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03

Allergies on the increase

The frequency of allergic diseases is increasing in many countries in such a way as to make scientists refer to these as diseases of civilisation. This is particularly evident from the statistics on

atopic eczema, allergic rhinitis and allergic bronchial



asthma. However, children and adults are affected differently by allergic conditions. Atopic eczema is primarily found in children and young people while allergic rhinitis tends to occur in adults ...





The prevalence of allergies is increasing particularly in industrialised countries (most markedly in Anglo-Saxon nations), although the geographical distribution is highly varied. There is a north-south divide as well as an east-west differential. This means that in countries such as Finland or the UK, the frequency of allergies is higher than in Mediterranean countries or eastern Europe. Significant results are expected from the ISAAC study (International Study of Asthma and Allergies in Children), which was started a few years ago and to date has questioned and examined 750,000 children in sixty different countries.

The 1997 European Allergy White Paper indicated that in Europe about 35% of the population showed a sensitivity or allergy. This has a considerable effect on the economy: lost working hours or time out of school, reduced productivity and costs for both outpatient and in-patient treatment. Throughout Europe at the present time, annual allergy-related costs are some €45bn. For example, the Berlin Centre for Public Health found that annual expenses for atopic dermatitis (neurodermatitis) in children totalled €4,400 per year and child.

Comparable findings were reported from the USA: 3.5 million working days and 2 million schooldays are lost every year. Between 1994 and 1997, costs for anti-allergic agents alone increased by 67% in the USA and went up by a further 89% from 1997 to 1999. In terms of chronic diseases, allergies rank sixth in the USA and generate annual costs of €18.4bn.

PREVALENCE OF ASTHMA, ALLERGIC RHINITIS AND ATOPIC ECZEMA IN SELECTED COUNTRIES

Country	Prevalence of asthma
1 Australia	33.5
2 New Zealand	32
3 UK	32
4 USA	25
5 Finland	20
6 Germany	15
7 Japan	13

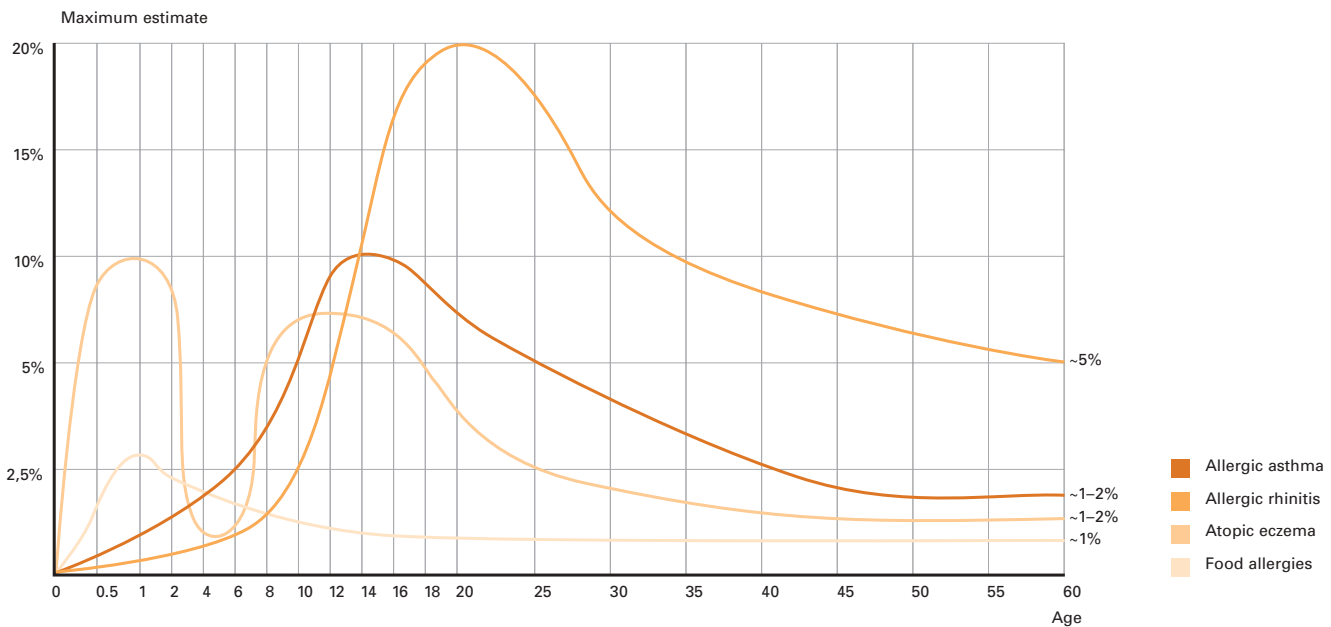
Country	Prevalence of dermatitis
1 UK	17
2 Finland	15
3 New Zealand	14
4 Japan	12
5 Australia	11
6 USA	10
7 Germany	7

Country	Prevalence of rhinitis
1 Australia	23
2 New Zealand	20
3 USA	17
4 UK	16
5 Finland	16
6 Japan	15
7 Germany	14

All figures in %

ISAAC study on children between 13 and 14 years old

FREQUENCY OF ALLERGIES



An underactive immune system strikes back

Various hypotheses have already been developed to explain the clear increase in the prevalence of allergic diseases, even though the phenomenon is still not completely understood. Actually plausible although somewhat amusing is the **"JUNGLE HYPOTHESIS"** positing that in earlier times of poor hygiene the human immune system was concerned with fighting off parasites, for which a specialised subgroup of lymphocytes (Th2 lymphocytes) was responsible. Originally these Th2 lymphocytes, which are suspected of excessive immune responses (e.g. in the form of allergies), were actually blocked and switched off. **EXPOSURE TO INFECTION DURING CHILDHOOD**, which has been reduced in the meantime, may also play an important role. Tuberculosis pathogens (*Mycobacterium tuberculosis*) seem to be of particular significance in

reducing allergic reactivity so that British researchers have recently been considering inoculating children with inactive tubercle bacteria.

There are impressive data for the **INFECTION HYPOTHESIS** from East Germany, stemming from those born after 1950: at the time of the German Democratic Republic, many children were given into **CHILDREN'S CRÈCHES** when they were only a few months old and the early contact with other children stimulated their immune systems. Compared with West German children of the same age, they suffer considerably less often from allergies later on in life. Since reunification in 1990, the prevalence is slowly equalising out between the east and west of the country.



Crèche outing in the GDR (Neuholland, Brandenburg, 1968).

Greenhouse effect and allergies

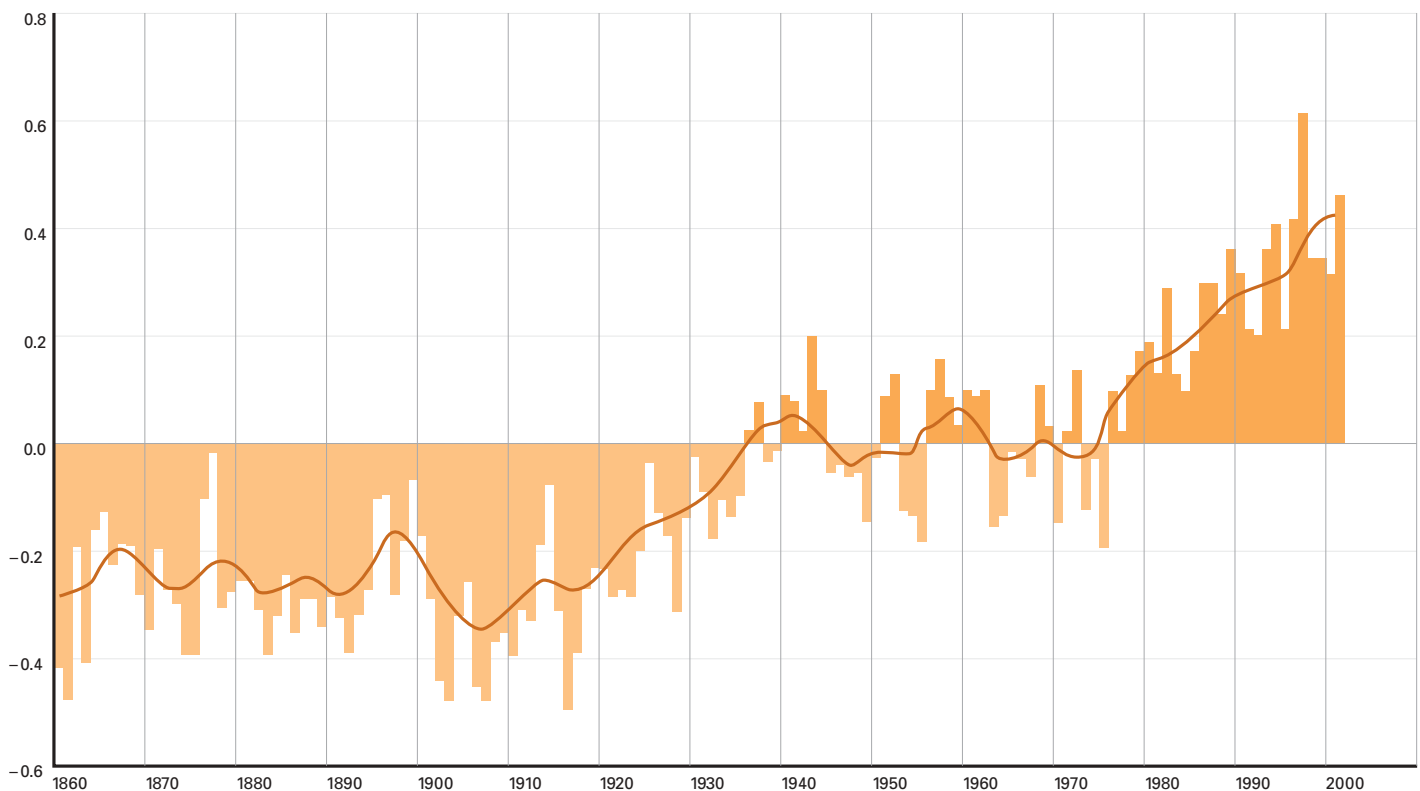
Long-term studies by climatologists have shown that global warming is taking place. This will continue in the years to come, and will become more and more obvious if we fail to control the emission of toxic substances such as nitrogen oxide and carbon dioxide. Even if the immediate increase in temperature (greenhouse effect) is only minimal, it will mean firstly that ozone levels near the earth will rise further and secondly that we can expect humidity and precipitation to become greater in many regions of the world.

Ozone promotes the release of allergens, and penetrates into the lower respiratory tract (bronchi) where it leads to an inflammatory effect by setting mediators free. One American study on children carried out in 2001 even indi-

cated that ozone not only intensifies existing asthmatic conditions, but also initiates the development of bronchial asthma.

Changes in the climatic and environmental conditions will mean the growth of plants that produce previously unknown pollens. Not only pollens, but moulds with corresponding allergic potential will also flourish in a higher humidity. In addition, it is feared that high pollen counts will start earlier and last longer so that the symptom-free interval for patients suffering from hay fever may be shortened to just a few months or even weeks.

MEASUREMENT OF GLOBAL MEAN TEMPERATURE – FROM 1860 TO 2002*



Source: Munich Re, Geo Risks Research 2002.

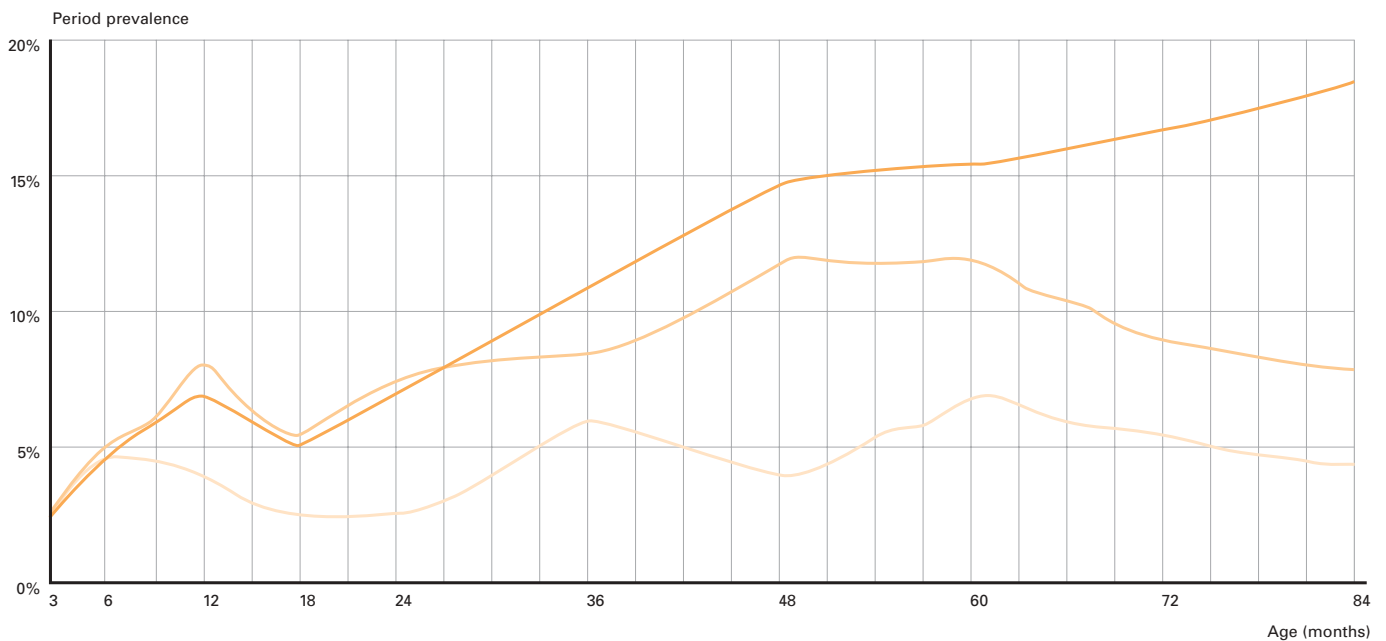
*Deviations from average readings 1951–1980

A question of inheritance?

For a long time, it was thought that environmental factors were mainly responsible for the development of allergic bronchial asthma. This hypothesis came into question when long-term observations of people with allergies showed that very often not only one but many members of their families were also affected by this type of illness. Hence, there had to be an inherited risk (genetic predisposition) of allergies that were partially responsible for the appearance of allergic conditions at a later stage. In Germany, the risk of atopy in children with two healthy parents is 5–15%. If one parent has an atopic condition of any description, the risk to the child is increased to

20–40%. Should both parents suffer from atopy with the same organic manifestations, the risk to the child is an impressive 60–80%. The recognition that the development of allergies depends on both genetic and environmental factors shows how important early allergy prophylaxis is for children with predisposed parents.

INHERITED RISK AND FREQUENCY OF ASTHMA IN CHILDREN



- Among immediate relatives:**
- Two allergy sufferers
 - One allergy sufferer
 - No allergy sufferers

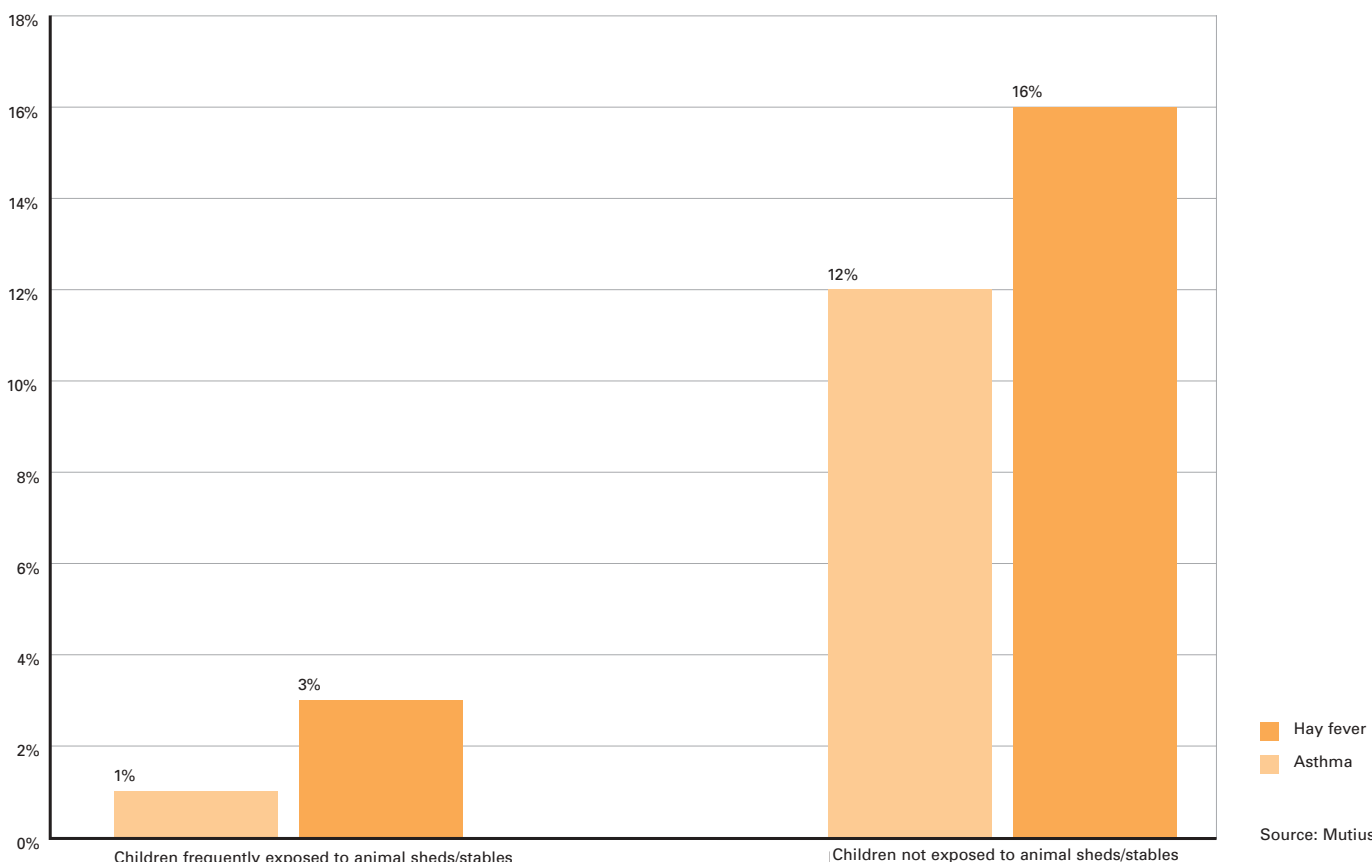


Does animal dung protect from allergies?

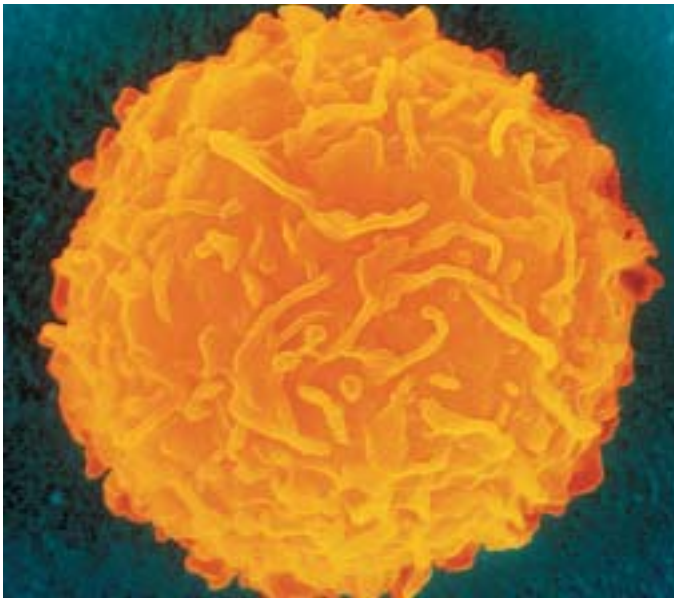
There have been some interesting results from recent studies carried out in Austria, Switzerland and southern Germany on children from farming communities. These have shown that the corresponding protection from later allergic diseases such as bronchial asthma or hay fever is built up if the child is exposed to farm animals and poultry in the first year of life and drinks cows' milk. The lowest rates of allergy were found when the mother had continued to work daily in the stables and cowsheds during the pregnancy. Also if the child came into contact with

sheds and stables at a later stage – but still within the first three years of life – there was still a clear protective effect in comparison with children who had not grown up on a farm. Components of Gram-negative bacteria are thought to be the decisive immune stimulants, as these are found in great numbers in animal dung.

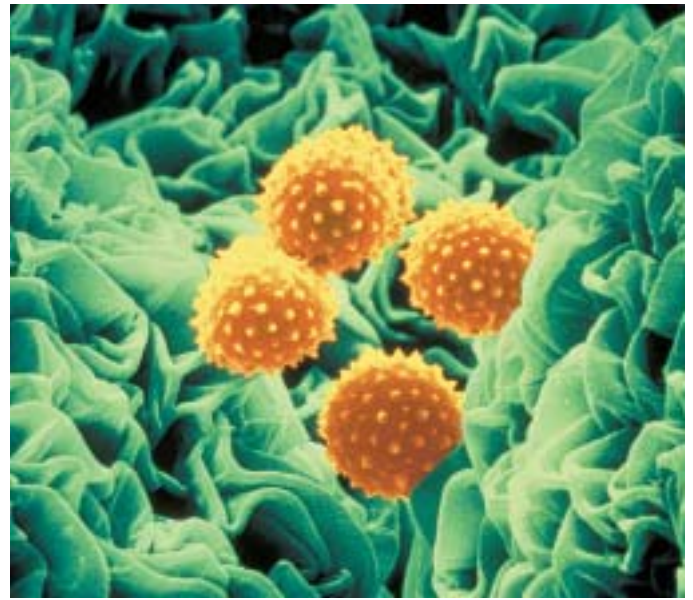
ALLERGY PREVALENCE IN CHILDREN FROM FARMING/NON-FARMING COMMUNITIES



Source: Mutius, E., Munich 2001.



Mast cell.



Pollen.

The immune system

All cells associated with immune defence are formed in the bone marrow. They later migrate and move freely between bone marrow, thymus, spleen, lymph nodes, blood/lymphatic channels and the liver. B and T lymphocytes are particularly important for a specific immune response in reaction to contact with an allergen. These comprise a subgroup of the leucocytes (white blood cells), with B lymphocytes being responsible for the production of immunoglobulins (antibodies). These are subdivided into five classes of immunoglobulin: IgA, IgD, IgE, IgG and IgM.

T lymphocytes have a mediating role between specific and non-specific immune responses and a distinction can be made between T helper cells (Th), T suppressor cells (Ts) and cytotoxic T cells (Tc). The equilibrium of T helper cells, further divided into Th1 and Th2 cells, has a decisive role in the development of allergies. As soon as the Th2 lymphocytes are not "held in check" by the Th1 lymphocytes, an IgE-mediated immunological chain reaction starts, culminating in the release of histamine from the mast cells. Histamine is the most important mediator of inflammation in the body, and gives rise to the well-known clinical char-

acteristics of immediate hypersensitivity (Type I reaction), for example an acute asthma attack after re-exposure to allergen.

The human immune system is already active before birth. At the time of birth, T cells can be demonstrated in the neonate. As children normally suffer from various infectious diseases of childhood, a healthy resistance is built up and the immune system trained. Through these early encounters, the immune system learns to distinguish between harmless and damaging antigens and thus builds up immunological tolerance.

Allergic diseases

ATOPIC DERMATITIS (ATOPIC ECZEMA, NEURODERMATITIS)

An important clinical picture from the group of atopic diseases is atopic dermatitis, suffered by up to 10% of the German population. The peak of this disease is seen in infants and children, although rare cases may still occur in adults as late manifestations. Eczematous skin changes are characteristic; depending on the age of the patient, these are found in various sites and associated with marked itching. Atopic dermatitis is not caused by allergens, although those affected frequently have raised levels of IgE antibodies and not a few of them suffer a further atopic disease such as allergic rhinitis or allergic bronchial asthma at a later stage. Besides basic treatment with moisturising ointments and creams, topical steroids are also used in atopic dermatitis, as they counteract itching and inflammation. In particularly severe cases, the external use of the immunosuppressive tacrolimus may be successful (see Medletter 02). Often the atopic dermatitis is cured years later although the patient often has a tendency to hypersensitive skin and, when choosing a career, should avoid jobs working in wet conditions.

ALLERGIC RHINITIS/ RHINOCONJUNCTIVITIS (HAY FEVER)

Allergic rhinitis also belongs to the atopic symptom complex. Up to 15% of the German population suffer from hay fever, an IgE-mediated inflammation of the nasal mucosa which, depending on the allergen responsible, may be seasonal or perennial, i.e. occur throughout the entire year. The group of seasonal allergens includes grass and tree pollens, while house dust mite or animal hair allergens may be responsible for perennial rhinitis. Often allergic conjunctivitis occurs in addition to the nasal symptoms, so a patient's performance may be greatly affected. Many of those affected do not know the allergic genesis of their condition, so valuable time is often lost before adequate allergic diagnostics are carried out. With the aid of an adequate history and prick test, it is possible to identify the allergens responsible in most cases. Then worthwhile measures can be taken to achieve allergen avoidance as completely as possible. Allergic rhinitis is treated either symptomatically with antihistamines, mast cell stabilisers and/or topical steroids or with specific immunotherapy (hyposensitisation). Where allergen specific immunotherapy is carried out consistently over a sufficiently long period (three years) success rates of up to 80% have been reported.

ALLERGIC BRONCHIAL ASTHMA (EXTRINSIC ASTHMA)

Allergic bronchial asthma – also referred to as extrinsic asthma – likewise belongs to the atopic group of conditions. Bronchial asthma is an inflammatory disease of the lower respiratory tract, characterised by bronchial hyperreactivity and reversible airways obstruction. Up to 10% of children and up to 5% of adults suffer. The allergic form is seen primarily in children while most adults have a mixed form, i.e. a combination of intrinsic (non-allergic) and extrinsic (allergic) asthma. The decisive factor in the development of allergic bronchial asthma is an IgE-mediated release of mediators (e.g. histamine). These mediators cause immediate bronchial obstruction with the clinical picture of attacks of breathlessness. Early symptoms may take the form of a distressing irritant cough. Anti-asthmatic therapy is based on three strategies: extensive allergen avoidance, anti-inflammatory drugs (e.g. glucocorticoids) and bronchodilators (e.g. theophylline). Since bronchial asthma is an episodic condition, lung function may be normal in the intervals between attacks. In this case, bronchial hyperreactivity can be demonstrated by a positive provocation test.

ANAPHYLAXIS

Anaphylaxis is understood as IgE-mediated immediate hypersensitivity (Type I reaction), with often only a few minutes between allergen contact and the first clinical symptoms. Anaphylactic reactions are divided into five degrees of severity which require different therapeutic interventions. In the case of anaphylactic shock, the patient must receive intensive medical care immediately. One of the most important groups of allergens that trigger severe anaphylactic reactions, including anaphylactic shock, are insect venoms. Food allergens may also provoke serious allergic reactions. Patients with insect venom or food allergies are often provided with an emergency set that contains a fast-acting antihistamine, a glucocorticoid and adrenaline. A highly efficient method of treating insect venom allergy is specific immunotherapy, formerly known as hyposensitisation. If this is given consistently over a period of five years, the success rate is more than 90%.

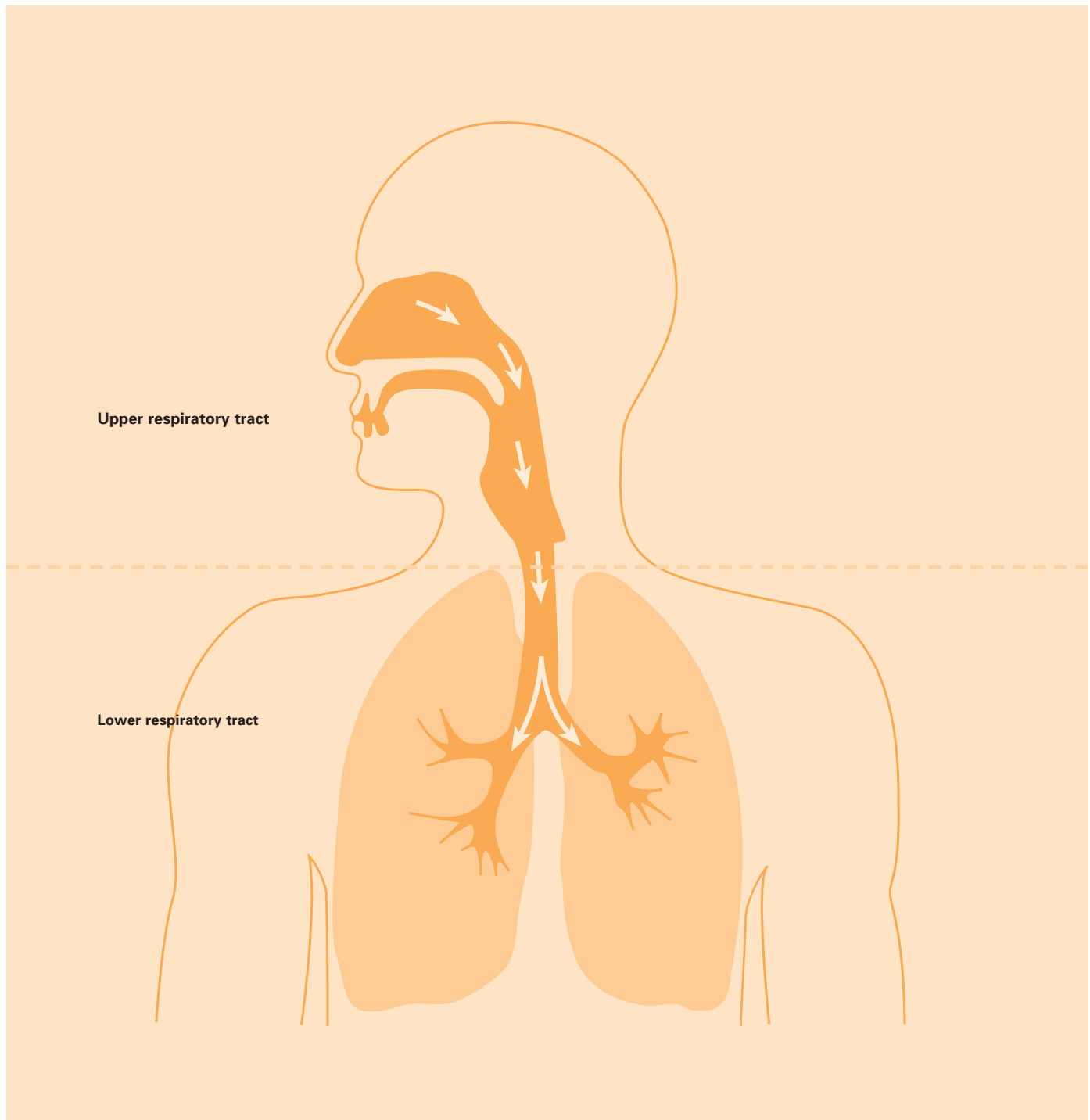


House dust mite.

Some face a difficult journey – The allergy march

“Allergy march” depicts a progression from atopic skin disease in early childhood through hay fever to allergic asthma over the course of time. According to the literature, this anatomical spread is reported in about 30% of people with hay fever, although the mechanism behind the phenomenon is not yet completely understood. The development of bronchial hyperreactivity seems to play a decisive role. This means there is an increased readiness of the bronchial system to react with clear bronchial obstruction

in response to subthreshold stimuli. To prevent this complication and protect hay fever patients from developing allergic bronchial asthma, many allergologists recommend that these patients be treated early and consistently.



ALLERGY DIAGNOSTICS TODAY

STAGE 1

The first step in well-founded allergy diagnostics is always to take a careful history (by questioning the patient). This will provide valuable information about the nature of symptoms and possible causative factors (allergens). If the patient consents and there are no contraindications (e.g. pregnancy or current immunosuppressive therapy), the next step is to carry out a prick test (see Stage 2) to determine any immediate hypersensitivity (Type 1 allergy). There are special tests for patients with eczematous or very reactive skin (e.g. people with neurodermatitis or young children) that prevent false positive reactions.

STAGE 2

The **PRICK TEST** today is standardised, includes twenty allergens, and has become the most important skin test. The allergens used range from tree and grass pollens through house dust mite and moulds to animal hair. The test thus covers the majority of allergens which induce an allergic rhinoconjunctivitis or allergic bronchial asthma. Drops of standardised allergens are placed on the inner aspect of the forearm and a sterile lancet used to prick the skin through the allergen solution, without causing any bleeding. There are also standard allergen extracts for the diagnosis of food allergies. Controls for the prick test are 0.9% saline and histamine. The skin is inspected after 20 minutes for evidence of reactions in the form of erythema or a weal with further checks for delayed reactions after six hours and again after 48 hours if appropriate. If there is a suspicion of an allergic contact dermatitis (Type IV reaction or delayed hypersensitivity), a similarly standardised **EPICUTANEOUS TEST** (patch test) is carried out. It is particularly important to investigate and clarify occupational skin reactions, e.g. with suspicion of latex allergy in a healthcare worker or of hairdresser's eczema. The epicutaneous test is also the method of choice if allergies to cosmetics, jewellery, clothing or cleaning agents are suspected. The substances are not pricked into the skin, but applied on the back with a plaster, and the results read after 48 and 72 hours.

STAGE 3

Should the results of the prick test not correlate with the clinical picture, or it is not possible to perform a skin test, a **RAST** (radioallergosorbent test) or an **EAST** (enzyme-allergosorbent test) may be carried out. These involve blood tests to demonstrate specific IgE antibodies. If an allergy is suspected, the total IgE concentration in the blood is only an indication of this, since IgE is raised not only in allergies but also in non-allergic conditions such as parasite infestations or congenital immune deficiencies. If, however, an allergy has already been clearly diagnosed on the basis of symptoms and results of skin testing, it is neither required nor justified to carry out expensive blood tests such as RASTs or EASTs.



STAGE 4

To elucidate questionable bronchial hyperreactivity, bronchial provocation (e.g. with acetyl choline) is performed, by inhaling the substance. Objective measurement of possible bronchial obstruction is performed by spirometry (measurement of the exhaled volume of gas) or by whole body plethysmography (measurement of airway resistance).

Prevention and treatment

Even though medicine today offers a wide variety of anti-allergic drugs, primarily the greatest efforts are made to achieve allergen avoidance as completely as possible. For treating allergies there are various active ingredients which have very different points of action, depending on the class of substance.

ANTIHISTAMINES are cornerstones of anti-allergic therapy and have been used for many years. They suppress the effects of histamines and prevent the symptoms of allergy. Unfortunately these preparations – at least the older first-generation drugs (e.g. Tavegil®) – have marked sedative effects (calming and making the patient feel sleepy) which limit the ability to drive or work efficiently. Over the past few years newer specific H1-antagonists of the second and third generations (e.g. Zyrtec®, Lisino®, Aeries®) have been developed to overcome these adverse drug reactions, and the sedative effect has been virtually eliminated. Antihistamines are indicated for acute and chronic allergic diseases such as urticaria, hay fever and allergic conjunctivitis. As for most anti-allergic drugs, these are available as tablets, eye drops, nasal sprays or solutions for injection so that optimal treatment can be given depending on the disease and the needs of the patient.

GLUCOCORTICOIDS also play a leading role in the treatment of various allergies, since they have an anti-inflammatory mechanism of action. These drugs were first synthesised in the mid-50s and have been extensively available since then. At first they were used somewhat indiscriminately, and as their side effects are not inconsiderable, a justified “cortisone” anxiety developed. Possible adverse reactions range from gastric problems and a tendency to oedema to high blood pressure and glucose intolerance. If the indications for therapy are correctly established and the appropriate form and dosage administered, the use of corticosteroids is an absolutely safe and highly efficient form of treatment. The development of turbuhalers, for example, has almost completely prevented undesirable systemic effects. Like antihistamines, glucocorticoids are available in different dosage forms such as nasal sprays, turbuhalers (e.g. Pulmicort®), ointments (Volon® A), tablets (e.g. Decortin®, Celestamine®) and solutions for injection.

In order to inhibit the IgE-mediated degranulation of mast cells and thus prevent the release of histamine, substances referred to as **MAST CELL STABILISERS** were developed. These are applied topically as powders, metered aerosols (e.g. Aarane®, Allergospasmin®), sprays (e.g. Irtan®) or eye drops to treat mild hay fever, allergic conjunctivitis and allergic bronchial asthma in children. Since their effects are of slow onset, mast cell stabilisers are only for prophylactic use – these drugs are no longer effective once

histamine has been released and the allergic reaction is in process.

The newest class of anti-allergic drugs is that of **LEUKOTRIENE ANTAGONISTS**. In Germany to date only drugs that act as receptor antagonists (LTRAs) have been licensed for use (e.g. Singulair® tablets). Approved indications are mild to moderate as well as exercise-induced bronchial asthma and analgesic intolerance. However, there are still no long-term studies of these active ingredients. Although it would appear to be the case at the moment that they are free of side effects, it remains to be seen whether this will change over time.

ALPHA-SYMPATHOMIMETIC DRUGS are suitable only for the symptomatic treatment of specific complaints (nasal obstruction, rhinorrhoea) and are used as nasal drops (e.g. Otriven® and Nasivin®) or nasal sprays. These should be used only over short periods as they may damage the nasal mucosa or the patient becomes accustomed to them (rebound phenomenon).

Effective secondary prevention can be found in allergen **SPECIFIC IMMUNOTHERAPY**, which was earlier also known as hyposensitisation or desensitisation. The tendency to react in an allergic manner to a particular allergen should be reduced by the repeated administration of gradually increasing doses of a specific substance. This type of treatment makes great demands on the patient, however. Firstly it has to be carried out for several years (3–5 years, depending on the allergy) and secondly it carries the risk of an allergic reaction (including that of anaphylactic shock). So that such complications are not missed, the patient always has to be observed for some time after the injection of the allergen. The success rates of specific immunotherapy are impressive for the correct indication. It is 70–80% for allergic rhinoconjunctivitis, and 90–95% for allergies to insect venom. At the present time, specific immunotherapy is still most commonly administered by subcutaneous injection, although several international studies are examining whether sublingual or oral administration of the allergen concerned is similarly effective. These new, more patient-friendly forms of administration could greatly increase the acceptance of specific immunotherapy in those affected.

In the case of occupational allergy, it is often not possible to achieve complete allergen avoidance or to bring about freedom from symptoms with anti-allergic therapy. It is then essential to direct the patient into a new profession, something that may be regarded as tertiary prevention.



Fully blown rhinoconjunctivitis with blepharodema.



INTERVIEW WITH AN EXPERT

Dr. Sebastian Gallenberger, pulmonologist and allergologist, senior consultant at the Munich Bogenhausen City Hospital agreed to be interviewed one afternoon recently in Munich Re's Department of Insurance Medicine. He advised on frequently-asked questions that arise in association with allergies, and on current problems relating to underwriting in connection with insurances of the person.

Munich Re: In the information supplied by applicants, the expression "bronchial hyperreactivity" often appears, even though there are no clinical symptoms. How should this diagnosis be assessed?

Dr. Gallenberger: Firstly, we should make it clear that there are still many unanswered questions about bronchial hyperreactivity. In contrast to allergies, we do not know precisely how bronchial hyperreactivity is mediated. Bronchial challenge or provocation (e.g. with hypertonic saline, histamine or acetyl choline) will trigger a bronchial reaction in anyone – including healthy people – depending on the dose of the irritant substance. Some people respond to viral infections with a hyperreactive bronchial system but are again symptom-free when the acute infection is over. Whether these people later develop asthma can only be seen with time – it is not possible to predict this with any degree of certainty. Bronchial hyperreactivity shows a genetic predisposition, but as long as the person affected has no clinical symptoms he/she is not considered to be ill and no treatment is given.

Munich Re: A significant increase in allergies over the last 50 years has been reported in the literature. Are there differences in geographical distribution?

Dr. Gallenberger: Geographically speaking, there is a clear north/south divide as well as a east/west differential. That is to say, in northern countries such as Scandinavia and western countries such as the UK allergies are much more common than, for example, in Mediterranean lands or eastern Europe. In her comparative study of children in Munich, Leipzig and Halle, Dr. von Mutius showed that there are also clear differences within Germany. In a further study she found that the prevalence of atopy is 10% in children in Estonia, 13.6% in Poland and 30% in Sweden.

Munich Re: In assessing the disability of applicants with hay fever, there is often the consideration that there may be what we call "allergy march". Is this concern justified?

Dr. Gallenberger: The term "allergy march" is not entirely undisputed. If one believes the literature, up to 30% of patients with hay fever undergo such an anatomical spread within about ten years and suffer progressively from allergic bronchial asthma. On the basis of clinical experience, however, it can safely be said that this anatomical shift is

more a problem in younger people. After the age of 30 years, it no longer has to be taken into consideration.

Munich Re: Is there a clear-cut age at which (allergic/non-allergic) asthma first presents?

Dr. Gallenberger: Unfortunately not. Asthma can theoretically occur at any time from birth until death – there is no age when the onset of this disease is not possible. Young children who show marked sensitisation certainly have a greater risk of suffering from allergic asthma at a later stage. If a child already suffers from allergic asthma, the history (severity of initial symptoms, number of hospital admissions, necessity for year-round treatment) gives meaningful indications of later developments – that is to say, the more marked the original symptoms, the more complicated the course of the disease will be. Medical experience has shown us that patients who first develop intrinsic (non-allergic) asthma at a more advanced age have a worse prognosis.

Munich Re: Allowing for the fact that – for purposes of disability insurance in particular – we must keep the possible long-term consequences of allergic diseases in mind, the question arises how problematic allergic asthma can really become?

Dr. Gallenberger: Fortunately, in the majority of cases, allergic asthma is an easily treatable disease. A patient with allergic asthma who is stabilised on treatment and shows good compliance has a normal life expectancy. He has no increased risk of the structural damage to the lungs (e.g. emphysema, right cardiac failure) that we know from patients with chronic obstructive pulmonary disease (COPD).

Munich Re: And how should people with asthma be viewed if they do not give up smoking?

Dr. Gallenberger: These patients do not respond so well to the various treatments available and have clearly worse lung function. The more severe course of their disease compared with non-smokers can sometimes be ascribed to chronic obstructive pulmonary disease that arises at the same time. And let me add that smoking during pregnancy or in the vicinity of children is an important risk factor for the child to develop allergies and asthma.

Munich Re: How do you regard hyposensitisation? Can we believe applicants when they tell us that they have been completely symptom-free since this treatment?

Dr. Gallenberger: Therapeutic success depends greatly on the right indication being established. When is hyposensitisation successful? To date, definite effects have been shown for the following diseases: hay fever due to pollen, mite and animal hair allergies, mild allergic bronchial asthma and insect venom allergies. Standardised allergens have been used with great success for treatment of the last-mentioned – some 95% of people with insect venom allergy speak favourably of such therapy. However, hyposensitisation in these patients – who are usually not atopic subjects – is not without risk and so treatment has to be given as an inpatient. Hyposensitisation in hay fever and allergic asthma has success rates of about 70–80%. There can be no objection, however, if a patient with hay fever that is well-controlled on medication decides not to have allergy shots.

Munich Re: What is the significance of the diagnosis “atopic” for those affected?

Dr. Gallenberger: Atopy means an inherited tendency to develop Type I allergies against inhalation antigens and eczema. Atopy thus includes atopic eczema, allergic rhinitis and allergic bronchial asthma. Atopic subjects have raised levels of IgE, although most of them remain healthy throughout their entire lives. What is important for these people, is to take this atopic tendency into account when considering their choice of career and rule out occupations which could cause problems, such as baker or florist. If a job with the corresponding allergic potential is still chosen, the first clinical symptoms usually appear relatively early – within the apprenticeship period. If an atopic person has been in a “problematic” working environment without any difficulties for more than ten years, it is most unlikely that symptoms will begin after this length of time.

Munich Re: What do you think of the claim that children often grow out of childhood asthma?

Dr. Gallenberger: In earlier times no consistent distinction was made between allergic asthma and obstructive bronchitis. It has to be said that the majority of young children who wheeze do not suffer from allergic asthma but from obstructive bronchitis (“happy wheezers”). The prognosis of obstructive bronchitis is good, the bronchi become

wider with age and the mucus therefore causes less obstruction. If a child suffers from allergic asthma, however, the symptoms are actually more marked during childhood and adolescence and decrease in adulthood.

Munich Re: What order of diagnostic investigations do you follow if you suspect an allergy?

Dr. Gallenberger: The first emphasis must be placed on taking a good history. That means asking the patient for information about the onset, nature and intensity of the allergic symptoms, in order to recognise any possible trigger factors and to establish the primary differential diagnosis. I would then perform a prick test (= skin test). Today there are standardised prick tests that cover 20 allergens (from grass pollens to animal hairs). With the aid of this recent test, it is possible to identify the majority of people with allergies. For patients with very reactive skins (e.g. those with neurodermatitis, young children) there are special tests to prevent false positive reactions. A RAST (radioallergosorbent test) is only required if the clinical picture does not correlate with the results of the prick test. In special cases (e.g. a suspicion of allergens in the workplace) a provocation test may be carried out. In view of the possible delayed reactions, it is important to monitor the patient for a sufficiently long period afterwards. Bronchial provocation gives the most significant results as the symptoms which may arise can be measured objectively with lung function tests.

Munich Re: Dr. Gallenberger, thank you very much for talking to us.

ALLERGOLOGY GLOSSARY

ALLERGEN Substance (protein) foreign to the body which causes an allergic reaction, e.g. of natural origin (plants, animals, microbes) or synthetic nature (cosmetics); acts as antigens and stimulates the production of antibodies (immunoglobulins). Allergens may be seasonal (e.g. grass pollens) or year-round/perennial (e.g. mite or food allergens)

ALLERGEN AVOIDANCE Exposure prophylaxis through avoiding the allergen(s)

ALLERGIC RHINITIS (RHINITIS ALLERGICA) Symptoms (hay fever) limited to a particular time of year (seasonal) as the reaction to pollen, or year-round (perennial) symptoms as the reaction to house dust mite; if the eyes are also affected, it is referred to as allergic rhinoconjunctivitis

ALLERGIC RHINOCONJUNCTIVITIS (RHINOCONJUNCTIVITIS ALLERGICA) Allergy-induced inflammation of the conjunctiva and nasal mucosa; also called hay fever

ALLERGY Excessive immune reaction in response to allergens

ALLERGY MARCH Progression from atopic skin disease in early childhood through hay fever to allergic asthma over the course of time

ANAPHYLAXIS Severe (IgE-mediated) immediate hypersensitivity (Type I allergic reaction), e.g. following a wasp sting

ANTIBODIES Immunoglobulins, produced by the human immune system in response to antigen contact (see **ALLERGEN**)

ATOPIC ECZEMA (NEURODERMITIS, ATOPIC DERMATITIS) Allergy-related skin disease with severe distressing itching

ATOPY Inherited tendency to develop IgE-mediated allergies; includes atopic eczema, allergic rhinitis (hay fever) and allergic (extrinsic) asthma as well as anaphylaxis

AVOIDANCE Prophylaxis by avoiding exposure to allergen(s)

B LYMPHOCYTES Special group of white blood cells which produce immunoglobulins; most important for the development of allergies is IgE

BRONCHIAL ASTHMA Allergic or non-allergic chronic inflammatory disease of the respiratory tract, with attacks of breathlessness due to bronchial obstruction

CROSS-ALLERGY Pollen-associated food allergy e.g. birch pollen allergy and cross-reaction with apple, kiwi, nuts, celery, etc.

CYTOKINES Substances which mediate interactions between cells, including interleukins, growth factors, interferons, tumour-necrosis factor

EAST (ENZYMEALLERGOSORBENT TEST) Blood test to demonstrate specific IgE antibodies

ECZEMA Chronic inflammation of the skin with involvement of the epidermis (outer layer of skin)

EPICUTANEOUS TEST Standardised skin test used to determine delayed hypersensitivity (Type IV reaction)

EXTRINSIC ASTHMA Allergic asthma (in response to allergens from the environment or workplace)

HAY FEVER See **ALLERGIC RHINITIS** and **ALLERGIC RHINOCONJUNCTIVITIS**

HISTAMINE Messenger substance released by mast cells, the most important naturally occurring trigger of allergic reactions (associated with itching, pain and bronchial narrowing)

HYPERREACTIVITY (BRONCHIAL) Increased readiness of the bronchial system to react with significant bronchial obstruction to even subthreshold stimuli

HYPOSENSITISATION Targeted immunotherapy by the repeated administration of increasing doses of a specific allergen, in order to reduce the readiness to react in an allergic manner to this allergen (also referred to as allergen specific immunotherapy, allergy shots and sometimes as desensitisation)

IMMUNOGLOBULINS Substances (proteins) produced by B lymphocytes following antigen contact, as part of the body's defence mechanism; five classes identified in humans to date: IgA, IgD, IgE, IgG, IgM

INTERFERONS Substances naturally occurring in the body as a defence against viruses (see also **CYTOKINES**)

INTERLEUKINS Signal transmitters between cells (see also **CYTOKINES**)

INTOLERANCE Inability of the organism to react adequately to external stimuli, or an enzyme defect (e.g. lactose intolerance)

INTRINSIC ASTHMA Non-allergic asthma

LATEX ALLERGY Allergy to natural latex after skin or mucosal contact or following inhalation of latex particles

MAST CELLS Cells that contain inflammatory mediators (e.g. **HISTAMINE**); found in the blood, skin and mucous membranes

MEDIATOR Substance responsible for the genesis of the allergic reaction (see also **HISTAMINE**)

NEURODERMATITIS (ATOPIC ECZEMA, ATOPIC DERMATITIS) Allergic skin disease with severe distressing itching

PERENNIAL ALLERGENS Year-round allergens

POLLEN Male spores of flowering plants, scattered by insects or the wind

PREVALENCE Frequency of a disease found in a particular population, data expressed as number of those affected per 100,000 persons

PRICK TEST Standardised skin test to determine immediate hypersensitivity (Type I allergic reaction)

PROVOCATION TEST Test to establish bronchial hyperreactivity, e.g. through nasal or bronchial challenge

PSEUDOALLERGY Intolerance similar to an allergy, often not clinically distinguishable from an allergy; known with acetyl salicylic acid (Aspirin) or red wine, for example

RAST (RADIOALLERGOSORBENT TEST) Blood test to demonstrate specific IgE antibodies

RHINITIS Inflammation of the nasal mucosa

SENSITISATION Increase in sensitivity following repeated contact

SPECIFIC IMMUNOTHERAPY Also allergen specific immunotherapy (see **HYPOSENSITISATION**)

SPIROMETER Apparatus for measuring respiratory gas volumes

SPIROMETRY Determination of ventilation capacity using a spirometer

T LYMPHOCYTES Responsible for cell-mediated immunity (e.g. T helper cells and cytotoxic T lymphocytes)

WHOLE BODY PLETHYSMOGRAPHY Apparatus for measuring airways resistance

A CASE IN PRACTICE

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CASE DESCRIPTION

29-year-old industrial salesman

Desired cover: Life insurance and additional disability benefits insurance

The applicant was diagnosed as having allergic bronchial asthma in 1998 after he had already suffered from a marked pollen allergy for many years. On more careful questioning, it also became clear that in his youth he had suffered from exercise-induced and cold-induced asthmatic symptoms. Since 1998, the applicant had received long-term treatment with Pulmicort® metered aerosol, also taking Zyrtec® (an antihistamine) as necessary during times of high pollen count. Hyposensitisation was carried out from 2000 to 2001, and no further asthmatic symptoms had occurred since 2000. The applicant had no restrictions on his everyday life although mild respiratory problems sometimes occurred on physical exercise and in winter. Lung function tests performed in March 2002 were normal – with no evidence of obstructive airways disease.

V

OUR VOTE IS:

Life insurance: Borderline standard

Additional disability benefits: + 50%

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COMMENT

The applicant suffers from pollen-associated allergic rhinitis, which is kept well under control by taking an antihistamine when required. The asthmatic symptoms that he has had since his youth, triggered by exposure to cold or induced by exercise, may be interpreted as intrinsic (non-allergic) bronchial asthma. In addition, since 1998, there has been an extrinsic (allergic) component of the bronchial asthma, so that there is now a mixed form, as suffered by some 80% of adults with asthma.

Risk assessment: Allergies in disability insurance (TPD own/any)

Allergic dermatitis, neurodermatitis, atopic eczema, occupational dermatitis:

Present, occupational	Excl. dermatitis
Non-occupational	0
In history	0

Allergic rhinitis, allergic syndrome, hay fever:

Severe	Excl. rhinitis
Otherwise	0

Allergic/non-allergic bronchial asthma:

Non-occupational, < 45 y, mild and moderate	50–100
Non-occupational, < 45 y, severe	Decline
Non-occupational, > 45 y, mild and moderate	25–50
Non-occupational, > 45 y, severe	Decline
Occupational, < 45 y or > 45 y, mild, moderate and severe	Decline

Exclusion clause for dermatitis (allergic dermatitis, neurodermatitis, atopic eczema, occupational dermatitis): No claim shall be admitted in respect of any disease, disability, disorder, injury, any operation, or treatment, whether directly or indirectly caused by dermatitis.

Exclusion clause for rhinitis (allergic rhinitis, allergic syndrome, hay fever): No claim shall be admitted in respect of any disease, disability, disorder, injury, any operation, or treatment, whether directly or indirectly caused by allergic disorders of the skin (including neurodermatitis), mucous membranes (e.g. the bronchi as well) and the gastrointestinal tract.

You will find a user-friendly presentation in our internet-based reference manual MIRA.

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Münchener Rückversicherungs-Gesellschaft
Königinstrasse 107
80802 München
Germany
Tel.: +49(0)89/3891-0
Fax: +49(0)89/389056
<http://www.munichre.com>

Responsible for content

Divisional Unit Life/Insurance Medicine

Your contact person

Dr. Ulrike Hartwagner
Tel.: +49(0)89/3891-9635
Fax: +49(0)89/3891-79635
E-mail: uhartwagner@munichre.com

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