.

Table 3.30: Lupine: characteristics of included studies

Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Demmel (2011)	Lupine	Crude extract	Flour wheat flour	Spiked **Source of spike** Sweet lupine flour from L angustifolius, (Chemical and Veterinarian Research Institute Freiburg, Freiburg, Germany)	PCR
Holden (2005)	Lupine	Crude extract	Bread hot dog Pasta	Spiked **Source of spike** Lopino (Lupina, Visbeck, Germany)	ELISA
Holden (2007)	Lupine	Crude extract	Bread, lupine-free **Field** Foods sampled cakes, bread/rolls, pasta, chocolate spread, biscuits, flour/mix, chips	Spiked Field **Source of spike** Processed proteins from L. albus seeds, in the form of tofu-like product (Lopino;Lupina, Visbek, Germany) native proteins from L.angustifolius seeds, in the form of lupine flour, (Soja Austria)	ELISA

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Kaw (2008)	Lupine	Crude extract	Cake corn muffin Meat Sausage Frankfurter	Spiked **Source of spike** purchased from a local grocery store Standardisation protein concentration was assessed using Lowry method	ELISA

Table 3.31: Lupine: description of assay

Study ID	Allergen	Assay details	Additional information
Demmel (2011)	Lupine	Test 1 Real time PCR	
Holden (2005)	Lupine	Test 1 *ELISA*	**ELISA** Polyclonal capture antibody Polyclonal detection antibody Sandwich In-house
Holden (2007)	Lupine	Test 1 *pAb-mAb*	**ELISA** Polyclonal capture antibody Monoclonal detection antibody Sandwich In-house
Kaw (2008)	Lupine	Test 1 ELISA	**ELISA** Sandwich In-house

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Table 3.32: Lupine: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Demmel 2011	lupine	crude	1 PCR real time	dough			0.1 µg/g	
Demmel 2011	lupine	crude	1 PCR real time	flour			0.1 µg/g	
Demmel 2011	lupine	crude	1 PCR real time	pizza (cooked)			0.1 µg/g	
Holden 2005	lupine	crude	1 ELISA	bread	1-1000 µg/g	80-116	0.2 µg/g	
Holden 2005	lupine	crude	1 ELISA	chocolate spread	1-1000 µg/g	61-84	0.4 µg/g	
Holden 2005	lupine	crude	1 ELISA	pasta	1-1000 µg/g	88-116	0.1 µg/g	
Holden 2005	lupine	crude	1 ELISA	sausage vegetarian	1-1000 µg/g	60-64	0.1 µg/g	
Holden 2007	lopino	crude	1 ELISA pAb-mAb	bread	1-1000 µg/g	85-150		
Holden 2007	lupine	crude	1 ELISA pAb-mAb	bread	1-1000 µg/g	44-88		
Kaw 2008	lupine	crude	1 ELISA	muffin (corn)	1-1000 ppm	91-118	1 µg/g	
Kaw 2008	lupine	crude	1 ELISA	sausage	1-1000 ppm	97-117	1 µg/g	

3.2.11. Milk

A range of assays were investigated including commercial and in house ELISAs, direct automated optical biosensor , mass spectrometry and dipsticks in a range of food matrices such as pasta sauce, sausage, cereals, biscuits, sorbet, dark chocolate and wine (Table 3.33). The assays tended to be directed against specific components of milk for example casein, kappa-casein and beta-lactoglobulin and these correspond to the major allergenic proteins (Table 3.34).

The in- house ELISA developed by Hefle (2004) was directed against casein and gave a limit of detection of 0.5 ppm equivalent to 0.5 µg/g in the food matrices ice-cream and dark chocolate.

The commercial ELISA kit FASTKIT tested by Akiyama (2002) (Table 3.35) gave between 5-95 percent recoveries for detecting casein and beta-lactoglobulin in food matrices spiked with as little as 5 ng/ml, these recoveries varied considerable in different foods matrices. Recovery was under 40% for casein in various sauces whereas the cereals, cookies and sausage mix gave better recoveries. The same FASTKIT directed against beta-lactoglobulin gave more consistent recoveries, 49-95% in the same range of foods. The FASTKIT directed against crude milk extract gave poorer recoveries of between 24-48%. Presumable this later assay was directed against a range of milk proteins including beta-lactoglobulin and caseins.

The RIDASCREEN ELISA for detecting casein tested by Khuda (2012a) gave very poor recovery in dark chocolate, this study was of high quality and the researchers ensured that they attempted to recover the milk proteins from tempered chocolate. The same RIDASCREEN assays for detecting casein and beta-

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lactoglobulin gave negligible recoveries from sugar cookie mixture, Khuda (2012b). However none of the assays tested in this study showed good recovery from the sugar cookie matrix.

The Veratox ELISA for casein tested by Khuda (2012a) gave excellent recovery from dark chocolate at 122%, and like the previous assay poor recovery from sugar cookie at 7%.

The ELISA ICP-MS for casein, was shown to have a limit of detection of 0.5µg/g in dark chocolate and ice cream matrices by Hefle (2004), the percent recovery was not shown.

The ELISA BIOKIT showed recoveries of only 2% from dark chocolate for beta-lactoglobulin but better at 50% for casein, Khuda (2012a). As mentioned in the previous paragraphs recovery from sugar cookie was poor at 0%, (Khuda 2012b).

The ELISA Systems kits gave recoveries of 50 and 40% for casein and beta-lactoglobulin respectively from chocolate, Khuda (2012a), and again negligible recoveries of 6% or less from sugar cookie, Khuda (2012b).

A novel, direct automated optical biosensor (Biacore 3000) was tested with a monoclonal antibody specific for cows' milk kappa-casein contaminating sheep or goats' milk, Haasnoot (2004). This specificity may not be useful for allergen testing as human IgE tends to show cross reactivity with caseins from different species. The assay limit of detection was given as 0.7% - 0.08 w/w which converts to 700-800µg/g for detecting bovine proteins in sheep and goat milk. This validation experiment while being suitable for species contamination was not suitable for allergy testing.

Morishita (2006) developed and tested an IC - dipstick method that provided a good limit of detection 5µg/g in a range of foods. This assay has the advantage over the ELISA systems that complex laboratory systems and equipment are not required.

Monaci (2008) developed and tested a detection system using mass spectrometry to detect the milk protein alpha lactalbumin. This gave recoveries of 73-79% for fruit juice spiked with as little as 5µg/ml.

Table 3.33: Milk: characteristics of included studies

Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Akiyama (2004)	Milk	Specific protein/peptide or gene Beta-lactoglobulin Casein	Cereal Cookie Sauce Pasta sauce Sausage	Spiked Dose of spike 5-20 ng/mL **Source of spike** Milk: provided by Nippon Meat Packers, Inc. Fresh Milk from Holstein.	ELISA
Eissa (2012)	Milk and dairy	Specific protein/peptide or gene Beta-lactoglobulin	**Field** Foods sampled Cake, biscuit and crisps	Field **Source of spike** Abcam (Cambridge, USA)	ELISA Electrochemical Immunosensor
Haasnoot (2004)	Cow's milk	Specific protein/peptide or gene	Milk Ewes and goats milk	Spiked **Source of spike** Cow's milk was made by	ELISA Biosensor immunoassay

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
		bovine kappa-casein		reconstituting a bovine skimmed milk powder (1g + 9ml of water)	
Hefle (2004)	Milk and dairy	Specific protein/peptide or gene Casein	Chocolate Ice cream Lemon sorbet	Spiked **Source of spike** Not reported	ELISA
Khuda (2012a) milk	Milk and dairy	Specific protein/peptide or gene Beta-lactoglobulin Casein	Dark Chocolate	**Source of spike** Non-fat dry milk-NIST SRM 1549 (National Institute of Standards and Technology, Gaithersburg, MD, USA)	ELISA
Khuda (2012b) Peanut	Milk and dairy	Crude extract	Cookie	Spiked **Source of spike** Non-fat dry milk-NIST SRM 1549 (National Institute of Standards and Technology, Gaithersburg, MD, USA)	ELISA
Khuda (21012b) milk	Milk and dairy	Specific protein/peptide or gene Beta-lactoglobulin Casein	Cookie	Spiked **Source of spike** Non-fat dry milk, NIST SRM 1549 (National Institute of Standards and Technology, Gaithersburg,MD, USA)	ELISA
Monaci (2008)	Milk and dairy	Specific protein/peptide or gene Whey proteins	Juice Apple, apricot, banana, passion fruit, guava, grape, kiwi, lemon, mango, orange, papaya, peach, pear, pineapple	Spiked **Source of spike** LG A, LG B (purity 92%) and x-LA (purity 98%) and formic acid, (98-100% purity grade) (FA) (Sigma-Aldrich St Louis, MO, USA).	Mass spectrometry
Monaci (2011)	Milk and dairy	Specific protein/peptide or gene Casein	Wine	Spiked **Source of spike** Not reported	Mass spectrometry
Morishita (2006)	Milk and dairy	Crude extract	Carrot Sherbet Cookie Jam Pickles (Soy Sauce, vinegar) Potato Salad Sauce Tomato Soup	Spiked	ELISA Immuno-chromatographic test kits Dip stick

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
			Steamed and fried Chinese dumpling Hamburger		
Weber (2006)	Milk and dairy	Specific protein/peptide or gene Casein	Chicken Chicken hot dog sample Cookie	Spiked **Source of spike** Local retail market	ELISA kit Mass spectrometry

Table 3.34: Milk: description of assay

Study ID	Allergen	Assay details	Additional information
Akiyama (2004)	*Milk*	Test 1 *Milk protein Casein ELISA kit* Test 2 *Milk protein beta-Lactoglobulin ELISA kit* Test 3 *FASTKIT Milk ELISA Kit*	Not stated ELISA Commercial company
Eissa (2012)	Milk and dairy	Test 1 Electrochemical *Immunosensor, (Dropsens, Inc, Spain)* Test 2 *beta-lactoglobulin ELISA, ELISA systems (Queensland Australia)(used as gold standard)*	Test 1 Electrochemical Immunosensor *Graphene modified screen/printed carbon electrodes (Dropsens, Inc, Spain) with Autolab PGSTAT302N* *Test 2* *Commercial ELISA*
Haasnoot (2004)	*Cows' milk*	Test 1 *direct automated optical biosensor (Biacore 3000) Mab 6A10* Test 2 *direct automated optical biosensor (Biacore 3000) Mab 4G10* Test 3 *inhibition automated optical biosensor (Biacore 3000) Mab 6A10* Test 4 *inhibition automated optical biosensor (Biacore 3000) Mab 4G10*	**Biosensor immunoassay** Direct Inhibition
Hefle (2004)	Milk and dairy	Test 1 *ELISA*	**ELISA** Polyclonal capture antibody Polyclonal detection antibody Sandwich In-house
Khuda (2012a)	Milk and dairy	Test 1 *RIDASCREEN FAST peanut, egg, and casein from*	**Test 1,2 and 3** ELISA

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Study ID	Allergen	Assay details	Additional information
milk		*R- Biopharm (RB, Washington, MO, USA)* Test 2 *Veratox peanut, egg, and total milk allergen quantitative test kits from Neogen (NE) Corp. (Lansing, MI, USA)* Test 3 *Morinaga (MO) peanut, egg, and milk (casein and BLG) protein ELISA kits (Crystal Chem, Downers Grove, IL, USA)* Test 4 *Tepnel (TE) BIOKITS peanut, egg, casein, and BLG assay kits (Neogen Corp.)* Test 5 *ELISA Systems (ES) peanut, egg, casein, and BLG residue kits (BioMerieux, Durham, NC, USA)*	Commercial company
Khuda (2012b) Peanut	Milk and dairy	Test 1 *RIDASCREEN FAST peanut, egg, and casein from R- Biopharm (RB, Washington, MO, USA)* Test 2 *Veratox peanut, egg, and total milk allergen quantitative test kits from Neogen (NE) Corp. (Lansing, MI, USA)* Test 3 *Morinaga (MO) peanut, egg, and milk (casein and BLG) protein ELISA kits (Crystal Chem, Downers Grove, IL, USA)* Test 4 *Tepnel (TE) BIOKITS peanut, egg, casein, and BLG assay kits (Neogen Corp.)* Test 5 *ELISA Systems (ES) peanut, egg, casein, and BLG residue kits (BioMerieux, Durham, NC, USA)*	**ELISA** Polyclonal capture antibody Commercial company
Monaci (2008)	Milk and dairy	Test 1 Mass spectrometry *xLA* Test 2 Mass spectrometry *LGA* Test 3 Mass spectrometry *LGB*	
Monaci (2011)	Milk and dairy	Test 1 Mass spectrometry	**Mass spectrometry** Ultima triple quadrupole mass spectrometer *HPLC coupled with single-stage Orbitrap mass spectrometry*
Morishata (2006)		Immuno-chromatographic test kits- Dip stick	Immunoassay In house
Weber (2006)	*Casein*	Test 1 *VERATOX kit ELISA* Test 2 **Mass spectrometry** Time of flight-mass spectrometry	Test 1 Neogen, Lansing, MI Test 2

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Table 3.35: Milk: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Akiyama 2004	milk	Casein	1 ELISA FASTKIT	cereals	5-20 ng/ml	63-65		
Akiyama 2004	milk	Casein	1 ELISA FASTKIT	cookie	5-20 ng/ml	82-91		
Akiyama 2004	milk	Casein	1 ELISA, FASTKIT	pasta sauce	5-20 ng/ml	34-35		
Akiyama 2004	milk	Casein	1 ELISA, FASTKIT	sauce	5-20 ng/ml	5-8.		
Akiyama 2004	milk	Casein	1 ELISA, FASTKIT	sausage	5-20 ng/ml	50-63	1 ng/ml	2 ng/ml
Akiyama 2004	milk	Beta-Lactoglobulin	2 ELISA, FASTKIT	cereals	5-20 ng/ml	53-67		
Akiyama 2004	milk	Beta-Lactoglobulin	2 ELISA, FASTKIT	cookie	5-20 ng/ml	85-93		
Akiyama 2004	milk	Beta-Lactoglobulin	2 ELISA, FASTKIT	pasta sauce	5-20 ng/ml	61-94		
Akiyama 2004	milk	Beta-Lactoglobulin	2 ELISA, FASTKIT	sauce	5-20 ng/ml	49-59		
Akiyama 2004	milk	Beta-Lactoglobulin	2 ELISA, FASTKIT	sausage	5-20 ng/ml	74-95	1 ng/ml	5 ng/ml
Akiyama 2004	milk	Standard milk protein	3 ELISA, FASTKIT	cereals	5-10 ng/ml	23-25		
Akiyama 2004	milk	Standard milk protein	3 ELISA, FASTKIT	cookie	5-10 ng/ml	34-41		
Akiyama 2004	milk	Standard milk protein	3 ELISA, FASTKIT	pasta sauce	5-10 ng/ml	27-40		
Akiyama 2004	milk	Standard milk protein	3 ELISA, FASTKIT	sauce	5-10 ng/ml	41-43		
Akiyama 2004	milk	Standard milk protein	3 ELISA, FASTKIT	sausage	5-10 ng/ml	41-48		
Haasnoot 2004	milk	kappa-casein	1 Biosensor direct Mab 6A10	milk (goat or sheep)	0.25-2 %	80-108	0.07 % w/w	
Haasnoot 2004	milk	kappa-casein	2 Biosensor direct Mab 4G10	milk (goat or sheep)	0.25-2 %	84-110	0.06 % w/w	
Haasnoot 2004	milk	kappa-casein	2 Biosensor direct Mab 4G10	milk (goat or sheep)	0.25-2 %	77-112	0.08 % w/w	
Haasnoot 2004	milk	kappa-casein	3 Biosensor direct Mab 6A10	milk (goat or sheep)	0.25-2 %	77-112	0.08 % w/w	
Hefle 2004	milk	casein	1 ELISA, ICP-MS	chocolate dark			0.5 ppm	
Hefle 2004	milk	casein	1 ELISA, ICP-MS	ice cream			0.5 ppm	
Khuda 2012a	milk	casein	1 ELISA, RIDASCREEN FAST	chocolate dark	linear regression	2		

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Khuda 2012a	milk	casein	2 ELISA, Veratox	chocolate dark	linear regression	122		
Khuda 2012a	milk	casein	3 ELISA, Morinaga	chocolate dark	linear regression	69		
Khuda 2012a	milk	BLG	3 ELISA, Morinaga	chocolate dark	linear regression	357		
Khuda 2012a	milk	casein	4 ELISA, BIOKITS	chocolate dark	linear regression	54		
Khuda 2012a	milk	BLG	4 ELISA, BIOKITS	chocolate dark	linear regression	2		
Khuda 2012a	milk	casein	5 ELISA Systems	chocolate dark	linear regression	50		
Khuda 2012a	milk	BLG	5 ELISA Systems	chocolate dark	linear regression	44		
Khuda 2012b	milk	casein	1 ELISA, RIDASCREEN FAST	sugar cookie	linear regression	0		
Khuda 2012b	milk	BLG	1 ELISA, RIDASCREEN FAST	sugar cookie	linear regression	0		
Khuda 2012b	milk	casein	2 ELISA, Veratox	sugar cookie	linear regression	7		
Khuda 2012b	milk	casein	3 ELISA, Morinaga	sugar cookie	linear regression	4		
Khuda 2012b	milk	BLG	3 ELISA, Morinaga	sugar cookie	linear regression	12		
Khuda 2012b	milk	casein	4 ELISA, BIOKITS	sugar cookie	linear regression	3		
Khuda 2012b	milk	BLG	4 ELISA, BIOKITS	sugar cookie	linear regression	0		
Khuda 2012b	milk	casein	5 ELISA Systems	sugar cookie	linear regression	6		
Khuda 2012b	milk	BLG	5 ELISA Systems	sugar cookie	linear regression	1		
Monaci 2008	milk	alpha LA	1 Mass Spectrometric	fruit juice	5-20 µg/ml	73-79		
Monaci 2008	milk	LGA	2 Mass Spectrometric	fruit juice		68-74		
Monaci 2008	milk	LGB	3 Mass Spectrometric	fruit juice		75-78		
Monaci 2011	milk	casein	1 LC-MS	white wine	10-1000 µg/ml-1		39 $\mu g/mL^{-1}$	
Morishita 2006	milk	crude	1 IC - dipstick	chicken meatball or burger			5 µg/g	
Morishita 2006	milk	crude	1 IC - dipstick	cookie			5 µg/g	
Morishita 2006	milk	crude	1 IC - dipstick	Dumplings fried/ steamed			5 µg/g	

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Morishita 2006	milk	crude	1 IC - dipstick	jelly			5 µg/g	
Morishita 2006	milk	crude	1 IC - dipstick	Pickles in Vinegar/soy			5 µg/g	
Morishita 2006	milk	crude	1 IC - dipstick	Potato salad			5 µg/g	
Morishita 2006	milk	crude	1 IC - dipstick	sauce			5 µg/g	
Weber 2006	milk	casein	2 Mass Spectrometric	chicken hot dog			5 ppm	

3.2.12. Peanut

Twenty studies investigated detection systems for peanut, perhaps as a result of the severity of symptoms reported by people with peanut allergy.

An in-house ELISA was developed and tested by Akiyama (2004b) and performed with similar sensitivity as the commercial FASTKIT, providing recoveries of 50-182% with spiked concentrations as low as 5 ng/ml. The matrix with the lowest recovery was chocolate with 50-54%. Deng (2012) developed an assay for peanut agglutinin and found recoveries in chocolate were variable depending on the concentration of the spike. Ehlert (2009) developed and tested an in-house ELISA and showed a limit of detection of between 5 and 100 mg/kg^{-1} equivalent to 50-1000µg/g . Khuda (2012b), tested the Morinaga ELISA in a sugar cookie matrix and found that recoveries were low at 12%. Kiening developed and tested an ELISA against crude peanut extract and when cereals, cookies, ice-cream or chocolate were spiked achieved recoveries of 87-123%. Yeung (1996) developed a similarly effective assay and extraction system for snacks, oils and sauces spiked with 2.5-20µg/g peanut extract.

Commercial ELISA systems were evaluated in a number of studies. The FASKIT ELISA gave good recoveries in the region of 65-97 % when butter, chocolate, pasta sauces were spiked with between 2-20ng/ml. RIDASCREEN gave good recoveries with dark chocolate but poor recoveries, 11 % from sugar cookie, Khuda (2012a), Park (2005) did not show recovery but did indicate that the limit of detection was 5 µg/g for chocolate, cereals, cookie and ice-cream. BIOKITS gave under 10% recovery for dark chocolate and sugar cookie Khuda (2012a). The Veratox ELISA gave limited recoveries from spiked chocolate, 30%, Khuda (2012a), poor from sugar cookie, 15%. Park (2005) did not give the recovery, but did show a limit of detection of 5µg/g for chocolate, cereals, cookie and ice-cream. Some companies and researchers have developed systems to increase the sensitivity of the ELISA system further. Speroni (2010) evaluated an ELISA system incorporating antibody coated magnetic micro particles, for the detection of the peanut allergens Ara h 3,4. This assay had a limit of detection of 0.8µg/g when cereals were spiked with peanut flour and a good recoveries, 80-95%.

Mass spectrometry and Electrospray mass spectrometry provided limit of detections for as low as 0.1µg/g in chocolate cereal snacks, Careri (2007b), these methods have the advantage that they can be directed against a range of peanut proteins, however the use will be limited as the equipment involved is expensive. The *Ligation dependent probe amplification (LPA) tested by Ehlert (2009)* did not give good limit of detection for cookie as a matrix at 5 mg/kg^{-1} (equivalent to 50µg/g) and gave a very poor limit of detection from walnut mixtures spiked with peanut. PCR evaluated by Ehlert (2009) sowed a similar limit of detection and was also not able to give good suitable limits of detection in walnut mixtures (100mg/kg^{-1}).

The IC-dipstick tested by Morishita (2006) gave consistent limit of detection of 5µg/g, and had the advantage of ease of use and did not require specialist equipment.

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Table 3.36: Peanut: characteristics of included studies

Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Akiyama (2004b)	Peanut	Crude extract	Biscuit Butter Chocolate Sauce	Spiked **Source of spike** Morinaga Institute of Biological Science, Virginia Peanuts Standardisation Protein measured BCA protein assay kit and adjusted to a concentration of 100-300 µg/ml	ELISA
Ben Rejeb (2005) peanut	Peanut	Crude extract	Chocolate	Spiked Roasted defatted peanuts and nuts extracted, dialysed Source of spike Not reported Standardisation Made up to 1mg/ml-1 protein content measured using BCA test	ELISA
Careri (2007a)	Peanut	Specific protein/peptide or gene **Ara h,**1,3,4	Breakfast cereals Cornflakes	Spiked **Source of spike** Leibniz-Centre for Medicine and Biosciences at the Research Centre Borstal (Borstal, Germany)	ELISA
Careri (2007b)	Peanut	Specific protein/peptide or gene Ara h 2 and Ara h 3/4	Breakfast cereals Rice crispy/cacao	Spiked **Source of spike** Red skin Peanuts Standardisation Not stated	Mass spectrometry
Careri (2008)	Peanut	Specific protein/peptide or gene **Ara h,** 2, 3	Chocolate snack	Spiked Field **Source of spike** Ara h2 was purified from toasted peanuts, Ara h 1 and Ara h3/4 were provided by the Leibniz Centre for Medicine and Biosciences at the Research Centre Borstal, Germany Standardisation Not explained	ELISA non-competitive sandwich Mass spectrometry
Deng (2012)	Peanut	Specific protein/peptide or gene Peanut agglutinin	Milk **Field** Foods sampled Range of products and peanut oil without	Spiked Field **Source of spike** Peanut agglutinin, (Sigma, St. Louis, MO, USA)	ELISA

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
			peanut protein.		
Ehlert (2009)	Peanut	Specific protein/peptide or gene DNA	Cookie Pesto cashew	Spiked **Source of spike** Nut materials, sesame seeds, ingredients of self-prepared DNA plant and animal materials used to test the specificity of the method and spike samples of chocolate, were obtained from the Bavarian Health and Food Safety Authority (Oberschleibheim, Germany)	ELISA PCR Ligation-dependent probe amplification
Hird (2003)	Peanut	Specific protein/peptide or gene Ara h2 gene	Biscuit Cake Chocolate Meat Pastry or dough	Spiked. **Source of spike** Biscuit prepared by Central Science Laboratory Food Analysis Proficiency Assessment Scheme, spiked with 2ppm peanut powder.	PCR
Khuda (2012b) Peanut	Peanut	Crude extract	Cookie	Spiked **Source of spike** light-roasted peanut flour, 12% fat light roast, product 521271, lot 109FA (Golden Peanut Co., Alpharetta, GA, USA)	ELISA
Khuda (2012a) peanut (dark chocolate)	Peanut	Crude extract	Chocolate	Spiked Spray dried whole egg powder NIST RM 8445 (NIST), non-fat milk powder, and light-roasted peanut flour, 12% fat light roast, product 521271, lot 109FA **Source of spike** Peanut (Golden Peanut Co., Alpharetta, GA, USA) Standardisation FDA	ELISA
Kiening (2005)	Peanut	Crude extract	Breakfast cereals Chocolate Cookie Ice cream **Field** Foods sampled cookie, cereals and chocolate	Spiked Standard peanut butter as peanut reference material (SRM 2387) National Institute of Standards and Technology (NIST, Gaithersburg, MD) Field **Source of spike** Standard peanut butter (SRM 2387) (National Institute of	ELISA

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
				Standards and Technology, NIST, Gaithersburg, MD).	
Morishita (2006)	Peanut	Crude extract	Carrot Sherbet, Cookie, Jam Pickles (Soy Sauce, vinegar),Potato Salad, Sauce Tomato, Soup Steamed and fried Chinese dumpling Hamburger	Spiked	ELISA Immuno-chromatographic test kits Dip stick
Park (2005)	Peanut	Crude extract	Cereal Chocolate Cookie Ice cream	Spiked **Source of spike** Peanut butter (National Institute for Standards and Technology (NIST); Gaithersburg, MD), Standard Reference Material (SRM) No.2387	ELISA
Pomes (2003)	Peanut	Crude extract Veratox Specific protein/peptide or gene Ara h 1	Cookie Flour pancake mix **Field** Peanut products: including peanut cookies, peanut butter sandwich cookies, peanut sweets, and peanut butter . Non-peanut products: including cookies and a group of nuts, beans, and seeds.	Spiked Field **Source of spike** Ground peanut	ELISA
Pomes (2004)	Peanut	**Specific protein/ peptide or gene** Ara h 1	Chocolate	Spiked Field **Source of spike** Oil-roasted Virginia peanuts (Planters Company, East Hanover, N.J.)	ELISA
Speroni (2010)	Peanut	Specific protein/peptide or gene Ara h 3,4	Biscuit Breakfast cereals	Spiked **Source of spike** Roasted peanuts purchased at a local food store	ELISA
Stephan (2002)	Peanut	Crude extract Peanut	Chocolate Rausch Schokoladen Gmbh (Peine,	Spiked Field **Source of spike** Not reported	Dipstick

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
			Germany) **Field** Foods sampled Range of foods labelled as containing, not containing and may contain peanut or hazelnut		
Stephan (2004)	Peanut	Crude (ELISA) Specific protein/peptide or gene **Ara h** 2 (PCR)	Chocolate Milk **Field** Foods sampled industrially manufactured samples of milk and semisweet chocolates	Spiked Field **Source of spike** Not reported	ELISA PCR
Wen (2005a)	Peanut	Specific protein/peptide or gene Ara h 1	Chocolate	Spiked **Source of spike** raw peanuts purchased from a local food market	Lateral Flow Assay
Yeung (1996)	Peanut	Crude extract	Chocolate Cookie Crisps Ice cream Oil Sauce Pasta sauce Snack Sesame snaps, wafers	Spiked **Source of spike** 3 peanut preparations (roasted, raw, denatured, unfolded raw peanuts) purchased in local stores	ELISA

Table 3.37: Peanut: description of assay

Study ID	Allergen	Assay details	Additional information
Akiyama (2004b)	Peanut	Test 1 Peanut protein ELISA Kit (Morinaga Institute of Biological Science) Test 2 FASTKIT Peanut ELISA kit	Test 1 ELISA In-house Test 2 ELISA Commercial (Nippon Meat Packers Inc.)
Ben Rejeb (2005) peanut	Peanut	Test 1	**ELISA** Polyclonal detection antibody Competitive inhibition In-house

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Study ID	Allergen	Assay details	Additional information
Careri (2007a)	Peanut	**ELISA** ICP-MS	**Inductively coupled plasma-mass spectrometry** using both direct competitive and non-competitive immunoassays
Careri (2007b)	Peanut	Test 1 Time of flight mass spectrometry (LC-ESI-Q-TOF) Test 2 Liquid chromatography-triple quadruple mass spectrometry LC-QqQ-MS-MS	Test 1 Mass spectrometry Test 2 Mass spectrometry
Careri (2008)	Peanut	Test 1 Europium (Eu)-tagged inductively coupled plasma mass spectrometry (ICP-MS) immunoassay ELISA ICP-MS Test 2 Electroliquid chromatography/electrospray ionization tandem mass spectrometry (LC/ESI-MS/MS) with a triple quadrupole mass analyzer. Ara h 3/4 Test 3 Electroliquid chromatography/electrospray ionization tandem mass spectrometry (LC/ESI-MS/MS) with a triple quadrupole mass analyzer. Ara h 3/4	
Deng (2012)	Peanut	Test 1 ELISA sandwich	**ELISA** Polyclonal capture antibody Polyclonal detection antibody Sandwich In-house
Ehlert (2009)	Peanut	Test 1 Ligation dependent probe amplification Test 2 Hazelnut and peanut: real-time PCR Surefood allergen kit (Congen Biotechnology GmbH, Berlin, Germany) cashew real time PCR In house Test 3 Hazelnut and peanut: ELISA Ridascreen (R-Biopharm AG, Darmstadt, Germany)	
Hird (2003)	Peanut	Test 1 Real time PCR	**PCR** Real-time PCR
Khuda (2012a)	Peanut	Test 1 RIDASCREEN FAST peanut, egg, and casein from R- Biopharm (RB, Washington, MO, USA) Peanut protein including Ara h1 Test 2 Veratox peanut, egg, and total milk allergen quantitative test kits from Neogen (NE) Corp. (Lansing, MI, USA) Test 3 Morinaga (MO) peanut, egg, and milk (casein and BLG) protein ELISA kits (Crystal Chem, Downers Grove, IL, USA) Test 4 Tepnel (TE) BIOKITS peanut, egg, casein, and BLG assay kits (Neogen Corp.)	**ELISA** Polyclonal capture antibody Polyclonal detection antibody Commercial company

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Study ID	Allergen	Assay details	Additional information
		Test 5 ELISA Systems (ES) peanut, egg, casein, and BLG residue kits (BioMerieux, Durham, NC, USA)	
Kiening (2005)	Peanut	Test 1 ELISA	**ELISA** Monoclonal capture antibody mouse Y70 Polyclonal detection antibody rabbit R695 In-house
Morishita (2006)	Peanut	Test 1 Immunochromatographic test kits, dipstick. Test 2 ELISA: FASTKIT	Test 1 IC dipstick Test 2 ELISA Commercial
Park (2005)	Peanut	Test 1 Veratox Assay for peanut Test 2 RIDASCREEN Assay for peanut Test 3 BioKits Assay for peanut	**Test 1** ELISA, commercial, Neogen **Test 2** ELISA, commercial, R-Biopharm RIDASCREEN FAST Peanut **Test 3** ELISA, commercial, Tepnel Biokits
Pomes (2003)	Peanut	Test 1 Test 1 ELISA In house Test 2 Veratox	Test 1 ELISA**,** Monoclonal capture antibody mAb 2C12, Monoclonal detection antibody mAB 2F7, in-house. Test 2 ELISA, commercial, Neogen Corporation, Lansing, Mich
Pomes (2004)	Peanut	Test 1 ELISA	**ELISA** Monoclonal capture antibody Monoclonal detection antibody Sandwich In-house
Speroni (2010)	Peanut	Test 1 Protein A-Pn-b ELISA Veratox Quantitative peanut allergen test Test 2 MP-NH2-PAMAM G 1.5-Pn-b ELISA	ELISA format based on antibody coated magnetic micro particles. The immune support are coated with Protein A-Pn-b and MP-NH2-PAMAM G1.5-Pn-b
Stephan (2002)	Peanut	Test 1 Dipstick: in-house	Dipstick method In house. Polyclonal capture antibody Polyclonal detector antibody.
Stephan (2004)	Peanut	Test 1 ELISA Test 2 Real-time PCR	**Test 1** **ELISA** Polyclonal capture antibody Polyclonal detection antibody Sandwich

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Study ID	Allergen	Assay details	Additional information
			Test 2 **PCR** Real-time PCR In-house
Wen (2005a)	Peanut	Test 1 Lateral Flow Assay	
Yeung (1996)	Peanut	Test 1 ELISA	**ELISA** Polyclonal capture antibody Polyclonal detection antibody Competitive inhibition In-house

Table 3.38: Peanut: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Akiyama 2004b	peanut	crude	1 ELISA	biscuit	5-20 ng/ml	74-76		
Akiyama 2004b	peanut	crude	1 ELISA	buffer	5-20 ng/ml		2 ng/ml	8 ng/ml
Akiyama 2004b	peanut	crude	1 ELISA	butter	5-20 ng/ml	68-70		
Akiyama 2004b	peanut	crude	1 ELISA	chocolate	5-20 ng/ml	50-54		
Akiyama 2004 b	peanut	crude	1 ELISA	sauce	5-20 ng/ml	66-68		
Akiyama 2004b	peanut	crude	2 ELISA, FASTKIT	biscuit	5-20 ng/ml	122-182		
Akiyama 2004b	peanut	crude	2 ELISA, FASTKIT	buffer			2.5 ng/ml	5 ng/ml
Akiyama 2004b	peanut	crude	2 ELISA, FASTKIT	butter	5-20 ng/ml	65-70		
Akiyama 2004 b	peanut	crude	2 ELISA, FASTKIT	chocolate	5-20 ng/ml	72-82		
Akiyama 2004b	peanut	crude	2 ELISA, FASTKIT	sauce	5-20 ng/ml	79-97		
Ben Rejeb 2005	peanut		1 ELISA	chocolate dark			1 ppm	
Careri 2007a	peanut	Ara h 1 and Ara h3/4	1 ELISA ICP-MS	cereals			2 mg/kg^{-1}	
Careri 2007b	peanut		1 LC-ESI-Q-TOF	cereal chocolate snack				
Careri 2007b	peanut	m/z 695 Ara h 3/4	2 LC-QqQ-MS-MS	cereal chocolate snack			1 $\mu g/g^{-1}$	3.7 µg/g-1
Careri 2007b	peanut	m/z 807 Ara h 2	2 LC-QqQ-MS-MS	cereal chocolate			5 $\mu g/g^{-1}$	14 µg/g-1

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
				snack				
Careri 2008a	peanut		1 ELISA Mass Spec	cereal chocolate snack	5 µg/g-1	86	2.2 µg/g^{-1}	5 µg/g-1
Careri 2008a	peanut	Ara h 3	2 Electrospray mass spec	cereal chocolate snack			1 µg/g^{-1}	3.7 µg/g-1
Careri 2008a	peanut	Ara h 2	3 Electrospray mass spec	cereal chocolate snack			5 µg/g^{-1}	14 µg/g-1
Deng 2012	peanut	peanut agglutinin	1 sandwich ELISA	milk	1-60 ng/mL	0- 69		
Ehlert 2009	peanut	DNA	1 LPA	chocolate	nr	nr	5 mg/kg^{-1}	
Ehlert 2009	peanut	DNA	1 LPA	cookie			5 mg/kg^{-1}	
Ehlert 2009	peanut	DNA	1 LPA	walnut cookies			1000 mg/kg^{-1}	
Ehlert 2009	peanut	DNA	2 PCR real time	chocolate			5 mg/kg^{-1}	
Ehlert 2009	peanut	DNA	2 PCR real time	cookie			0.5 mg/kg^{-1}	
Ehlert 2009	peanut	DNA	2 PCR real time	walnut cookies			1 mg/kg^{-1}	
Ehlert 2009	peanut	crude	3 ELISA	chocolate			5 mg/kg^{-1}	
Ehlert 2009	peanut	crude	3 ELISA	cookie			5 mg/kg^{-1}	
Ehlert 2009	peanut	crude	3 ELISA	walnut cookies			100 mg/kg^{-1}	
Hird 2003	peanut	Ara h2	1 PCR real time	biscuit			2 ppm	> 2 ppm
Khuda 2012a	peanut	crude	1 ELISA, RIDASCREEN FAST	chocolate dark	linear regression	73		
Khuda 2012a	peanut	crude	2 ELISA, Veratox	chocolate dark	linear regression	35		
Khuda 2012a	peanut	crude	3 ELISA, Morinaga	chocolate dark	linear regression	11		
Khuda 2012a	peanut	crude	4 ELISA, BIOKITS	chocolate dark	linear regression	3		
Khuda 2012a	peanut	crude	5 ELISA Systems	chocolate dark	linear regression	29		
Khuda 2012b	peanut	crude	1 ELISA, RIDASCREEN FAST	sugar cookie	linear regression	11		
Khuda 2012b	peanut	crude	2 ELISA, Veratox	sugar cookie	linear regression	15		
Khuda 2012b	peanut	crude	3 ELISA, Morinaga	sugar cookie	linear regression	12		

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Khuda 2012b	peanut	crude	4 ELISA, BIOKITS	sugar cookie	linear regression	0		
Khuda 2012b	peanut	crude	5 ELISA Systems	sugar cookie	linear regression	2		
Kiening 2005	peanut	crude	1 ELISA	cereals	1-10 mg/kg	105-117		
Kiening 2005	peanut	crude	1 ELISA	chocolate dark	1-10 mg/kg	87-101		
Kiening 2005	peanut	crude	1 ELISA	chocolate milk	1-10 mg/kg	113-123		
Kiening 2005	peanut	crude	1 ELISA	cookie	1-10 mg/kg	92-107		
Kiening 2005	peanut	crude	1 ELISA	ice cream	1-10 mg/kg	94-110		
Morishita 2006	peanut	crude	1 IC - dipstick	chicken meatball or burger			5 µg/g	
Morishita 2006	peanut	crude	1 IC - dipstick	cookie			5 µg/g	
Morishita 2006	peanut	crude	1 IC - dipstick	Dumplings fried/steamed			5 µg/g	
Morishita 2006	peanut	crude	1 IC - dipstick	jelly			5 µg/g	
Morishita 2006	peanut	crude	1 IC - dipstick	Pickles in Vinegar/soy			5 µg/g	
Morishita 2006	peanut	crude	1 IC - dipstick	Potato salad			5 µg/g	
Morishita 2006	peanut	crude	1 IC - dipstick	sauce			5 µg/g	
Park 2005	peanut	crude	1 ELISA Veratox	cereals			5 µg/g	
Park 2005	peanut	crude	1 ELISA Veratox	chocolate			5 µg/g	
Park 2005	peanut	crude	1 ELISA Veratox	cookie			5 µg/g	
Park 2005	peanut	crude	1 ELISA Veratox	ice cream			5 µg/g	
Park 2005	peanut	crude	2 ELISA RIDASCREEN	cereals			5 µg/g	
Park 2005	peanut	crude	2 ELISA RIDASCREEN	chocolate			5 µg/g	
Park 2005	peanut	crude	2 ELISA RIDASCREEN	cookie			5 µg/g	
Park 2005	peanut	crude	2 ELISA RIDASCREEN	ice cream			5 µg/g	

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Park 2005	peanut	crude	3 ELISA BioKits	cereals			5 µg/g	
Park 2005	peanut	crude	3 ELISA BioKits	chocolate			5 µg/g	
Park 2005	peanut	crude	3 ELISA BioKits	cookie			5 µg/g	
Park 2005	peanut	crude	3 ELISA BioKits	ice cream			5 µg/g	
Pomes 2003	peanut	Ara h 1	1 ELISA in house	chocolate	0.006-0.01667 g/g	0-0		
Pomes 2003	peanut	Ara h 1	1 ELISA in house	cookie	0.006-0.01667 g/g	7-100		
Pomes 2003	peanut	Ara h 1	1 ELISA in house	flour	0.006-0.01667 g/g	54-94		
Pomes 2003	peanut	Ara h 1	1 ELISA in house	range of products				
Pomes 2004	peanut	Ara h 1	1 ELISA	chocolate			0.16 % w/w	
Speroni 2010	peanut	Ara h3/4	1 ELISA	biscuit				
Speroni 2010	peanut	Ara h 3/4	1 protein A-Pn-b ELISA	biscuit	5-15 mg/kg	93-94	0.8 mg/kg	2.4 mg/kg
Speroni 2010	peanut	Ara h 3/4	1 MP-NH2-PAMAM G 1.5-Pn-b ELISA	biscuit	5-15 mg/kg	114	0.8 mg/kg	2.4 mg/kg
Speroni 2010	peanut	Ara h3/4	1 ELISA	breakfast cereal				
Speroni 2010	peanut	Ara h 3/4	1 protein A-Pn-b ELISA	cereals	5-15 mg/kg	80-95	0.8 mg/kg	2.4 mg/kg
Speroni 2010	peanut	Ara h 3/4	1 MP-NH2-PAMAM G 1.5-Pn-b ELISA	cereals	5-15 mg/kg	84	0.8 mg/kg	2.4 mg/kg
Stephan 2004	peanut	Crude	1 ELISA	chocolate milk	10-200 ppm	64-111		
Stephan 2004	peanut	Crude	1 ELISA	milk	10-200 ppm	81-142		
Wen 2005a	peanut	Ara h1	1 LFA	chocolate			158 µg/g	
Yeung 1996	peanut	crude	1 ELISA	chocolate	2.5-20 µg/g	83-88		
Yeung 1996	peanut	crude	1 ELISA	cookie	2.5-20 µg/g	62-75		
Yeung 1996	peanut	crude	1 ELISA	crisps/ Thai cracker	2.5-20 µg/g	53-100		
Yeung 1996	peanut	crude	1 ELISA	ice cream	2.5-20 µg/g	45-81		
Yeung 1996	peanut	crude	1 ELISA	oil	2.5-20 µg/g	71-84		

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Yeung 1996	peanut	crude	1 ELISA	sauce	2.5-20 µg/g	84-92		
Yeung 1996	peanut	crude	1 ELISA	snack	2.5-20 µg/g	66-80		
Yeung 1996	peanut	crude	1 ELISA	snack	2.5-20 µg/g	80-95		
Yman 2006	peanut	crude	1 RIE	chocolate			70 µg/g	
Yman 2006	peanut	crude	2 SPR immunoassay	chocolate			1 µg/g	
Yman 2006	peanut	crude	3 Ridascreen	chocolate			1 µg/g	
Yman 2006	peanut	crude	4 BioKit (Tepnal BioSystems)	chocolate			1 µg/g	

3.2.13. Sesame

ELISA and one PCR method were assessed in the included studies for detecting sesame in matrices such as wheat cracker, cookie, muesli, crisp toast and bread (Table 3.39).

There were two studies evaluating in-house ELISAs. Hussain (2010) showed good recoveries from bread, cookies and snacks when spiked at a relatively high concentration of 24-200 µg/g. Redle (2010) showed similar results for their in-house ELISA.

There was only one study that evaluated PCR, Coisson (2010), and this was directed against the DNA for sesame mannitol dehydrogenase (Table 3.40). The limit of detection was given as 10% w/w for sausage meat samples spiked with sesame, which is equivalent to 10,0000µg/g (Table 3.41).

Table 3.39: Sesame: characteristics of included studies

Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Coisson (2010)	Sesame	Specific protein/peptide or gene DNA sesame mannitol dehydrogenase Si2S	Meat meat balls	Spiked **Source of spike** S. indicum (sesame seeds) and A. graveolens L (celery leaves). Samples purchased from commercial stores in Italy.	PCR
Husain (2010)	Sesame	Crude extract	Breakfast cereals Bread roll, wholegrain bread Cookie Crisp toast crisp toast 1, crisp	Spiked Field **Source of spike** purchased from local supermarkets Standardisation Protein concentration	ELISA

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
			toast 2, multigrain crisp toast Snack Wheat cracker **Field** Foods sampled sesame snack, sesame balls, crisp flakes, sesame flakes, cookies, crisp toast, sesame oil, biscuits, crackers, muesli, cereal	assessed using Bradford assay	
Redl (2010)	Sesame	Crude extract	Bread whole grain bread, whole wheat bread, crisp toast Cookie whole wheat Snack **Field** Foods sampled muesli, vegetarian, processed foods, crisp toast, snacks	Spiked Field **Source of spike** White peeled, unpeeled, and black sesame seeds were bought from different producers.	ELISA

Table 3.40: Sesame: description of assay

Study ID	Allergen	Assay details	Additional information
Coisson (2010)	Sesame	PCR with multiplex	PCR Multiplex with Lab-on-chip (R)-based detection capillary electrophoresis
Husain (2010)	Sesame	*ELISA*	**ELISA** Polyclonal detection antibody Competitive inhibition In-house
Redl (2010)	Sesame	ELISA	**ELISA** Sandwich In-house

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Table 3.41: Sesame: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Coisson 2010	sesame	DNA	1	meatball or burger	PCR	nr	10 % w/w	
Husain 2010	sesame	crude	1 ELISA	bread	25-200 µg/g	70-85		
Husain 2010	sesame	crude	1 ELISA	cookie	25-200 µg/g			
Husain 2010	sesame	crude	1 ELISA	Crisp toast	25-200 µg/g	92-103		
Husain 2010	sesame	crude	1 ELISA	muesli	0.001-1%	80-300		
Husain 2010	sesame	crude	1 ELISA	snack	25-200 µg/g	76-126		
Husain 2010	sesame	crude	1 ELISA	Wheat cracker	0.001-1 %	80-300		
Redl 2010	sesame	Crude	1 ELISA	Crisp toast	25-200 µg/g	89-145	5 µg/L	
Redl 2010	sesame	Crude	1 ELISA	snack	25-200 µg/g	48-108	3 µg/L	
Redl 2010	sesame	Crude	1 ELISA	white bread	25-200 µg/g	85-120		
Redl 2010	sesame	seeds	1 ELISA	Whole wheat cookies	0.001-0.5 %	80-200		
Redl 2010	sesame	Crude	1 ELISA	Whole-wheat bread	0.001-1%	20-220	5 µg/L	

3.2.14. Soy

There were seven recent studies investigating assays for soy proteins (Table 3.42). The assays studies include ELISA and PCR (Table 3.43) and the findings for limit of detection (Table 3.44) highlight that there are assay available that can to less than 5µg/ml.

Cuco (2012) compared their in-house ELISA to the commercial kit, KTI-ELISA. While there were good recoveries for cookie as a matrix, 83-118% for both assays the cookie mixtures spiked before baking had poor recoveries at only 0-32% recovery. This is an important finding for any products that could become contaminated with heat stable allergenic foods. Ma (2010) developed an ELISA against the major allergenic proteins of soy, glycinin. This assay detected the allergen in processed soy products spiked with glycinin and found recoveries of between 96-103%.

L'Hocine (2007) evaluated the Tepnal and ELISA systems kits and found the limits of detection in milk was good at 1 and 0.1µg/ml respectively.

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Table 3.42: Soy: characteristics of included studies

Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Cucu (2012)	Soy	Crude extract	Cookie	Spiked **Source of spike** Alpro (Wevelgem, Belgium) and Cargill (Mechelen, Belgium). A mixture of equal amounts of each kind.	ELISA
Espineira (2010)	Soy	Specific protein/peptide or gene DNA Lectin gene	Fish Canned fish	Spiked **Source of spike** Not reported	PCR
Hei (2012)	Soy	Specific protein/peptide or gene B-conglycinin	Defatted soybean **Field** soybean, soybean meal, soybean protein concentrate, soybean protein isolate, extruded soybean fermented soybean meal	Spiked Field **Source of spike** Not reported	ELISA
L'Hocine (2007)	Soy	Crude extract	Milk Cows milk (2% fat)	Spiked Field **Source of spike** Commercial soy flour (SF), soyprotein concentrate (SPC), and soy protein isolate (SPI) provided by "Aliments Newly Weds" (Boucherville, Que., Canada). Commercial soy protein hydrolysate (SPH) purchased from "Aliments UFL" (Boucherville, Que., Canada). Texturized soy protein (TSP) (Beef "Not!") was from DixieDiners' Club	ELISA
Ma (2010)	**Soy** Glycinin	Specific protein/peptide or gene Glycinin	Soybean Soybean products such as seed, meal and fermented paste	Spiked **Source of spike** Crude extracts of glycinin (Professor Shuntang Guo of China Agricultural University) further purified by the researchers Standardisation 92% pure assessed using SDS PAGE	ELISA
Morishita (2008)	Soy	Glycinin Soybean Gly m Bd 30k	Carrot Sherbet Cookie Jam	Spiked Field **Source of spike**	ELISA

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
			Pickles (Soy Sauce,vinegar) Potato Salad Sauce Tomato Soup Steamed and fried Chinese dumpling Hamburger	The soybeans (Glycine max var.Enrei, Haruyutaka, Nattosyoryu and Toyomusume) (Kinki University) were used to make defatted soybean powder (DSP) Standardisation DSP in the model processed foods was calculated, taking into account the protein content of the DSP and the change in weight of the model processed foods during their preparation. **Field** Purchased at local supermarkets (Ibaraki, Japan) in 2006	

Table 3.43: Soy: description of assay

Study ID	Allergen	Assay details	Additional information
Cucu (2012)	Soy	Test 1 Soybean- ELISA Test 2 KTI-ELISA	Test 1 ELISA In-house Test 2 Commercial company
Espineira (2010)	Soy	Test 1 End-point PCR Test 2 Real-time PCR	
Hei (2012)	Soy	Test 1 ELISA	**ELISA** Polyclonal capture antibody Monoclonal detection antibody Sandwich In-house
L'Hocine (2007)	Soy	Test 1 Tepnel Biosystems kit (Tepnel Biosystems Ltd., Flintshire, U.K.) Test 2 ELISA Systems kit(Elisa Systems,Windsor, Australia)	**ELISA** Commercial company
Ma (2010)	Soy Glycinin	Test 1 ELISA	**ELISA** Monoclonal detection antibody

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Study ID	Allergen	Assay details	Additional information
			Competitive inhibition
Morishita (2008)	Soy Glycinin	Test 1 ELISA	**ELISA** Polyclonal capture antibody Polyclonal detection antibody Sandwich

Table 3.44: Soy: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Cucu 2012	soy	crude	1 ELISA	cookie	21-84 µg/g	1.5-24		
Cucu 2012	soy	crude	1 ELISA	cookie (spiked after baking)	10-100 µg/g	94-115		
Cucu 2012	soy	KTI	2 KTI ELISA	cookie	21-84 µg/g	0-32		
Cucu 2012	soy	KTI	2 KTI ELISA	cookie (spiked after baking)	10-100 µg/g	83-118		
Espineira 2009	soy	crude	1 PCR real time	fish			0.05 % w/w	
Espineira 2009	soy	DNA	1 PCR real time	flour			100 mg/kg	
Espineira 2009	soy	crude	2 End-point PCR	fish			0.06 % w/w 25	
Espineira 2009	soy	DNA	2 End-point PCR	flour			10 mg/kg	
Hei	soy	beta-conglycinin	1 ELISA	Soybean protein concentrate	50-200 mg/g-1	88.1-106.6		
L'Hocine 2007	soy	crude	1 ELISA, Tepnel kit	milk	0.5-25 µg/ml	104.5-286	1 µg/ml	3 µg/ml
L'Hocine 2007	soy	crude	2 ELISA systems kit	milk	0.1-20 µg/ml	103.0-280.2	0.01 µg/ml	0.23 µg/ml
Ma 2010	soy	glycinin	1 ELISA	Extracted soybean meal	10-40 µg/ml	96-99		
Ma 2010	soy	glycinin	1 ELISA	Extruded soybean meal	10-40 µg/ml	98-103		
Ma 2010	soy	glycinin	1 ELISA	Fermented soybean paste	10-40 µg/ml	97-105		
Ma 2010	soy	glycinin	1 ELISA	Roasted full fat soybean	10-40 µg/ml	97-102		

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Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Ma 2010	soy	glycinin	1 ELISA	Soybean protein concentrate	1-4 µg/ml	95-103		
Ma 2010	soy	glycinin	1 ELISA	soybean seed	50-200 µg/ml	102-103		
Morishita 2008	soy	glycinin	1 ELISA	buffer	10 µg/g (one conc. only)		0.19 µg/ml	0.38 µg/ml
Morishita 2008	soy	glycinin	1 ELISA	croquette	10 µg/g (one conc. only)	92.8		
Morishita 2008	soy	glycinin	1 ELISA	rice gruel/ porridge	10 µg/g (one conc. only)	97.6		
Morishita 2008	soy	glycinin	1 ELISA	sauce	10 µg/g (one conc. only)	89.7		
Morishita 2008	soy	glycinin	1 ELISA	sausage	10 µg/g (one conc. only)	87.7		
Morishita 2008	soy	glycinin	1 ELISA	soup	10 µg/g (one conc. only)	98.7		

3.2.15. Walnut

Assays that detected walnut proteins in a range of foods such as biscuit, cake, chocolate, cashew pesto, cereals, cakes and flour (Table 3.45) were included in this review. The assays investigated included ELISA and PCR (Table 3.46). Doi (2008) used roasted walnut flour as the spike for a wide range of foods (Table 3.45), the results were shown for only one concentration of spike, and this was at 10µg/g (Table 3.47). In all foods matrices tested the recovery was good at 83-123 %. A study by Niemann (2009) showed the development and validation of an in-house ELISA that for chocolate demonstrated good recovery at 95-100% and a limit of detection of 1ppm or 1µg/g.

The study by Wang (2009) evaluated a real time PCR in a wheat matrix. The limit of detection was shown to be 0.001% w/w which equates to 1 µg/g. So this assay shows similar findings to the ELISA tests.

Table 3.45: Walnut: characteristics of included studies

Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Doi (2008)	Walnut	Specific protein/peptide or gene Walnut 2S protein	Biscuit Breakfast cereals Bread, Cake, sponge cake Jelly, Juice Meat chicken meatballs **Field**	Spiked Field **Source of spike** Defatted walnut powder (Chandler, Haward and Chinese Walnut) (Tabata Inc, Chiba, Japan and Mitsuboshi Boeki Ltd, Kobe, Japan) all	ELISA

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
			Foods sampled Variety of commercial foods such as chocolate and biscuits	were roasted 120 ^{o}C for 15 min.	
Niemann (2009)	Walnut	Crude extract roasted	Breakfast cereals Cake Chocolate Cookie	Spiked **Source of spike** Several brands of English walnuts and black walnuts, finely ground roasted (non-defatted)	ELISA
Wang (2009)	Walnut	Specific protein/peptide or gene Walnut vicilin-like seed storage protein	Flour wheat powder	Spiked **Source of spike** Juglans regia bought from local markets. All nuts were roasted/baked.	PCR

Table 3.46: Walnut: Description of Assay

Study ID	Allergen	Assay details	Additional information
Doi (2008)	Walnut	Test 1 ELISA	**ELISA** Polyclonal capture antibody Polyclonal detection antibody Sandwich In-house
Ehlert (2009)	Walnut	Test 1 Ligation dependent probe amplification Test 2 Hazelnut and peanut: real-time PCR Surefood allergen kit (Congen Biotechnology GmbH, Berlin, Germany) cashew real time PCR In house Test 3 Hazelnut and peanut: ELISA Ridascreen (R-Biopharm AG, Darmstadt, Germany)	
Niemann (2009)	Walnut	Test 1 ELISA	**ELISA** Polyclonal capture antibody Polyclonal detection antibody Sandwich In-house
Wang (2009)	Walnut	Test 1 Real-time PCR In-house	

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Table 3.47: Walnut: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Doi 2008	walnut	soluble protein	1 ELISA	biscuit	10 µg/g	83		
Doi 2008	walnut	soluble protein	1 ELISA	bread	10 µg/g	123		
Doi 2008	walnut	soluble protein	1 ELISA	cake	10 µg/g	100		
Doi 2008	walnut	soluble protein	1 ELISA	chicken meatball or burger	10 µg/g	120		
Doi 2008	walnut	soluble protein	1 ELISA	jelly	10 µg/g	102		
Doi 2008	walnut	soluble protein	1 ELISA	juice	10 µg/g	101		
Doi 2008	walnut	soluble protein	1 ELISA	rice gruel/ porridge	10 µg/g	115		
Niemann 2009	walnut	crude	1 ELISA	cake	1-100ppm	not clear		
Niemann 2009	walnut	crude	1 ELISA	cereals	1-100ppm	not clear		
Niemann 2009	walnut	crude	1 ELISA	chocolate	1-100ppm	95-104	0.5 ppm	1 ppm
Niemann 2009	walnut	crude	1 ELISA	cookie	1-100ppm	not clear		
Wang 2009	walnut	crude	1 Real-Time PCR	wheat flour			0.001 % w/w	

3.2.16. Other

There were two studies that looked at allergenic foods not listed in the previous categories, the foods detected were the tree nuts macadamia and pecan and mustard (Table 3.48). Lee (2008) evaluated an ELISA to detect mustard, the antibodies were directed against whole/crude mustard proteins (Table 3.49). Sausage was spiked with between 1 and 1000 ppm and gave good recoveries of between 80-107% (Table 3.50). The same study evaluated a commercial ELISA Systems kit, and this achieved only 13-20% recovery under the same conditions.

The Ligation dependent probe amplification gave a very poor limit of detection of 1000 mg/kg^{-1} (100 µg/g), for both macadamia and pecan in a walnut cookie matrix, Ehlert (2009) (Table 3.50).

Table 3.48: Other: characteristics of included studies

Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
Ehlert (2009)	Pistachio	Crude extract Specific protein/peptide	Cookie Pesto cashew	Spiked **Source of spike** Nut materials, sesame seeds, ingredients of self-prepared DNA plant and animal	ELISA PCR Ligation-dependent probe

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Study ID	Allergen	Assay developed to detect	Food matrix tested	Spiking experiments or field samples tested and source of spike	Type of assays tested
		or gene DNA		materials used to test the specificity of the method and spike samples of chocolate, were obtained from the Bavarian Health and Food Safety Authority (Oberschleibheim, Germany)	amplificatio n
Ehlert (2009)	Pecan	Crude extract Specific protein/peptide or gene DNA	Cookie Pesto cashew	Spiked **Source of spike** Nut materials, sesame seeds, ingredients of self-prepared DNA plant and animal materials used to test the specificity of the method and spike samples of chocolate, were obtained from the Bavarian Health and Food Safety Authority (Oberschleibheim, Germany)	ELISA PCR Ligation-dependent probe amplificatio n
Lee (2008)	Mustard	Crude extract	Meat Sausage Cooked Frankfurter **Field** Foods sampled baked beans, salad dressing, sauce and marinade, seasoning mix, sausage	Spiked Field **Source of spike** Not reported Standardisation Unclear	ELISA

Table 3.49: Other: Description of Assay

Study ID	Allergen	Assay details	Additional information
Ehlert (2009)	Pistachio	Test 1 Ligation dependent probe amplification Test 2 Hazelnut and peanut: real-time PCR Surefood allergen kit (Congen Biotechnology GmbH, Berlin, Germany) cashew real time PCR In house Test 3 Hazelnut and peanut: ELISA Ridascreen (R-Biopharm AG, Darmstadt, Germany)	
Ehlert (2009)	Pecan	Ligation dependent probe amplification	
Lee (2008)	Mustard	Test 1 ELISA sheep Test 2 ELISA rabbit	**ELISA** Sandwich Polyclonal capture using rabbit or sheep antibody

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Table 3.50: Other: accuracy and limit of detection and quantification

Study ID	Allergen	Specific protein	Test Type	Matrix	Conc. for recovery	% recovery	Limit of detection units	Limit of quantification units
Ehlert 2009	macadamia	DNA	1 LPA	walnut cookies			1000 mg/kg^{-1}	
Lee 2008	mustard	crude	1 ELISA	sausage	1-1000 ppm	80-107		
Lee 2008	mustard	crude	2 ELISA systems	sausage	1-1000 ppm	12.6-20.0		
Ehlert 2009	pecan	DNA	1 LPA	walnut cookies	nr	nr	1000 mg/kg^{-1}	

3.2.17. Quality of studies

The quality of the included studies was assessed using predetermined criteria as outlined in the methods section (Table 3.2). For some food matrix types it would seem that the allergen spike was not mixed with the food in a way that would reflect real world situations. For example grinding up foods that were already cooked to make a powder and then mixing with the powdered allergen extract. These studies were therefore marked as a risk of bias for the spiking procedure. A few of the studies used standardised extracts from a trusted source and they received a low risk of bias grading for this item. While nearly all studies indicated that they repeated the assay procedure, only a few of the studies showed their findings for repeat spiking and extraction processes and these were graded as low risk of bias for this item. (Table 3.51)

Table 3.51: Quality of the included studies

Short Title	Spiking procedure	Source of extract for spike	Extraction repeated
Akiyama (2003)	High risk of bias	Low risk of bias	High risk of bias
Akiyama (2004a)	High risk of bias	Low risk of bias	High risk of bias
Akiyama (2004b)	High risk of bias	Low risk of bias	High risk of bias
Akkerdaas (2004)	Low risk of bias	High risk of bias	High risk of bias
Allred (2012)	Low risk of bias	Low risk of bias	High risk of bias
Ben (2003)	Unclear risk of bias	Low risk of bias	Unclear risk of bias
Blais (2001)	High risk of bias	Low risk of bias	High risk of bias
Brzezinski (2006)	High risk of bias	High risk of bias	High risk of bias
Ben Rejeb (2003)	High risk of bias	High risk of bias	Unclear risk of bias
Ben Rejeb	High risk of bias	Unclear risk of bias	Unclear risk of bias

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Short Title	Spiking procedure	Source of extract for spike	Extraction repeated
(2005)			
Brzezinski (2007)	High risk of bias	High risk of bias	Low risk of bias
Careri (2007a)	Unclear risk of bias	Low risk of bias	Unclear risk of bias
Careri (2007b)	Spiked or field Unclear risk of bias	High risk of bias	Unclear risk of bias
Careri (2008)	Unclear risk of bias	Unclear risk of bias	High risk of bias
Coisson (2010)	Low risk of bias	Low risk of bias	High risk of bias
Costa (2012)	High risk of Bias	Low risk of bias	High risk of bias
Cucu (2012)	Low risk of bias	Low risk of bias	Unclear risk of bias
Cucu (2012)	Low risk of bias	High risk of bias	Unclear risk of bias
Demmel (2011)	Low risk of bias	Unclear risk of bias	Low risk of bias
Deng (2012)	Low risk of bias	Unclear risk of bias	Low risk of bias
Cai	Unclear risk of bias	High risk of bias	Unclear risk of bias
Doi (2008)	Unclear risk of bias	Unclear risk of bias	Low risk of bias
Drs (2004)	Unclear risk of bias	Unclear risk of bias	Unclear risk of bias
Ehlert (2009)	Low risk of bias	Low risk of bias	Unclear risk of bias
Eissa (2012)	Unclear risk of bias	Unclear risk of bias	Unclear risk of bias
Espineira (2010)	Unclear risk of bias	Unclear risk of bias	Unclear risk of bias
Faeste (2006)	High risk of Bias	Low risk of bias	Low risk of bias
Faeste (2008)	Low risk of bias	High risk of bias	Unclear risk of bias
Fuller (2006)	Unclear risk of bias	High risk of bias	High risk of bias
Garber (2010a) almond	Low risk of bias	High risk of bias	Unclear risk of bias
Garber (2010b) hazelnut	Low risk of bias	High risk of bias	Unclear risk of bias
Gaskin (2011)	Low risk of bias	High risk of bias	Low risk of bias
Haasnoot (2004)	Unclear risk of bias	High risk of bias	Unclear risk of bias
Hefle (2001)	Low risk of bias	High risk of bias	High risk of bias
Hefle (2004)	Low risk of bias	Unclear risk of bias	Low risk of bias

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Short Title	Spiking procedure	Source of extract for spike	Extraction repeated
Hei (2012)	Unclear risk of bias	Unclear risk of bias	Unclear risk of bias
Hird (2003)	Low risk of bias	Low risk of bias	Unclear risk of bias
Holden (2005)	High risk of Bias	Low risk of bias	Low risk of bias
Holden (2007)	Low risk of bias	High risk of bias	Unclear risk of bias
Holzhauser (1999)	Unclear risk of bias	Low risk of bias	Unclear risk of bias
Holzhauser (2002)	Unclear risk of bias	Unclear risk of bias	Unclear risk of bias
Husain (2010)	Unclear risk of bias	Low risk of bias	Unclear risk of bias
Kaw (2008)	Low risk of bias	Low risk of bias	Unclear risk of bias
Khuda (2012a) egg	Low risk of bias	Low risk of bias	Low risk of bias
Khuda (2012a) milk	Low risk of bias	Low risk of bias	Low risk of bias
Khuda (2012b) Peanut	Low risk of bias	Low risk of bias	Low risk of bias
Khuda (2012b) egg	Low risk of bias	Low risk of bias	Low risk of bias
Khuda (2012a) peanut	Low risk of bias	Low risk of bias	Low risk of bias
Khuda (21012 b) milk	Low risk of bias	Low risk of bias	Low risk of bias
Kiening (2005)	High risk of bias	Low risk of bias	Unclear risk of bias
Lacorn (2011)	Low risk of bias	Low risk of bias	Low risk of bias
Lee (2008)	Low risk of bias	Unclear risk of bias	Unclear risk of bias
L'Hocine (2007)	Unclear risk of bias	Unclear risk of bias	Unclear risk of bias
Ma (2010)	High risk of bias	Low risk of bias	Low risk of bias
Mena (2012)	Unclear risk of bias	Unclear risk of bias	Unclear risk of bias
Monaci (2008)	Unclear risk of bias	Unclear risk of bias	Low risk of bias
Monaci (2011)	Low risk of bias	Unclear risk of bias	Unclear risk of bias
Morishita (2006)	Unclear risk of bias	Unclear risk of bias	Unclear risk of bias
Morishita (2008)	Low risk of bias	Low risk of bias	High risk of bias

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Short Title	Spiking procedure	Source of extract for spike	Extraction repeated
Niemann (2009)	Low risk of bias	High risk of bias	Unclear risk of bias
Panda (2010)	Low risk of bias	Unclear risk of bias	Unclear risk of bias
Park (2005)	Low Risk of bias	Low risk of Bias	Low risk of bias
Piknova (2008)	Low Risk of bias	High risk of Bias	Unclear risk of bias
Pomes (2003)	High Risk of Bias	High risk of Bias	Unclear risk of bias
Pomes (2004)	Unclear risk of bias	High risk of Bias	Unclear risk of Bias
Redl (2010)	Low Risk of bias	High risk of Bias	Unclear risk of bias
Roeder (2010)	Low risk of bias	High risk of bias	Low risk of bias
Roeder (2011)	Low risk of bias	Low risk of bias	Unclear risk of bias
Roux (2001)	High risk of bias	High risk of bias	High risk of bias
Schneider (2010a)	High risk of bias	High risk of bias	Unclear risk of bias
Schneider (2010b)	High risk of bias	Unclear risk of bias	Unclear risk of bias
Sealey-Voyksner (2010)	High risk of bias	Unclear risk of bias	Unclear risk of bias
Sharma (2009)	Unclear risk of bias	High risk of bias	Unclear risk of bias
Shibahara (2007)	High risk of bias	High risk of bias	Unclear risk of bias
Shibahara (2013b)	Unclear risk of bias	High risk of bias	Low risk of bias
Shon (2010)	Unclear risk of bias	Unclear risk of bias	Unclear risk of bias
Speroni (2010)	Unclear risk of bias	High risk of bias	Unclear risk of bias
Stephan (2002)	Unclear risk of bias	Unclear risk of bias	Unclear risk of bias
Stephan (2004)	Unclear risk of bias	Source of spike Source Not reported	Unclear risk of bias
Taguchi (2011)	Unclear risk of bias	High risk of bias	Unclear risk of bias
Wang (2009)	Low risk of bias	High risk of bias	High risk of bias
Wang (2011)	Low risk of bias	High risk of bias	Low risk of bias
Weber (2006)	Unclear risk of bias	High risk of bias	Unclear risk of bias
Wen (2005a)	Unclear risk of bias	High risk of bias	Unclear risk of bias

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Short Title	Spiking procedure	Source of extract for spike	Extraction repeated
Werner (2007)	Unclear risk of bias	Unclear risk of bias	High risk of bias
Wu (2010)	Unclear risk of bias	High risk of bias	High risk of bias
Yeung (1996)	Unclear risk of bias	High risk of bias	Unclear risk of bias
Yeung (1997)	Unclear risk of bias	Unclear risk of bias	Unclear risk of bias

3.3. Discussion and Conclusions

This review revealed that there are a large number of studies that have investigated the effectiveness of assays for detecting allergens in foods since 2004. There was variability in the types of experiments carried out, the format and statistical analysis of the data presented and in specific techniques such as the method of spiking and in the source of extracts used to validate the assay in the studies retrieved for this review. In a large proportion of studies there was a potential high risk of bias for at least one item. There are a range of criteria that could be used to validate assays and ensure that there is consistent quality control across institutions. We focused on the accuracy as determined by the percentage recovery of a spiked sample and the limit of detection of each allergen within a suitable food matrix; this is just one aspect of quality control.

The range of quality criteria that should be assessed in the validation of any assay to detect a chemical or biologically active compound and these are outlined by the International Conference on Harmonisation of Technical Requirements for Registration of Pharmaceuticals for Human Use (ICH) (1995). However these guidelines require adaptation for the specific requirements of detecting allergens in foods. There are a number of standards and guidelines produced throughout the world to facilitate this. Within Europe there are two standards that apply to the detection of allergens in foods **EN 15633-1: 2009** for immunoassays and **EN 15634-1: 2009,** for molecular biological, these standards are produced and published by the European Committee for Standardization (2013). In Japan official detection assays were adopted by the government and the method used to validate this assays published (Akiyama, Imai and Ebisawa; 2011). In addition to assay quality criteria those developing and using the tests must be aware of current research and guidelines on the types of foods found to be allergenic and the quantities could potentially cause symptoms. The Codex Alimentarius Commission, established by Food and Agriculture Organization of the United Nations and the World Health Organization develops harmonised international food standards, guidelines and codes of practice and so should be a useful source for this type of information.

Before funding or adopting an assay and extraction procedure it is recommended that all key quality and validation data are reviewed in accordance with the relevant standards and that each laboratory carry out their own validation experiments to assess the performance of the assay within their specific context.

The results section within this review show the percentage recovery and the limit of detection and quantification for each assay when different food matrices were spiked with the allergenic food. This information is grouped by allergen. It was apparent that for many of the allergenic foods there were assays that could detect down to 1 µg/g. Data was available for the following allergenic foods: almond, Brazil nut, Buckwheat, cashew nut, celery, egg, fish and shell fish, hazelnut, lupine, milk, mustard, peanut, pecan, sesame, soy and walnut.

The immunoassays generally gave a similar limit of detection as the PCR assays. Although PCR is extremely sensitive for detecting tiny quantities of DNA we were reporting the ability to detect contamination with crude preparations of the allergenic foods for example peanut flour, rather than extracts of peanut DNA.

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The individual findings can be found in the results sections however there are several points to consider when looking at these findings as a whole. These include:

- The limit of detection reported by some of these studies showed that the values reported by manufacturers are not always achieved in practice. Limits of detection for the allergen extracted from a similar food to the intended use are essential.

- The food matrix contaminated with an allergenic food is highly likely to affect the performance of the extraction processes and limit of detection of the assay. Chocolate in particular could mask the allergen, and decrease the percentage recovery and increase the limit of detection. Users should ensure that the assay is validated for the specific food matrix.

- Consideration should be made as to whether users need to know the limit of detection for a specific protein, for example a food additive such as lysozyme or presence of any protein from the allergenic food.

- Processing, for example baking, can reduce the percentage recovery and increase the limit of detection. If the contamination could have occurred prior to processing then the validation experiments should include this processing step.

- Internationally agreed standards for the allergenic food source used in the spiking experiments the concentration of specific proteins will vary, and this in turn will lead to differences in the measured limit of detection by ppm or weight/weight.

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3.5. List of Excluded Studies

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ABBREVIATIONS

DBPCFC	Double blind placebo controlled food challenge
HN	Hazelnut
IgE	Immunoglobulin -E
OAS	Oral allergy syndrome
OFC	Open Food Challenge
PCR	Polymerase Chain Reaction
PBS	Phosphate Buffered Saline
SPT	Skin Prick Test

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