Allergy Today

Allergy UK's publication for healthcare professionals

Winter 2019/20

AllergyUK

Allergy News - The Facts about Anaphylaxis
Practical Management of Atopic Eczema - Grass Pollen
Indoor Aeroallergens - Adult Food Allergy

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Allergy UK is the operational name of the British Allergy Foundation. We are the leading national charity for people living with allergic disease, providing advice and support on all types of allergy. Allergy UK acts as the 'voice' of the millions of people who live with allergic disease, representing the views and healthcare needs of those affected by this multi-organ disease.

Our vision is for everyone affected by allergy to receive the best possible care and support and we work with healthcare professionals, health organisations and government towards our mission to raise the profile of allergy at all levels.

Contents

05 **Foreword**
Dr. Adam Fox Chair of the Allergy UK Health Advisory Board; Consultant Paediatric Allergist, Evelina London Children's Hospital

06 **Welcome**
Amena Warner, Head of Clinical Services, Allergy UK and Carla Jones, CEO, Allergy UK

07 **Allergy News**
News from the world of Allergy

08 **Anaphylaxis - The Facts**
James Gardner

12 **Atopic Eczema (Dermatitis) – Practical Management**
Julie Van Onselen

15 **Grass Pollen, Environmental DNA and Respiratory Health: An Introduction to the PollerGEN Project**
Dr Nick Osborne, Dr Francis Rowney

19 **Indoor Aeroallergens and their Impact on Health**
Professor Chris Corrigan

22 **An Introduction to Adult Food Allergy**
Kate Roberts MSc RD, Allergy UK Clinical Dietitic Advisor

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Foreword

Dr. Adam Fox
Chair of the Allergy UK Health Advisory Board
Consultant Paediatric Allergist, Evelina London Children's Hospital

Welcome to this edition of Allergy Today.

Since the last edition of this magazine when I talked about the need to instigate changes to make a difference to the lives of people living with allergy, the death of another young person from a catastrophic reaction to food hit the headlines. After the inquest into the death of 13 year old Karanbir Singh Cheema, who died after another pupil at his school threw a piece of cheese at him, the Coroner raised a number of matters of concern. In fact, these concerns were similar to those she had raised following the inquest into the death of 14 year old Nasar Ahmed three years ago following an anaphylactic reaction while he was at school. They focused on the need for robust school policies and staff training in allergy management, as well as targeted education for pupils to create a better understanding of the realities and risks of living with a food allergy.

These concerns have now been expressed in two Prevention of Future Deaths report from the same Coroner. This Coroner had heard the full and horrifying details of the circumstances surrounding these two tragic deaths and saw very clearly how an embedded school policy around allergy management, coupled with improved education of staff and pupils, could help prevent further avoidable deaths in a place where young people should feel safe – at school.

While the change in legislation that allows schools to obtain "spare" AAI devices without prescription certainly goes some way towards creating a safer school environment, this is only the start of the journey. There has been no movement by government to proceed with mandatory guidance and training for anaphylaxis management, despite the fact the fact that this measure has been successfully implemented in other countries such as Australia and the USA.

Now a collaboration of BSACI, Allergy UK, the Anaphylaxis Campaign and other stakeholders has been pressing the Department for Education to heed the Coroner's concerns and take action to work with us to develop mandatory policy and training for schools. This is long overdue.

We will continue to press our case strongly and we hope that the Secretary of State for Education will give this their urgent attention.

Welcome

At this time of year we will have completed a full review of our activities and achievements over the last financial year. Our 2018/2019 Annual Report, which you can read on our website, provides a powerful insight into our work towards our mission of improving the lives of people living with allergic disease. As the leading patient charity in this space we are committed to raising awareness and understanding of allergic disease and the impact that it has on people's lives. It's good to report that, for the first time, we are seeing what we believe to be signs of positive change. Our engagement with government and policy makers, whether it is around improving food labelling, considering the challenges of allergic people travelling by air or acknowledging the effects of air quality on our allergic community has been significant over the last year and we are hopeful that legislative changes around the care of allergic pupils in school may now be on the horizon.

In this edition of Allergy Today there is news of some of the key projects we have been involved with over the last year, together with articles on a number of topics, including allergic conditions that are exacerbated by indoor air quality. We hope you will enjoy reading this Allergy Today.

Finally, we want to acknowledge the huge contribution of time, knowledge and advice that Professor Adam Fox has made to Allergy UK as a Trustee over the last ten years. His counsel and support has been a significant factor in the charity's growing influence over the decade. While Professor Fox stood down from his Allergy UK Trustee role this autumn he will, we are very glad to say, remain Chairman of the Allergy UK Health Advisory Board. We want to convey our sincere thanks for his work as one of our charity's Trustees and our appreciation that he will still be involved with Allergy UK through his Health Advisory Board chairmanship.

We do hope you enjoy reading this issue.

Carla Jones, CEO

Amena Warner, Head of Clinical Services

Allergy News

Food Labelling

Allergy UK was closely involved in workshops and meetings around the public consultation on allergen information labelling on food pre-packed for direct sale, including a meeting with the then DEFRA Secretary of State Michael Gove. The consultation was launched by DEFRA, in collaboration with the FSA, following the tragic death of Natasha Ednan-Laperouse. Our direct engagement with the allergic community through our social media, website and mailing lists means that we were able to share information on the consultation to encourage and empower those living with food allergy to make their views heard on this important subject.

We strongly advocated for full allergen information to be provided on pre-packed direct sale food and were very pleased that this option was successful and will come into effect in 2021. These tougher labelling laws will provide improved safety for the two million people food-allergic consumers and allow them to make more informed decisions when purchasing food. However, there is still more to be done to improve food labelling and Allergy UK continues to be closely involved in discussions and initiatives around this topic.

International Life Sciences Institute Project

- A framework to help define an appropriate level of protection for consumers with food allergies

The aim of this paper is to describe the current situation in the management of unintended allergen presence. A critical element missing from current discussions is the absence of any transparent consideration of what level is tolerable in relation to the consequences of unintended allergen presence at an individual and public health level. Allergy UK and other stakeholders, including the FSA, food industry experts and food scientists will discuss the obstacles to defining a tolerable risk, and therefore an appropriate level, and suggest a way forward.

Air Quality

There has been a considerable amount of activity from government and policy makers around the quality of the air we breathe, both outside and inside our homes. We have been advocating for improved air quality for a number of years because of its impact on people living with allergic disease and this year we have made air quality our campaign theme, focusing on outdoor air quality for our Allergy Awareness Week in the spring and indoor air quality for our second awareness week in autumn, 2019. As well as responding to DEFRA's 'Draft Clean Air Quality' consultation last year Allergy UK takes part in the APPG (All Party Parliamentary Group) for Healthy Homes and Buildings and a number of other groups working on the improvement of the quality of the air we breathe. Most recently our CEO was a topic advisor representing the allergic community in discussions around the NICE Guidelines on Indoor Air Quality which will be published later this year.

Allergy Management in Schools

In a collaborative letter to the Department for Education, with Anaphylaxis Campaign and BSACI, Allergy UK has joined the call for robust policies and staff training

to improve the safety of allergic pupils at schools, as well as education for pupils to better understand the realities of living with a food allergy. Also, as a member of the Health Conditions in Schools Alliance, we have added our voice to the broader cause of more stringent measures for the protection of children with a range of medical conditions while they are at school.

Our own SAAG (School Allergy Action Group) free online toolkit, which helps secondary schools develop and implement an effective allergy management policy, has signed up over 100 schools since its launch in May, 2019, which indicates a growing awareness by schools that a robust policy for allergy management is essential for the protection of pupils.

Allergy UK Research and Development Nurse

In 2015, Allergy UK provided a grant to the University of Edinburgh for a piece of research which aims to highlight the positive impact that a nurse-led service can have on the diagnosis, treatment and referral to secondary and tertiary care, with the ultimate aim of improving the quality of, life of patients living with allergic disease. Nurse-led clinics in NHS Lothian are run out of two GP surgeries, now covering 19 surgeries in the area which can refer patients. As well as helping patients manage their condition, the nurse is also a nurse-prescriber with authority to prescribe appropriate treatments and medicines for patients.
The first phase outcomes this important piece of research, which will include both quantitative and qualitative data, are due to be published in early 2020.

A Matter of Fact

During the last financial year nearly 80,000 Factsheets were downloaded from the Allergy UK website (an increase of 28% over the previous year) and over 8,000 people contacted us through our direct communication channels. Understanding their allergies can be confusing for people who live with allergy, or who suspect that they have an allergy. This is testament to Allergy UK's vital role in providing help and advice to these people in response to a direct need, as well as the importance of our role in creating awareness of allergic disease and its impact on people's lives. As we work on our next five year strategy supporting those living with allergic disease in every aspect of their lives remains at the top of our strategic aims.

ANAPHYLAXIS
The Facts

By James Gardner

James Gardner is currently the Children's and Young Persons Allergy Nurse Consultant at the Great North Children's Hospital in Newcastle and Associate Clinical Lecturer at Newcastle University. He has worked in many of the allergy centres in London and completed his MSc in Allergy from the University of Southampton. He is passionate about allergy and raising awareness and is a keen tweeter @allergynurseuk!

Anaphylaxis has been a high profile news item over the past 12 months due to sadly some high profile deaths including that of Natasha Ednan-Laperouse who sadly died following anaphylaxis to sesame and Megan Lee who had a fatal anaphylaxis from ingestion of peanut from a takeaway that had not disclosed ingredient. More recently we have heard of 13 year old Karanbir

Singh Cheema who died after anaphylaxis to cheese whilst at school.

All these tragic cases have raised awareness of allergy and the risks of anaphylaxis.

What is Anaphylaxis and its Clinical Features?
The Resuscitation Council, NICE and World Allergy Organisation [1,2,3] define anaphylaxis as a severe, life-threatening, generalised or systemic hypersensitivity reaction.

It can be characterised by rapidly developing, life-threatening problems involving the airway (pharyngeal or laryngeal oedema) and/or breathing (bronchospasm with tachypnoea) and/or circulation (hypotension and/or tachycardia). In most cases, there are associated skin and mucosal changes.

Symptoms and signs of anaphylaxis usually occur within two hours of exposure to the allergen [4] usually within 30 minutes for a food allergen and even faster with medication or insect stings.

Prevalence?
In the UK, between the years 1992 and 2012, Hospital admissions from all-cause anaphylaxis increased by 615% however fatal anaphylaxis had not increased. [5]
An estimated 1 in 1,333 of the English population have at some point in their lives experienced anaphylaxis [6]

Triggers / Causes
Food is a particularly common trigger in children, while medicinal products are much more common triggers in older people. In the UK it is estimated that 500,000 people have had a venom-induced anaphylactic reaction and 220,000 people up to the age of 44 have had a nut-induced anaphylactic reaction [7].

Risk Factors
Co-existing asthma is a risk factor for anaphylaxis, especially if severe and uncontrolled [8,9].

Mast cell disorders and underlying cardiovascular disease, are also associated with an increased risk of severe or fatal anaphylaxis [10,11,12].

Co-factors which increase the risk of an allergic reaction occurring or its severity include exercise, fever, acute infection, premenstrual status and emotional stress [13]

NSAID and alcohol have been shown to enhance some food allergic reactions [14].

How it is Treated?
For patients having an anaphylactic reaction the minimum treatment is:

1. Recognition that they are seriously unwell.
2. An early call for help.
3. Initial assessment and treatments based on an ABCDE approach.
4. Adrenaline therapy if indicated.
5. Investigation and follow-up by an allergy specialist.

Adrenaline must be administered to all patients experiencing anaphylaxis. It is the first line for treatment. It should also be administered to those with clinical features that are likely to evolve into anaphylaxis.

The Resuscitation Council guidelines note that Adrenaline helps in 3 different ways by working on (i) α-1 receptors causing peripheral vasoconstriction thereby reversing hypotension and mucosal oedema, (ii) β-1 receptors by increasing both the rate and force of cardiac contractions thereby reversing hypotension, and (iii) β-2 receptors reversing bronchoconstriction and reducing the release of inflammatory mediators.

The intramuscular (IM) route is the best for most individuals who require adrenaline to treat an anaphylactic reaction. The best site for IM injection is the anterolateral aspect of the middle third of the thigh.43resus

The needle used for injection needs to be sufficiently long to ensure that the adrenaline is injected into muscle.

The subcutaneous or inhaled routes for adrenaline are not recommended for the treatment of an anaphylactic reaction because they are less effective.

There are no absolute contra-indications to treatment with adrenaline in a patient experiencing anaphylaxis; benefits outweigh the risks in the elderly and patients with pre-existing cardiovascular disease (6resus).

Patients experiencing anaphylaxis should be kept still and positioned according to their presenting symptoms. With a presentation of respiratory distress (the most common) - position sitting up. When there is circulatory instability, position lying on back with the lower extremities elevated.

Second line treatments in anaphylaxis would be: Oxygen, Fluid support (in patients with cardiovascular instability), inhaled short-acting beta-2-agonists (salbutamol), antihistamines and corticosteroids which may help prevent or shorten protracted reactions. They may prevent a biphasic reaction.

After anaphylaxis

Adrenaline auto-injectors should be offered to people after emergency treatment for suspected anaphylaxis, as an interim measure before they have an allergy appointment.

It is important to use an adrenaline auto-injector as soon as possible if an anaphylactic reaction is suspected. Ensuring that people know when and how to use their device will help ensure timely and correct use if they have a further anaphylactic reaction.

Healthcare professionals should be familiar with the use of the most commonly available auto-injector devices. There are three devices available in the UK: Epipen, Jext and Emerade. Epipen and Jext are available in 0.15mg (Junior) and 0.3mg (Standard) doses. Emerade is also available in a 500mcg dose.

The BSACI guidelines show the doses of adrenaline available for self-administration:

Group	Dose adrenaline
Adult or child> 12 years	* 0.5 mg
Adult, adolescent or child > 30 kg	0.3 mg
Children 15–30 kg	** 0.15 mg
Children < 15 kg (unlicensed)	0.15 mg

*0.3 mg more appropriate for a smaller child > 12 years.
**0.3 mg may be more appropriate for some children, for example over 25 kg.

Following a European review of all adrenaline auto-injectors approved in the EU, the MHRA recommend that 2 adrenaline auto-injectors are prescribed, which patients should carry at all times. This is particularly important for patients with allergic asthma, who are at increased risk of a severe anaphylactic reaction. Patients with allergies and their carers should be trained to use the particular auto-injector they have been prescribed and encouraged to practise using a trainer device. Patients are advised to check the expiry date of the adrenaline auto-injectors and obtain replacements before they expire.

Follow up is important

NICE clearly state that patients with anaphylaxis should be referred to an allergy service.

An allergy service will provide a definitive diagnosis and identify the cause of an anaphylactic reaction and will ensure that people receive the correct advice and treatment.

If people are not referred to a specialist allergy service their safety might be compromised and they may receive inappropriate management, have an increased risk of recurrent anaphylactic reactions and feel anxious about possible recurrence.

An Anaphylaxis Emergency Action Plan with likely presenting symptoms and how to respond to each should be issued to the patient and are easily available on the BSACI, RCPCH, RCGP and Allergy UK websites. Studies have shown that after the inception of a management plan, accidental reactions are less common, at least in children with peanut or tree nut allergies [15,16]. A management plan used by a multi-disciplinary allergy clinic has a positive effect on parental knowledge of avoidance measures and emergency treatment of reactions [17].

Differential diagnosis

Sometimes an anaphylactic reaction can present with symptoms and signs that are very similar to anaphylaxis such as life-threatening asthma – this is commonest in children.

• A low blood pressure (or normal in children) with a petechial or purpuric rash can be a sign of septic shock.

Non life-threatening conditions (these usually respond to simple measures):

• Faint (vasovagal episode).
• Panic attack.
• Breath-holding episode in child.
• Idiopathic (non-allergic) urticaria or angioedema

Guidelines

There are a number of guidelines available for in regards to anaphylaxis for further reading:
NICE, Resuscitation Council UK, BSACI, EAAC

REFERENCES

1. NICE Anaphylaxis: assessment and referral after emergency treatment Clinical guideline [CG134] Published date: December 2011
2. Resuscitation Council (UK) Emergency treatment of anaphylactic reaction: Guidelines for healthcare providers 2008
3. World Allergy Organization Guidelines for the Assessment and Management of Anaphylaxis World Allergy Organization Journal, 2010; 4(2):13-37, February 2011
4. de Silva IL, Mehr SS, Tey D, Tang MLK. Paediatric anaphylaxis: a 5 year retrospective review. Allergy 2008;63:1071-1076
5. Turner, Paul J. et al. Increase in anaphylaxis-related hospitalizations but no increase in fatalities: An analysis of United Kingdom national anaphylaxis data, 1992-2012 Journal of Allergy and Clinical Immunology, Volume 135, Issue 4, 956 - 963.
6. Sheikh A, Hippisley-Cox J, Newton J, Fenty J. Trends in national incidence, lifetime prevalence and adrenaline prescribing for anaphylaxis in England. J R Soc Med. 2008;101(3):139–143. doi:10.1258/jrsm.2008.070306
7. P. Ewan, N. Brathwaite, S. Leech, D. Luyt, R. J. Powell, S. Till, S. Nasser and A. Clark, Clinical & Experimental Allergy, 2016 (46) 1258–1280.
8. Calvani M, Cardinale F, Martelli A, Muraro A, Pucci N, Savino F et al.; Italian and members Members of the Italian Society of Pediatric Allergy and Immunology (SIAIP) Anaphylaxis Study Group. Risk factors for severe pediatric food anaphylaxis in Italy. Pediatr Allergy Immunol 2011;22:813-819.
9. Gonzalez-Perez A, Aponte Z, Vidaurre CF, Rodriguez LAG. Anaphylaxis epidemiology in patients with and patients without asthma: A United Kingdom database review. J Allergy Clin Immunol 2010;125:1098-1104
10. Brown SGA. Clinical features and severity grading of anaphylaxis. J Allergy Clin Immunol 2004;114:371-376
11. Triggiani M, Patella V, Staiano RI, Granata F, Marone G. Allergy and the cardiovascular system. Clin Exp Immunol 2008;153:7-11.
12. Wimazal F, Geissler P, Shnawa P, Sperr WR, Valent P. Severe life-threatening or disabling anaphylaxis in patients with systemic mastocytosis: a single-center experience. Int Arch Allergy Immunol 2012;157:399-405.
13. Hompes S, Kohli A, Nemat K, Scherer K, Lange L, Rueff F et al. Provoking allergens and treatment of anaphylaxis in children and adolescents - data from the anaphylaxis registry of German-speaking countries. Pediatr Allergy Immunol 2011;22:568-574
14. Cardona V, Luengo O, Garriga T, Labrador-Horrillo M, Sala-Cunill A, Izquierdo A et al. Co-factor-enhanced food allergy. Allergy ;67:1316-1318
15. Ewan PW, Clark AT. Long-term prospective observational study of patients with peanut and nut allergy after participation in a management plan. Lancet 2001;357:111-115.
16. Ewan PW, Clark AT. Efficacy of a management plan based on severity assessment in longitudinal and case-controlled studies of 747 children with nut allergy: Proposal for good practice. Clin Exp Allergy 2005;35:751-756.
17. Kapoor S, Roberts G, Bynoe Y, Gaughan M, Habibi P, Lack G. Influence of a multidisciplinary paediatric allergy clinic on parental knowledge and rate of subsequent allergic reactions. Allergy 2004;59:185-191

Atopic Eczema (Dermatitis)

– Practical management

Julie Van Onselen, Lecturer Practitioner in Dermatology, Dermatology Education Partnership Ltd and Stirling University, Nurse Adviser, National Eczema Society

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Atopic eczema (or dermatitis) is a very common skin condition, which can affect people of all ages. It is called both atopic eczema and atopic dermatitis – essentially the two terms mean the same. The word 'eczema' (from the Greek 'ekzema', meaning eruption) describes a skin condition caused by inflammation. The word "dermatitis" means inflammation of the skin. 'Atopic' means out of place and refers to an inherited allergic tendency, to develop asthma, eczema, rhinitis and some food allergies.

What causes atopic eczema?

Atopic eczema generally starts in the first year of life. It is estimated that atopic eczema affects 20% of children under age 11; but 60% of all children with atopic eczema develop it in the first 6 months of life.[1] Atopic eczema can improve in childhood, but we now understand that many children do not 'grow out of eczema', as approximately 50% have an the altered gene (known as a filaggrin mutation), which means they have a faulty skin barrier for life[2], so these individuals will always be susceptible to eczema returning at any stage in adulthood. It is estimated that 75% of children improve but 25% continue to have atopic eczema in adulthood, which is a long term relapsing and remitting chronic condition; and for some can be a severe and debilitating chronic disease.[3] The main cause of atopic eczema is genetic, which is complicated by an altered immune system, in addition to being triggered by irritants and allergens. Atopic eczema is a long-term condition with no cure.

Atopic eczema and atopy/allergy (the atopic march)

Atopic eczema involves a complex interaction between the environment (including irritants and allergens) and genes, which leads to skin barrier dysfunction and local systemic immune dysregulation.[4] The atopic march describes the development of atopic disease, which has a family history and commences in infancy/childhood with the development of atopic eczema; then a potential for IgE-mediated food allergy; then in turn, potential for childhood asthma and rhinitis.5 The prevalence of all atopic disease is rising and the interrelationships between atopic diseases can be explained by genetic risks, such as the mutation of filaggrin. The role of an atopic immunologically dysfunctional skin barrier is likely to be central to the evolution of allergic responses in atopic patients with relevant environmental exposure. [5]

Irritants	Allergies
Soaps, detergents (cosmetic washes) Fragrance Sweating Skin infections Stress	Animal dander (fur, hair, saliva) Diet (more likely under 2 years) House dust mite (and droppings) Pollen Preservatives in cosmetics and some topical treatments (causing contact dermatitis)
Environmental irritants	**Allergy testing in atopic eczema**
Seasonal changes (hot climates, cold and wind in winter) Central heating (moving between hot and cold temperatures) Low humidity Clothing (synthetic/wool Grass, pollen and Moulds Dust and chemical agents	Patch tests (contact dermatitis) Prick tests (animal allergy and HDM) Blood tests (Diet)

Table 1: Irritants and Allergies in atopic eczema

Treating atopic eczema

All infants, children, young people and adults with atopic eczema should have regular reviews with a health care professional. When atopic eczema is diagnosed a holistic assessment should include full skin examination, with areas of the body affected by eczema and severity noted. A skin history to include general health, past medical history, current medications and allergies. The skin assessment should include questions on individual eczema triggers (see Table 1). The health care professional should ask about past and current topical treatments, as well as asking general questions on skin care, including any

use of health remedies and personal hygiene products. Questions should be asked about how skin treatments are applied and effectiveness of current treatments.

Emollients

Daily management with complete emollient therapy – that is emollients used for washing and as 'leave-on' moisturisers is crucial. Parents/carers and children need to establish a good daily routine. A daily tepid emollient bath/shower is important for cleansing the skin (all soaps and bubble baths should be avoided). Emollients repair the skin barrier by occluding the stratum corneum to trap water and reduce epidermal water loss. This effect may last for a few hours with emollient creams, or longer with grease-based emollients, depending on skin dryness6. Emollients with humectant actions draw water into corneocytes (skin cells) to give additional skin barrier repair. Humectant emollients contain natural moisturising factors (NMFs), for example urea or glycerol.[6]

In addition, there are emollients containing both humectants and ceramides, which repair and replace lipids. There are also emollients containing antimicrobials (to help reduce staphylococcus aureus in infected eczema and emollients containing lauromacrogols (local anesthetics) helps reduces very itchy skin. It is recommended that 500 g/week is prescribed for a older child with eczema and 250g/week for an infant/toddler.[7,8] All people with eczema should be offered a choice of emollients; they may use one emollient for both washing and moisturizing or need a selection of products. The best emollient is the one that suits the individual, repairs the skin barrier by preventing dry skin and does not cause any irritant or allergic reaction. Emollients should be available on prescription as atopic eczema is a chronic skin condition.

Topical corticosteroids

Topical steroids are the first-line treatment for eczema flares, when the skin becomes, red, very itchy and sore (inflammation). They used as short burst therapy to reduce symptoms of inflammation [7,8] . Topical steroids need to be used as short-burst therapy, which is generally a two week treatment course, to gain control) and for up to 2 weeks in moderate to severe eczema) [8]. Topical steroids applied in correct amounts, to minimize side effects. Using 'sparingly' is an outdated direction and often results in under-dosing, the actual medical direction in the British Drug Tariff is 'apply thinly' and the finger tip unit (FTU) method of application is recommended and instructions on FTUs included in all topical corticosteroid packaging information. Current evidence advises that topical steroids are effective with once daily use, to minimize potential side effects – although the direction on the labeling may be twice a day.[9] There should always be a gap of around 30 minutes between applying topical steroids and emollients[8], the actual order does not matter, but when eczema is flaring, topical steroid applied firstly after bathing/showering and patting dry, is preferable, as the topical steroid will be applied to moisturized skin and inflamed areas will be clearly visible. Emollients can then be soothed onto the skin in a downwards stroking action 30 minutes later.

Topical steroids should be selected according to the age of the individual, area of the body and severity of eczema. Dilution should be avoided [7,8].

There are four groups of topical steroid in the UK:

- Group 1: mild
- Group 2: moderate
- Group 3: potent
- Group 4: very potent

A selection of topical steroids may be required, for example, in a child: mild for the face, and moderate or potent for the body; very potent topical steroids are only in children on a dermatologist's direction). Adults often require potent for the body and mild-moderate for the face, for hands and feet, adults are often prescribed very potent to gain control of these areas with thicker skin. Adults and older children with more severe eczema, who frequently relapse, may be recommend proactive treatment, which involves using moderate or potent topical steroids on two consecutive days a week (known as 'weekend therapy') to prevent flares[10].

Other eczema treatments

Emollients for long term prevention of dry skin and constant repair of the skin barrier and short term treatment with topical steroids for eczema flares, while avoiding known individual irritants and allergies is the standard treatment for atopic eczema and recommended on NHS guidelines.

Second-line atopic eczema treatments include topical calcineurin inhibitors. These are immunomodulator

treatments for moderate to severe atopic eczema (aged 2 years plus), where topical steroids have not gained eczema control or if there is a risk of side effects [7,8].Topical calcineurin inhibitors can be used in all body areas but are particularly useful for sensitive areas (including face (eyelids), neck, and flexures), which may be more susceptible to the adverse effect of topical corticosteroids, particularly skin thinning, with long-term topical corticosteroid use. Potential side effects from topical calcineurin inhibitors include skin irritation, infection and photosensitivity.

Antihistamines are often wrongly thought to treat itch in eczema, but itch in eczema is due to dry skin and an abnormal skin barrier not histamine release. There is no place for non-sedating antihistamines in atopic eczema and sedating antihistamines are generally prescribed at night to help with sleep[8]. Unless, a person also has hay fever, emollients should be first-line treatment for itch.

Eczema frequently becomes infected, if topical steroids are not working and the eczema is becoming worst, inflamed, sore and weepy (with yellow crusts), bacterial infection should be suspected. Infected atopic eczema is generally treated with a course of oral antibiotics, topical antibiotics are only recommended for small areas of infected skin.[7,8] The NHS is mindful of reducing antibiotic use and a recent study, conducted in children with atopic eczema under 8 years with less severely infected eczema should not be treated with antibiotics and continue with topical steroids, but this advice does not apply to children /or adults with more severe signs of infection.[11] Eczema herpiticum is a rare viral infection, the individual will become ill, feverish and their skin will be painful with punch out blisters – this is a medical emergency and treatment is required at hospital within 48 hours.

Bandages, dry and wet wraps and clothing are recommended for comfort and reducing damage caused by scratching, however these adjuvant treatments are not routinely prescribed in the NHS.

Dermatology referral and hospital based treatments

Individuals with atopic eczema that is not responding to topical treatment and allergy should be referred to a dermatologist. The Primary Care Dermatology Society recommend anyone with atopic eczema should be referred to a dermatologist if there is[8]:

- Diagnostic uncertainty
- Severe eczema
- Moderate-to-severe eczema not responding to treatments as described
- Steroid atrophy or concerns regarding the amount of topical steroids / immunomodulators being used
- Possible case of allergic contact dermatitis – for patch testing (older children and adults

In addition, NICE referral guidelines for children with atopic eczema add additional referral criteria[7]:

- Eczema not controlled based on subjective parent/child assessment
- Atopic eczema causing significant psychological/social problems
- Severe eczema not responding after 1 week of topical therapy and recurrent infection
- Atopic eczema of the face which has not responded to treatment
- Suspected food allergy
- Children who fail to grow at expected growth trajectory
- Eczema herpeticum or this is suspected

A referral to a dermatologist will be for assessment, further investigations/patch and allergy testing (this may include joint referral with Allergy consultants, depending on area and referral) and options of hospital-based treatments, including systemic (drug) treatments, phototherapy (adults) and new biologic therapies (adults).

Systemic drug treatments are immunosuppressants, these options include azathioprine, ciclosporin, methotrexate and mycophenolate mofetiel. Ciclopsorin is the only systemic drug licensed for children over 2 years. All systemic drugs are need careful monitoring, usually by dermatology departments as they all have side effects.

Phototherapy, also called light therapy, involves the use of ultraviolet (UV) wavelengths of light as a medical therapy. Phototherapy penetrates the skin, where it reduces T-cell activity and inflammation. A short-term course of phototherapy is recommended for young people and adults, who can tolerate UV light and who are not well controlled with topical
therapy alone. Phototherapy involves 2-3 times a week hospital visits.

Dupilumab is the only biologic agent approved for the treatment of adults with moderate to severe atopic eczema. Multiple additional biologic therapies are currently in development and Dupilumab will soon be approved for use in young people (13-17 years). Dupilumab acts on the specific immunological pathway that control the inflammatory activity of eczema by blocking the interleukin (IL)-4 and IL-13 signaling pathways, so it is a targeted therapy and different to systemic drugs which suppress the immune system. It is self administered as a twice monthly injection.

Conclusion

Atopic eczema is a long-term condition and individuals need to learn how to manage the skin on a daily basis. A good skin care routine, with daily use of emollients to repair the skin barrier and prevent dry skin and itching is essential. When eczema flares, prompt treatment, usually with topical steroids, used for a short burst will treat eczema. Topical steroids need to be respected and side effects can occur but this is unlikely if the correct potency is used matching to age, severity and areas of the body. A reason for atopic eczema not being controlled is often due to under treatment and not over use of topical steroids. It is also important to recognise irritants and allergens and try to avoid individual trigger factors, as well as being aware of eczema complications and recognising infection.

Finally, the impact of eczema on an individual and their family can be huge, living with eczema can be very challenging. It is important to see a health care professional regularly for help with management and if appropriate referral to dermatology.

Key Messages

Emollients are for every day and should be used for washing and moisturising

Topical steroids are first-line eczema treatments and should be used to control eczema flares intermittently in short treatment bursts

Atopic eczema triggers are very individual, not everyone with eczema has allergies but skin irritants are universal

References

1. Schofield J, Grindlay DJC, Williams HC. Skin Conditions in the UK: a Health Care Needs Assessment. 2009. Nottingham: Centre of Evidence-Based Dermatology, University of Nottingham; 6: 85-88.
2. Irvine AP, McLean WH, Leung DYM Filiaggrin mutations associated with skin and allergic diseases. New England Journal of Medicine. 2011; 365 (14):1315-1327
3. Thomsen S. Atopic dermatitis: natural history, diagnosis and treatment. ISRN Allergy. 2014; 2014:354250.
4. Cork MJ, Danby SG, Vasilopoulos Y et al. Epidermal barrier dysfunction in atopic dermatitis. Journal of Investigative Dermatology.2009; 129(8): 1892-1
5. Shaker M. New insights into the allergic march. Current Opinion Paediatrics. 2014; 26 (4): 516-20
6. Cork MJ., Danby S., Skin barrier breakdown: a renaissance in emollient therapy. British Journal of Nursing. 2009; 18(14):872-877
7. National Institute for Health and Care Excellence (2007) Atopic eczema in children. [Online] Available from http://www.nice.org.uk/CG57 [Accessed August 2019]
8. PCDS (2016) Guidelines for the management of atopic eczema. Primary Care Dermatology Society.[Online] Available at: www.pcds.org.uk/clinicalguidance/atopic-eczema [Accessed August 2019]
9. Williams HC. Established corticosteroid creams should be applied only once daily in patients with atopic eczema. British Medical Journal. 2007; 334:1272
10. Berth-Jones J, Damstra RJ, Golsch S et al. Twice weekly fluticasone propionate added to emollient maintenance treatment to reduce risk of relapse in atopic dermatitis: randomized, double blind, parallel group study. British Medical Journal. 2003; 326: 1367.
11. Francis NA, Ridd MJ, Thomas-Jones E et al. CREAM team. A randomised placebo-controlled trial of oral and topical antibiotics for children with clinically infected eczema in the community: the ChildRen with Eczema, Antibiotic Management (CREAM) study. Health Technology Assessment. 2016; 20(19):1-84

Grass Pollen, Environmental DNA and Respiratory Health:

An Introduction to the PollerGEN Project

Dr Nick Osborne is associate professor of environmental epidemiology at UQ School of Public Health. He is an environmental epidemiologist and toxicologist with research interests in using environmental epidemiology to examine aetiology and pathological pathways of disease.

He is currently working on projects using molecular approaches to predict grass pollen-related health outcomes in the UK and examining CKD in Sri Lanka and its relationship to pesticides and/or the water:heat stress nexus.He completed his PhD at the then named National Research Centre for Environmental Toxicology and the School of Preventive Medicine, UQ.

Dr Francis Rowney is a postdoctoral research associate at the European Centre for Environment and Human Health (University of Exeter Medical School, Truro), researching the links between grass pollen and human respiratory health, particularly asthma. He has a background in palaeoecology, and studied the ecology and climates of Middle Pleistocene interglacials during his PhD, using fossil pollen, insects and fungal spores.

As the spring and summer advance, days become warmer and longer and many people enjoy spending time outdoors. However, for people with asthma and respiratory allergies, these seasons can be less welcome, particularly when grasses start to flower and produce pollen. In Europe, grass pollen is the single most important outdoor aeroallergen, and the population prevalence of sensitivity to grass pollen is generally greater than for other pollen types. In severe cases, exposure to high levels of grass pollen can even lead to hospitalisation.

These allergic reactions are a result of the body's immune response to otherwise benign proteins contained within pollen grains. On first exposure to pollen, the body decides if some of these otherwise harmless proteins are dangerous. If it decides they are, the immune system produces immunoglobin E (IgE) antibodies: a process called sensitisation. Next time the body is exposed to these proteins, the IgE antibodies detect them and cause cells to release histamine and other chemicals. These may then result in a range of symptoms, including the production of mucus,

itchy eyes and nose, sneezing and inflammation of the respiratory system.

However, whilst we know that grass pollen is important, there are many different species of grass and we don't know which ones contribute more (or less) to the incidence of hay fever symptoms and other health outcomes. This is one of the key issues being addressed by the PollerGEN Project.

PollerGEN (pollergen.bangor.ac.uk/) is a multi-disciplinary project led by researchers at Bangor University and funded by the Natural Environment Research Council (NERC). Other project researchers are based at Aberystwyth University, the University of Worcester, the University of Exeter, The University of Queensland (Australia), the Met Office and the National Botanic Garden of Wales.

A New Approach: Genetics-based Pollen Monitoring

In the UK, pollen monitoring is undertaken between March and September, coordinated by the Met Office. Rooftop samplers draw air into a chamber containing a slowly rotating spool, which is covered in wax-coated tape that pollen grains (and other airborne particles) stick to. This tape is then cut into sections, and each is studied under the microscope to produce daily time series of local pollen concentrations.

With sufficient expertise, pollen from different types of plants can be readily identified and counted in this manner. Pollen from an oak (Quercus) tree looks different to pine (Pinus) pollen, which looks different to nettle (Urtica) pollen, and so on. However, visually distinguishing pollen from closely related plants (for example, different species of oak) is often difficult at best, requiring highly specialist knowledge, or virtually impossible at worst. Identifying the pollen of different types of grasses (family Poaceae) falls into the latter category, and as a result we don't know whether some grasses are more significant than others in a public health context. This is where PollerGEN comes in.

The revolution in the analysis of environmental DNA (eDNA), genetic material derived from samples of water, soil, sediment and air, is proving very useful in this context. PollerGEN researchers have been using specially redesigned pollen samplers to collect air samples and subject them to genetic analyses: an approach that has allowed the monitoring of different types of grass pollen for the first time. The results of the first year of this monitoring, tracking changes in airborne grass pollen diversity through the pollen season, have recently been published in Nature Ecology and Evolution. The research has confirmed what was expected – different species of grass release pollen at different times and the release patterns are mirrored in the air. The aerial distribution of pollen types follows a sort of "sliding window" of shifting types of pollen communities. The results also showed that all grass types had specific incidence peaks of pollen, which varied depending on location, with grasses further north releasing their pollen later. Work is currently on-going to analyse similar datasets for two more years to look at year to year variation.

Linking with Respiratory Health and Improving Forecasting

We're now using the data produced by PollerGEN's eDNA specialists to investigate whether different types of grass are more influential than others on respiratory health. We are combining these datasets with public health data, producing statistical models that allow us to explore the linkages between different grasses and indicators of population respiratory health, such as prescribing rates of respiratory antihistamines and asthma-related hospital admissions. Being aware of which grasses may be more significant in this context will benefit people with asthma and respiratory allergies, as well as healthcare providers and professionals, who can take preparatory actions when they are likely to flower and release pollen locally.

PollerGEN researchers have also been producing detailed source maps for different grass species in the UK, and combining these with the genetic data to develop integrated aerobiological models. These models are allowing the investigation of how pollen grains likely move across landscapes, and may lead to new approaches to pollen forecasting in the future.

PollerGEN Website: pollergen.bangor.ac.uk

The Future

In the long term, the vision is to develop tools for providing specific pollen forecasts for grass, and to unravel which species are most likely causing allergic responses. More broadly, we also want to provide information to healthcare professionals and charities, who can translate this information to help people with asthma and respiratory allergies live healthier, happier lives.

Indoor Aeroallergens and their Impact on Health

Chris Corrigan, Professor of Asthma, Allergy & Respiratory Science, King's College London Faculty of Life Sciences & Medicine

Chris Corrigan is Emeritus Professor of Asthma, Allergy & Respiratory Science in the King's College London Faculty of Life Sciences and Medicine, based at Guy's Hospital in South London and a Principal Investigator in the UK Medical research Council Centre for Allergic Mechanisms of Asthma based in London. He led in building the large adult allergy service, particularly the allergen immunotherapy service, and the severe asthma service based at Guy's Hospital, London.

Foreword

In the UK the principal indoor aeroallergens arise from house dust mites, moulds (fungi) and domestic animals. In mites and domestic animals the majority of these allergens are found in bowel or urinary proteins (in the saliva, urine and faeces): hair and inorganic dust particles are not allergens, although they may harbour them. Mould allergens are typically disseminated in spores. Aeroallergens are so named because, in a subset of individuals who manufacture IgE against certain of their protein components, cross-linking of this IgE bound to the high-affinity IgE receptor FcεRI on the surface of respiratory and conjunctival mucosal mast cells following airborne exposure results in the release of histamine and other mediators, resulting in acute symptoms of allergic rhinoconjunctivitis and potential exacerbation of asthma. Indoor aeroallergens are "perennial" allergens, typically present all year round although not necessarily always at the same concentrations, depending on the precise circumstances.

Is my patient clinically sensitised to indoor aeroallergens?

It is important to appreciate that not all patients who manufacture IgE against any aeroallergen develop symptoms of acute histamine release on exposure because their presumably sensitised mast cells do not release significant amounts of histamine: the mechanism of this variability in clinical responsiveness remains elusive. Thus, positive allergy diagnostic tests such as skin prick tests and blood tests for IgE specific for any allergen suggest only that symptoms on exposure are possible, not inevitable. It is therefore important to establish a clear clinical history of acute, perennial symptoms of histamine release (itchy, runny and sore eyes, itchy and runny nose with sneezing and possible acute exacerbation of symptoms in asthmatic patients) on likely exposure to the suspected allergen before assuming that the patient is clinically sensitised. House dust mites are microscopically small creatures which breed in soft furnishings such as mattresses, duvets, pillows, thick curtains, carpets and cuddly toys. They thrive in warm, humid conditions (which is unfortunately how most people like to keep their bedrooms these days). Patients with clinically significant sensitisation to house dust mite allergens typically wake up every morning with symptoms (typically most exposure occurs while in bed). Chronically uncontrolled symptoms may result in a degree of persistent nasal blockage but it should be noted that persistent nasal blockage in isolation, for which there are multiple potential causes, does not indicate significant house dust mite allergy even in an IgE-sensitised individual. Fungal allergens disseminate in the spores of ubiquitous, environmental fungi (Aspergillus, Alternaria, Cladosporium and others) which grow widely in the soil. They may cause seasonal symptoms in September when soil-dwelling fungi sporulate (the mushroom and toadstool season), or more rarely perennial symptoms in circumstances of high indoor exposure (for example, living in damp, mouldy bedrooms). Significant symptoms on exposure to domestic animals (cats, dogs, hamsters etc.) are usually obvious. They do not diminish with repeated exposure, although individuals may to an extent acclimatise themselves to living with them. As with dust mites, patients are sensitised to their bowel and urinary proteins, and not, as is commonly believed, their hair.

Is there a role for allergen avoidance?

In patients whose symptoms of perennial allergic rhinoconjunctivitis are caused principally or solely by house dust mite allergy, allergen avoidance is theoretically likely to be beneficial but is hard to realise within a typical UK domestic environment. Some patients anecdotally derive benefit from avoidance measures such as frequent washing of their bedclothes and the use of closely woven barrier bedcovers (lists of these measures are available from organisations such as Allergy UK and the British Society for Allergy & Clinical Immunology), although this benefit is not universal since exposure to house dust mite and domestic animal allergens may be very specific to the individual, for example from the wearing of old, infrequently washed outer clothing [1]. Although some studies[2] have linked exposure to house dust mite allergens in sensitised individuals with clinical activity of asthma, and others have shown that, with careful and sometimes extreme, individualised measures of environmental control, including repeated house dust mite eradication, amelioration of asthma symptoms is possible[3,4], overall most "real life" studies of house dust mite avoidance have failed to show any significant impact on asthma control [5]. Similarly, the only completely effective way to remove domestic pet allergens from the home environment is to exclude the animal(s) of origin completely, although this may take 6 months or more [6]. Frequent washing of the animals coupled with abolition of potential reservoirs such as carpets, and other measures such as air filtration may

reduce household pet allergens, at least partially and temporarily, but the clinical benefit is uncertain [7-9], while the act of vacuuming, even with an efficient air filter, may actually increase personal exposure [10].

Allergens and the natural history of "atopic" diseases

The long established tenet of the childhood "atopic march", in which it was postulated that, through exposure to allergens, children with the inherited propensity to develop IgE responses against aeroallergens were at risk of developing a "march" of atopic diseases through childhood and adolescence (atopic eczema, food allergy, allergic rhinoconjunctivitis and asthma), with the underlying implication that avoidance of allergen exposure might modify this "march", has been subject to increasing scrutiny in recent years. Although children with atopic eczema are at increased risk of other diseases of the "march", progression in any individual is by no means certain and likely depends on additional, individual responses to the effects of multiple environmental influences acting at mucosal surfaces [11-13]. While attempting to reduce exposure of clinically sensitised children to indoor aeroallergens suspected of exacerbating atopic eczema, allergic rhinoconjunctivitis and asthma seems logical, there is no formal evidence that a priori allergen avoidance reduces the incidence of "atopic march" diseases such as atopic eczema [14].

Immunotherapy for indoor aeroallergen-induced symptoms

For clinically sensitised patients whose symptoms of perennial allergic rhinoconjunctivitis are inadequately controlled by allergen avoidance, insofar as it is possible, and regular, adequate dosages of anti-histamines, topical steroids and other measures such as regular douching, allergen immunotherapy delivered subcutaneously or sublingually is of proven, long-lasting benefit in a majority of patients in reducing symptoms, although continuation of "conventional" therapy is usually necessary since the treatment is not curative and seldom abolishes symptoms entirely. At present allergen immunotherapy is available for allergy to house dust mites, cats and dogs, although not (yet) moulds. Many NHS Allergy services have a policy of denying immunotherapy in situations where allergen avoidance is eminently possible (for example by handing over pets to other carers). House dust mite immunotherapy has also been shown to contribute to asthma control in sensitised, atopic asthmatics with significant clinical house dust mite allergy [15].

References

1. Exposure to mite and cat allergens on a range of clothing items at home and the transfer of cat allergen in the workplace. De Lucca SD, O'Meara TJ, Tovey ER. J Allergy Clin Immunol. 2000 Nov;106(5):874-9.
2. Exposure to house dust mite allergens and the clinical activity of asthma. Custovic A, Taggart SC, Francis HC, Chapman MD, Woodcock A. J Allergy Clin Immunol. 1996 Jul;98(1):64-72.
3. Results of a home-based environmental intervention among urban children with asthma. Morgan WJ, Crain EF, Gruchalla RS, O'Connor GT, Kattan M, Evans R 3rd, Stout J, Malindzak G, Smartt E, Plaut M, Walter M, Vaughn B, Mitchell H; Inner-City Asthma Study Group. N Engl J Med. 2004 Sep 9;351(11):1068-80.
4. Effect of mattress and pillow encasings on children with asthma and house dust mite allergy. Halken S, Høst A, Niklassen U, Hansen LG, Nielsen F, Pedersen S, Osterballe O, Veggerby C, Poulsen LK. J Allergy Clin Immunol. 2003 Jan;111(1):169-76.
5. House dust mite control measures for asthma. Gøtzsche PC, Johansen HK, Schmidt LM, Burr ML. Cochrane Database Syst Rev. 2004 Oct 18;(4):CD001187. Review. Update in: Cochrane Database Syst Rev. 2008;(2):CD001187.
6. The effect of cat removal on allergen content in household-dust samples. Wood RA, Chapman MD, Adkinson NF Jr, Eggleston PA. J Allergy Clin Immunol. 1989 Apr;83(4):730-4.
7. The effect of air filtration on airborne dog allergen. Green R, Simpson A, Custovic A, Faragher B, Chapman M, Woodcock A. Allergy. 1999 May;54(5):484-8.
8. Airborne cat allergen (Fel d I). Environmental control with the cat in situ. de Blay F, Chapman MD, Platts-Mills TA. Am Rev Respir Dis. 1991 Jun;143(6):1334-9.
9. Washing the dog reduces dog allergen levels, but the dog needs to be washed twice a week. Hodson T, Custovic A, Simpson A, Chapman M, Woodcock A, Green R. J Allergy Clin Immunol. 1999 Apr;103(4):581-5.
10. High-efficiency particulate arrest-filter vacuum cleaners increase personal cat allergen exposure in homes with cats. Gore RB, Durrell B, Bishop S, Curbishley L, Woodcock A, Custovic A. J Allergy Clin Immunol. 2003 Apr;111(4):784-7.
11. Exposure to microbial agents in house dust and wheezing, atopic dermatitis and atopic sensitization in early childhood: a birth cohort study in rural areas. Karvonen AM, Hyvärinen A, Gehring U, Korppi M, Doekes G, Riedler J, Braun-Fahrländer C, Bitter S, Schmid S, Keski-Nisula L, Roponen M, Kaulek V, Dalphin JC, Pfefferle PI, Renz H, Büchele G, von Mutius E, Pekkanen J; PASTURE Study Group. Clin Exp Allergy. 2012 Aug;42(8):1246-56.
12. To what extent is allergen exposure a risk factor for the development of allergic disease? Custovic A. Clin Exp Allergy. 2015 Jan;45(1):54-62.
13. Atopic Dermatitis and Respiratory Allergy: What is the Link. Belgrave DC, Simpson A, Buchan IE, Custovic A. Curr Dermatol Rep. 2015;4(4):221-227.
14. Dust mite avoidance for the primary prevention of atopic dermatitis: A systematic review and meta-analysis. Bremmer SF, Simpson EL. Pediatr Allergy Immunol. 2015 Nov;26(7):646-54.
15. Immunotherapy for house-dust mite allergy. Nelson HS. Allergy Asthma Proc. 2018 Jul 1;39(4):264-272.

An Introduction to Adult Food Allergy

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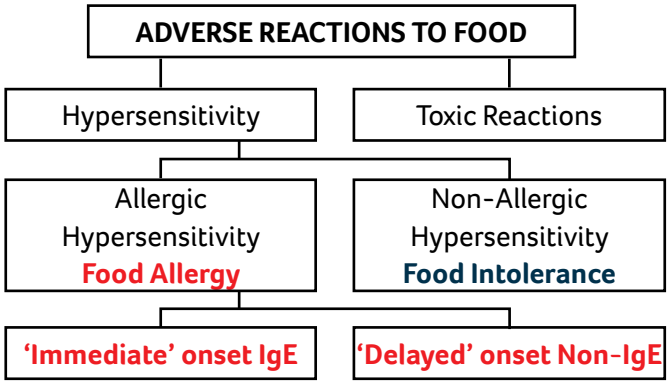
Kate is an HCPC registered dietitian and Allergy UK's Clinical Dietetic Advisor. She graduated in 2010 and has a wide range of dietetic experience both with adults and children and is a specialist in paediatrics and food allergy. Kate's interest in food allergy was magnified in 2015 when her second child developed food allergies. Kate also works part-time for the Newcastle upon Tyne Hospitals NHS Trust as a Specialist Paediatric Community Dietitian. She gained a Master's degree in Advanced Dietetic Practice in summer 2019.

Food allergy is an immune mediated response to a culprit protein in food. It can be divided into those reactions which involve Immunoglobulin E and produces reactions that happen quite soon after the food has been ingested (so called immediate onset) and those that happen sometime after the food has been ingested (so called delayed) that do not involve Immunoglobulin E. As you can see from the diagram below, Food intolerance is not an allergic response and should not be referred to as Allergy.

IgE Mediated food allergy is a type I allergic response which occurs when the body produces Immunoglobulin E (IgE) antibodies against dietary proteins. Non-IgE mediated allergic reactions tend to be delayed and are less easy to diagnose.



IgE Mediated (Immediate onset - up to 2 hours after ingestion)	Non-IgE Mediated (2-72 hours after ingestion)
Itchy mouth, tongue and throat	Itchy mouth tongue and throat
Swelling of lips, around the eyes or face	Swelling unlikely
Red raised itchy rash (often called nettle rash, hives or urticaria)	Non-specific rash
Vomiting, nausea, abdominal pain or diarrhoea	Vomiting, nausea, abdominal pain, constipation or diarrhoea
Runny nose and sneezing	Persisting upper and/ or lower respiratory symptoms and signs
Eczema flare	Persistent moderate eczema
Anaphylaxis	

Common triggers for adults include fruit, vegetables, seafood, peanuts and tree nuts (Skypala, 2011).

Prevalence

The prevalence of adult food allergy is not well established and it is difficult to say how many cases persist from childhood and how many are new cases in adulthood (Skypala, 2011). With adults, there is a strong link between other allergic conditions such as eczema and hay fever (allergic rhinitis). The prevalence of Allergic Rhinitis has trebled within the last 30 years and therefore it is very likely that food allergy in adults will be seen more in primary care (Skypala, 2011).

Fruit and vegetables

The most common food allergens to affect adults are fruit and vegetables and the reason for this is cross reactivity, the proteins in fruit and vegetables can be similar to other allergenic proteins such as pollen. Oral Allergy Syndrome (OAS) is an umbrella term used to describe cross-reactive plant food allergies. The most common of these allergies is Pollen Food Syndrome (PFS).

Pollen Food Syndrome

Approximately 2% of the adult population across the UK suffer with PFS (Skypala, et al., 2013). It most commonly affects adults who have Allergic Rhinitis; reactions are caused due to proteins in the fruit and vegetables that are similar to those in pollen. They are mistaken for allergens within the body and this causes an allergic reaction. Bet v 1, the main allergen in Birch pollen is highly cross reactive to many plant foods (Allergy UK, 2017). It is estimated that 50-90% of people who are sensitised to birch pollen also have PFS (Skypala, et al., 2013).

Fruit and vegetables that people react to depend on the pollen that they are sensitised to. Reactions only occur when foods are raw or lightly cooked. This often causes confusion. Table 1 shows the main foods associated with allergic reactions. The most common UK pollens have been highlighted as pollens vary in different countries.

Table 1. Pollen and associated food (Kelava, et al., 2014)

Pollen	Fruit	Vegetable	Nuts	Grains
Birch*	Kiwi, peach, apple, nectarine, apricot, banana, pear, plum, avocado, cherry, fig, strawberry, dried plum	Potato, carrot, celery, chicory, cilantro, fennel, pepper (green), parsley, parsnip, dill, cumin, tomato, bean sprouts, coriander, mange tout, tomato	Hazelnut, almond, walnut, brazil nut	Soybeans, wheat, lentils, peas, beans, peanuts
Mugwort / Wormwood*	Apple, melon, watermelon	Carrot, celery, parsley, coriander, pepper, cilantro, fennel, aniseed, celery salt, mustard, parsley, spices		Sunflower seeds
Grass*	Fig, melon, orange, kiwi, watermelon	Tomato, potato, swiss chard		Peanut, wheat
Ragweed	Banana, melon, honey dew, watermelon	Pepper, squash, cucumber, artichoke, hibiscus, chamomile tea		Sunflower seeds
Weeds	Melon, watermelon, orange, kiwi	Tomato		
Alder	Apple, cherry, peach, pear, strawberry, raspberry	Celery, parsley	Hazelnut, almond, walnut	
Parietaria	Cherry, melon			

**common in the UK*

Symptoms of PFS usually occur within 5-10 minutes after eating a trigger food and commonly involve: itching, tingling or a burning sensation within the mouth, ears or throat and occasionally swelling of the lips, tongue and throat (Allergy UK, 2017). Less common reactions include rash, nausea, vomiting, sneezing and a blocked nose. More serious reactions could occur if large amounts of trigger foods are consumed and in very rare cases PFS can cause anaphylaxis.

Latex fruit syndrome

Natural rubber latex is another protein which cross-reacts with food, approximately 20% of latex allergy sufferers will react to foods including: avocado, buckwheat, banana, chestnut, kiwi, melon, plums, strawberries and tomatoes (Allergy UK, 2017; Moneret-Vautrin & Morisset, 2005)

LTP allergy

Non-specific lipid transfer protein (LTP) allergy is much more common in the Mediterranean than the UK and can result in severe allergic reactions due to the structure of the proteins being resistant to heat and pepsin digestion (Asero, et al., 2018; Skypala, 2013). Fruits from the Rosaceae family such as peach, apricot, apple, cherry, pear and plum and nuts are the main plant food allergens involved (Asero, et al., 2018; Azofra, et al., 2016).

Seafood allergy

Seafood allergy can encompass fish, crustacean and mollusc and different people can react to different proteins in different species. Fish allergy is more common in adults than children and parvalbumin, a protein in cod, salmon and carp accounts for 70-100% of reactions (Ruethers, et al., 2018). Shell fish allergy is more prevalent than fish, especially reactions to prawn and crab. Mollusc allergy is less common. Reactions to seafood can be severe and it is an allergy that does not tend to be grown out of (Ruethers, et al., 2018).

Wheat

IgE wheat allergy tends to originate in childhood and is often grown out of before adulthood; however it is a common trigger for food-dependent exercise-induced anaphylaxis in adults (Skypala, 2011). This rare condition only occurs when a specific food trigger in combination with exercise causes IgE-mediated hypersensitivity causing anaphylaxis (Minty, 2017). Other food triggers include shell fish, tomatoes, peanuts, strawberries, cheese, celery, corn and soy (Skypala, 2011).

Food additives

Food additives are a common trigger in adults for non-immune mediated reactions to food. There is a huge range of symptoms and offending foods and therefore it can be very confusing.

Sulphites

Sulphites are added usually as preservatives to extend the life of packaged foods and drinks such as fish, cured meat, dried fruit and vegetables, sauces, alcoholic and soft drinks. Foods with over 10ppm (10mg per kg) are required by laws in the EU and USA to be labelled. Reactions to sulphites are most common in people with asthma but can also cause angio-oedema and urticaria either from ingestion or topical application (Skypala, et al., 2015).

Benzoates

Benzoic acid is again produced by plants and animals and added to foods because of antimicrobial properties, it is also a product of digestion. Therefore it is present in a large variety of foods and drinks from all food groups. There is good evidence in literature showing that benzoates are linked to: asthma, chronic urticaria, rhinitis, atopic dermatitis and anaphylaxis (Skypala, et al., 2015).

Monosodium Glutamate (MSG)

MSG is a common food additive as well as being naturally occurring in foods such as dried or cured items. The most commonly reported symptoms caused by ingesting large amounts of MSG are headaches, rhinitis and urticaria. However, study results are not conclusive on the effectiveness of an MSG free diet (Skypala, et al., 2015).

Histamines

Histamine is a vasoactive amine which is produced by bacteria and is present naturally in many foods, alcohol and certain drugs; histamine intolerance is caused by a lack of diamine oxidase (DAO), the key enzyme which metabolises histamine. An excess of histamine consumed can result in similar symptoms to an allergic reaction: flushing, pruritus, urticaria, rhinoconjunctival symptoms, GI symptoms and tachychardia. The foods usually involved are chocolate, cheese, loosely cooked eggs, cured meats, fish, dried fruit fermented soya products, and caffeinated drinks. This list is by no means exhaustive (Maintz & Novak, 2007; Skypala, et al., 2015).

Histamine levels can accumulate to dangerous levels in fish that has been processed or stored improperly; this is referred to as Scombroid poisoning and can cause a severe reaction including the symptoms already mentioned as well as bronchospasm and vasodilatory shock (Stratta & Badino, 2012).

Diagnosis of adult food allergy

It is important if possible for patients to be referred to a specialist allergy service, although specialist adult allergy services are not as common as paediatrics. Diagnosis should be based on an allergy-focused clinical history. Validated tests such as skin prick testing and serum specific IgE assays should be used in conjunction with the allergy-focused clinical history translated by an allergy specialist, not in isolation (National Institute for Health and Clinical Excellence, 2016). The gold standard of food allergy diagnosis is an oral food challenge (Skypala, et al., 2013).

Management

In general, the management of food allergy and intolerance is to avoid the food which causes symptoms. In the cases of multiple allergies a diet can become restricted leading to nutritional deficiencies and therefore a referral to a dietitian is best practice. Dietitians can suggest alternatives to allergenic food and ensure that a patient's nutritional requirements are being met.

Patients are often provided with a prescription or advice on antihistamines to treat symptoms and in rare cases an adrenaline auto-injector (AAI). It is very important that asthma is well controlled as people with asthma are more at risk of severe reactions.

Conclusion

Adult food allergy is on the rise and can be a severe and life threatening illness. Even in mild to moderate cases it can make a huge impact on quality of life and therefore should not be taken lightly. Allergy UK have over 60 factsheets on food allergy and a helpline to support both members of the public and healthcare professionals.

Works Cited

Allergy UK, 2017. Natural Rubber Latex Allergy. s.l.:s.n.

Allergy UK, 2017. Oral Allergy Syndrome or Pollen-Food Syndrome. [Online]
Available at: https://www.allergyuk.org/assets/000/001/548/Oral_Allergy_Syndrome_original.pdf?1512985580
[Accessed 09 July 2019].

Asero, R., Piantanida, M., Pinter, E. & Pravettoni, V., 2018. The clinical relevance of lipid transfer protein. Clin Exp Allergy, 48(1), pp. 6-12.

Azofra, J. et al., 2016. Lipid Transfer Protein Syndrome in a Non-Mediterranean Area. Int Arch Allergy Immunol, Volume 169, pp. 181-188.

Kelava, N. et al., 2014. Oral Allergy Syndrome – the need of a multidisciplinary approach.. Acta Clin Croat, Volume 53, pp. 210-219.

Maintz, L. & Novak, N., 2007. Histamine and histamine intolerance. Am J Clin Nutr, Volume 85, p. 1185–96.

Minty, B., 2017. Food-dependent exercise-induced anaphylaxis. Canadian Family Physician, 63(1), pp. 42-43.

Moneret-Vautrin, D. & Morisset, M., 2005. Adult Food Allergy. Current Allergy and Asthma Reports, Volume 5, pp. 80-85.

National Institute for Health and Clinical Excellence, 2016. Food allergy (QS118), London: NICE.

Ruethers, T. et al., 2018. Seafood allergy: A comprehensive review of fish and shellfish allergens. Molecular Immunology, Volume 100, pp. 28-57.

Skypala, I., 2011. Adverse Food Reactions—An Emerging Issuefor Adults. Journal of the American Dietetic Association, Volume 111, pp. 1877-1891.

Skypala, I. et al., 2013. The prevalence of PFS and prevalence and characteristics of reported food allergy; a survey of UK adults aged 18–75 incorporating a validated PFS diagnostic questionnaire. Clinical & Experimental Allergy, Volume 43, p. 928–940.

Skypala, I. J. et al., 2015. Sensitivity to food additives, vaso-active amines and salicylates: a review of the evidence. Clinical and Translational Allergy, 5(34), pp. 1-11.

Stratta, P. & Badino, G., 2012. Scombroid poisoning. Canadian Medical Association journal = journal de l'Association medicale canadienne, 184(6), p. 674.

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