Record-breaking Success in Barcelona
Bacteria and Allergy
Th17-cells
and more...
Dear EAACI-members,

This year’s meeting, the EAACI 2008 Congress in Barcelona, attracted more than 7600 delegates from over 100 countries and more than 1800 abstracts were presented! This clearly shows the growing interest in allergy – an epidemic of the 21st century.

When and how did this epidemic start? From page 6 and onwards you can read more about the three current hypotheses and the many questions that still need to be answered. More than 500 million people worldwide suffer from allergic rhinitis and more than 300 million people of all ages suffer from asthma. Globally, 250 000 people die of asthma every year. Consequently, the revised GINA guidelines stated that asthma is the most serious of allergic diseases and that it is disabling and occasionally fatal.

You can also read about the influence of nutrition on inducing and maintaining allergic disease and the increasing knowledge on the link between infection and allergy. Next to the endotoxins of gram-negative bacteria, the enterotoxins of gram-positive bacteria, especially superantigens released by Staphylococcus aureus gain more and more focus. They are perfect candidates to severely impact allergic airway diseases and bronchial hyperreactivity, from induction to amplification of disease. An update on the subject is included in this issue.

More exciting news are the understanding of T-cells and their role, which more and more abandons the TH1/TH2 paradigm and introduces T-regulatory, TH17 and other cells to the picture. These “new kids on the block” clearly make immune responses even more complex, but they also provide new opportunities for an explanation of the inflammatory pattern we observe.

Those of you with a special interest in upper airway immunology are cordially invited to the 7th SERIN-meeting in Dubrovnik, November 2008. And the next EAACI Congress in Warsaw 2009 will definitely update you on the exciting research, which keeps our specialty so interesting.

Immunology is the name of the game!

Claus Bachert
The congress held in Barcelona, Spain was very successful, with a total number of 7,700 delegates and close to 1,900 abstracts, breaking all records for any congress to date. Again, I would like to express my thanks to Ignacio Ansotegui, Vicky Cardona and the Spanish Society, and also to Cezmi Akdis and Christian Virchow for all their efforts and the years of hard work they have put into preparing for the congress. Barcelona was truly The City of Wonders!

One of our goals at the Barcelona Congress was to improve the bonds of friendship with all National Societies (NS). To achieve this, we held a get-together with NS officials and extended an invitation to NS chairs to the Presidential Dinner. These initiatives were appreciated and should form the basis of a better – and also more formal – kind of interaction between the EAACI leadership and the National Societies. We liaised with speakers and officials coming from many countries and organisations outside Europe. We held an international reception to demonstrate our enthusiasm for cooperating with organisations from other areas, and also to acknowledge the contribution they make to our Academy. I was particularly pleased and happy to meet the many hundreds of delegates from South and Central America.

As I reported in the previous Newsletter, we have launched the EAACI Exam and we can expect to hear more about this initiative from Professor Werner Pichler, the driving force behind the venture. However, at the moment I can tell you that the first Exam more than met our expectations and appeared to have a high standard. The feedback from participants was positive, and the initiative is certainly one we will continue to promote.

News from the General Assembly

The General Assembly was held on the 9th of June and took several important decisions. First of all, Professor Jan Lötvall – our current Secretary-General – was elected President-Elect. I am very happy to offer him my congratulations. The new system of selecting a President-Elect one year before they take up the position gives the nominee some opportunities to elaborate on the new programme during the following term. The General Assembly confirmed our proposals to extend the Executive Committee by two members representing the Interest Groups, and those chosen were Pascal Demoly and Stephan Vieths, who will make excellent ambassadors from the Interest Groups. Finally, we took a vote on our name. The Executive Committee proposed changing “Allergology” to “Allergy” and also including the word “Asthma” in the title. The General Assembly decided otherwise. Indeed, the majority voted for the change to “Allergy” but not for the inclusion of “Asthma”. Although the outcome differs from our proposal, I was satisfied that the resulting decision was taken democratically. We launched an online discussion, giving a balanced overview of the benefits and disadvantages during the Assembly meeting. We did not ask the General Assembly simply to approve our decisions. As a result, the name of our Society is now changed to the European Academy of Allergy and Clinical Immunology.

Coming Events

Two Allergy Schools took place after the congress in Barcelona. The Allergy School in Porto Helio, Greece in June focused on Allergy, Asthma & Sports, and the School in Venice, Italy in July covered the topic of paediatric asthma. Both schools were well attended and successful. Unfortunately, we were obliged to cancel the Allergy School planned for Tbilisi, Georgia in August as a result of insufficient applications, although war broke out one day after the cancellation. I sincerely hope for better times for our colleagues in Georgia.

The two schools mark the start of a new series of initiatives. In September, we are supporting the 12th International Paul Ehrlich Seminar in Bad Homburg, Germany. In October, we will contribute to the annual meeting of the Portuguese Society; while in the same month the AAAI and EAACI will organise a PRACTALL consensus meeting on food challenges in New York, US. In November, the EAACI and WAO leadership will join at the XIX World Congress of Asthma in Monte Carlo, Monaco. The 7th SERIN Congress takes place in Dubrovnik, Croatia, and finally, the Allergy School on Epidemiology of Allergy and Respiratory Diseases will be held in London, UK. In December, EAACI will support the 3rd International Consensus Meeting on Urticaria in Berlin, Germany. These meetings are an expression of the dynamics of our organisation. I invite all EAACI groups to present new proposals for meetings and task forces in 2009. We have successful yearly congresses, but do not sit idle between those events.

Roy Gerth van Wijk
EAACI President
The Congress attracted more than 7600 delegates (new record!) from over 100 countries and the total number of abstracts was 1851 (another record!). The total number of symposia/workshops was 93 and the size of the exhibition area was 1527 sqm.

The Congress was covered by Spanish as well as international media, and over 80 journalists were present at the meeting.

Photos, press releases and more information about the EAACI 2008 Congress can be found on www.eaaci2008.com

Join the EAACI 2009 Congress in Warsaw, 6–10 June 2009! More information about this meeting will be published in the next EAACI Newsletter.
The New International Pediatric Allergy and Asthma Consortium (iPAC)

A group of leading academic Pediatric allergists met scientists from the industry in Guincho, Portugal on 18–20th October 2007.

Their aim was to instigate state-of-the-art research in Pediatric allergy and immunology and to define new ideas and trends

Initiated by the EAACI Section on Pediatrics, the meeting was financed by EAACI, AAAAI, and the EAACI-Clemens von Pirquet Foundation, with unrestricted grants from the industry (Mead Johnson, Nestlé Nutrition Institute, Phadía, and Schering Plough Spain).

Small working groups addressed issues including basic research and atopy prevention, atopic dermatitis, IgE and non IgE-mediated food allergy, anaphylaxis, and respiratory allergy. Plenary discussions developed the ensuing ideas for future research. The discussions were summarised in a supplement to Pediatric Allergy and Immunology in August 2008.

All participants acknowledged the need to illuminate areas in the field of pediatric allergy and immunology, and to support interventional studies that would reinforce knowledge in early allergic events, most of which is currently based on observational data. The meeting also recognised that networking is essential for good science. Several initiatives exist but overwhelmingly address specific diseases such as food allergy and respiratory diseases. The participants work in Australia, Canada, Europe, and the U.S. and stressed the necessity of improving cross-border collaboration.

The meeting took the decision to found an international consortium to promote research in Pediatric allergy. The international Pediatric Allergy and Asthma Consortium (iPAC) will provide a research platform for promoting specific research projects in the field of Pediatric allergy. iPAC is a joint initiative of the pediatric sections of EAACI and AAAAI, and will be managed by the EAACI-Clemens von Pirquet Foundation. It will favour collaboration with industry, aim to provide public funding for research in the field, and also promote private donations.

iPAC is a unique tool that will promote research in early life events and the prevention of allergic diseases within the European Research Framework Programmes.

Philippe Eigenmann
Allergy – an Epidemic of the 21st Century

When and how did this epidemic start?

The Origins of Hay Fever
The first description of hay fever was made by Blackley in 1873. By 1900, outbreaks of hay fever had reached epidemic levels. In 1911, Noon published a paper on immunotherapy for hay fever, not because it was a rare disease but because it was already commonly diagnosed. In 1955, the hospital housing the hay fever clinic run by Bill Frankland asked him to close down – it was distorting the statistics of the hospital because he had so many patients.

The Origins of Asthma
By contrast, asthma is an old disease, well recognised since antiquity. Salter described asthma in his book published in 1870, although incidences were still not very common. Variations of asthma became noted from 1960, and showed a tremendous increase in the decades between 1960 and 2000. The following three hypotheses are the main ones explaining the increased prevalence of asthma:

Hypothesis 1. The Hygiene Hypothesis
The decline in infectious diseases included fewer instances of parasitic worms. Animals have not been part of many people’s domestic environment for more than 100 years. The article The Prevalence of Asthma by Eder, Ege, and Von Mutius in the New England Journal of Medicine (2006;355:21) showed the increases in asthma in many different countries and their scales of prevalence.

An article by Armstrong et al in JAMA (1999;281:61–66) showed a decline in infectious diseases and mortality in the same period starting in 1900, when people aimed to increase levels of hygiene. By 1910, public facilities included clean water, many more people wore shoes, food supplies were under control, and vaccination programmes were in place. The prevalence of asthma started to increase in the U.S., and became very obvious in the 1960s.

Between 1979 and 1990, there was a 20-fold increase in the hospitalisation of asthmatic children from poor backgrounds in the U.S. Before the 1970s, asthma was considered to be a disease that did not occur in families receiving welfare funding but this perception has changed in the last 30 years.

Hypothesis 2. Changes in Lifestyle
In 1955, most children spent their days playing outdoors, while contemporary children spend more time indoors and are less active. Whether this inclination by the majority of children toward a more sedentary lifestyle is relevant to the increase in asthma prevalence remains unknown. Some studies show a relationship with obesity. Research by Firrincielli et al in Paeds Pulm (2005) and Shaaban et al in Thorax (2007) suggests that the main factors involved in the increase of asthma prevalence include a decrease in overall physical activity.
activity and failure to expand the lungs regularly with high-energy air consumption. Further research by Fredberg in AJRCCM (1999) and Hark et al in Annals (2005) points to consequential changes in diet, while work published by Camargo et al in Am J Clin Nutr (2007) attests to a decrease of sunlight, supported by research into the prevalence of asthma among African-Americans who moved to northern states in the U.S. and mainly stayed indoors, for whom the level of vitamin D became critical. In the 1980s, associations between obesity and asthma were shown. Changes in physical activity and diet are difficult to measure, but the results of activity and eating patterns are easily measured, for example through obesity patterns.

**Hypothesis 3. Increased Exposure Allergens**

One mite fecal particle contains Der p 1 ~ 0.1ng Der p 2 ~ 0.2ng, as revealed by Tovey et al in Nature (1982). The par-ticulate matter contains Der p 1 ~ 0.2ng Der p 2 ~ 0.1ng as revealed by Tovey et al in Nature (1982). The particle also contains endotoxin, and bacterial and dust mite DNA, according to Satinover et al in AAIAA (2007). The highest prevalence and severity of asthma relates to mite and cockroach exposure. High prevalence (≥20%) is observed in Australia, New Zealand, Taiwan, the U.K., and inner cities in the U.S., while low prevalence (≤10%) is observed in Albania (2%), Germany (6%), Greece (4%), and Sweden (8%). In those countries or areas with high dust mite exposure, cockroach, or Alternaria pathogen exposure, both the prevalence and titre of IgE antibodies are high. By contrast, in countries where mite exposure is low and cats and dogs are the dominant allergens, such as Germany and Scandinavia, the prevalence and titre of IgE antibodies are lower. The differences between Russia (2%) and Finland (5%) or New Zealand (≥20%) and Sweden (8%) are sufficient to influence the prevalence of asthma.

**Where do we stand today?**

The World Health Organization Assembly held its annual meeting in Geneva, Switzerland this year with the participation of 193 health ministers. The assembly adopted an agenda for the next 10 years that comprises four priorities: cardio-vascular diseases, cancer, and chronic respiratory diseases (CRDs) such as allergies and diabetes. CRDs are responsible for 7% of total mortality. According to the revised Global INitiative for Asthma (GINA), asthma is the most serious of allergic diseases in that it is disabling and occasionally fatal.

**Globally, 250,000 people die of asthma every year.** The frequency with which allergy affects people varies largely in different countries, being as low as 1% or as high as 40%. In many places, the prevalence of allergic sensitisation is often higher than 50% for some age groups. Allergy is often underestimated, under-diagnosed, and under-treated despite its high prevalence and effect on quality of life, according to the Allergic Rhinitis and its Impact on Asthma (ARIA) study in 2007, and 300 million people of all ages have asthma. The prevalence of asthma worldwide varies highly (1–18%) and has increased following changes brought about by the modern urban lifestyle. Using a conservative estimate, more than 500 million people worldwide have allergic rhinitis.

In Australia, Europe, New Zealand, and the U.S., the prevalence of IgE sensitisation to Aero-allergens (by allergen-specific IgE in serum or skin tests) is more than 40–50%.

Epidemiologic studies consistently show that asthma and rhinitis often co-exist in some patients. The prevalence of asthma in subjects without rhinitis is usually less than 2%.

The prevalence of asthma in patients with rhinitis varies at 10–40%, depending on studies, according to ARIA in 2007.

**How can the situation be improved?**

There is a need for different thinking – lateral thinking. The term “atopic march” refers to the characteristic sequence of appearance and disappearance of atopic manifestations with time.

**There is a misconception of the term atopic march, that it starts mildly at birth or in infancy and worsens to end up as a chronic disease in adulthood.** There are many different thoughts on this topic. The term “atopic march” refers to the characteristic sequence of appearance and disappearance of atopic manifestations with time. There is a need for different thinking – lateral thinking. The term “atopic march” refers to the characteristic sequence of appearance and disappearance of atopic manifestations with time.

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Cont’d Allergy – an Epidemic of the 21st Century

atopic condition. Cohort studies show clear windows that can be used for primary and secondary prevention:

• **Primary prevention in high-risk and low-risk infants (1–6 months)** with no clinical signs, the following measures can be used: diet in early infancy, breast-feeding, hypo-allergenic formulae (high risk), probiotics (lactobacilli), prebiotic formula (low risk and high risk) and avoidance of tobacco smoke exposure.

• **Secondary prevention in infants/young children (6–36 months)** with early wheeze, atopic dermatitis, sensitisation to egg or milk, early sensitisation to indoor allergens, or any combination of these risk factors.

• **Disease modification (school age)** children with SAR.


The **hypo-allergenic approach** is based on the concept of avoiding exposure to certain allergens. The three-year results of the German Infant Nutritional Intervention Study by von Berg et al in the J Allergy Clin Immunol (2007;119:718–25) confirms that certain hydrolysed formulas reduce the incidence of atopic dermatitis, although this is not the case with asthma. The studies showed that using probiotics (lactobacilli) was effective in reducing eczema if mother and child get sufficient amounts of lactoba-
cilli. Prebiotic formula that promotes lactobacteria to grow is the use of oligosaccharides instead of bacteria in children.

The **hygiene hypothesis continues** to play a role in studies of TLR receptors. Farm milk consumption that contains viable bacteria, bacterial endotoxins, and proteins LPS was shown to be effective in prevention. Some approaches of adjuvant use for immunotherapy result from LPS relation with the expression of toll-like receptors. Allergen-induced tolerance is a method widely used in the form of sublingual immunotherapy and could be important for food allergy.

Combining **family history and early phenotypes with specific gene mutations** may help to identify a child with persistent asthma within the first year of life. Prediction instead of risk assessment – that is the new aim! Recent studies show a number of genetic markers that can help to determine atopic phenotype type. Prediction on an individual basis is the new era of prevention.

Dr Michael Rudenko MD, PhD
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That rates of food allergy vary is generally accepted. A meta-
analyses of studies found that the incidence of self-reported food allergies is in the range of 3–35%. However, very few studies employed oral challenge procedures to confirm diagnosis. The ones that did found that some 1–4% of individuals suffer from real food allergy. To address these issues, the EuroPrevall partnership is undertaking a series of studies using common protocols to obtain good estimates of the prevalence of food allergy, and to identify the major foods and clinical patterns of food allergy across Europe (Mills, Mackie et al 2007).

Dr Montserrat Fernandez-Rivas from Spain outlined several fac-
tors that seem to predispose the development of food allergy, including genetics, the host’s intestinal flora, timing, dosage, frequency of exposure to various dietary allergens, and the allergenicity of various food proteins. Immaturity of the intestinal mucosal barrier, abnormalities in the induction, and maintenance of oral tolerance may play a major role. In addition, reduced exposure to early childhood infection may also have a key role in the development of allergy and tolerance, as food reactions tend to be more severe when the lungs are involved. Environmental factors also seem to be important triggers in provoking food allergy or intolerance, and may be the cause of different clinical patterns of food allergy across Europe.

Food allergy is the result of either genuine reactivity to comestibles through the gastrointestinal tract (Class I food allergens) or secondary

Fernandez-Rivas also spoke about allergy to a plant food resulting from either direct sensitisation to that particular food or from primary sensitisation to pollen, latex, or to another food. The age distribution of a food allergy shows striking differences that may reflect the possibility that food allergies that are caused by pollen occur later than allergies that have no relation to pollen.

For example, two patterns of apple allergy exist across Europe: one pattern in the Netherlands, in Austria, and in 90% of (northern) Italian patients and another pattern in Spain and in remaining Italian patients. In the first group, apple allergy is mild and related to birch pol-
linosis and sensitisation to Bet v 1 and its apple homologue, Mal d 1. In Spain, apple allergy is severe and related to peach allergy and sensitisation to the nonspecific lipid transfer protein Mal d 3 (Fernandez-Rivas, Bolhaar et al 2006). There is also a strong association between soybean and birch pollen allergy. In Central Europe, soy allergy seems to be mainly encountered in birch pollen allergic patients, but subjects with primary soy allergy have also been identified (Ballmer-Weber, Viets 2008).

The molecular basis of food allergy was presented by Dr Barbara Ballmer-Weber from Switzerland. Food allergy is the result of either genuine reactivity to comestibles through the gastrointestinal tract (Class I food allergens) or secondary
sensitisation to cross-reactive food allergens as a consequence of initial reactivity to homologous pollen-related allergens (Class II food allergens). Stable Class I food allergens have the potential to induce severe reactions, whereas easily degradable Class II food allergens tend to induce milder reactions often limited to oral allergy symptoms. Progress in biochemistry and molecular biology allowed for the identification, cloning, and recombinant production of allergenic proteins, as well as the synthesis of IgE epitope-emulating peptides of a number of food allergens. The increasing availability of allergen panels derived from various sources enables a detailed analysis of the sensitisation profile for individual patients. This concept is defined as component-resolved diagnostics (CRD). Examples of such allergen components useful for CRD studies include Mal d 1, 2, 3, and 4 from apple (Bolhaar, van de Weg et al 2005) and Bos d 4, 5, 6, and 8 from cow milk (Shek, Bardina et al. 2005). CRD can also be based on panels of homologous allergens sharing similar structures and a high degree of sequence identity, detecting different levels of co-recognition by specific IgE antibodies (e.g. Bet v 1 homologues from hazelnut, apple, and celery or vicilins from different legume seeds).

Ballmer-Weber also focused on birch pollen-related allergenic foods and especially on soybeans. Birch pollen-related food allergies are mainly mediated by cross-reactions between the PR-10 protein Bet v 1 or the profilin Bet v 2 and homologous proteins in plant food. Bet v 1 homologous soy protein named Gly m 4 has been cloned and produced, as has Gly m 3, the profilin from soybeans. In a recent study, 21 of 22 Swiss birch pollen-allergic patients with soy allergy were sensitised to Gly m 4 and six patients to Gly m 3 (Mittag, Vieths et al 2004), showing that Gly m 4 is the major soy allergen for patients allergic to birch pollen with soy allergy. In summary, apart from primary sensitisation, allergy to legumes may be acquired in birch pollen allergic patients by cross-reaction mediated by PR-10 proteins present in birch pollen (Bet v 1) and legumes (i.e. Gly m 4).

Finally, current methods of managing food allergy were presented by Professor Sampson. The primary therapy for food allergy is to avoid the causal food or foods. Patients and caregivers should be encouraged to read food labels and recognise high-risk situations, to learn how to detect early signs of allergic reaction, to use self-injectable epinephrine, and to activate emergency services. Comprehensive educational materials are also available through organisations (http://www.foodallergy.org). Novel therapies for IgE-mediated food allergy have been reviewed. Injections of anti-IgE antibodies (TNX-901) for treatment of patients with peanut allergy showed an increase in the average amount of peanut tolerated, but 25% of the group showed no improvement (Leung, Sampson et al 2003). Traditional Chinese herbs showed efficacy in a murine model of peanut-induced anaphylaxis (Li, Zhang et al 2001). Standard immunotherapy for pollen-induced rhinitis might also improve pollen-food allergy syndrome (Bolhaar, Tiemessen et al 2004) and significant increases in tolerance to hazelnuts after sublingual immunotherapy have been assessed by double-blind, placebo-controlled food challenge (Enrique, Pineda et al 2005). Modified peanut proteins have also been produced (mArah2) for desensitisation of peanut-allergic patients, and have showed encouraging results (King, Helm et al 2005).

To conclude, the knowledge about and interest in the field of food allergy has grown tremendously in recent years. Food hypersensitivity affects up to 6% of children under the age of three years and approximately 4% of the general population, with different clinical patterns across countries. Current studies of allergen characterisation and immunologic mechanisms should provide a better understanding of the immunopathology of these disorders and new, more specific forms of diagnosis and therapy.

Maria Xatzipsalti

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Maria Xatzipsalti

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Allergy

EAACI Newsletter

- Issue 16
Hygiene, Infection and Allergy

The relationship between infection and allergy is an intriguing topic, one in which both researchers and clinicians recognise the great potential for the development of novel preventive and therapeutic strategies in the future.

The link between exposure to microbial sources and allergic illness has been the subject of allergy research for many years. The hygiene hypothesis has been updated and modified to merge the various elements of this multifaceted relation. These elements compose a complex enigma, comprising several aspects of allergy research including time, interaction between genetic variability and environment, and affected phenotype. Indeed, in clinical practice, manifestations of allergic disease sometimes appear rather uniform, but it is important to recognise the diverse underlying mechanisms and causes that may be involved. One example is the increasing evidence of the heterogeneous disease of the asthma syndrome appearing in recent years.

A vast number of studies clearly shows that the effect of given exposure depends on timing. During the stages of development and maturation, which start from the prenatal period over childhood to adolescence, predefined processes display windows of accessibility and vulnerability to extrinsic influences. Genetic factors are undoubtedly involved in these processes, and it becomes clearer that no single gene can be responsible for the clinical manifestations of allergic diseases.

One mechanism frequently associated with the hygiene hypothesis is the skewing of the Th1/Th2 balance away from allergy-promoting Th2 cells towards Th1 cells, mediated by living in a less hygienic environment. However, conflicting data on this particular issue cannot be disregarded, as not only Th2 diseases, such as allergies, have been increasing in frequency over recent decades, but also inflammatory diseases, such as Crohn’s disease and diabetes mellitus. Moreover, it has been shown that Th2 type immune responses induced by helminthic infections protect against allergic diseases. Finally, in vitro and animal data show that the activation of the innate immune system does not necessarily promote a Th1 response, as Th2 responses might also occur depending on the experimental protocol. It becomes more clear that although the mechanisms related to the Th1/Th2 balance are undeniably linked to the development of allergic diseases, they might not suffice to explain the effects related to the hygiene hypothesis.

Role of infections

Atopy is an important discriminating feature to bear in mind when studying the relation between viral infections and asthma. It appears that non-atopic wheeze is mainly associated with recurrent chest infections at the age of two years, while atopic wheeze is related to allergic illness in the child and family. Moreover, it has been known for some considerable time that virus-associated wheeze takes a milder course and offers a better prognosis than allergic asthma. The increased exposure to viruses in a child’s environment might lead to a milder form of wheezing by suppressing the atopic component. The type of infecting virus, its virulence, the severity of the infection, and the viral load must be taken into account.

With bacterial infections, it is clear that there is not one micro-organism that accounts for the observed protection to the development of allergic diseases. However, there is a particular interest in mycobacteria, as these micro-organisms show remarkable potentially immunomodulatory characteristics. Murine research into allergic asthma shows a suppression of several allergic features by treatment with mycobacteria.

Exposure to parasites in industrialised societies may be confined. However, interesting evidence from endemic areas shows a strong inverse relation between parasitic infections and the development of atopy.

Role of environmental exposure

Environmental exposure to non-viable microbial products occurs with children living in farming communities, mimicking a natural experiment. Indeed, many studies document the lower prevalence of hay fever and atopic sensitisation in children raised on farms. These children are exposed to animal sheds and haylofts, and drink unpasteurised milk. Interestingly, the timing of exposure and a clear maternal effect was also observed here.

Scientists continue to unravel the mysteries of the complex relationship between hygiene, infection, and allergy. A truly unifying concept remains missing, but the role of the host immune response, the characteristics of invading micro-organisms, the level and variety of environmental microbial exposure, and the genetic background become more apparent. Although practical implications are difficult to deduce from these findings, they provide great potential for novel preventive and therapeutic strategies.

References

Superantigens (SAGs) are a class of immunostimulatory and disease-causing proteins with the ability to activate large sections of the T cell population. Unlike conventional antigens, SAGs bind to certain regions of major histocompatibility complex (MHC) class II molecules of antigen-presenting cells (APCs) outside the classical antigen-binding groove and concomitantly bind in their native form to T cells at specific motifs of the variable region of the beta chain (Vβ) of the T cell receptor (TCR). This cross-linking triggers the non-specific activation and proliferation of T cells and induces the production of high levels of a variety of cytokines. The T cell response to SAGs is polyclonal, Vβ specific, involves both CD4+ and CD8+ cells, and is MHC II dependent but not MHC II restricted. Bacterial superantigens, and here we focus on the classical and newly described egc-locus enterotoxins, have the following common characteristics: They are among the most potent pyrogens known, and they are capable of inducing a highly lethal toxic shock syndrome. The chronic disease is characterised by T cell activation, with expansion of Vβ-specific T cells.

Enterotoxins derived from *Staphylococcus aureus* (SAEs) can act as conventional antigens and also as superantigens to exert influence on chronic inflammatory airway diseases such as nasal polyposis (NP) and asthma. These enterotoxins act as disease modifiers, as a local immune response is seen in 50% of all NP patients, comprising increased levels of enterotoxin-specific IgE. This percentage increases even to 88% if these patients suffer from concomitant asthma and aspirin hypersensitivity.

**There is evidence for the involvement of SAEs in eczema and wheezing by young children.** Previous research shows that higher levels of SAE sIgE are found in children with asthma and atopic eczema compared to controls. Moreover, children with more severe wheezing and atopic eczema more frequently have SAE sIgE. SAEs act also as disease modifiers in asthma at an adult age, and in particular in severe late-onset asthma, where again higher levels of SAE sIgE are found compared to control patients. Similar findings were reported with poorly controlled asthmatics.

**Moreover, there is evidence for a role for SAEs in other allergic diseases** such as allergic eczema and allergic rhinitis, and even beyond the scope of allergic illness, significantly higher levels of sIgE are described in the serum of patients with chronic obstructive pulmonary disease (COPD) compared to healthy controls, similar to severe asthma.

**The hypothesis of aggravating effects of SAEs has recently been shown in a murine model of allergic asthma, where nasal and bronchial SEB aggravate the phenotype of allergic airway inflammation through the enhanced expression of both Th2 and Th1 cytokines in bronchi, systemic cytokine release, and the stimulation of IL4 production and IgE synthesis.** Moreover, and in line with the above-cited human data on wheezing children, interesting and challenging data are found on the effects of SAEs in sensitisation to otherwise inert allergens.

**Staphylococcus aureus enterotoxins and other superantigens** are thus perfect candidates to severely impact allergic illnesses such as airway and skin diseases from early childhood to adult age, and clearly merit further attention. Nearly one third of the population is lifelong carriers of these germs, and nearly all have the potential to produce these toxins. Superantigens may have the answers to many questions, especially for severe airway and skin diseases, in the near future.

**Wouter Huvenne**

Presented at the EAACI 2008 Congress in Barcelona, Spain.
Asthma can be caused by many types of interaction between factors that are environmental or genetic. It is clear that hereditary elements are important to this disease, since the risk of asthma increases significantly for anybody that has close relatives with asthma. However, individual disease genes almost never trigger the disease exclusively. The disease is often a result of the interaction between both risk-increasing and protective genes. The balance between these can determine whether the person is sensitive to disease-causing risk factors in the environment, or is resistant to the environmental factors.

Professor Juha Kere’s group identified a gene on chromosome 7 that in a certain form increases the risk of asthma (Laitinen et al. Nat Genet 2001). In 2004, GPRA (G protein-coupled receptor for asthma susceptibility) was discovered as susceptibility for asthma on the chromosome 7 region mapped earlier. In three cohorts from Finland and Canada, single nucleotide polymorphism-tagged haplotypes associated with high serum immunoglobulin E or asthma, implicating GPRA in the pathogenesis of atopy and asthma (Laitinen et al. Science 2004). Recently, a whole-genome association study identified the gene ORMDL3 as a promising candidate for asthma in Caucasian populations. The association in three genetically diverse populations provides strong support for its role in asthma susceptibility (Galanter et al. Am J Respir Crit Care Med 2008).

Environmental triggers in utero and early life: “the home front”

Most asthma has its origins in the early years of life. Professor John Warner explained why it is critical to understand the early life origins of asthma to identify targets for prevention and early intervention.

First of all, maternal genes appear to be more likely to influence the allergic status of the child in comparison with inherited allergy genes from the father. This suggests that maternal allergy in some way primes the foetus to be more likely to react in an allergic way. Normal pregnancy can only proceed if the maternal Th1 cell-mediated immune response to foetal paternal antigens is suppressed. A T cell balance in favor of Th2 and T regulatory cells (Tregs) is very likely during a normal pregnancy, suppressing Th1 responses. Indeed, Th2 cytokines such as IL4, IL13, and Treg-related cytokines IL10 and TGFβ can be detected in amniotic fluid during the second trimester of pregnancy, but not Th1 cytokines such as IL2, IL12, or IFNγ. IgE and maternal allergens can also be detected at levels of about 10% of that in the maternal circulation.

When a mother is allergic, she has a higher level of IgE in her circulation and in her amniotic fluid. Furthermore, the amniotic fluid of allergic mothers also contains higher levels of IL10 than does the fluid of non-allergic mothers. The timing and concentration of allergen exposure during pregnancy appears to be critical. Low-dose exposure in the second
trimester is likely to sensitize, while high-dose exposure has the opposite effect.

**Maternal nutrition has also been implicated** in protection against asthma development. Based on a Europe-wide study, reduced intake of anti-oxidants (fresh fruit and vegetables) was thought to be associated with a higher rate of allergic sensitisation. Current studies show that antioxidants might influence asthma severity rather than asthma prevalence. Lipids are considered important in the immune ontogeny. Fish oil supplementation in pregnancy affects the neonatal immune response. Whether this affects allergic sensitisation remains to be established. Maternal pregnancy fish intake 2–3 times a week or more is associated with reduced food sensitisation for the offspring (Calvari et al Ped Allergy Immunol 2006). Two fish oil supplement trials have shown some effects on early allergic manifestations (Peat et al JACI 2004).

**Several studies have shown** reduced prevalence of allergy and allergic diseases with the feeding of human milk in comparison to cow milk in infancy. Exclusive breastfeeding for at least four months after the child is born reduces the risk of eczema and perhaps asthma.

**The commitment towards an allergic response** might also be influenced by the use of antibiotics. There is an inverse relationship between the use of antibiotics during both pregnancy and infancy, and the prevalence of allergy. As antibiotics influence the maternal and the infant’s intestinal flora, interest in the use of probiotics increased. However, no effect could be shown of the use of single probiotics on allergy prevention. As probiotic bacteria might not achieve long-lasting colonisation, the use of pre-biotics might give beneficial results by modulating the diet and achieving permanent colonisation.

**Maternal smoking severely influences foetal health,** and results in an increase of recurrent wheeze and doctor-diagnosed asthma until the child is two years old. In addition, grand-maternal smoking certainly needs to be avoided.

**Prevention of allergy and asthma: conclusions**

- **Choose your maternal genes!**
- **No maternal or grand-maternal smoking**
- **Maternal good nutrition**
- **Maternal avoidance of antibiotics**
- **No allergen avoidance. High-dose exposure?**
- **Microbial “exposure”**
- **All strategies require further studies!**

Nicholas Van Bruaene

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**Asthma**

**Treatment of Pediatric Asthma**

*Asthma is the most common chronic childhood disease that occurs in most countries.* Early diagnosis, monitoring, and proper treatment of its symptoms are essential. Short-acting inhaled beta2 agonists are used widely as relief bronchodilator therapy, while and inhaled corticosteroids (ICS) are used as prevention therapy. New therapeutic strategies are suggested as being more effective in controlling children’s asthma.

**Professor Giovanni Rossi from Italy** highlighted the potential beneficial effects of long-acting inhaled beta2 agonists (LABA) in childhood asthma. Single doses of LABA can be used in bronchoprotection for exercise-induced bronchoconstriction (EIB), as it has been shown that they cause prolonged bronchodilatation (>12h) and extended bronchoprotection (Bisgaard 2000). The onset of action of formoterol is comparable to salbutamol or terbutaline, while salmeterol has a slower onset of action. Regarding the regular use of LABA for asthmatic children, Rossi concluded that despite some potential damaging effects including tachycardia and arrhythmia, tremor, hypocalaemia, and tolerance/tachyphylaxis, these treatments can be used as additional therapy for children older than five years and for patients whose asthma is not controlled on low to high doses of ICS (Akpinarli, Tuncer et al 1999). In most studies, LABA has been found to make significant improvements in lung function measurements (van der Woude, Winter et al 2001; Pohsun, Kuna et al 2006) and has a good safety profile, but not to make any significant reduction in the frequency of asthma exacerbations (Bisgaard 2003).

**The role of leukotrienes (LTRAs)** in pediatric asthma therapy was reviewed by Dr Antonio Nieto from Spain. Asthma phenotype in children, based on PRACTALL consensus, may be divided into the categories of virus-induced, exercise-induced, allergen-induced, and unresolved (Bacharier, Boner et al 2008). The effect of different therapeutic agents may vary as a result of the heterogeneous entity of asthma phenotype. The release of cysteinyl leukotrienes may be the most important cause of bronchoconstriction after inhaled environmental allergen in subjects with atopic asthma and EIB (Carraro, Corradi et al 2005).

**Several therapeutic options** are now available for treating childhood exercise-induced asthma.
although each has its limitations (Stelmach, Grzelewski et al 2008). A simple, long-acting regimen in the home is likely to lead to better compliance and to be more effective than short-acting regimens that require timely administration. Physical activities for children take place mainly at school, where they perhaps do not carry medication, or may feel it is inconvenient to use their inhalers in front of their peers. Inhaled short-acting and long-term beta2-agonists are effective prevention for EIB, although long-term use is associated with tolerance over time and life-treating asthma (Weinberger, Abu-Hasan 2006). 

Inhaled or oral corticosteroids may require combination therapy with other drugs for the control of EIB, while LTRAs can prevent EIB when given control of EIB, while LTRAs may require combination therapy with other drugs for the control of EIB, while LTRAs can prevent EIB when given long-term use is associated with tolerance over time and life-treating asthma (Weinberger, Abu-Hasan 2006). Inhaled short-acting and long-term beta2-agonists are effective prevention for EIB, although long-term use is associated with tolerance over time and life-treating asthma (Weinberger, Abu-Hasan 2006).

**Dr Mathias Kopp from Germany** outlined the possible role of anti-IgE (omalizumab) for pediatric asthma. Omalizumab is a recombinant humanized monoclonal antibody directed against immunoglobulin E to inhibit the immune system’s response to allergen exposure. It prevents free serum IgE from attaching to mast cells and other effector cells and prevents IgE-mediated inflammatory changes. It has already been demonstrated that omalizumab is an effective treatment for add-on therapy in adults and adolescents under 12 years of age with poorly controlled, moderate-to-severe allergic asthma and allergic rhinitis (Morjaria, Gnanakumaran et al 2007). This novel therapy seems to be beneficial in combination with specific immunotherapy for the treatment of allergic diseases (Parks, Casale 2006), offers improved efficacy, limited adverse effects, and potential immune-modifying effects. Preliminary studies demonstrated the efficacy of omalizumab in patients with atopic dermatitis, urticaria (Sheikh 2008), and food allergy.

In summary, asthma is a heterogeneous disease with different clinical phenotypes that influence the variability of patient responses to therapy. New therapeutic tools have been suggested but the most important consideration in asthma treatment is the therapeutic response of each patient, as asthma therapy should always be individualised.

**Maria Xatzipsalti**

**References**


Garcia Garcia ML, Wahn U et al. (2005) Montelukast, compared with flixotide, for control of asthma among 6 to 14-year-old patients with mild asthma: the MOSAIC study. Garcia, Garcia Wahn et al. (2005). Nieto concluded that LTRAs can be used as first line monotherapy for children with mild asthma, at ages under 10 years, in asthma induced by virus and exercise, and when there is strong suspicion of inadequate compliance with the inhalers.

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Regulation of the Allergic Immune Response

Central versus peripheral tolerance

The immune system is a highly specialised system, carefully balancing tolerance against self-antigens, and immunity against pathogens. Inappropriate responses may occur against harmless environmental antigens resulting in allergic diseases, and a failure in the recognition of auto antigens as self results in auto-immunity. No response to pathogens might result in immunodeficiency.

Central tolerance is the mechanism by which newly developing T cells and B cells are rendered non-reactive to self. The development and selection of T cells in the thymus biases the naive T cells against T cell reactivity. Central tolerance is distinct from peripheral tolerance in that it occurs while cells are still present in the primary lymphoid organs (thymus and bone marrow), prior to export into the periphery, while peripheral tolerance is generated after the cells reach the periphery. Mature T cells are subject to secondary selection (deletion, anergy) in lymphoid and non-lymphoid organs. These include the suppression of autoreactive cells by regulatory T cells (Tregs) and the generation of hyporesponsiveness (anergy) in lymphocytes which encounter antigen in the absence of the co-stimulatory signals that accompany inflammation. In addition, regulatory T cells can be considered both central tolerance and peripheral tolerance mechanisms, as they can be generated from self-reactive T cells in the thymus during T cell differentiation, but they exert their immune suppression in the periphery on other self-reactive T cells that escape selection.

Recent elucidation of the role of central tolerance in preventing organ-specific auto-immunity has changed the concepts of self/nonself discrimination. This paradigmatic shift is largely attributable to the discovery of promiscuous expression of tissue-restricted self-antigens (TRAs) by medullary thymic epithelial cells (mTECs). TRA expression in mTECs mirrors virtually all tissues in the body, irrespective of developmental or spatio-temporal expression patterns.1,2

Th1, Th2, Th17, Treg: why stop at four?

To function properly, the immune system must keep life going in harmony with the internal and external environment. It must recognise and neutralise danger (infections), tolerate “self” and “non-infectious non-self”, and must keep tissue integrity. The immune system is a highly interactive network, which makes its decisions on the basis of input from all organs and tissues, infections, normal flora bacteria, and many or even any environmental agents.

T cells play a central role in cell-mediated immunity. Different T cell subsets have been described, each with distinct functions. Initially, only two subsets of T effector or T helper (h) cells were described: Th1 cell types, important in the inflammatory delayed type hypersensitivity producing IFNγ, TNFα, and TNFβ; and Th2 cells characterised by IL3, 4, 5, 9, and 13 secretion. The existence of a dedicated population of suppressive T cells was the subject of controversy for many years. Recent advances in the characterisation of this T cell population, called regulatory T cells (Tregs), firmly established their existence and the critical role they play in the immune system. They are characterised by the expression of forkhead box p3 (FOXP3) in the CD4 and CD25 positive population. Tregs are a specialised subpopulation of T cells that actively suppress activation of the immune system and thereby maintain immune system homeostasis and tolerance to self-antigens.3

A subset of T cells producing interleukin 17 (Th17 cells) has been identified that is highly pro-inflammatory and could play an important role in immunity and disease. The role of TH17 cells in allergy is still largely unclear, but experimental models suggest that TH17 cells may be important for neutrophilic inflammation in acute airway inflammation. Many functions that were initially attributed to Th1 cells are shown to be part of Th17 responses.4

It is very likely that in addition to the currently identified T cell subsets (Th1, Th2, Tregs and Th17), other T cell subsets will emerge, characterised by the expression of dominant cytokines such as IL25, IL27, IL31, IL32, and IL35.

Nicholas Van Bruaene

References

Presented at the EAACI 2008 Congress in Barcelona, Spain.
Sport and allergic diseases

GA²LEN Olympic study

First pan-European study on allergy and asthma in athletes launched

GA²LEN centres will be following more than 2,000 athletes selected for the Olympic Games 2008 to assess the prevalence and diagnosis rates of asthma and allergies among top athletes in summer sports.

This study is the first pan-European study on allergy and asthma in athletes, designed as part of GA²LEN joint research activities on sports and allergic diseases.

It was initiated in Norway on request of the National Olympic Committee, to follow athletes and provide optimal care if needed. The scientists were also interested in learning more about the effect of air quality and pollution on the athletes.

Nine research centres will participate to the study in agreement with the National Olympic committees of each country. They represent the geographical areas of Europe: Denmark, Finland, Germany, Greece, Italy, Norway, Poland, Portugal and Spain.

The protocol of the study was designed by GA²LEN work package on Sport and Asthma (WP 2.8.2) and will be applied in the nine participating centres. This will allow scientists to collect comparable data on the degree of asthma and allergies in European athletes and to validate tools for further studies.

Scientists from Poland and Norway, including the lead researcher Kai-Hakon Carlsen, travelled to Beijing to set-up a respiratory laboratory in the Olympic Village and to provide care for athletes in need, in accordance to doping regulations. Clinical follow-up should also allow assessment of the impact of the local environment on potential symptoms.

In the long run, the study will contribute to a better understanding of exercise-induced asthma.

Objective & Expected Outcome

The scientists are looking to substantiate a number of information, including:

- Specify the prevalence of asthma, exercise induced asthma and other allergic diseases among European athletes qualified for the Beijing Olympics.
- Assess the impact of environmental pollution on asthma symptoms and lung function, identify athletes who may develop symptoms.

Next steps

- Early 2009: Expected results and publication
- 2010: Winter Olympics Vancouver for a follow-up study

Sport, asthma and allergic diseases

It is suspected that physical activity may trigger symptoms both in allergic athletes and in non-professional exercisers. In endurance sports, higher levels of asthma may be due to the prolonged periods with highly increased ventilation and the duration of high level physical activity performed. The highly increased ventilation of endurance top athletes is adequate and in relationship to the demands of their exercising body. This is different to the hyperventilation asthma patients can experience: an increase ventilation out of relationship to the demand.

Up to 20 percent of summer sports athletes have asthma. Endurance sports in particular such as runners, swimmers, and cyclists have been reported to have a higher prevalence.

A Backgrounder on the Olympic study is available on GA²LEN website. www.ga2len.net
Allergic rhinitis

NEW GUIDELINES FOR PRIMARY CARE PROFESSIONALS

Two practical publications to support GPs in diagnostics and assessment of allergic rhinitis

Allergic rhinitis is one of the most common chronic diseases with over 600 million people affected worldwide. More than 200 million of them also suffer from concomitant asthma. However, allergic rhinitis is generally under-diagnosed and under-treated.

Most patients who seek medical advice are seen first in primary care practices. GPs therefore have an essential role to play in the adequate diagnosis and treatment of allergic rhinitis.

Two complementary guidelines on the diagnosis and treatment of allergic rhinitis were published in the August issue of the Allergy journal. These guidelines are the result of a close cooperation between scientists, primary care professionals’ organisations IPCRG, WONCA and IPAG, and EFA, the European Federation of Allergy and Airway Diseases Patients Association. GA2LEN contributed to the guidelines representing European research in the field.

The guidelines review best practices worldwide and propose practical questionnaires for history taking, which is at the core of diagnosis, including advice on how to differentiate allergic rhinitis from other common diseases such as the common cold and non-allergic rhinitis.

Classifications will allow doctors to assess the severity of the disease and the impact on the patients’ quality of life. Primary care professionals will also find a list of ‘red flags’ suggesting that urgent referral is needed and a glossary of rhinitis medications.

These guidelines complement GA2LEN’s campaign Does rhinitis lead to asthma?, launched in 2007.

1. Primary care: the cornerstone of diagnosis of allergic rhinitis


Prof. Jean Bousquet, GA2LEN Vice-President and Chairman of ARIA, Allergic Rhinitis and Its Impact on Asthma, stresses that “neither allergic nor non-allergic rhinitis are trivial disorders. They significantly impair patients’ daily quality of life, school and work performance. Moreover, people with allergic rhinitis have a greater risk to develop asthma and many patients with rhinitis already have asthma as well. Although patients come with a complaint about their nose, asthma too must be checked by the doctor.”

GA2LEN, Global Allergy and Asthma European Network, is a Network of Excellence that brings together leading European research teams in the field of allergy and asthma. GA2LEN fosters a holistic, multidisciplinary approach to allergic diseases, including its genetic basis, clinical treatment, environment aspects and social causes throughout age groups.

www.ga2len.net
Today’s Allergies: from Acute Interventions to Chronic Management

According to GA²LEN, allergic diseases are the most common chronic diseases in Europe and their prevalence is growing, with an estimated 80 million Europeans experiencing some form of allergic disease. It is estimated that the total financial impact in Europe of allergic diseases is 100 billion every year and it has been suggested that workplace productivity losses due to allergic rhinitis are higher compared to other medical conditions like stress, diabetes, and coronary heart disease.

Clearly allergic diseases represent a serious challenge to patients and healthcare systems. The reach of this effect is likely to increase with continued global change in the forms of urbanization, industrialization, increased hygiene and global travel and trade. Yet despite these factors, the World Allergy Organization identified this summer that the number of healthcare professionals trained in the diagnosis and treatment of allergy has fallen. For this reason, among others, it is vital that conferences such as this June’s XXVII Congress of the European Academy of Allergology and Clinical Immunology (EAACI) continue to bring together healthcare professionals with an interest in allergic diseases to share best practices, forge research relationships and discuss current thinking and disease management issues relating to allergic diseases.

Not surprisingly, the first clinical plenary symposium of EAACI 2008 was entitled “Allergy, an Epidemic of the XXIst Century”. This session paved the way to topics related to treatments of today’s allergies, changes in today’s understanding of diagnosis and the need for monitoring of allergies, novel developments in the genetics of allergic diseases, novel approaches in immunotherapy, and the role of hygiene and infection in allergy. UCB was pleased yet again to be a founding sponsor of this congress and was delighted to support the symposium “Today’s allergies: from acute interventions to chronic management”. Chaired by Dr. Joaquim Mullol, Hospital Clinic Barcelona, Spain, this well-attended symposium was part of the main congress programme and provided some indications as to why allergy may be an epidemic of our time.

Alessandro Fiocchi MD from the Fatebenefratelli-Melloni University Hospital, Italy, delivered an enlightening presentation on poly- and cross-sensitization and suggested that nowadays, allergy experts have to be ‘detectives’. This is because reactions have become unpredictable, diagnosis more arduous and symptoms more severe and persistent due to factors such as the growing extent of cross-sensitization between new and classical allergens. These trends suggest that whilst allergen avoidance is an essential part of managing allergic diseases, it tends to be difficult, time consuming and expensive to implement and may not always lead to clinical improvement. On top of this, the World Allergy Organization, in its State of World Allergy Report 2008, has suggested that the increasing complexity of allergies results in patients frequently experiencing multiple allergic disorders. For example, a study of allergy patient group members found that asthma, eczema, food allergy and urticaria are concomitant conditions for, respectively, 43, 32, 29 and 19% of allergic rhinitis patients.

Professor Mullol’s presentation showed allergic rhinitis prevalence levels ranging from 17 – 29% of the population in European countries. Moreover, certain allergy patient groups have reported that allergic rhinitis is a long-term, and often persistent, disease, where symptoms tend to be moderate to severe and daily activities and sleep are often impacted.

Questioning how disease management should be approached, Joaquim Mullol suggested that continuous treatment, rather than on-demand, may offer patient benefits and underlined that QoL and cost savings should be also considered when choosing treatment options for persistent allergic rhinitis. Professor Todor Popov, Medical University Sofia, Bulgaria, highlighted that differences between anti-histamines reported in pharmacodynamic and pharmacokinetic models
are being increasingly con-
firm-in comparative head-
to-head clinical trials where
CIU patients report different
efficacy outcomes when treated
with different anti-histamines.9

The “Today’s allergies…” sym-
posium also discussed the need
for acute interventions in severe
allergic reactions, with Profes-
sor Pascal Demoly, University
of Montpellier, France, review-
ing data and issues relating
to anaphylaxis. Demoly was
keen to communicate that in
order to prevent anaphylaxis
and fatalities, firm diagnosis
is required and more doctors
should consider prescribing
self-administered epinephrine
auto-injectors. He outlined
that whilst anaphylaxis is a
rare but life-threatening event,
it is often under-recognized,
go undiagnosed and inci-
dence is increasing (perhaps
due to issues discussed by Ales-
sandro Fiocchi). For this rea-
son, Pascal Demoly suggested
that increased education about
anaphylaxis and epinephrine as
a first-line treatment option is
required amongst the public,
patients and healthcare profes-
sionals. He also outlined the
often biphasic nature of ana-
phylaxis and how these attacks
may necessitate the admin-
istration of a second dose of
epinephrine.

The UCB sponsored symposium
at EAACI 2008 brought together
renowned speakers in their field
of expertise to share knowledge
about current issues facing
our allergic patients and the
treatment options available to
doctors to manage these condi-
tions. To make this information
available to more physicians,
UCB has produced a webcast of
the symposium presentations,
which can be viewed at www.
aaci-ucb.com, and where
questions can be posted to each
of the presenters. The webcast
is free and will be available
until the end of October 2008.
We encourage you to visit the
site to consider how allergic
diseases are changing and why
this means that your patients
may require timely and long-
lasing medical support.
Join the 7th Symposium on Experimental Rhinology and Immunology of the Nose (SERIN)

Programme details and updates can be found on www.hdorl.net/serin2008