Psoriasis from Gene to Clinic meeting

Genome-wide association studies advance understanding of genes

The 5th International Congress Psoriasis from Gene to Clinic took place Dec. 4-6, 2008, in London, UK. Held every three years and chaired by Professor Jonathan Barker and Professor Christopher Griffiths, this meeting comprised four plenary sessions dedicated to important areas of psoriasis research: genetics; immunology and immunity; co-morbidities and outcome measures; and targeted therapeutics. The conference was a combination of invited lectures and free communications from submitted abstracts. Here, we have summarized select findings from the Psoriasis from Gene to Clinic meeting.

Genetics

Genome-wide association studies (GWAS) have enabled rapid advances in our understanding of complex genetic diseases. GWAS allow the examination of approximately 100,000 to 1 million single nucleotide polymorphisms (SNPs) across the genome in large sets of cases and controls. These comprehensive surveys allow for the identification of common variants that have relatively small effects. When coupled with clinical and phenotypic information, this is a powerful tool to increase our understanding of complex genetic diseases such as psoriasis. Data from the Collaboration Association Study of Psoriasis (CASP) were presented.

This GWAS study examined 1,409 cases and 1,436 controls for approximately 450,000 SNPs. Follow-up studies were conducted in a replication cohort of 5,048 cases and 5,041 controls, and at least seven loci were implicated in psoriasis susceptibility. The greatest signal was seen in the major histocompatibility complex (MHC) region with HLA-C. Additional areas of association included interleukin (IL) 23R, IL4/IL13, TNFAIP3 (tumor necrosis factor-α-induced protein 3), TNIP1 (TNFAIP3 interacting protein 1), IL12B, and IL23A. Components of the IL-12/IL-23 pathway that have immunological relevance were implicated at the genetic level, consistent with earlier reports. In addition, IL23A which encodes p19, the subunit of IL-23 not shared with IL-12, was a novel finding in this study (Figure 1, page 5). TNIP1 and TNFAIP3 are inhibitors of TNF-α, and TNFAIP3 has been associated with other autoimmune diseases and is an important regulator of the NF-κB pathway.

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Dear Colleagues,

On behalf of the International Psoriasis Council (IPC) and this issue’s co-editors, Giampiero Girolomoni, M.D., and Kenneth Gordon, M.D., I am pleased to present the December 2008 edition of our clinical newsletter, *IPC Psoriasis Review*.

Published several times per year, *IPC Psoriasis Review* appraises important current clinical and research publications and provides commentary on those that we believe make the greatest contribution to our understanding of the disease and its treatment. In this issue, we include meeting reviews from the 17th European Academy of Dermatology and Venereology (EADV) Congress, Sept. 17-20, 2008, in Paris, France, and the 5th International Congress Psoriasis from Gene to Clinic, Dec. 4-6, 2008, in London, UK. We also present a case from our Sept. 20, 2008, IPC Meet the Experts (MTE) program, chaired by Professor Wolfram Sterry and held in Paris. Three additional cases from the MTE program can be viewed online for CME credit.

December has been a productive month for IPC. Our consensus on improving adherence to topical therapies was published in the JAAD.¹ Professor Jonathan Barker chaired the multi-disciplinary *Methotrexate Safety and Monitoring Consensus Conference*, Dec. 3, 2008, prior to the Psoriasis from Gene to Clinic meeting in London. This consensus conference was the result of a request from the European Medicines Agency (EMEA) to the IPC Board of Directors for recommendations regarding the use of methotrexate in clinical practice.

As 2008 draws to a close, we would like to thank our members and other medical professionals who have contributed to our many programs. We also acknowledge our donors and sponsors who make our work possible. IPC has a robust agenda for 2009 and we look forward to sharing our progress with you. We will continue to publish *IPC Psoriasis Review* three times per year and hold Meet the Experts symposia at international medical meetings. IPC will continue its leadership role in the co-morbidities field, building from our Sept. 5-6, 2008, consensus conference, *Psoriasis Interdisciplinary Conference on Co-morbidities and Lifestyle Modification*, held in Dallas. Finally, our Systemic Therapy Working Group and Topical Therapy Working Group will continue their efforts in these essential areas of psoriasis research and treatment.

We hope this newsletter is informative and valuable to you in treating your psoriasis patients. On behalf of the IPC Board of Directors and staff, I wish you a happy and healthy 2009.

Sincerely,

Alan Menter, M.D., President
International Psoriasis Council

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* The term “councilor” is used by IPC to honor those who have made extraordinary contributions to our understanding and treatment of psoriasis.

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**IPC Psoriasis Review**
Gene to Clinic

Continued from page 1

ZNF313/RNF114, a putative psoriasis susceptibility gene identified by Capon et al., has been independently replicated in a case control sample by Nair and colleagues. Work continues to elucidate the biological function of RNF114, which has recently been shown to be upregulated by cytokines relevant to psoriasis pathogenesis. Copy number variation is another area of interest in psoriasis genetics. Increased genomic copy number of the beta-defensin cluster on chromosome 8p23 was associated with psoriasis susceptibility. In addition, deletion of a portion of chromosome 1q21, containing late cornified envelope genes LCE3B and LCE3C, was associated with psoriasis susceptibility. LCE3C, while not expressed in normal skin, was expressed in the epidermis of psoriasis lesions.

Immunology

The Th17 pathway has generated great interest in diseases of the immune system, including psoriasis. Naive T cells differentiate into Th1, Th2 or Th17 subsets in response to stimulation by specific cytokines (Figure 1). Th17 cells are a recently identified T-cell subset that produces IL-17 and IL-22 in response to stimulation by IL-23 and are implicated in the pathogenesis of autoimmune diseases (Figure 2). In contrast, Th1 cells are stimulated by IL-12 and produce INF-γ and IL-2. While Th17 cells and Th1 cells are believed to promote the formation of psoriasis lesions, the cytokine pattern of individual T cells of the psoriasis lesion has not yet been determined. In one study, all T-cell clones isolated from psoriasis lesions expressed TNF-α but not IL-4. More than half of the clones had a cytokine pattern of IL-2, IFN-γ, IL-17 and IL-22, suggestive of Th17 cells. Other clones showed different combinations of IFN-γ, IL-17 and IL-22, suggesting that other, yet to be determined, T-cell subsets may also contribute to psoriasis development. Another study examined skin biopsies of psoriasis lesions and found that Th17 cells, identified using CCR4, CCR6, and IL-23 receptor, were present in greater amounts in psoriasis lesions (as many as half of T cells) than in peripheral blood (<5% of T cells) of psoriasis patients. A third study confirmed increased CD4+, IL-17+ and CD8+, IL-17+ T cells in psoriasis lesions. Stimulation with IFN-γ resulted in expansion of IL-17+ T cells through IL-1 and IL-23, suggesting a novel mechanism for IFN-γ in Th17 cells, as previous reports have shown INF-γ inhibits Th17 cell development. Another T-cell subset, the γδ T cell, was examined in peripheral blood and lesions of psoriasis patients. The function of γδ T cells in human skin is not well-understood, however, these cells are involved in immunosurveillance and immunoregulation in the epidermis of mice. The number of γδ T cells was reduced in the blood of psoriasis patients compared to controls. Vγ9 T cells were present in psoriasis lesions and produced IFN-γ, TNF-α, IL-4 and RANTES, but not IL-17, IL-22, and IL-6 among other cytokines.

Therapeutics

There is vast interest in understanding cellular and molecular changes in response to psoriasis treatment. IL-17 is produced by Th17 T cells, important cells in the pathogenesis of psoriasis. IL-17 isoforms (IL-17A–F) and p38 mitogen-activated protein kinase (MAPK) activity were investigated in psoriasis lesions before and after treatment with the anti-TNF-α therapy adalimumab. IL-17A, IL-17C and IL-17F were elevated in psoriasis lesions. Four days after treatment, levels of IL-17C, IL-1β and activation of p38 MAPK decreased. IL-17A, IL-17F, IL-22, IL-23 (p40 subunit) and IL-6 decreased 14 days after treatment, and IL-23 (p19) levels decreased 84 days after initiation of therapy. In similar experiments, biopsies taken four days after treatment with adalimumab showed a decrease of TNF-α in lesional skin by 50%, and P38 MAPK activity was significantly inhibited. The levels of TNF-α in biopsies were similar to that of non-lesional skin at day 84. Taken together, these results suggest that adalimumab reduces levels of p38 MAPK and IL-17 isoforms in psoriasis lesions, and inhibition of p38 MAPK may be a potential mechanism of action for adalimumab in psoriasis.
In the ACCEPT trial (described on page 5), biopsies were taken in patients treated with ustekinumab. Expression of p19 and p40, subunits of the IL-23 cytokine, were elevated in psoriasis lesions. After treatment with 90 mg ustekinumab, levels of multiple cytokines were decreased, including I-NOS, IL-23(p40), IL-23(p19), IFN-γ, IL-17 and IL-22, cytokines of Th17 cells. It was also determined that resident T cells are retained within psoriasis lesions, but the inflammatory cells are eliminated in response to therapy. These results give insight into the biology of response to anti-IL-23/23 p40 antibodies, treatments that target important cytokines of the Th17 pathway.

The PRESTA trial, a randomized, double-blind, multi-center study of psoriasis and psoriatic arthritis, compared administration of twice-weekly 50 mg etanercept to once-weekly administration. At week 12, 55% of the 50 mg twice-weekly group had achieved a PASI 75, compared to 36% of the once-weekly group. At 24 weeks, these increased to 70% and 63% respectively, but the differences were not statistically significant. The improvement in joint symptoms and safety profiles were similar with both doses. Those taking the 50 mg twice-weekly dose showed greater improvement in skin symptoms after 12 weeks.

Pharmacogenomics
Pharmacogenomics continues to be an area of scientific interest. To be able to predict the responders to a particular therapy would greatly enhance treatment decisions. Polymorphisms of the TNF promoter region have been reported to predict response to anti-TNF-α therapies in rheumatoid arthritis, ankylosing spondylitis and Crohn’s disease. One study in the United States examined polymorphisms of TNF-α and HLA-Cw6 in 683 psoriasis patients receiving systemic and biologic therapies, and some weak trends were identified. A smaller Italian study examined HLA-Cw6 status in 82 patients with psoriasis treated with efalizumab or etanercept and found that response to efalizumab but not etanercept was linked to HLA-Cw6. The presence of the HLA-Cw6 allele predicting response to efalizumab has also been reported in the Newfoundland founder population. A retrospective study of 1,200 patients on efalizumab examined polymorphisms in HLA-Cw6 but no association was seen between HLA-Cw6 and response to therapy. Additional studies were presented on searching for predictive variants of efalizumab response. More research is needed to further elucidate if polymorphisms in HLA-Cw6 or other genes can predict response to therapy.

**COMMENTARY**

The 5th International Congress Psoriasis from Gene to Clinic was superb. Our understanding of psoriasis genetics was increased with the advent of new technologies. The immunology of psoriasis, particularly the importance of Th17 cells, was further elucidated. Furthermore, clinical research studies enhanced our understanding of psoriasis pathogenesis. We certainly look forward to the next Psoriasis Gene to Clinic in London in 2011.

**References**

1. Nair et al. Genome-wide scan reveals association of psoriasis with interleukin-23, Th2 and NF-kB pathways. FC03.
2. Nair et al. QTL of psoriasis with interleukin-23, Th2 and NF-kB pathways. FC03.
3. Nair et al. Replication of association of ZNF313/RNF114 locus on chromosome 20q13 with psoriasis. FC02.
5. Schalkwijk et al. Psoriasis is associated with copy number variation of genes involved in innate immunity and skin barrier function: evidence for genetic interaction with HLA-Cw6. FC04.
7. Lewis et al. Phenotype and functional confirmation that Th17 cells constitute the major CD4 population in psoriatic lesions and are likely to have a major pathogenic role. FC13.
11. Brodmerkel et al. Ustekinumab decreases pathological immune infiltrates while normal leucocyte subsets are retained in skin and peripheral circulation. FC03.
12. Sterry et al. Results of a randomized, double blind study to evaluate the efficacy and safety of etanercept in patients with psoriasis and psoriatic arthritis: the PRESTA trial. FC05.
14. Costanzo et al. HLA-Cw6 allele confers sensitivity to efalizumab treatment in psoriasis. FC07.

**Additional reading**

The 17th European Academy of Dermatology and Venereology (EADV) Congress was held Sept. 17-20, 2008 in Paris, France. The anti-IL-12/IL-23 p40 molecules continue to show promise in clinical studies. IL-23 is composed of two subunits, p19 and p40 (Figure 1). The p40 subunit is shared with the IL-12 cytokine, which also has a p35 subunit. IL-12 and IL-23, both produced by dendritic cells and macrophages, are functionally distinct. IL-12 promotes the differentiation of naïve T cells to Th1 cells, while IL-23 promotes proliferation and survival of Th17 cells. These cytokines have been shown to play an important role in the pathogenesis of psoriasis, and polymorphisms in IL12B and IL23R have been linked to psoriasis susceptibility.

**Ustekinumab**

The ACCEPt study is the first head-to-head trial of biologic treatments in psoriasis. Here, ustekinumab, a human monoclonal antibody against the p40 subunit of IL-12/IL-23, was compared to etanercept, a receptor fusion protein that blocks TNF-α in patients with moderate to severe psoriasis. The study had the following groups: etanercept 50 mg twice weekly, 45 mg ustekinumab given at week 0, week 4, and every 12 weeks thereafter, and 90 mg ustekinumab given at week 0, week 4, and every 12 weeks thereafter. At week 12, 57% of patients treated with etanercept achieved PASI 75 compared to 68% of patients treated with 45 mg ustekinumab and 74% of patients treated with 90 mg of ustekinumab. Ustekinumab and etanercept were well-tolerated, and safety profiles were similar to those published previously for both agents.

**ABT-874**

ABT-874, another monoclonal antibody against the p40 subunit of IL-12/IL-23 released 48-week results from a phase II trial. A 36-week, blinded, observation/retreatment phase followed the 12-week placebo-controlled phase. In patients who achieved PASI 75, ABT-874 was discontinued. When response fell below PASI 50 on or before week 36, subjects received retreatment with the same dosing regimen they received in the initial 12-week period. Retreatment lasted for 12 weeks. Of the 130 patients who were responders, 72 maintained response, and 58 patients that lost response were retreated. Of those who were retreated, a majority of patients were able to again achieve a PASI 75. The percent achieving PASI 75 originally and at retreatment were as follows: 63% vs. 55% with one 200 mg dose, 93% vs. 94% with 100 mg every other week (eow), 90% vs. 69% with 200 mg weekly for 4 weeks, 93% vs. 75% 200 mg eow, and 90% vs. 83% with 200 mg weekly. Treatments were well-tolerated and appeared to have a favorable safety profile over 48 weeks.

**Current Status**

Ustekinumab is manufactured by Centocor, Inc., and ABT-874 is manufactured by Abbott Laboratories. Ustekinumab was submitted to the U.S. Food and Drug Administration (FDA) and European Medicines Agency for approval for psoriasis in December 2007. Ustekinumab (Stelara) was approved for moderate to severe psoriasis in Canada on Dec. 15, 2008. Phase III trials for ABT-874 in psoriasis are ongoing.

**COMMENTARY**

In this first head-to-head biologics trial, more patients receiving ustekinumab achieved PASI 75 at week 12 compared to those receiving etanercept; however nearly 60% of those in the etanercept group achieved the clinical end point. Both treatments were well-tolerated. ABT-874 continued to show significant efficacy in a phase II, 48-week retreatment study.

**References**


**Additional reading**

CASE 1: A 46-year-old woman with flexural psoriasis

History and presentation
This patient has a 15-year history of psoriasis with episodes of erythroderma and 30% body surface area of involvement, including with significant involvement of flexural areas and genitalia. She also has a history of psoriatic arthritis including the distal interphalangeal (DIP) joint and right knee.

What is the most appropriate treatment option?

PANELIST 1: In cases of flexural psoriasis, it is important to exclude candida. If candida is present, it should be treated. I would then initiate therapy with methotrexate to improve both skin and joint symptoms.

When beginning therapy with methotrexate, what dose do you use?

PANELIST 1: We usually start with 7.5 mg of methotrexate and then rapidly increase. We hope to be, within four weeks, at the dose of at least 15 mg per week. By starting methotrexate this way, a major improvement will not take place within the first four weeks of treatment. In the situation where a major improvement is needed and there are no risk factors, we may start with 10 or 15 mg of methotrexate, and we see the patient more frequently. Furthermore, all our patients will be taking methotrexate in combination with folates.

PANELIST 2: Unless there are risk factors, we would start methotrexate at 5 mg weekly, and we would increase in 5 mg increments to 15 mg a week. If there are no problems, we would leave her on that dose for the first 12 weeks of therapy to assess progress. We also always use folic acid, although you could argue about what dose of folic acid should be given. In any case, I think the patient should always have folic acid supplementation.

What screening do you complete prior to beginning methotrexate therapy?

PANELIST 3: We take a complete history, screen for hepatitis B and C, perform liver function tests, and place a PPD prior to initiating therapy with methotrexate.

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Faculty Disclosures
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CLINICAL COURSE
Candida superinfection was excluded. Methotrexate was started and increased to 22.5 mg/week. The arthritis improved substantially, but flexural psoriasis did not, and hair loss was a significant problem.

What intervention would you use now?

PANELIST 3: I would have initially started with a steroid/anti-yeast combination and then once the flexural psoriasis was responding, I would have transitioned the patient to a topical calcineurin inhibitor.

PANELIST 2: I agree, but I would also decrease the methotrexate dose to mitigate the hair loss.

CLINICAL COURSE
Methotrexate was reduced to 15 mg/wk and hair loss subsided. Calcitriol ointment and hydrocortisone acetate 0.1% were added. The flexural psoriasis significantly improved.

How long would you use the steroid and vitamin D combination?

PANELIST 1: I would suggest a period of three to four weeks, but no longer than eight weeks. I would then switch to tacrolimus.

PANELIST 2: I agree with a maximum of eight weeks on the steroid and vitamin D combination.

PANELIST 1: There were actually various episodes in this patient. There were times that the patient could do without any topical treatment, and from time-to-time, we gave a combination of a topical corticosteroid and an antimycotic preparation. And at other times, a vitamin D treatment alone was effective. This was all given with the methotrexate for the arthritis and also the skin.

What strategies would you use to decrease irritation of flexural regions?

PANELIST 1: Tacrolimus or pimecrolimus are good options to reduce burning and irritation. Pimecrolimus is somewhat less irritating as it is a cream formulation. If there is just slight irritation, I usually advise the patient to discontinue perhaps for one or two days and then just to try to continue the treatment.

What treatment would you use?

The audience was asked how they would treat this patient, and the choices were methotrexate, phototherapy, anti-TNF agent, anti-T cell agent, or a systemic agent with topical therapy. More than half of respondents would treat the patient with a combination of systemic and topical therapy.

![Image of bar chart showing treatment choices with systemic therapy + topical therapy at 55%, anti-T cell agent at 5%, anti-TNF agent at 18%, phototherapy at 12%, and methotrexate at 10%]

Figure 3: Audience response on how they would treat this patient

We hope this case review was informative in your assessment and treatment of psoriasis patients.