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Zahava Vadasz MD and Elias Toubi MD

Division of Allergy and Clinical Immunology, Bnai-Zion Medical Center affiliated with Rappaport Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel

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The International Congress on Autoimmunity, organized for the eighth time by Prof. Yehuda Shoenfeld, has become a tradition and is one of the most important scientific events in the field of autoimmunity worldwide. This time, the congress took place in the scenic city of Granada and was the most attended to date, with the participation of 2500 clinical immunologists, rheumatologists, scientists, and many clinicians in different fields, coming from 71 different countries. Eighty world-renowned companies supported this event by displaying their pharmaceutical and technical developments. The meeting was co-chaired by Prof. Ricard Cervera (Spain), Prof. Angela Tincani (Italy), and Prof. Carlos Vasconcelos (Portugal). At the opening ceremony, continuing the tradition of awarding a prize for lifetime achievement in autoimmunity, the three recipients this year were Dona Jackson Nakazawa (United States) for her contribution to increasing awareness of the role of the environment in the development of autoimmune diseases, Prof. Piere Youinou (France) for his contribution in the field of B cells and autoimmunity, and Prof. Allan Wiik (Denmark) for his contribution to the development of new and well-standardized diagnostic tools for autoimmune diseases. As in the past, new books were distributed: *Gender, Sex Hormones, Pregnancy and Autoimmunity* edited by Profs. Y. Shoenfeld, A. Tincani and M.E. Gershwin, and *The General Practice Guide to Autoimmune Diseases* edited by Profs. Y. Shoenfeld and. P.L. Meroni. During the four days of the meeting the participants enjoyed the extensive scientific program as well as the special atmosphere of Granada, infused by its native son, the poet Lorca, and the Alhambra.

More than 500 talks were given, covering a wide spectrum of issues [1-20] such as: the regulation of autoimmunity, the pathophysiology of systemic lupus erythematosus, scleroderma, systemic sclerosis and others. Organ-specific autoimmune diseases of the skin, intestinal tract and the eye were included in the program. Also discussed were novel therapies for autoimmune diseases and new diagnostic technologies for an earlier and better diagnosis of these diseases. One of the innovations at this meeting was screen presentations, enabling young participants to present their first posters on a screen and discuss them with clinicians and basic researchers. These sessions facilitated productive interaction between clinicians and basic immunologists and provided the opportunity for future international collaborations. Of the many important topics, a select few are summarized below.

CONTROLLING AUTOIMMUNITY

Effector CD4+ T lymphocytes are continuously induced in order to react against both non-self pathogens and self antigens. On the other hand, the induction of Foxp3+ regulatory T cells (Tregs) is crucial to prevent or limit inflammatory reactions. The functional failure of these regulatory mechanisms leads to the development of autoimmune and inflammatory disorders. Prof. A.K Abbas (USA) opened this meeting with a fascinating talk on the continuous balance between effector CD4+ T cells and Treg cells, discussing the many regulatory mechanisms that control the development of Treg cells in autoimmunity. In his laboratory, transgenic mouse models were developed in order to examine T cell responses to systemic and tissue-restricted self antigens and to explore how these responses are controlled. Exposure of naïve CD4 T cells to systemic or tissue antigens under various conditions leads to the development of pathogenic effector cells and inflammatory diseases. The disease resolves spontaneously in association with the generation and activation of Tregs. Tregs that encounter tissue antigen increase their suppressive activity, and a fraction of these Tregs persists as a memory population that continues to control subsequent inflammatory reactions in the tissue. Thus, Tregs undergo a sequence of processes: development in the thymus, activation in the periphery to perform their function and then survive as memory cells whose life history is fundamentally similar to that of all lymphocyte populations. The generation and maintenance of pathogenic effector T cells vs. protective Tregs is determined by the duration of antigen exposure and cytokines, especially interleukin-2 (IL-2), which, at low concentrations, preferentially expands and maintains Tregs. Elucidating the stimuli that generate and

maintain functional Tregs in the periphery will likely be valuable for manipulating immune responses in inflammatory diseases and for optimal vaccination and cancer immunotherapy.

Dr. A.S. De Groot (USA) presented another point of view on the fundamental role of Treg cells in preventing autoimmunity. "Tregitopes" are T cell epitopes naturally located in immunoglobulins that bind to multiple major histocompatibility complex class II alleles and induce regulatory Treg responses. Harnessing the tolerogenic effects of Tregitopes provides a novel tool to suppress unwanted immune responses and maintain antigen-specific tolerance, thus changing treatment paradigms in autoimmunity. Evidence was provided that antigen-presenting cells (APCs) present Tregitopes to natural (n) Treg cells, engage feedback mechanisms that promote a tolerogenic APC phenotype, reinforce Treg activation/expansion, and modulate antigen-specific effector T cell responses. Proportions of activated APC were suppressed, consistent with the previously reported beneficial effects of intravenous immunoglobulin (IVIG). Moreover, Dr. De Groot noted the significant increase in the proportion of IL-10-producing CD4+CD25+ FoxP3 expressing nTregs in the presence of Tregitopes. These studies are an exciting first step towards understanding the basic mechanism of Tregitope tolerance induction, and propose the following mechanisms: a) APCs present Tregitopes to nTregs; b) nTregs undergo activation and proliferation, producing increased amounts of IL-10; c) nTregs provide tolerogenic feedback signals to APCs, modulating the APC phenotype; and d) nTregs and tolerogenic APCs together suppress antigen-specific T cell responses. These data suggest a future role for Tregitopes in providing possible therapeutic applications.

A different controlling mechanism in the process of preventing autoimmunity was suggested by Prof. J. Weinstock (USA), involving helminthic infection in the gut. The exclusion of helminths from our environment strongly affects our host immune responses and may contribute to the emergence of immune mediated disease. Clinical trials suggest that helminths or their products have therapeutic potential in the management of autoimmune illnesses. Their mechanisms of action probably involve induction of several independent immune regulatory pathways. At least part of the protection depends on parasite induction of regulatory-type cytokines in the host. Interactions with cells of the innate immune system like dendritic cells and macrophages may be part of the regulatory process. Moreover, helminths like *Heligmosomoides bakeri* activate Foxp3+ T cells, making them highly regulatory, probably through induction of GATA-3. Identification of some helminths as potential commensals offers new options for therapeutics. Moreover, a better understanding of our natural relationship with the organisms around us could lead to the development of better immune modulatory vaccines that could help prevent the development of these diseases in susceptible people.

THE PATHOGENESIS OF AUTOIMMUNE DISEASES

The role of circadian and circannual rhythms in autoimmune diseases, mainly rheumatoid arthritis (RA), was discussed by Prof. M. Cutolo (Italy). He stated that early symptoms of arthritis either resolve spontaneously or progress into erosive disease. One predictive factor, according to a new multicenter study, is the season when the first symptoms emerge. So how does the earth's movement influence disease progression? Diurnal fluctuations in joint inflammation are familiar to patients with RA; the intensity of pain and stiffness varies consistently as a function of the hour of day and is greater upon waking in the morning. To explain such rhythms, the production of important circadian nocturnal hormones, such as the pro-inflammatory hormone melatonin and the anti-inflammatory adrenal hormone cortisol, has been implicated in the control of inflammation, at least in the context of RA. In particular, basal melatonin concentrations are higher, and the nocturnal rhythm shows peak levels earlier, higher and of longer duration in patients with RA as compared to healthy individuals. Accordingly, cytokines secreted by type I T helper cells such as IL-2, IL-6, IL-12, and tumor necrosis factor (TNF) reach peak production during the late night and early morning when melatonin serum levels are highest and plasma cortisol lowest. Thus, symptoms of RA might well worsen during the night and early morning. The influence of sunlight on the onset of RA is less defined than the effects of the circadian rhythm but is nevertheless supported by substantial clinical data. For example, in the northern hemisphere the onset of RA from October to March is as frequent as in other months. Recently, down-regulation of Toll-like receptor 4-mediated production of IL-6, interferons and TNF by peripheral blood mononuclear cells was observed in summer, as compared with winter, in healthy individuals. He concluded his talk by stating that the seasonality of first symptoms might therefore act as an independent predictive factor of radiographic outcome and disease severity. If replicated in other early arthritis cohorts, these results might offer additional ways of assessing which patients should receive intensive early therapy for RA.

Prof. Miri Blank (Israel) demonstrated the significance of antiglycan antibodies in the pathogenesis of antiphospholipid syndrome (APS). Glycans are predominant surface components of various cells. Researchers in her laboratory evaluated the presence of antiglycan antibodies (aGA) in patients with APS, and the association between aGA and clinical features of

the disease. Sera from patients with primary APS, with systemic lupus erythematosus (SLE) without APS, and healthy controls were analyzed retrospectively for levels of aGA by ELISA. Analysis of the association of aGA with clinical manifestations of APS was performed. Screening of sera from APS patients showed significantly high levels of antibodies against the following glycans: GlcNAc-β, GalNAc-β, GalNAc-α, Neu5Ac-α and Gal(β1,4) GlcNAc(β). There was an association between anti-GalNAc-β Abs and recurrent pregnancy loss. No specific association was found between any aGA and either thrombosis or central nervous system manifestations. In naive mice infused intravenously with anti-GalNAc-β the rate of fetal resorption increased. In conclusion, APS sera contain significant levels of aGA directed against several glycans. Anti-GalNAc-β Ab is specifically associated with recurrent pregnancy loss in both human patients and experimental animal models. The pathogenic effects of anti-GalNAc-β include amelioration of trophoblast cell invasiveness accompanied by decreased MMP2 and MMP9 secretion.

Pointing to new avenues in the study of the pathogenesis of SLE, Dr. Z. Vadasz (Israel) reported on the association between semaphorin 3A (sema3A) and SLE disease activity. In her recent study she found serum sem3A to be inversely correlated with SLE disease activity. She also demonstrated that sema3A has regulatory properties, inducing altered TLR9 expression on ODN-stimulated B cells from SLE patients. This suggests that sema3A may become an immunomodulatory agent in treating SLE.

THE ROLE OF B CELLS IN AUTOIMMUNE DISEASES

The importance of B cells in autoimmunity was discussed by many meeting participants, pointing to the fact that B cells being antigen-presenting cells and also pro-inflammatory producers contribute to the development of autoimmunity. Prof. P. Youinou (France), a leading researcher in the field of B cells, discussed the role of B cell-activating factor (BAFF) in B cell homeostasis. By rescuing autoreactive B cells, excessive BAFF favors the development of autoimmune diseases. Given the numbers of variants of this B cell-specific cytokine, caution must be exercised when determining its serum level. Alternate splice isoforms, such as Δ 3 BAFF and Δ 4 BAFF (BAFF fractions), have been identified, raising the possibility that their overproduction impacts the synthesis of full-length BAFF. In line with the above, Dr. G.J. Tobon discussed the mechanisms of induction and regulation of Δ 4 BAFF. Incubation of cell lines with IFNγ showed induction of Δ 4 BAFF transcripts. This induction increased the expression of membrane-bound and soluble forms of BAFF, thereby contributing to autoimmunity. Aiming to better understand CD5+ B cell functions, Dr. J.O. Pers (France) revealed that CD5 promotes constitutive activation of multiple signaling pathways including extracellular signal-regulated kinases (ERK1/2), phosphatidylinositol 3-kinase (PI-3K), mammalian target of rapamycin (mTOR) and calcineurin-NFAT signaling pathways. These data provide a framework for understanding how CD5 impacts B cell biology and responses. Aspects of B regulatory cells were also discussed in this session. Prof. E. Toubi (Israel) presented data showing that Breg cells can be identified as CD25high CD1d high and IL-10 high. These cells were shown to inhibit the proliferation of CD4 T cells and increase Treg properties (increasing Foxp3+ and CTLA +) following the co-culture of Bregs with autologous Treg cells. In seeking new identifying markers for human Breg cells it was found that semaphorin 3A is a good marker for this subset of cells. Studying the role of B cells of salivary glands in Sjogren's syndrome, Dr. J.O. Pers observed that direct human salivary gland B cell contacts were able to induce apoptosis in epithelial cells. This B lymphocyte-mediated cell death could not be ascribed to Fas-Fas ligand interactions but required translocation of PKC-δ into the nucleus of epithelial cells. Ultimately, activation of PKC-δ resulted in histone H2B phosphorylation on serine 14 and poly (ADP-ribose) polymerase cleavage.

INFECTION/VACCINATION AND AUTOIMMUNITY

This controversial topic was presented by Prof. Y. Shoenfeld from Israel. He summarized the data on the role of infections such as cytomegalovirus, parvoviruses, hepatitis C, and others in preceding or even inducing many autoimmune diseases. He also related to the possible role of other infections such as helmentics and *Helicobacter pylori* in protecting or preventing the development of autoimmune diseases. However, less than viral infections, and quite rarely, the use of silicone and many adjuvanted vaccines in medicine can also induce the development of autoimmune diseases, mainly in susceptible individuals, a phenomenon that he terms autoimmune/inflammatory syndrome induced by adjuvants (ASIA). A wide spectrum of clinical symptoms – musculoskeletal, fatigue, neurological – and the development of elevated titers of autoantibodies are proposed to become the criteria for ASIA and are useful for raising the awareness of physicians who follow patients with similar post-vaccination events. In his talk and later in several others, aluminum was suggested to be the most common adjuvant associated in many studies with the development of autoimmunity. Therefore, the question of using safer adjuvants such as the newly introduced TLR-ligands is crucial. Of interest was his com-

ment that narcolepsy was recently recognized as possibly autoimmune in origin, suggested by many clinical case reports and even animal models in which adjuvanted vaccines preceded the development of narcolepsy. On the same subject, the neurotoxicity of aluminum was further discussed by Prof. C.A. Shaw (Canada). He stated that aluminum is well recognized as a neurotoxin, both in experimental models and human neurological diseases. Humans are exposed to aluminum from various sources: food, water, and medicinals being the major contributors to exposure. Increasing attention is now being directed to the impact of aluminum used as an adjuvant in the numerous vaccines for various diseases. The causal role of aluminum in the development of autoimmunity should be established. In line with the above was the contribution by Dr. L. Tomljenovic from Canada, who further discussed the question of adjuvants such as mercury and aluminum, both of which are designed to hyperstimulate the immune system, hence their ability to induce both autoimmunity and neuroimmune disorders.

Autoimmunity following hepatitis B vaccine as part of the spectrum of ASIA was documented in 93 cases and discussed by Drs. Y. Zafrir and N. Agmon-Levin from Israel. They presented data showing that the mean latency period from the last dose of hepatitis B virus (HBV) vaccine and onset of symptoms was 43.2 days. Commonly reported manifestations include neuropsychiatric, musculoskeletal and fatigue. Interestingly, all the above speakers stated that vaccination is still safe for the vast majority of vaccines but that we need to be alert to the appearance of symptoms included in ASIA. Among the several talks on vaccination followed by autoimmunity, Dr. N. Agmon-Levin (Israel) pointed to the increasing awareness of ASIA and the need to use these criteria for accurate diagnosis in future case reports describing the wide spectrum of this syndrome

NOVEL THERAPIES IN AUTOIMMUNE DISEASES

One of the main topics at this conference was the array of novel treatments for autoimmune diseases. Prof. David D'cruz (London, UK) began his review by mentioning standard protocols for SLE treatment. These protocols include treatments for various stages of the disease: antimalarial therapies for the early stages, steroids, and later the addition of cytotoxic drugs for advanced stages and relapses. Prof. D'cruz also described the new biologic drugs that were developed based on understanding of the disease's pathophysiologic mechanisms. Belimumab, already approved by the U.S. Food & Drug Administration and in many other countries, is one of the newest drugs for SLE. Several other new biological drugs are now undergoing clinical trials; they include CTLA-4 molecules (abatacept), anti-CD11 (efalizumab) and two new human anti-interferon-alpha drugs.

Prof. Chaim Putterman (New York) talked about the new TWEAK/Fn14 pathway blockade that attenuated renal disease in a mouse model of autoantibody-induced nephritis. TWEAK belongs to the TNF superfamily members that are instrumental in the pathogenesis of lupus nephritis. TWEAK-mediates activation through its receptor Fn14, which stimulates the secretion of MCP-1, RANTES, IP-10 and KC by mesangial cells and podocytes and also modulates renal cell survival and proliferation. Nephrotoxic serum nephritis, a model for lupus nephritis, was used to study the role of the TWEAK/Fn14 pathway in the pathogenesis of renal disease induced by pathogenic antibodies. To confirm the protective effect of TWEAK inhibition with a pharmacological approach, researchers at his center induced nephrotoxic nephritis in 129 Fn14 WT mice and initiated treatment with an anti-TWEAK mAb or control immunoglobulin. Similar to results in Fn14KO mice, significant amelioration of proteinuria and improvement in renal histology were observed in mice treated with anti-TWEAK mAb. Anti-TWEAK mAb treatment did not appear to affect the systemic immune response since no alteration in murine anti-rabbit IgG subclass antibody titers was evident. This may enable a new strategy for lupus nephritis treatment.

Dr. A. Kazav (Israel) demonstrated the efficacy of IVIG in experimental autoimmune neuritis, an animal model for acute inflammatory demylinating neuropathy, similar to the human Gullain-Barre Syndrome (GBS). The researchers used several doses of IVIG and IVIG mimetics compared to vehicle treatment. They demonstrated efficacy of treatment with 40 mg/kg using the motor performance test performed on a Rotarod. The present findings support the potential use of recombinant mimetics of IVIG in diseases such as GBS. The study may thus serve as the basis for a clinical trial in humans with GBS.

Dr. G. Zandman-Goddard (Israel) summarized the B cell depletion strategy for APS and catastrophic APS. The data on this treatment in the literature are limited. Rituximab appeared to have a beneficial clinical effect in 24 case reports of treated patients. In the majority of the cases, lupus anticoagulant, anticardiolipin antibodies and β2GPI antibody levels either decreased or normalized after treatment with rituximab. Improvement in hematological, neurological, renal, pulmonary, gastrointestinal, and cutaneous manifestations was also noted. In addition, rituximab seemed to have a beneficial therapeutic effect in six of seven case reports of patients with catastrophic APS.

IVIG IN AUTOIMMUNE DISEASES

Prof. S. Kaveri from France gave a talk on the history of IVIG and its increasing use in many autoimmune diseases, becoming a well-established mode of therapy. He discussed the many new mechanisms by which IVIG immuno-

modulates inflammation and autoimmunity and why IVIG is considered unique and continues to attract the attention of rheumatologists, autoimmunologists, neurologists and others. IVIG exerts beneficial effects in autoimmune and inflammatory diseases via several mutually non-exclusive mechanisms.

While IVIG can directly modulate the functions of both innate and adaptive immune cells such as dendritic cells (DCs), macrophages, B and T cells, several reports have also stressed that the regulation of immune responses by IVIG can be indirect. Indirect regulation of immune cells by IVIG-educated DCs was assessed. Whereas human B cells are resistant to IVIG-educated DCs, IVIG at therapeutic concentrations can directly inhibit B cell activation and proliferation.

The immunomodulatory properties of IVIG were further discussed by Prof. A.G. Tzioufas from Greece. He referred to a recent study suggesting that an active idiotypic-anti-idiotypic network (id/anti-id) operates and regulates the production of autoantibodies. In neonatal lupus, mothers with high anti-idiotypic antibody activity against anti-La autoantibodies are at lower risk of giving birth to a diseased child, as compared to mothers without anti-idiotypic antibodies. These findings suggest that anti-idiotypic antibodies may confer protection from the harmful effect of pathogenic autoantibodies in neonatal lupus. He stated that following IVIG treatment, serum titers of anti-La decreased in 80% of the mothers, while in 60% the anti-idiotypic antibodies were increased. The Id/anti-Id ratio was significantly higher in mothers whose offspring developed neonatal lupus compared to mothers who gave birth to a healthy child ($P < 0.0001$). Thus, a high Id/anti-Id ratio in both the IVIG preparation and the maternal serum may explain the lack of an effect of IVIG in preventing recurrent neonatal lupus in some cases. An elegant study was presented by Prof. B. Mazer (Canada), in which IVIG was shown to inhibit airway inflammation by bridging innate and adaptive immunity to induce regulatory T cells. In this study, C57Bl/6 mice were sensitized and challenged by intranasal OVA exposure. IVIG or albumin control was administered 24 hours prior to the challenge. Treg cells were tracked using reporter mice expressing Foxp3, and Treg and dendritic cell phenotype and activity were elucidated by co-culture and flow cytometry. IVIG treatment of OVA-sensitized and challenged mice induced antigen-specific Foxp3+ Treg cells that home to lungs and draining lymph nodes, and are highly suppressive compared to Treg purified from controls. Induction of Treg cells is mediated by tolerogenic DC generated following IVIG exposure. Thus, the anti-inflammatory effects of IVIG therapy may be mediated by the immunomodulation of DC, creating a bridge that induces antigen-specific highly suppressive regulatory T cells. Another elegant mechanism by which IVIG is beneficial in immunomodulating autoimmunity was presented by Dr. S. Hillion from France. The study was designed following the notion that IVIG may render autoreactive B cells tolerant through induction of a functionally unresponsiveness to B cell receptor (BCR) stimulation. Tonsil mononuclear cell suspensions were enriched in B cells. B cells were stimulated using anti-IgM in the presence of different fractions of IVIG. Responses of BCR stimulation were analyzed using confocal microscopy, western blot analysis, and flow cytometry. IVIG prevented the in vitro activation of B cells and induced some features characteristic of functional silencing. Upon stimulation it was demonstrated that IVIG down-regulated BCR-induced early phosphorylation, leading to the suppression of PI3K signaling. It was also confirmed that ligation of BCR in the presence of IVIG led to a failure of calcium mobilization, culminating in an aberrant nuclear factor-kB translocation into the nucleus. Taken together, these data suggest that IVIG could induce B cells to enter a state of functional silencing and anergy. This holds promise for a new therapeutic avenue in B cell-related autoimmune diseases.

RESISTANCE TO TRADITIONAL THERAPIES IN AUTOIMMUNE DISEASES

The introduction of new biological therapies and off-label drugs raised the need to define resistance to traditional therapies and when to define an autoimmune disease as refractory. Defining the issue of refractory disease in autoimmune diseases, Prof. C. Vasconcelos (Portugal) stated that this process should be regarded as dynamic, according to the availability of new drugs. It should be differentiated from severe disease and damage definitions and it must take into account duration of adequate therapy and patient compliance. It can be related to inadequate or inefficacious treatment or to pathogenesis. Refractory disease definition has multiple implications for clinical guidelines and for the use of off-label or new biological drugs.

On the same topic, Prof. E. Pras (Israel) pointed to the fact that about 5% of patients with familial Mediterranean fever will not respond to colchicines. He found that genotyping enables us to study the response rates according to specific mutations. In this case, a large number of M694V homozygotes did not respond well to colchicines despite treatment with maximal sustained doses. He summarized his talk by stating that the prevalence of complete responders among M694V homozygotes is much lower than previously appreciated.

NOVEL DIAGNOSTIC APPROACHES IN AUTOIMMUNE DISEASES

Many diagnostic autoantibodies and cellular subtypes are assessed today with automated technologies. Many of these procedures require careful validation, standardization, and well-established performance. At this meet-

ing many presentations introduced some of these innovations with the aim of encouraging their wider utilization. One of these presentations was by Dr. S.S. Copple (USA) who compared the performance of the NOVA View automated immunofluorescent (IFA) microscope to that of manual viewing of ANA HEP-2 slides by an experienced certified medical technologist. Overall agreement of 98% was demonstrated comparing the NOVA View titers with those read by the medical technologist on a fluorescent microscope. Negative sera yielded 100% and positive sera 97% agreement.

In another talk, Dr. T. Matthias (Germany) discussed the development and validation of an IFA processor with an integrated optical system for automated IFA slide processing and reading to perform a positive/negative sample discrimination. The development resulted in an IFA processing and evaluating system featuring an integrated optical system suitable to fully automate the IFA process. The validation of the system with over 1000 serum samples showed a 98.4% correlation of results (positivity/negativity determination) when compared to the manual IFA method with visual interpretation. Furthermore, the system reduces process time significantly and provides more consistent results for the clinician. The Bioplex 2200 platform was challenged against a previous algorithm of both ELISA and IFA systems. In order to do so, Dr. M. Barak (Israel) tested 158 sera samples from well-defined patients suffering from autoimmune disease and found that the BioPlex results are highly concordant with the ELISA-based algorithm. Its use will provide more information and save technician time. In summary, new automated techniques are gradually replacing many of the old traditional and time-consuming methods.

The Eighth International Congress on Autoimmunity closed with a farewell dinner, at which participants expressed that this was one of the best meetings they had attended. The preparations for the ninth congress to take place in Nice, France, have already begun and participants are enthusiastic for the work ahead to ensure exciting and challenging results and present good science two years hence.

Corresponding author:
Dr. E. Toubi
Division of Allergy and Clinical Immunology, Bnai-Zion Medical Center, Haifa 31048, Israel
email: elias.toubi@b-zion.org.il

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Erratum

In the article "Inducible clindamycin resistance among methicillin-sensitive *Staphylococcus aureus* infections in pediatric patients," which appeared in the 2011 October issue (volume 13, number 10, pages 605-8), one of the author's names was spelt incorrectly. It should be Bilavsky, and not Bilvasky as printed.

We are not here to see through each other but to see each other through

Anonymous