RESOURCES
The International Psoriasis Council is pleased to bring you the following educational opportunities to advance your knowledge of treating patients with psoriasis.

UPCOMING IPC EVENTS
May 1, 2014
IPC Meet the Experts, Santiago, Chile, 32nd Annual Meeting of Latin American Dermatologists (RADLA)

August 15, 2014
IPC Meet the Experts, Vancouver, Canada, 68th Meeting of the Pacific Dermatology Association

September 10 – 13, 2014
IPC Scientific Symposium, Copenhagen, Denmark, 44th European Society for Dermatological Research

September 25 – 27, 2014
IPC Meet the Experts, Montevideo, Uruguay, 16th Meeting of the Uruguay Society of Clinical and Surgical Dermatology

October 10, 2014
IPC Meet the Experts, Amsterdam, Netherlands, 23rd Congress of the European Academy of Dermatology & Venereology

For more information, please visit our website at www.psoriasiscouncil.org

Included in this Issue
P1 A Letter from the President
P4 Top 5 Clinical Papers Review
P9 Focus on Psoriasis: A Report from the 22nd Congress of the European Academy of Dermatology & Venereology, Istanbul, Turkey
P21 Meet the Experts goes to Istanbul
P24 IPC News

A Letter from the President
Dear Friends,

It is with a mixture of sadness and pride that I put pen to paper for this, my final time as IPC’s president. The occasion has afforded me the opportunity to reflect upon my three-year tenure. I believe we have accomplished much in that time, while overcoming periods of adversity.

I am proud that we have established a strategic agenda for IPC and, indeed, progressed toward accomplishing its objectives. I recall with fondness the vibrant discussions we dermatologists had with some of the world’s leading geneticists in Montreal in 2011 to get them to focus on psoriasis. Today, in collaboration with geneticists in the U.S., Germany and UK, we have successfully typed more than 20,000 samples, and are now embarking on a bioinformatics phase with the potential to deliver profound data on the inherited susceptibility to the condition.

IPC is a pioneer in associating psoriasis with various comorbidities. Our journey began with a multidisciplinary meeting in Rhodes in 2006, followed by a second interaction in Dallas in 2008. These meetings ultimately resulted in IPC initiating and completing a 600-patient trial investigating the relationship between body mass index and psoriasis in the pediatric population. Led by Professor Amy Paller at Northwestern University Feinberg School of Medicine, the study has now been published in the Archives of Dermatology, and it extends our understanding of psoriasis as it relates to various comorbidities. Indeed, in November, at IPC’s annual Think Tank, we again addressed the theme in a stimulating symposium that explored the interrelationship among genetics, inflammation, stress, psychology, psoriasis and various comorbid outcomes. Thus, we continue advancing toward our aspiration of evaluating the psoriasis patient from a holistic health-and-well-being perspective.

Cont., Page 2
A LETTER FROM THE PRESIDENT

Together with our late CEO, Karen Rodman, the board and staff changed IPC from a dedicated task group to a professional organization. During my term as president, we have worked on a structure that allows our 83 councilors to provide major input into IPC policies and projects. We have started the yearly Think Tank meetings in London, Amsterdam and Boston. The active discussions on the ICD11 proposal and the recent e-mail storm regarding the scoring system as proposed by Robert Chalmers are examples of how we, together, are passionate, realistic, and effective. At the last Think Tank meeting in Boston, I was delighted with the active role of the councilors in the organization, with constructive discussions on our strategic plan and a meeting with international experts presenting on the association of comorbidities in psoriasis. Also, the installation of working groups of councilors and psoriasis experts on topical and systemic treatments, juvenile psoriasis, health economics, and internationalization has become an important feature of our organization.

During my time as president, IPC overcame significant challenges. The loss of CEO Karen Baxter Rodman on Oct. 1, 2012, affected all of us in the organization both personally and professionally. Karen was a revered colleague, a leader and a personal friend. She was beginning to have a profound impact on IPC’s global leadership and the resources we access to support our goals. We miss a great leader and close friend. We are so grateful to you, my dearest Karen.

Today, I can say that we have weathered this difficult time and are beginning to blossom with renewed enthusiasm to reach even higher. Interim CEO Christy Langan, together with scientific director Paul Tebbey, have made the impossible possible. We realized our targets with respect to our Meet the Expert symposia worldwide, our research projects, and projects with major organizations, including International League of Dermatological Societies (ILDS), International Federation of Psoriasis Associations (IFPA), European Dermatology Forum (EDF), and European Academy of Dermatology and Venereology (EADV).

Our founding president, Dr. Alan Menter of Dallas, Texas, ended his three-year term as past president. We owe Alan the great friendship that characterizes IPC. We share a mutual passion: enhancing care by advancing knowledge. We have professionalized IPC, but we will always remember our early days. Alan will remain as emeritus founding president.

As is customary, I am proud, for the last time, to introduce the latest issue of the Psoriasis Review and its co-editors. For this issue, the co-editors are Dr. Francisco Kerdel of the University of Miami Hospital, Miami, Florida, USA, and Dr. Robert Sabat of the University Hospital Charité, Berlin, Germany. In this issue, we have included a review of the advances discussed at the 22nd EADV Congress that took place in Istanbul, Turkey, in October. We also present a summary of IPC’s Oct. 5 Meet the Experts program, which I was honored to lead in association with my colleagues and fellow IPC councilors Drs. Kristian Reich, Elke de Jong, and Anthony Ormerod.

For each issue of IPC’s Psoriasis Review, IPC councilors enjoy nominating and selecting the top five, semi-annual clinical and research articles. In this issue, we present articles selected from the first half of 2013. To qualify, manuscripts had to be published either in print or electronically (epub) between January 1 and June 30, 2013. This issue’s selections reflect the scope of IPC’s strategic agenda. Two of the selected manuscripts further our understanding of the genetic basis of psoriasis as it relates to the resultant...
A LETTER FROM THE PRESIDENT

On that note, I am pleased to introduce you to my friend, colleague and fellow IPC board member, Professor Christopher Griffiths, who will take the reins in 2014 as our next president. Professor Alexa Kimball will serve as president-elect.

Chris is foundation professor of dermatology at the University of Manchester, UK, and director of the Manchester Academic Health Science Centre. Among his many accomplishments, Chris developed the hub-and-spoke model of dermatology services for Greater Manchester and introduced a multidisciplinary clinic for severe psoriasis, for which he received numerous awards. You will find out more about Chris in our next (July 2014) issue of Psoriasis Review. I have full confidence that Chris will bring the IPC to a new level in many respects. I know that, under his leadership, IPC will continue to follow an aggressive agenda that will continue to make significant impacts on psoriasis care. I wish him the best in this new role.

Finally, to my fellow councilors, colleagues and friends within the IPC family, I say that it has been an honor to serve and now I wish you “tot ziens en veel geluk!”

Sincerely,

Professor Peter van de Kerkhof, MD, PhD
President, International Psoriasis Council

For additional copies of IPC’s Psoriasis Review Newsletter, or to learn more about IPC, please visit www.psoriasiscouncil.org.
1. Lower risk of undesirable cardiovascular outcomes in severe psoriasis patients treated with TNF-α inhibitors or methotrexate: a nationwide study


Summary
It has been known for several years that psoriasis is associated with an increased risk of cardiovascular morbidity and mortality. This might be due in part to the mutual amplification of the immune-inflammatory processes underlying psoriasis on the one hand and atherosclerosis, insulin resistance, endothelial dysfunction and thrombus formation on the other. Thus, anti-inflammatory treatment of psoriasis could positively influence the course of cardiovascular diseases. This study evaluated rates of cardiovascular disease events in patients with severe psoriasis treated with biologic agents, methotrexate, or other therapies, including retinoids, cyclosporine and phototherapy. The researchers used individual-level linkage of nationwide Danish databases from 2007 to 2009 and performed a retrospective, longitudinal cohort study totaling 2,400 patients with severe psoriasis. Of those, 693 patients were treated with biologic agents. The majority (>80%) received TNF-α inhibitors, and no events were related to use of interleukin (IL)-12/IL-23 inhibitors. Patients treated with biological agents were younger and included a higher percentage of men compared with those treated with non-biological therapies. The mean follow-up of this study was approximately 18 months. The main outcome measure consisted of a composite of death, myocardial infarction, and stroke. The results displayed an incidence rate per 1,000 patient years of 6.0 (95% CI 2.7–13.4) for those treated with biologic therapies, 17.3 (95% CI 12.3–24.3) for those treated with methotrexate, and 44.5 (95% CI 34.6–57.0) for patients treated with other therapies. Age- and sex-adjusted hazard ratios (HRs) followed a similar pattern, wherein biological agents and methotrexate reduced the risk of hazard with ratios of 0.28 (95% CI 0.12–0.64) and 0.65 (95% CI 0.42–1.00), respectively, relative to patients using other therapies as the reference cohort. Thus, in this nationwide study of patients with severe psoriasis, treatment with biologics (mostly TNF-α inhibitors) or methotrexate was associated with lower cardiovascular disease event rate compared to patients treated with other systemic therapies.

COMMENTARY The data presented in this study deliver “real-world” experience and impact from the use of systemic therapies in psoriasis in a population contained within the Danish Civil Registration System. The conclusion that TNF-α inhibitors and methotrexate are beneficial to patients in terms of risk of cardiovascular endpoints such as death, myocardial infarction and stroke are consistent with results that we highlighted in the June 2013 issue of IPC’s Psoriasis Review (“TNF-inhibitor therapy statistically reduces the risk for myocardial infarction in patients with psoriasis,” Volume 9, Number 1, Page 5). These data support the early use of TNF-α inhibitors in patients with severe psoriasis and at high risk of cardiovascular mortality. However, additional research and further real-world registry data remain necessary to determine which treatment option has the biggest positive impact on the cardiovascular comorbidities.
2. Streptococcal antigens might be directly involved in the pathological mechanisms that result in the appearance of psoriasis lesions


**Summary**

To elucidate how psoriatic lesions are initiated and triggered, Ferran et al studied cultures containing purified blood memory T cells expressing cutaneous lymphocyte–associated antigen (CLA) and autologous epidermal cells. An important technical point of the experiments was the purification of the CLA+ memory T cells, which was achieved via a series of negative and positive selection methods designed to deplete unwanted cell types and to enrich for the desired T cells. The purity of the cell types was assessed by flow cytometric analysis and was found to be above 95%. In the initial experiments, the addition of streptococcal extracts (SEs) to the co-cultures led to activation of the cells and to the production of Th1, Th17, and Th22 cytokines, as measured via real-time PCR. Additionally, epidermal cell mediators (CXCL8, CXCL9, CXCL10, and CXCL11) were documented in culture supernatants as measured by enzyme-linked immunosorbent assay (ELISA). Importantly, the CLA+ memory T cells were also activated when they were cultured with non-lesional autologous epidermal cells and SEs. The occurrence of activation was independent of HLA-Cw*0602 positivity of the donors. SEs did not induce any activation of CLA- memory T cells cultured together with autologous epidermal cells. Interestingly, in cultures containing CLA+ memory T cells, epidermal cells from healthy subjects, and SEs no activation was observed either. The supernatants of CLA+ memory T cells cultured with lesional autologous epidermal cells and SEs were found to be sufficient to reproduce the characteristics of psoriasis when injected into mice, as measured by histological evaluation and skin thickness. Moreover, the ex vivo, SEs-induced activation of CLA+ memory T cells correlated with the serum level of anti-streptolysin O antibodies in psoriasis patients. Steptolysin O is a streptococcus-derived hemolytic exotoxin that is indicative of bacterial infection. Taken together, the results demonstrate that streptococcal antigens can be directly involved in the pathological mechanisms that result in psoriasis lesions.

**COMMENTARY** This work extends previous observations related to the initiation of psoriasis and the potential involvement of infectious entities, particularly streptococcus. In fact, a previous study identified a correlation between streptococcal infection and psoriasis, and demonstrated that chronic plaque psoriasis can be exacerbated after streptococcal throat infections using clinical and epidemiological data. Additionally, T cells were found in the blood of psoriatic patients that recognized streptococcal M-proteins and potential auto-antigens like cytokeratin 17. IPC reported a related study in the December 2012 issue of Psoriasis Review ("Clinical support for infectious molecular mimicry as a pathogenic mechanism for the initiation and propagation of psoriasis," Volume 8, Number 2, Page 5). Experiments in that study assessed the association of tonsillectomy with both a reduced frequency of circulating specific T cells that recognized cytokeratin 17 as well as streptococcal M protein and disease improvement in the range of 30 to 90%. However, the current study by Ferran et al demonstrates direct and specific interaction of circulating CLA+ memory T cells, epidermal cells, and streptococcus antigens. One of the big questions arising now is why the interaction is specific for keratinocytes from patients with psoriasis.
3. Distinguishing the genetic fingerprint of two subtypes of generalized pustular psoriasis (GPP)


Summary
Generalized pustular psoriasis (GPP) is a rare, inflammatory, potentially life-threatening skin disease. The characteristic skin manifestations of GPP are episodic sterile pustules on an erythematous background over wide areas of the body, GPP often presents in patients with existing or prior psoriasis vulgaris (PV). Accordingly, the authors subdivided 31 Japanese patients suffering from GPP into two groups: those who did not have PV (GPP-only; n=11) and those with PV (GPP with PV; 20) and searched for mutations in the gene (IL-36RN) encoding interleukin-36 receptor antagonist (IL-36RA). The analysis was performed via PCR amplification of DNA isolated from peripheral blood cells using oligonucleotide primers that were designed to capture exon/intron boundaries. DNA from normal, non-psoriatic Japanese individuals was examined as a control. The results showed that 5 out of 11 GPP-only patients but none in the GPP–with-PV group displayed homozygous mutations in IL-36RN. An additional 4 patients exhibited only GPP and only 3 patients in the GPP-with-PV group displayed compound heterozygous mutations. Thus, there was a significant difference in the frequencies of IL-36RN mutations between GPP-only versus GPP with PV: 9 out of 11 vs. 3 out of 20 (p<0.01). Consequently, epidermal expression of IL-36RA was minimal in GPP-only patients as measured by immunohistochemical staining of skin lesions with anti-IL-36RA antibody. In contrast, strong IL-36RA expression was seen in the upper spinous and granular layers of the epidermis of a patient with PV as the positive control. Interestingly, patients suffering from GPP with PV showed a positive IL-36RA staining in the granular layers of the epidermis. Thus, the data support the notion that GPP with mutations within IL-36RN is etiologically distinguished from the GPP that is typically observed with PV.

COMMENTARY
Much progress has been made recently with the elucidation of the genetic basis of GPP. In the June 2012 issue of IPC’s Psoriasis Review of Top 5 papers, we illuminated the work of Marrakchi et al (“Interleukin-36-Receptor Antagonist Deficiency and Generalized Pustular Psoriasis”), who reported a homozygous missense mutation based upon a leucine-to-proline amino acid transition in nine Tunisian families. In the current study, Sugiura and colleagues demonstrated that mutations in the IL-36RA-encoding gene (IL-36RN) are characteristic for only a subtype of Japanese GPP patients. This is important because it gives us a measurable marker to subdivide GPP in two subtypes: the “genuine” GPP and the severe PV with pustular eruptions. Given the capacity to definitively diagnose true GPP via mutational analysis in IL-36RN, there is an expectation for new therapy developments that will address this pathway and resolve the condition.
4. Effects of a specific single-nucleotide polymorphism in the IL-23 receptor-encoding gene: significance for psoriasis


Summary
The inherited basis of psoriasis has received much attention in recent years, and numerous susceptibility genes have been identified, more than 30 genes belonging to the IL-23/Th17 axis, the NF-kB pathway, and the epidermal differentiation complex. But are these genetic linkages explained in terms of psoriasis phenotype? In this article, Di Meglio et al dissect the effect of a single-nucleotide polymorphism (SNP) in the IL-23 receptor-encoding gene. In the respective DNA position, the guanine nucleotide (G) frequently appears that leads to arginine (Arg) at position 381 of IL-23R. The other variant of this SNP is the rarer adenine nucleotide (A) that results in glutamine (Gln) amino acid substitution. The IL-23R Arg381Gln substitution protected against several immune-mediated inflammatory diseases, such as psoriasis, Crohn’s disease, and ankylosing spondylitis. However, the authors did not detect any differences in the frequencies of Th17 cells between GG, GA, and AA genotypes of healthy donors. Instead, the Th17 cells were found to be attenuated in response to IL-23 signaling. For this experiment, memory CD4 cells from normal healthy individuals — heterozygous and homozygous carriers of Gln381 — were stimulated with anti-CD3/anti-CD28-coated beads in the presence or absence of IL-23 and culture supernatants of these cells were subsequently assayed for the presence of Th17 cell cytokines. The results demonstrate a nearly complete abrogation of enhanced IL-23 effect on Th cells from AA carrying healthy donors. Thus, the Gln381 allele impairs IL-23-driven survival and expansion of memory Th17 cells. As in the cases of healthy donors, there was no difference in Th17 cell frequency between GA and GG-carrying psoriasis patients. However, in psoriasis patients, the IL-23 mediated IL-17A and IL-22 production in memory Th cells was significantly reduced in GA compared to GG carriers. Interestingly, there was no correlation of the Gln381 allele with psoriasis severity, age of onset of disease or frequency of cases of severe psoriasis, implying a more profound relationship between the presence of the allele and psoriasis propagation. Furthermore, there was no significant difference in skin mRNA of IL-17A, IL-17F, IL-22, and IFN-γ in GA as compared with GG patients. However, a positive correlation of IL-23p19 with IL-17A and IL-22 mRNA levels was observed in GG patients that was absent in GA patients. Collectively, the results suggest that, although not affecting disease severity, the IL-23R SNP associated with Arg381Gln substitution still results in attenuated IL-23 signaling and impaired Th17 response.

COMMENTARY This study shows many aspects that are important for both psoriasis researchers and immunologists. It further substantiates the assumption that IL-23 has a minor impact, if at all, on the in vivo development of Th17 cells from naive Th cells. Rather, the significance of this cytokine might concern the enhancement of activation and expansion/survival of these cells. However, these IL-23 functions are shared by other immune mediators in the psoriatic lesion. The similar disease severity in patients with and without genetically dependent IL-23 signaling weakness stresses the fact that psoriasis is a multi gene-dependent disease and that environmental factors play an important role in its development and persistence. The pathogenetic mechanisms might result from interactions between genetic background, the changes that immune system and tissue cells experienced and memorized (for example, expressed as epigenetic changes), and external acute trigger factors. Finally, SNP inducing IL-23R Arg381Gln substitution may be a promising candidate for a biomarker for non-responders to anti-p40 or anti-IL-23 therapies. Larger studies are needed to test whether insights gleaned from “SNP-to-function” studies can be translated into patient-benefiting and cost-effective stratified medicine approaches.
5. Secukinumab: clinical profile of an anti–IL-17A monoclonal antibody in moderate to severe psoriasis patients.


Summary

Evidence is accumulating to support the notion that IL-17A is an important effector cytokine in the pathogenesis of psoriasis. First, IL-17A is over-expressed in psoriatic plaques and plays a profound role in some mice models of skin inflammation. Additionally, IL-17A is produced by Th17 cells, likely in response to activation by IL-23, a pro-inflammatory cytokine playing an essential role in maintenance of psoriatic lesions in many patients, as has been learned from clinical trials. The selective targeting of IL-17A thus represents a potential therapeutic strategy that could meet patients' needs, which continue to persist despite the availability of multiple systemic and biologic agents.

The objective of this study was to assess the efficacy and safety of secukinumab, a fully human, anti-IL-17A IgG1, monoclonal antibody, in patients with moderate to severe plaque psoriasis. The study was a phase II, randomized, double-blind, placebo-controlled, parallel-group design conducted at 19 centers in six countries (Canada, Estonia, Iceland, Japan, Latvia, and United States). Patients (n=125) were randomized to placebo (n = 22) or one of four secukinumab regimens: 1 x 25 mg (n = 29), 3 x 25 mg (n = 26), 3 x 75 mg (n = 21) or 3 x 150 mg (n = 27) at weeks 0, 4, and 8. After the 12-week treatment period, patients entered a follow-up period of 24 more weeks. The primary efficacy outcome was at least 75% improvement from baseline in the Psoriasis Area and Severity Index score (PASI 75).

After 12 weeks of treatment with secukinumab, PASI 75 response rates were 82% and 57% for the 150-mg and 75-mg doses, respectively. This compared favorably to a placebo response of 9%. The PASI 75 responses gradually declined during the follow-up period to 26% and 19%, for the 150-mg and 75-mg doses. The PASI 90 response rate was significantly higher in the 150-mg dose group at 52% vs. placebo (5%) at week 12, and this remained higher during the follow-up period.

A weight-based dose dependency was observed, since patients weighing <90 kg displayed better responses versus patients in the >90 kg group. The overall incidence of adverse events (AE) was higher in the 150-mg group (89%), compared with the other secukinumab dose cohorts (73–76%), which were similar in AE incidence to the placebo group. Of the AEs, 41.6% (n = 52) were mild, 26.4% (n = 33) were moderate and 9.6% (n = 12) were severe in intensity. The most frequently reported AEs across all cohorts were worsening of psoriasis (16.8%, n = 21), nasopharyngitis (12.0%, n = 15) and upper respiratory tract infection (6.4%, n = 8). AEs that led to permanent discontinuation of the study drug occurred in one patient in the 3 x 25 mg group (exacerbation of psoriatic arthropathy) and one patient in the 3 x 150 mg group (abnormal liver function test results). Overall, secukinumab was considered to be well tolerated and its monthly use in doses of either 3 x 75 mg or 3 x 150 mg was found to be statistically efficacious in moderate to severe psoriasis, as measured by the primary outcome of PASI 75 response at 12 weeks.

COMMENTARY The results of this phase II clinical study validate secukinumab as a potential new therapeutic option for moderate to severe psoriasis. Given that conventional systemic therapies do not fully meet patients' needs, the selectively targeted monoclonal antibody to IL-17A represents a promising solution. Secukinumab displayed high and sustained effectiveness after a 12-week treatment period and was administered with a convenient dosing profile. Furthermore, it was well tolerated during this short time. However, phase III trials and further observation are needed to determine the benefit-risk profile of this treatment. Additionally, in light of the fact that only half the treated patients reached PASI 90, there must be more pressure to identify biomarkers before therapy starts, signaling which patients will derive the greatest benefit from the therapy.
A REPORT FROM THE 22ND CONGRESS OF THE EUROPEAN ACADEMY OF DERMATOLOGY & VENEREOLOGY

Focus on Psoriasis: A Report from the 22nd Congress of the European Academy of Dermatology & Venereology, Istanbul, Turkey

By Sophie Momen, BMBS, MRCP

Contributing writer Sophie Momen is currently completing her general medical training at St. George's Hospital London, after which she hopes to pursue a career in dermatology. She graduated from the University of Nottingham in 2010.

The four-day EADV Congress convened under the theme “Dermatovenereology in a changing world” in historic Istanbul. The following is a summary of presentations about developments and challenges in diagnosing and treating psoriatic disease.

Secukinumab

Secukinumab, a fully human anti-IL-17A monoclonal antibody, was the topic of two presentations. Dr. Richard Langley of Dalhousie University, Halifax, Nova Scotia, presented the results of the FIXTURE (Full year Investigative eXamination of Secukinumab vs Etanercept Using two dosing Regimens to determine Efficacy in psoriasis) study, a randomized, double blind, phase III study in patients with moderate to severe psoriasis. The primary objectives were to assess the superiority of secukinumab (150 mg and 300 mg) versus placebo in achieving PASI 75 (Psoriasis Area and Severity Index) and IGA 0/1 (Investigator’s Global Assessment) at 12 weeks. Secondary endpoints included the superiority of secukinumab versus etanercept to achieve maintenance of PASI 75/ PASI 90/ IGA at 52 weeks.

A total of 1,264 patients with moderate to severe psoriasis were enrolled. The inclusion criteria included a minimum of PASI 12, IGA> 3, BSA >10% affected, and inadequate control with previous treatments, including biologics. Secukinumab 300 mg was shown to have a higher clinical efficacy compared with etanercept (Table 1). Almost complete skin clearance (PASI 100) was observed at week 12 in 24% of patients (secukinumab 300 mg) versus 4% (etanercept 50 mg). The greatest response of secukinumab 300 mg was observed at week 52, nearly twice as many patients treated with secukinumab 300 mg achieved a PASI 90 response (65%) compared with 33% of patients treated with etanercept. The side effect profile was similar to that of etanercept.

Candida infections were seen in patients treated with etanercept, secukinumab 150 mg and secukinumab 300 mg at 1.2%, 2.3% and 4.7% respectively.

Ulrich Mrowietz of the University Medical Centre Schleswig-Holstein, Kiel (Germany) presented the results of SCULPTURE (Study Comparing secukinumab Use in Long-term Psoriasis maintenance therapy: fixed regimens vs reTreatment Upon start of ReLapse), another phase III study of secukinumab. This study compared a fixed

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A REPORT FROM THE 22ND CONGRESS OF THE EUROPEAN ACADEMY OF DERMATOLOGY & VENEREOLOGY

regimen (monthly dosing) versus retreatment upon start of relapse (RTN) of patients who achieved PASI 75 after 12 weeks of therapy (Table 2). Relapse was defined as a loss of PASI 75 response. In both 150-mg and 300-mg secukinumab groups, a greater percentage of patients achieved PASI 75 at week 52 in the fixed-dose groups. Importantly, 67% of patients in the 300-mg RTN group achieved PASI 75 at this point, compared to only 62% in the 150-mg fixed-dose group (Table 3). The number of injections in the RTN groups was less than in the fixed-dose group and, therefore, a less costly regimen. Anti-drug antibodies developed in 5 of the 966 patients (0.52%). Interestingly, there was no difference in the frequency of patients with anti-drug antibodies between the fixed-dose groups and the RTN groups (n=2). There were no safety concerns. Similar side effect profile was seen in all groups; this was mild to moderate and did not stop treatment. In conclusion, it may be possible to use RTN dosing in some patients.

JAK 1inhibitor (INCB039110)

Dr. Robert Bissonnette, president of Innovaderm Research Montreal, Quebec, Canada, presented the findings of a double blind, placebo-controlled study, which explored the safety, efficacy, and tolerability of a 28-day course of an oral JAK 1 inhibitor (INCB039110) in escalating doses in patients with stable chronic plaque psoriasis. INCB039110 was assessed in four treatment groups (100 mg once daily, 200 mg once daily, 200 mg twice daily, 600 mg once daily) in parallel to placebo. Randomization occurred on a 3:1 basis (INCB039110: placebo). The primary endpoint was the mean change from baseline in Physician Global Assessment (PGA) at day 28. The secondary endpoint was the percentage of patients to achieve a static Physician Global Assessment (SPGA) 0/1 at day 28. Inclusion criteria were BSA >5%, PASI 5, PGA >3. A dose-dependent improvement was observed as measured by the primary and secondary endpoints (Table 4). There were no significant adverse effects. Reductions in platelet count were observed; however, these remained within normal range. Reductions in LDL and HDL were observed, but there was no change in the LDL/HDL ratio. Two patients withdrew due to non-compliance. The study acknowledged that, with short-term follow-up of 28 days, all effects of the drug might not be seen.

Methylation genes in CD3+ psoriatic cells

Our knowledge about epigenetic alterations in psoriasis are still limited. An important component of epigenetic regulation is DNA methylation. Dr. A. Lavrov presented findings related to the status of gene methylation of T lymphocytes in both psoriatic patients before treatment (n=10 with an average PASI of 24) and healthy individuals (n=11). The methylation levels of FRK, JAK1, AKAP7, DYRK1A, PRKCA, RPS6KA2 were detected with microchips Illumina.

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<th>Table 4. Results of primary and secondary endpoints reached with 28 days of varying dose of INCB039110:placebo.</th>
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Infinium Human Methylation 450 BeadChips. FRK (Fyn related kinase) belongs to a family of tyrosine protein kinases, which may suppress cell growth during G1 and S phase of the cell cycle. They found that the methylation level of FRK gene is nearly twofold higher in controls compared with patients with psoriasis. This means that patients with psoriasis have higher levels of demethylation of this gene and that it is transcribed more actively. The authors proposed that methylation of FRK in CD3+ cells might be able to predict at an early stage those patients who may develop psoriasis, potentially supporting early diagnosis.

Factors affecting response to treatment
Dr. Kim Papp, president of Probity Medical Research Inc, in Waterloo, Ontario, Canada, presented the results of a phase II, randomized, double blind, placebo-controlled, parallel group, dose-range study assessing the impact of body weight and prior biologic agent use on the effectiveness of ixekizumab (an anti IL-17A monoclonal antibody) in patients with moderate to severe psoriasis. In the study, 142 patients were treated with ixekizumab at varying doses (10 mg, 25 mg, 75 mg, 150 mg, placebo) over 20 weeks. Treatments were given at weeks 0, 2, 4, 8, 12, and 16. Patients were randomized according to weight (<100 kg, >100 kg [n=52]) and previous biologic use [n=59]. Post hoc analysis was performed at 12 weeks. The regression analysis of all patients revealed that baseline body weight was negatively associated with a PASI improvement (0.045) and a 12-week PASI 75 response rate (p=0.01). However, body weight and prior biologic use did not affect the efficacy of high-dose ixekizumab (75 mg, 150 mg), but they did affect the efficacy of low doses (10 mg, 25 mg). (Tables 5 & 6)

Use, economics, and safety of biologics
Safety data from the long-term use of biologics was a frequent topic at the 2013 EADV conference. Summarized here are the observations of Drs. Jonathan Barker, Luigi Naldi, Carle Paul, Lluís Puig and Kristian Reich.

Drs. Barker and Naldi led a session entitled “Biologics in psoriasis: Long-term therapy pros and cons.” They spoke against and for long term biologic therapy, respectively. Dr. Barker presented the following arguments. Biologics cost 12,000 euros per annum for each treated patient. In the US, $2.6 billion dollars were spent on biologics to treat psoriasis in 2010, and the cost is estimated to increase to $5.6 billion by the end of the decade. In the UK, biologics encompass 40%-60% of the current National Health Service expenditures for psoriasis. However, this money reaches fewer than 10% of patients suffering from psoriasis because 65%-80% of these patients are categorized as having a “mild” form of the disease. What impact does this have on our health budgets? Does this limit the resources for patients with psoriasis who do not qualify for such treatments?

The adherence to biologic therapy may diminish over time due to loss of efficacy and side effects. In fact, decreased efficacy of ustekinumab over five years of treatment was also observed in the PHOENIX 1 study. This raises the question: Can we identify long-term responders and non-responders early and, thus, minimize the drug’s expense for patients who do not respond? Both parties acknowledged that most studies follow patients for a few years, but when discussing “long-term therapy” we must decide on the definition of “long-term.” The average period of a patient’s life spent living with psoriasis may be up to 40 years; therefore, long-term follow-up studies are needed.

| Table 5 & 6. Effect of weight and previous biologic use in ixekizumab treatment. |
|-----------------|-----------------|-----------------|-----------------|-----------------|
| Week 12 | Low dose (10-25 mg) | High dose (75-150 mg) | Low dose (10-25 mg) | High dose (75-150 mg) |
| <100 kg | 61% | 86% | 13+/−7 | 15+/−5 |
| > 100 kg | 41% | 75% | 10+/−8 | 16+/−7 |
| P=0.178 | P=0.298 | P=0.111 | P=0.558 |

<table>
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<th>Table of PASI 75 at Week 12</th>
<th>Prior Biologic use</th>
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<td>Low dose</td>
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<td>High dose</td>
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Dr. Naldi presented the arguments for long-term biologic therapy. Psoriasis is a chronic disease that can significantly impact a patient’s quality of life, in addition to having many systemic associations. Thus, the optimal management of it is of paramount importance. Patients want therapies that are easy to administer and have a long duration of action – factors that biologics deliver.4 With interrupted biologic therapy, relapse of disease has been seen in some patients treated with adalimumab and ustekinumab.5,6

Number needed to treat
Dr. Richard Langley discussed the number of patients with moderate to severe psoriasis needed to treat (NNT) to achieve a reduction in PASI 75 with adalimumab 40 mg, infliximab 5 mg/kg, ustekinumab (weight-based dosing) and etanercept 25 mg/50 mg. He also discussed the incremental cost per PASI 75 responder (ICR) during the first 10 to 16 weeks of therapy. Randomized controlled trials (RCTs) in patients treated with systemic therapies for moderate to severe psoriasis were used to develop a Bayesian network meta-analysis to estimate the probability of achieving a PASI 75 score. The NNT and the incremental cost per responder (ICR = NNT x cost) were calculated. Importantly, adalimumab was the most cost effective when measured by ICR of PASI 75 responder (Table 7).

Safety of biologics
Speakers addressed the following aspects regarding the safety of biologics:
• Clinical trials provide high-quality data, but only follow patients for a short period of time.

• In order to assess safety accurately, such medications need to be tested in a population that truly represents “real life.” Of 1,042 patients receiving systemic psoriasis therapy, 30% were deemed ineligible for inclusion in randomized controlled trials due to exclusion criteria, including age >70 years, type of psoriasis, history of malignancy, infection, virus, liver/renal disease. In fact these patients had a higher risk of developing serious adverse events (SAE 2.7%).6

• The definition of moderate to severe psoriasis may vary worldwide, thus affecting inclusion criteria, which also may vary.

• Most post-marketing data is often of low quality, but may detect rare adverse events.

• Background risk for developing cancer may be greater in a patient with psoriasis and other inflammatory conditions than the general population. For example, lymphoma is more prevalent in patients with psoriasis and rheumatoid arthritis (RA) than the general population. Therefore, the control and reference population must be relevant.

• Results may vary depending on the comparator used (eg, active vs placebo – placebo is the safest option, but often the least effective). For long-term follow-up, there is no placebo.

• How should AE (adverse event) be defined? (eg, does this include opportunistic infection?) It is thought that one of the best predictors for the development of serious adverse events is age.

| Table 7. Number needed to treat (NNT) and incremental cost per responder (ICR) for adalimumab 40 mg, infliximab 5 mg/kg, etanercept 25 mg, ustekinumab (weight-based dosing) to achieve a PASI 75 in patients with moderate to severe psoriasis. |
|---|---|---|---|---|---|
| | Adalimumab 40 mg | Infliximab 5 mg/kg | Ustekinumab (weight-based dosing) | Etanercept 25 mg | Etanercept 50 mg |
| PASI 75 | 67% | 82% | 71% | 37% | 52% |
| NNT | 1.58 (1.44-1.81) | 1.28 (1.20-1.37) | 1.49 (1.39-1.61) | 3.05 (2.50-3.78) | 2.10 (1.86-2.38) |
| ICR | £6,130 (5,572-7,020) | £7,570 (7,091-8,101) | £7,221 (6,740-7,817) | £8,019 (6,573-9,930) | £10,188 (9,007-11,544) |
Safety per biologic agent

**Adalimumab.** Presented in this discussion were 10-year data of 19,041 patients exposed to adalimumab therapy for six diseases: rheumatoid arthritis (RA), juvenile idiopathic arthritis (JIA), psoriasis (Ps), psoriatic arthritis (PsA), Crohn’s disease and ankylosing spondylitis. Serious adverse events (SAE) per 100 patient years were lower for psoriasis (1.32) than for RA (4.65).7

**Etanercept.** The OBSERVE 5 study followed 1,890 patients and reported three-year incidence proportions of SAE (0.14) and serious infections (0.04). Observations of lymphoma, serious infectious events requiring hospitalization, non-melanoma skin cancer (NMSC), and malignancies excluding NMSC were no greater than the estimated number of cases from a large US administrative health claims database.8

**Infliximab.** Integrated data of 1,373 psoriatic patients treated with infliximab were compared with external databases of general and psoriasis populations, based upon 1,106 patient years and 116 patient years of follow-up. The standardized mortality ratio in the infliximab-treated patients was less than that of the general psoriasis population (0.17). Data were presented comparing the standardized incidence ratios (SIRs) for serious infection rates 1.28 (infliximab-treated) versus 1.47 (placebo). The incidence of tuberculosis (TB) was 0.18 per 100 patient years in infliximab-treated, while no TB cases were reported in the placebo group. Rates of internal malignancy (excluding NMSC skin cancers) were similar to those predicted in the general US population – SIR of malignancy (0.39) versus none in placebo.9

The use of TNF inhibitors in patients with psoriasis has been shown to significantly reduce the risk of myocardial infarction, compared to topical therapies.10

**Ustekinumab.** Safety data for patients using 45 mg/90 mg ustekinumab were pooled from four phase II/III RCTs. Four-year follow-up of 3,117 patients (6,791 patient years) revealed that rates of adverse events were similar to those in the general and psoriasis populations, and remained stable over time. There was no suggestion of a cumulative toxicity. Cumulative rates per 100 patient years for serious infections were 0.80 and 1.32, NMSC 0.70 and 0.53, and major adverse cardiac event (MACE) 0.56 and 0.46 (45 mg, 90 mg respectively).11

Psoriasis and the risk of malignancy

Dr. Carle Paul discussed the topic, “Cancer risk assessment in psoriasis: power and pitfall of registries.” A systematic literature review from 1980-2012 suggested that patients with psoriasis may have an increased risk of squamous cell carcinoma, SIR = 5.3, (95% CI 2.63-10.71) and basal cell carcinoma SIR = 2.00, (95% CI 1.83-2.20), with no increased risk of melanoma. Such findings may be due to previous 8-methoxypsoralen-ultraviolet-A (PUVA), cyclosporine, and possibly methotrexate exposure.12 Patients may be at an increased risk of developing solid cancers associated with smoking and alcohol, such as respiratory tract malignancies (SIR = 1.52, 95% CI 1.35-1.71), upper digestive tract malignancies (SIR = 3.05, 95% CI 1.74-5.32), urinary tract cancer (SIR = 1.31, 95% CI 1.11-1.55) and liver cancer (SIR = 1.90, 95% CI 1.48-2.44). The risk of non-Hodgkin lymphoma appears slightly increased in psoriasis (SIR = 1.40, 95% CI 1.06-1.86).13

Risk of infection

Dr. Luigi Naldi discussed the long-term risk of infection. The prevalence of latent TB in patients with psoriasis in one study was reported to be 29%.14 Results of tuberculin skin tests may be difficult to interpret in patients who receive anti-TNF therapy with underlying psoriasis. Reactivation of latent TB appears to be greater in patients receiving infliximab and adalimumab over etanercept that had precedence for extra-pulmonary sites.15 Clinicians must be vigilant for reactivation of latent TB, as screening tests...
may be difficult to interpret when considered with the prevalence of disseminated TB.

**Immunobiomarkers are needed for the implementation of personalized therapy strategies with biologics.**

**Major adverse cardiac events (MACE)**
Dr. Alice Gottlieb presented results of a study that assessed MACE rates in the Psoriasis Longitudinal Assessment and Registry (PSOLAR). The number of MACE events was small, and there was no large difference between ustekinumab and other biologics (ustekinumab 0.21 events per 100 years of patient observation [PYO] compared with non-sponsor biologics – adalimumab and etanercept - 0.31 PYO). It should be noted that randomized controlled trials are not powered for safety, and rare SAE incidences will only become prevalent with time. Thus, findings on long-term safety in “real life” patients are urgently needed.

**Switching biologics**
Dr. Ulrich Mrowietz presented the concept of switching to and from biologics. However, there is no evidence at present to support switching. Continuous therapy is generally thought to give a greater efficacy over time, compared with intermittent therapy. In clinical trials where primary responding drugs are reintroduced after cessation, up to 20% of patients fail to regain a PASI 75 response after the first reintroduction. This decreased efficacy may be greater with intermittent usage of medications. The first biologic used usually has the longest effectiveness. However, there may be various reasons for biologic cessation, including primary/secondary non-response, intolerance, or safety signals. Dr. Mroweitz also noted:

- When switching from conventional therapy, biologics can be commenced without a washout period, especially with cyclosporine, in order to reduce rebound of disease.
- When switching from one biologic to another because of treatment failure, the new biologic can generally be started without a washout when the next scheduled dosage is due. However, if discontinuation is due to an adverse safety profile, then a washout period is advised until safety parameters have normalized.

**Personalized biologics**
Clinicians are well aware that factors such as the extent of lesions, erythema intensity, and pustulation of psoriatic lesions do not predict response. Currently, patients are treated through trial and error; when one biologic fails, another is tried. Perhaps clinicians should use a more stratified approach. That was the basis of a discussion about “personalized biologics” by Professor Peter Van de Kerkhof. The choice of biologic can be stratified depending on a patient’s phenotype, comorbidities, endophenotype, pharmacokinetics, and, in the future, particularly immunobiomarkers.

**Patients’ comorbidities must be taken into account when prescribing biologics.**

Patient characteristics (secondary non-responders, disease duration <8.3 years and weight less than 102 kg) have been shown to predict those who will respond to dose escalation of adalimumab. Patients’ comorbidities must be taken into account when prescribing biologics. For example, anti-TNF therapy is contraindicated for patients with stage III (moderate) or stage IV (severe) congestive cardiac failure, as classified by the New York Heart Association. However, ustekinumab is permitted. The British Association of Dermatologists suggests that biologics should not be prescribed in patients with a history of malignancy within the previous five years or if they have a significant risk of developing malignancy (200 PUVA and/or >350 UVB treatments). However, recent studies have suggested that infliximab therapy may be beneficial in the treatment of renal cell carcinoma. Perhaps a personalized decision on prescribing biologics in patients with underlying malignancy might be appropriate.

Adalimumab drug levels at four weeks into treatment may help predict patients who will respond to therapy.
and, as a result, reduce exposure to further medication and cost. Professor Griffiths discussed the effects that pharmacogenetics and concurrent polypharmacy may have on the choice of biologic agent. Recently, Talamonti et al demonstrated that the HLA-Cw6 gene may act as a pharmacogenetic marker for response to ustekinumab. Approximately 96% of CW6 positive patients achieved a PASI 75 at 12 weeks, compared with 65.2% of CW6 negative patients.

Polypharmacy has been addressed as a growing issue in patients with psoriasis. Of 114,512 patients with psoriasis, 51% had more than one comorbidity. Patients with psoriasis who had comorbidities are more likely to need emergency department care (1.58), hospital admission (2.27), and outpatient appointments (1.53), thus highlighting the economic impact. In an aging population, polypharmacy will remain an escalating issue for dermatologists. They must be vigilant about drug-on-drug interactions and how treating one condition may improve or exacerbate psoriasis and comorbidities. Overall, the aim of treatment is effective, long-term management, with safe control and minimal monitoring.

Biologics with limited resources
Dr. Vidyadhar Sardesai discussed problems faced when prescribing biologics in locations with limited resources. Issues included the prescriptive cost of biologics; their side effect profile, especially with the high prevalence of TB; the need for patients to have a baseline education stature to understand the need for regular monitoring and compliance; and the so called “Indian mindset” that equates cost with cure.

Psoriasis and comorbidities

Inflammation, metabolic syndrome and cardiovascular disease: possible links
Psoriasis is an inflammatory condition known to have many associated comorbidities that can have a huge impact on quality of life and life expectancy. Drs. Peter Arenberger and Alice Gottlieb discussed its association with metabolic syndrome. Metabolic syndrome has a greater prevalence in both children and adults with psoriasis (30% of children with psoriasis/psoriatic arthritis had metabolic syndrome, compared with a 5% prevalence in children without the disease). One report showed that a 30-year-old patient with severe psoriasis has a threefold increased relative risk of having a myocardial infarction than the normal population. The relative risk of cardiovascular mortality in a 40-year-old and 60-year-old with severe psoriasis has been estimated at 2.69 and 1.92, respectively.

Regarding links between psoriasis and metabolic syndrome, it has been suggested that inflammation within the skin releases inflammatory mediators such as TNF-a into the systemic circulation that act at cellular levels and directly cause some metabolic alterations (eg, insulin resistance). It has also been suggested that activated adipocytes from adipose tissue release pro inflammatory cytokines (eg, IL-8 and TNF-α) that amplify skin inflammation. Furthermore, inflammation plays a key role in atherosclerosis.

One report showed that a 30-year-old patient with severe psoriasis has a threefold increased relative risk of having a myocardial infarction than the normal population.

Dr. James Krueger further explored the cardiovascular association with psoriasis. Approximately 3,000 genes are differentially expressed in psoriasis lesions, compared with matched biopsies of non-lesional skin. Many of these genes link to functional pathways associated with metabolic diseases. Dr. Krueger reported on a study in which PET/CT (positron emission topography/computed topography) scans were performed on 10 patients with moderate to severe psoriasis. The researchers used [18F]-fludeoxyglucose (FDG), which is taken up by macrophages, based on a study by Mehta et al. They observed increased uptake in psoriatic plaques and within vessels, suggesting that the vessels may be in an inflamed state. These patients were treated with etanercept for one year and had pre- and post-treatment PET/CT scans. After etanercept treatments, their psoriasis improved (average 45.9 to 12.2)
and there was a marked decrease in vascular inflammation. The subcutaneous fat of affected psoriatic plaques showed increased pixel density (suggesting inflammation) compared with unaffected fat. Decreased pixel density in affected areas of subcutaneous fat was noted after etanercept therapy.

Additionally, an elevated Renin-angiotensin-aldosterone pathway had been identified in patients with psoriasis, thus explaining why some patients have elevated diastolic and systolic blood pressures.\textsuperscript{31}

Obesity

Obesity was mentioned frequently throughout the EADV conference, particularly by Dr. Paolo Gisondi. Obesity can cause systemic inflammation. Macrophages and leukocytes are thought to infiltrate adipose tissue, which acts as a nutrient source. Following this, adipokines are released, which then triggers an inflammatory response body organs, producing a variety of clinical effects (in liver, non-alcoholic fatty liver disease [NAFLD]; in adipose tissue and skeletal muscles, insulin resistance; in brain, decreased sensitivity to leptin).\textsuperscript{32} In the skin, this response may fuel psoriatic inflammation.\textsuperscript{33} Prospective studies have shown that an increased body mass index (BMI) and weight gain are strong risk factors for developing psoriasis in women.\textsuperscript{34}

Furthermore, a BMI >30 is associated with an increased prevalence of liver function and renal function derangement with methotrexate and cyclosporine, respectively.\textsuperscript{35} It has been suggested that obesity may be associated with reduced drug effectiveness. In fact, biologics that are prescribed at fixed doses (etanercept and alefacept) may have decreased efficacy in obese patients.\textsuperscript{36} Naldi et al looked at the PSOCARE cohort study for patients on systemic therapies; they noted that an increased BMI was associated with a lower odds ratio to achieve a PASI 75 and, thus, reduced biologic effectiveness.\textsuperscript{37} This was independent for other risk factors, including type of treatment, age and psoriasis severity. Gisondi et al performed multivariate analysis looking at predictors for early drug discontinuation. BMI was an independent risk factor for early drug discontinuation due to ineffectiveness, independent of other risk factors. Possible interventions such as low-calorie diets may be helpful in such patients. A low-calorie diet for eight weeks reduced PASI and Dermatology Life Quality Index (DLQI) by -2.0 (95\% CI, 4.1 to -0.1; \(P = .06\)) and -2.0 (95\% CI, -3.6 to -0.3; \(P = .02\)), respectively. Additional RCTs investigating patients treated with cyclosporine +/- low calorie diet revealed a greater PASI reduction in those with combined diet and cyclosporine, thus providing support for dietitian input in the management of psoriasis.\textsuperscript{38} More extreme measures such as bariatric surgery may also be beneficial.\textsuperscript{39}

Non-alcoholic fatty liver disease (NAFLD)

NAFLD has been shown to have an increased prevalence in people with psoriasis and could influence treatment options (eg, methotrexate). Dr. Ella Van der Voort reported the results of a cross-sectional study she and colleagues conducted. It consisted of patients over 65 from a large, prospective, population-based cohort study, comparing the prevalence of NAFLD (diagnosed on ultrasonography) in patients with psoriasis versus those without. The study included 2,292 patients (5.1\% had psoriasis). NAFLD had a prevalence of 46.2\% in those with psoriasis versus 33.3\% in the control group (\(p=0.005\)). After correcting for possible confounding factors (including alcohol consumption, smoking history, metabolic syndrome, elevated liver enzymes), psoriasis poses a significant forecaster for the developments of NAFLD (adjusted OR = 1.7, 95\% CI 1.1-2.6). Patients with psoriasis over 65 are 70\% more prone to develop NAFLD than patients without the disease.

Lipids

Dr. April Armstrong presented “Lipid levels in the context of high-grade inflammatory states: old and new concepts.” Plaques within atherosclerosis are lipid-rich
and inflammatory. Within the arterial wall, VLDL (very low density lipoprotein) and IDL (intermediate density lipoprotein) penetrate into vessel walls. The smaller and more dense a lipid particle, the more potential they have to penetrate the vessel wall and form atherosclerosis. The total number of lipoproteins does not equate to atherogenicity. Approximately 50% of myocardial infarctions are in healthy individuals with normal LDL levels, and it has been suggested that C-reactive protein (CRP) may be a risk factor. Statins are known to lower both CRP and LDL and subsequent cardiovascular risk. The JUPITER (Justification for the Use of Statins in Primary Prevention: An Intervention Trial Evaluating Rosuvastatin) trial looked at the use of rosuvastatin 20 mg versus control in 17,802 healthy individuals without hyperlipidemia but with a CRP >2 mg. They were followed for the combined primary endpoint of myocardial infarction, stroke, arterial revascularization, hospitalization for unstable angina, or death from cardiovascular causes. LDL levels were reduced by 50% and C-reactive protein levels by 37% in rosuvastatin group. The trial was stopped after 1.9 years (maximum 5 years). Primary endpoint rates were 0.77 per 100 person years of follow-up in the treatment group versus 1.36 in placebo. Inflammatory mediators can change lipoproteins due to hepatic synthesis and change the function of lipoprotein, leading to atherosclerosis. In inflammation, there is an increase in triglyceride levels and VLDL, due to increased fatty acid synthesis in the liver and a reduction in LDL and HDL, inhibition of cholesterol synthesis, and increased clearance in the reticulo-endothelial system. Anti-psoriatic therapy has been shown to improve PASI score and improve HDL efflux independent of HDL levels. New guidelines from the European Society of Cardiology, expected in 2014, will encourage treatment as per LDL level, depending on individual risk (eg, high-risk inflammation).

Vascular function
Dr. Evangelia Papadavid discussed the similarities between patients with psoriasis and hypertension. A study she conducted with colleagues compared patients with psoriasis and no hypertension (n=59) versus patients with untreated hypertension (n=59) and normal healthy patients (n=40). Markers of subclinical atherosclerosis, vascular function and left ventricular myocardial deformation were measured. The researchers concluded that both patients with psoriasis and untreated hypertension had similarly impaired levels of the measured values in comparison to healthy controls. In addition, the degree of carotid atherosclerosis was found to be associated with the severity of psoriasis.

Psoriatic arthritis
Dr. Wolf-Henning Boehncke acknowledged that psoriatic arthritis (PsA) is notoriously difficult to diagnose. Therefore, dermatologists are in a position to identify such cases in order to introduce treatment early and minimize disease progression. Early intervention has been shown to result in better efficacy of treatments. It is not only patients with severe psoriasis who are at risk of developing PsA; those with scalp, nail or perianal involvement are also at high risk. Thus, screening for PsA should be routine for all patients with psoriasis. Several questionnaires are available for screening purposes. The Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) have validated three screening tools to assess for PsA: Toronto Psoriatic Arthritis Screening (ToPAS); Psoriasis Epidemiology Project (PEST); and Psoriatic Arthritis Screening and Evaluation (PASE) (Table 8). The development of enthesis seems to be mediated by IL-23 and...
T cells that produce IL-22.

**Topical treatments and adherence**

Poor adherence to topical treatments, a well-known phenomenon in medicine, was discussed by Drs. Steven Feldman, Andrew Finlay, Peter van de Kerkhof, and Ravi Ratnavel. It is acknowledged that severe disease does not necessarily equate to adherence. There are many reasons why patients do not adhere to therapy. The doctor-patient relationship is central to patient adherence, and many ways were discussed to aid adherence. Early treatment adherence is key to long-term compliance and, as a result, patient satisfaction.

The presentations stressed the importance of gaining early adherence, thereby developing an effective doctor-patient relationship. It is important how patients perceive clinicians. Patients want caring doctors who have time to listen to them and acknowledge the impact that their disease might have on their quality of life. Clinicians should arrange early initial follow-up to assess medication compliance. It is important to highlight realistic goals with the patients and their families, and to identify any concerns that they may have about treatment. Treatment should be personalized with individual plans, perhaps choosing less messy topical applications that suit a patient’s lifestyle. Patient education about their condition is key. Dr. Finlay suggested that using techniques such as the “finger tip rule” might make patients more confident when using topical therapies. Compliance has been observed to be inversely proportional to the number of therapies prescribed. Written information is often helpful. Factors such as financial, psychological, physical issues must be considered when formulating such plans. It can be beneficial to identify patients for whom compliance might be difficult and perhaps offer them extra support. Allied health care professionals can provide support, and the Internet has proven a useful adjunct to improving patient compliance.

Dr. Marieke Seyger noted that compliance in children can be particularly challenging. A recent prospective study of children treated with short-contact dithranol and receiving once-weekly Skype consultations along with personal consultations had a 73% success rate. Other support programs for children were presented, including SPECTRUM, which focuses on educating children and their families about psoriasis, highlighting trigger factors, and providing information on how to cope with and manage the disease in the long term. It is important to acknowledge that it is futile to prescribe a medication that patients will not take. It is a financial burden to both patients and the economy. Clinicians must be comfortable asking patients about compliance in a non-judgmental manner. Dr. Finlay suggested asking patients how much they managed to use the day before their visit, acknowledging that compliance is difficult when lives are so busy. Ultimately, more validated research with a clinical, evidence-based approach is needed to understand compliance. Although compliance with topical therapy is difficult, the following scenarios were highlighted when such treatments are key: mild to moderate disease; usage by children; patients with a history of multiple skin cancers who are not suitable for systemic therapies; patients who are immune-compromised or reluctant to use systemic therapies. It must be remembered that early compliance is key.

For an outcome measure to be realistic, clinicians must consider the efficacy, safety, effect on quality of life, tolerability, and cost effectiveness of interventions.

**Outcome measures**

From her previous EADV lecture in 2012, Dr. Phyllis Spuls’s takeaway message had been that validation and harmonization of outcome measures were needed in conjunction with long-term efficacy and safety data. For an outcome measure to be realistic, Dr. Spuls noted in her presentation at the 2013 conference, clinicians must consider the efficacy, safety, quality of life (QOL), tolerability, and cost effectiveness of interventions. The limitation of evidence-based medicine is the heterogeneity of outcome measures, patient populations, subjects, and treatment aspects. The COMET (Core Outcome Measures...
Effectiveness Trial)\textsuperscript{47} initiative has been introduced to define a set of outcome measures (OM) in trials. At present, too many OMs are used. This initiative has been introduced aiming to harmonize and ensure their clinical relevance.

The COSMIN (CO\textsuperscript{2}nsensus-based S\textsuperscript{t}andard\textsuperscript{3}ed for the selection of health M\textsuperscript{e}asurement I\textsuperscript{n}struments) aims to clarify and standardize the terminology and descriptions of outcome measures so that the terms used to describe outcome measures are easily understood and uniform throughout. Within dermatology, it is important to define outcome measures and domains. Domains include signs, symptoms and severity, whereas outcome measures include Psoriasis Area and Severity Index (PASI), Physician Global Assessment (PGA), and Body Surface Area (BSA).

For patients with psoriasis, there are many different outcome measures, which lack validation and standardization.\textsuperscript{48} For example a recent study of RCTs in patients with psoriasis from 2001-2006 found that 58\% used PASI and 8\% QOL.\textsuperscript{49} It is imperative that valid measures of outcome and severity are available to clinicians to aid research and evidence-based medicine.

PASI is the most commonly used clinical measure; however, it has many limitations, including: its inter-rater variability; it is inapplicable for mild disease; it does not include location; and it is not helpful for erythrodermic patients.\textsuperscript{48} In response, the Simplified Psoriasis Index (SPI) was created. As explained by Dr. Robert Chalmers at the EADV conference, this psoriasis measure encompasses current severity and psychosocial impact, as well as previous interventions and severity. There also are professionally assessed SPI (proSPI-s) and self-assessed SPI (saSPI-s). Comparison of SPI to PASI has recently been investigated. These studies supported the use of SPI in clinical practice. Compared with PASI, there was greater inter-rater concordance (0.81) and fewer methodological errors. PASI scores were closely related to SPI. A 75\% reduction in PASI equated to 85\% and 95\% reductions in proSPI-s and saSPI-s, respectively, and PASI scores for mild (PASI<10) and severe (PASI>20) psoriasis were <9 and >18 for proSPI-s (n=300) and <10 and >20 for saSPI-s (n=200; AUC=0.86-0.96), respectively.\textsuperscript{50}

References
A REPORT FROM THE 22ND CONGRESS OF THE EUROPEAN ACADEMY OF DERMATOLOGY & VENEREOLOGY


IPC’s Meet the Experts Program goes to Istanbul

In 2013, IPC sponsored Meet the Experts Programs (MTEs) in Punta del Este, Uruguay; Paris, France; Hong Kong, China; and, most recently in October, in Istanbul, Turkey. At each of these sessions, worldwide experts led discussions that included cases of difficult-to-treat psoriasis patients.

During the meeting of the 22nd Congress of the European Academy of Dermatology & Venereology (EADV) in Istanbul, IPC sponsored a Meet the Experts program, under the chairmanship of IPC president Peter van de Kerkhof, MD, PhD, Nijmegen, Netherlands. Contributing writer Sophie Momen attended the meeting. In the report below, she summarizes the session’s presentations, led by our panel of experts: Kristian Reich, MD, Hamburg, Germany; Elke de Jong, MD, PhD, Nijmegen, Netherlands; and Anthony Ormerod, MD, Aberdeen, United Kingdom. Discussions focused on developments in the treatment of psoriasis as well as three patients cases.

The program began with a comprehensive overview of developments in treating psoriasis and outlined what can be learned from this knowledge. Psoriasis is a T helper 1 (Th1)-mediated disease driven by tumor necrosis factor (TNF). This is supported with the injection of interferon gamma and a subsequent Koebner phenomenon of psoriatic plaques appearing at injection sites. It is thought that interleukins 12 and 23 are secreted from the skin cells. When psoriatic patients are treated with MK-3222, a specific inhibitor to IL-23 (anti-IL-23p19), the results show an incremental PASI 75 response over time. Studies in the PHOENIX 1 trial using ustekinumab (IL-12/23p40) antibody revealed full efficacy at week 24 and sustained until week 40. Th17 is thought to release IL-17, which has three different subsets (A,C,F) that are overexpressed in psoriasis. IL-17A and F use the same receptors (sharing the alpha chain of a major chain.)

IL-17 has proven difficult to detect in human skin biopsies. Within the skin, it is expressed in neutrophils and mast cells. IL-17 levels in T cells are low and there is no difference among levels in psoriatic plaques, psoriatic skin, or normal skin. Therefore, there must be other sources of IL-17. Neutrophils are full of IL-17. A spectrum of neutrophil-rich pathogenesis is seen in psoriasis ranging from micro-abscesses histologically in normal psoriatic lesions to macro-abscesses seen in pustular psoriasis. In summary, IL-17 is produced from neutrophils, mast cells and T helper cells. After administering secukinumab intravenously for two weeks (anti-IL-17), the presence of IL-17 in neutrophils has been shown to nearly vanish; however, its presence still remains in T cells.

Time-to-response Phase III trials using briakinumab (monoclonal antibody against the p40 molecule shared by


A REPORT FROM THE 22ND CONGRESS OF THE EUROPEAN ACADEMY OF DERMATOLOGY & VENEREOLOGY
IL-12 and 23) demonstrate a higher efficacy for briakinumab versus methotrexate in treating patients with moderate to severe psoriasis. (In a study by Reich and colleagues, 81.8% of patients treated with briakinumab achieved PASI 75 at week 24, compared with 39.9% in the methotrexate group.) Ixekizumab (anti-IL-17) has been shown to improve symptoms of psoriasis. It is thought to directly interfere with the neutrophil T-cell axis. Secukinumab has not yet been approved for the treatment of psoriasis and briakinumab is no longer being studied for use in psoriasis.

Discussion question: What is the role of IL-17 within the skin?

Genetic defects in the IL-17 pathway are seen in chronic mucocutaneous candida (CMC). These patients are unable to produce IL-17, and it is thought to be a defect in the alpha chain of the receptor (IL-17RA). CMC is thought to be an autosomal recessive deficiency of IL-17RA, which leads to absent cellular response to IL-17A and IL-17F, whereas, an autosomal dominant deficiency of IL-17F leads to reduced activity.

To summarize: In psoriasis, there are two adaptive pathways which are thought to overlap: TNF-α and IL-17/23. The dominant cells for TNF-α are dendritic cells and, for IL-17, neutrophils. There are many similarities and differences between IL-17/IL-23, which need to be better understood. Blocking one pathway could precipitate overactivity in the other pathway. This may explain why, in some cases, when we treat a flare of psoriasis, palmoplantar pustulosis (PPP) is exacerbated. These adaptive pathways (TNF, IL-12/23) also interact with innate pathways involving neutrophils and keratinocytes.

CASE 1: Psoriasis and schizophrenia, alcohol abuse

History and presentation
This 31-year-old male patient had a past history of schizophrenia, intravenous drug use, heavy smoking and alcohol consumption, and psoriasis. A wide range of therapies – topical, ultraviolet light, and systemic psoriatic therapies – had been tried, including the systemics fumaric acid (insufficient effect), cyclosporine (unclear why he stopped taking it) and acitretin (side effects and patient no longer wanted it). Methotrexate had not been given due to his alcohol intake and presumed liver problems. Patient was examined in February 2013 because of an exacerbation of his psoriasis. He had stopped treatments due to a number of issues, including that he was tired of his medication regimen. He presented with widespread psoriatic plaque-like eruption (PASI 22.5), which was significantly affecting his quality of life. Surprisingly, hematology and chemistry lab tests results were normal. After discussion with the patient about restricted alcohol use and the need for regular lab investigations, patient started treatment with subcutaneous injections of methotrexate along with oral folic acid. Compliance was initially promising and he showed clinical improvement (PASI from 22.5 to 3.4 within two months). However, he later decided to stop therapy due to personal problems and this was associated with another flare within three months.

Clinical question: What should his doctor have done next?
Consensus of the audience was to be cautious about prescribing a biologic drug due to possible noncompliance with laboratory monitoring. Still, as his condition was affecting his quality of life, intervention was required. Possible options might have included re-exploring cyclosporine therapy.

CASE 2: Psoriasis and schizophrenia

History and presentation
The patient, a 48-year-old male patient who had psoriasis since age 20, presented with psoriasis and schizophrenia. Patient was started on adalimumab, 40 mg every 2 weeks injected by his mother. Dosage was later increased to 40 mg weekly, along with folic acid and methotrexate 10 mg/week. His psoriasis improved, but, during a psychotic episode, he threw away his adalimumab (10,000 euros). He was hospitalized and is mentally stable at this time.

Clinical question: What should the next step be?
The audience consensus was that this patient should not be given such a large supply of medication and perhaps should be treated with methotrexate (a cheaper alternative) in weekly or every-two-weeks prescriptions, or with biologics such as infliximab at intervals 8 weeks apart with nurse administration.
Because schizophrenia is unpredictable, it may be preferable that subcutaneous injections be administered by a nurse, home care professional or someone in the family. In patients with complex psychological backgrounds, factors such as compliance, overdose and disposal of medications must be considered.

CASE 3: Psoriasis and psoriatic arthritis

History and presentation
The patient, a 43-year-old female, had a 20-year history of psoriasis and psoriatic arthritis. She had been well controlled on methotrexate, folic acid and adalimumab, when she presented to her dermatologist with a surprise pregnancy. Methotrexate therapy had been stopped by the patient one month prior to this due to disease control. Following discussion with her obstetrician, she stopped all medications. Three months into her pregnancy, she developed an extensive flare of her psoriasis reminiscent of generalized pustular psoriasis with associated dactylitis. She was still hesitant to start medications, but her symptoms continued to worsen. At 30 weeks, she was hospitalized for rest due to the severe impact of her disease on her life. When the fetus had grown to 50%, she and her medical providers considered resuming adalimumab.

Clinical question: Is it appropriate to use immunosuppressants and biologics during pregnancy?
Gastroenterologists are much more familiar with prescribing tumor necrosis factor-α (TNF) inhibitors during pregnancy. The consensus is that they are safe during the first two trimesters and during breastfeeding. TNF inhibitors are approved Class B drugs by the U.S. Food and Drug Administration. It was suggested that, as the flare seemed to be generalized pustular psoriasis, a flare during pregnancy might have been predicted. Etanercept is thought to be relatively safe and especially good due to its short half-life. Cyclosporine is well used and safe in renal transplant patients. A 2012 national survey of rheumatologists and obstetricians in the United Kingdom* assessed the use of DMARDs in pregnancy: 92% of rheumatologists had concerns over such prescriptions, compared with 7% of obstetricians. Conclusion: A drug with a short half-life is preferable, and biologics are generally thought to be safe in the first and second trimester and through breastfeeding.

– Sophie Momen

Psoriasis and its related comorbidities were the focus of the IPC Think Tank, which took place in November in Boston, Mass. The IPC Think Tank is a two-day event in which IPC’s board of directors, councilors and corporate partners come together to discuss important issues in the field of psoriasis. At this year’s gathering, attended by many of the world’s leading psoriasis experts, participants discussed the interrelationship among genetics, inflammation, stress, psychology, psoriasis and various comorbid outcomes.

The keynote speaker was Dr. Nehal Mehta of the National Institutes of Health, who spoke about “Inflammation and Cardiometabolic Diseases.” Speakers for the Think Tank scientific program were Dr. James Krueger of Rockefeller University, New York, USA (“The transcriptome of psoriasis, the ‘core’ pathogenesis of disease, and how it may relate to the comorbidities”); Dr. Abrar Qureshi of Harvard Medical School and Brigham Women’s Hospital, Boston, USA (“Interactions between adiposity and genetic polymorphisms on the risk of psoriasis”); and Dr. Johann E. Gudjonsson of the University of Michigan, USA (“Inflammation pathways in psoriasis and their possible relationships to the comorbidities”).

New councilors appointed
Since June, the IPC Board of Directors has added five new councilors to IPC’s membership. IPC councilors serve in an advisory capacity and lend their global expertise on psoriasis research, treatment, and education to support all IPC programs, events, and initiatives. They provide expert opinion on current psoriasis therapeutic and research-related issues, participate in roundtable conferences as well as contribute manuscripts to top-tier journals and make presentations before congresses around the world. The new councilors are:

**Cristina Echeverría**
*Buenos Aires, Argentina*
Dr. Cristina Echeverría graduated with honors from the University of Buenos Aires School of Medicine. She continued her studies, specializing in both internal medicine at the National Ministry of Health and dermatology at the University of Buenos Aires. She is an active member of the Argentine Society of Dermatology and at present is the acting Scientific Secretary of SOLAPSO (Latin American Society of Dermatology). She has been an assistant professor of dermatology at the University of Buenos Aires School of Medicine and worked at the Eva Perón Hospital in San Martin. She started her own practice, Consultos Echeverría, in 1999.

**André Vicente Esteves de Carvalho**
*Porto Alegre, Brazil*
Dr. André Vicente Esteves de Carvalho is responsible for the Psoriasis Ambulatory at the Complexo Hospitalar Santa Casa de Porto Alegre (Rio Grande do Sul State) and is 2013-14 coordinator of the Psoriasis Department of the Brazilian Society of Dermatology (SBD). Dr. Carvalho is a specialist in dermatology certified by the Brazilian Society of Dermatology. He completed specialization courses in Dermatologic Surgery and Mohs micrographic
TREATMENT

Topical Therapy Working Group meeting at EADV

Gaps in knowledge about the effectiveness, safety and modes of action of topical treatments were the focus of an October meeting of IPC’s Topical Therapy Working Group in Istanbul, Turkey. The meeting was part of IPC’s participation in the 22nd Congress of the European Academy of Dermatology and Venereology (EADV).

Discussion was based on the results of a survey of IPC councilors conducted earlier this year, as well as input from councilors attending the EADV conference. Both groups were asked to help identify these knowledge gaps. Overall, the councilors indicated that evidence is needed in the following areas:

Elise Kleyn
Manchester, United Kingdom
Dr. Kleyn is a senior clinical lecturer in dermatology at the Greater Manchester Dermatology Centre, University of Manchester, and an honorary consultant dermatologist at the Salford Royal NHS Foundation Trust. She received her MBChB degree from the University of Cape Town, South Africa, and pursued her postgraduate training in the United Kingdom. She trained in dermatology at the North West Regional Dermatology Training Scheme. In 2008, she received her doctorate from the University of Manchester, and in 2010 she was awarded her MMedSci degree from the University of Liverpool.

Helen Young
Manchester, United Kingdom
Dr. Young, a colleague of Dr. Kleyn, is a senior lecturer at the University of Manchester, Manchester Academic Health Science Centre, Department of Dermatology, and an honorary consultant in dermatology at Salford Royal Hospital. She received her MB, ChB and PhD degrees from The University of Manchester. She trained in dermatology on the North West Regional Dermatology Training Scheme (Manchester) and at Harvard Medical School, where she was a research fellow in dermatology at the Cutaneous Biology Research Center, Massachusetts General Hospital, Boston. Her Clinical Senior Lectureship, funded by the Higher Education Funding Council for England (HEFCE), was awarded in September 2008. Dr. Young is the first dermatologist to have received this award. She is also the pharmacovigilance lead for the British Association of Dermatologists Biologic Intervention Register (BADBIR).

Both Dr. Kleyn and Dr. Young are among the consultant dermatologists who lead the Manchester Psoriasis Service, a multidisciplinary clinic for patients with severe psoriasis, which received the Hospital Doctor Dermatology Team of the Year Award in 2002.
IPC NEWS

• Effectiveness, safety and mode of action of current topicals, such as dithranol, and the safety of long-term topical corticosteroids;

• Long-term effects of topical treatments, including their value as disease modifiers and their ability to control inflammation and comorbidities;

• Safety and efficacy of topicals in juvenile psoriasis.

They also said that developing new, small-molecule treatments should be a priority.

Discussion then turned to specific areas relating to topical therapies that need more study, including prescribing them for geriatric populations, combining them with systemic therapies, occlusion, and treating scalp psoriasis.

Other topics covered during the session were:

• The use of dithranol, a unique topical therapy that can result in long-lasting remission. Researchers need a historical view of the dithranol’s use and impact, and an understanding of the drug’s mechanism of action (MOA).

• Topicals and systemic inflammation. More information is needed about when to switch from a topical to a systemic treatment. Identifying biomarkers that would determine the need for systemic therapy would help researchers understand when and how to use topicals and systemics as monotherapy or in combination.

• New treatments. Researchers need to look for therapies that: prolong duration, do not have to be used every day, convert active into non-active psoriasis sites, and have fewer side effects. Also needed is a predictive animal model of psoriasis to test such compounds.

• Formulation and compliance: Studies are needed to find new topical treatments that support long-term use. Also needed are registries to study the location and responsiveness of psoriasis plaques at different areas of the body and in different psoriasis phenotypes. Industry databases may be leveraged to make these assessments.

RESEARCH

IPC in the BJD


EDUCATION AND OUTREACH

Meet the Experts around the world

In addition to the Meet the Experts Program in Istanbul in October, IPC sponsored three other international Meet the Experts meetings in 2013. Here is an overview of these meetings.

Punta del Este, Uruguay
IPC’s first program of the year, Meet the Experts: Case-based Learning, took place April 26, during the 31st annual meeting of Latin American Dermatologists (RADLA). Co-sponsored by IPC and Sociedad Latinoamericana de Psoriasis (SOLAPSO), the meeting was co-chaired by IPC board member and past president Dr. Alan Menter of Dallas, US, and current SOLAPSO president Dra. Nélida Raimondo of Buenos Aires, Argentina. Faculty for the program included IPC councilors Dr. Edgardo Chouela, Buenos Aires, Argentina; Dr. Ricardo Romiti, São Paulo,
Brazil; and local dermatologist Dr. Néstor Macedo, Montevideo, Uruguay.

**Paris, France**

This Meet the Experts: Case-based Learning program took place July 4 during the 4th Psoriasis International Network (PIN) Congress. Topics included “Management of Generalized Pustular Psoriasis,” “Psoriasis and Pregnancy,” “Erythrodermic Psoriasis,” “Severe Hand and Foot Pustular Psoriasis,” and “Cyclosporine-Induced Renal Impairment.” Co-chairs for this meeting were IPC board member Prof. Hervé Bachelez of Paris and Dr. Wayne Gulliver of Newfoundland, Canada. Faculty members included IPC councilors Dr. Nick Reynolds, Newcastle upon Tyne, United Kingdom, and Dr. Colin Theng, from Singapore. More than 300 people attended this meeting making it one of our most successful Meet the Experts programs to date.

**Hong Kong, China**

On July 11, IPC sponsored a Meet the Experts: Case Based Learning program as part of the 9th Asian Dermatological Congress. This program was chaired by IPC board member and incoming President Professor Christopher Griffiths, from Manchester, UK. Faculty presenters included IPC board member Dr. Alan Menter, Texas, US; IPC councilor Dr. Vermén Verallo-Rowell, Makati City, Philippines; and local dermatologist Dr. Steven Loo, Hong Kong. Topics included “Can Systemic Biologic Therapy for Psoriasis Prevent Cardiovascular Disease?”, “Nail Psoriasis: Targeting the Triggers,” and “Difficulty Choosing Systemic Agents in Patient with Hepatitis B.” An audience of more than 200 people participated in this program.

**IPC founder Alan Menter honored at ‘Commit to Cure’ gala**

IPC founder, past president and emeritus board member Dr. Alan Menter was honored for his lifelong commitment to psoriasis research and treatment at a celebratory gala sponsored by the National Psoriasis Foundation.

The Foundation’s annual Commit to Cure Gala took place in October in Dallas, where Dr. Menter heads the dermatology division at Baylor University Medical Center and is clinical professor of dermatology at the University of Texas Southwestern Medical School.

Dr. Menter, who helped found IPC in 2004, was honored for his many accomplishments, including his work to help discover in 1994 the first gene associated with psoriasis. He is involved in multiple clinical, investigative and drug research studies. In 2012, Dr. Menter served as president of the 3rd World Psoriasis & Psoriatic Arthritis Conference in Stockholm, Sweden.

The gala raised more than $1.2 million to support the foundation’s work.

We congratulate our friend and colleague for this well-deserved honor. Find a Dallas Morning News profile of Dr. Menter at www.psoriasiscouncil.org/news.htm.
Advancing Knowledge | Enhancing Care

The International Psoriasis Council is a dermatology led, global nonprofit organization dedicated to innovation across the full spectrum of psoriasis through research, education and care. Our vision is to improve scientific knowledge and bring the best care to all patients with psoriasis.

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