# PSORIASIS FROM GENE TO CLINIC

## 8TH INTERNATIONAL CONGRESS
THE QUEEN ELIZABETH II CONFERENCE CENTRE, LONDON, UK

## PROGRAMME & ABSTRACTS BOOK

### CO-CHAIRS

<table>
<thead>
<tr>
<th>Name</th>
<th>Location</th>
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<tbody>
<tr>
<td>Jonathan Barker</td>
<td>London, UK</td>
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<tr>
<td>Christopher Griffiths</td>
<td>Manchester, UK</td>
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</tbody>
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### LOCAL ORGANISING COMMITTEE

<table>
<thead>
<tr>
<th>Name</th>
<th>Location</th>
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<tbody>
<tr>
<td>David Burden</td>
<td>Edinburgh, UK</td>
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<tr>
<td>Catherine Smith</td>
<td>London, UK</td>
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<tr>
<td>Richard Warren</td>
<td>Manchester, UK</td>
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### SCIENTIFIC COMMITTEE

<table>
<thead>
<tr>
<th>Name</th>
<th>Location</th>
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<tbody>
<tr>
<td>Hervé Bachelez</td>
<td>Paris, France</td>
</tr>
<tr>
<td>James Elder</td>
<td>Ann Arbor, USA</td>
</tr>
<tr>
<td>Michel Gilliet</td>
<td>Lausanne, Switzerland</td>
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<tr>
<td>Lars Iversen</td>
<td>Aarhus, Denmark</td>
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<tr>
<td>Alexa Kimball</td>
<td>Boston, USA</td>
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<tr>
<td>James Krueger</td>
<td>New York, USA</td>
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<tr>
<td>Alan Menter</td>
<td>Dallas, USA</td>
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<tr>
<td>Errol Prens</td>
<td>Rotterdam, The Netherlands</td>
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<tr>
<td>Jörg Prinz</td>
<td>Munich, Germany</td>
</tr>
<tr>
<td>Peter van de Kerkhof</td>
<td>Nijmegen, The Netherlands</td>
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### ORGANISING SECRETARIAT

Psoriasis from Gene to Clinic  
Conference and Events Services  
British Association of Dermatologists  
4 Fitzroy Square  
London W1T 5HQ, UK

Tel: +44 (0) 20 7391 6358  
Email: conference@bad.org.uk  
Website: www.psoriasisg2c.com
Welcome

The outlook for patients with psoriasis has never been better. New medicines are being introduced into clinical practice that offers the prospect of long-term disease control. These advances are built upon significant insights into the immunopathogenesis of psoriasis and how it potentially relates to other conditions. But there is much more that needs to be done. For example, can immunology and genetics provide insight into the mechanisms underlying different forms of psoriasis? Can these insights translate into more targeted therapy for patients? How close are we to delivering the right treatment for the right patient at the right time?

Building on the success of our previous International Congresses, held every three years over the past 21 years, Psoriasis: from Gene to Clinic is designed to provide a forum for experts from around the world to present and discuss cutting edge issues. Delegates are anticipated to include clinicians, scientists and members of the biotechnology and pharmaceutical industries.

The Congress will be entirely plenary allowing attendees to listen to all invited and submitted oral presentations and meet all poster presenters. The programmed sessions will be dedicated to key areas of current scientific and clinical interest ranging from genetics and immune mechanisms to comorbidities and therapeutics. Stratification approaches to both the diagnosis and treatment of psoriasis will feature prominently. The scientific programme will include invited lectures from experts drawn predominantly from outside dermatology. These will include keynote lectures given by two internationally renowned experts: Professor Sir John Savill, London, UK and Dr Leroy Hood, Seattle, USA. Between these presentations there will be free communications chosen from submitted abstracts. Each day will feature sponsored lectures, poster presentations and opportunity for informal discussions.

The high quality of the meeting is reflected in our choice of venue. The Queen Elizabeth II Conference Centre is uniquely situated in the shadow of Big Ben and Westminster Abbey. The welcome reception will take place at the Conference Centre and the Congress Dinner will be held at the magnificent Natural History Museum, a unique, historic venue in the heart of London.

Winter is an excellent time to visit London and we look forward to welcoming you.

Jonathan N W N Barker
St John's Institute of Dermatology
Kings College London, UK

Christopher E M Griffiths
The Dermatology Centre
University of Manchester, UK
NOTES FOR SPEAKERS AND POSTER PRESENTERS

The Speaker Preview Room will be located in the East Long Room on the third floor. It is essential to the smooth running of the Congress that all speakers take their presentation to the Speaker Preview Room as soon as possible after their arrival at the Congress Venue but not later than 1 hour before the beginning of their session.

SCIENTIFIC POSTER DISPLAY

The scientific poster display will be situated in the Whittle Room on the third floor. The poster area will be available for presenters to mount their posters from 07:30 on Thursday 30th November. Posters are to be removed by 14:00 on Saturday 2nd December. Presenters of odd numbered posters are asked to be at their poster sites for discussion from 12:45 to 13:45 on Thursday 30th November and those with even numbers between the times of 12:15 to 13:15 on Friday 1st December.

AWARDS FOR BEST POSTER AND BEST ORAL PRESENTATION

Awards will be made to the presenters of the best oral and best poster presentation. The presentation of these awards will be made at the end of the last Congress session on Saturday 2nd December.

WELCOME RECEPTION

THURSDAY 30TH NOVEMBER  17:00 - 19:30

A welcome drinks reception will be held in the Britten Room at The Queen Elizabeth II Conference Centre at the end of the day’s scientific sessions on Thursday 30th November. All registered delegates are invited to attend. We hope this will be a perfect opportunity to relax, catch up with old acquaintances and form new friendships.

CONGRESS DINNER

FRIDAY 1ST DECEMBER  19:30 - 23:00

The Congress Dinner will be held at The Natural History Museum. With its outstanding history, the Natural History Museum is an iconic building in the heart of London. With many galleries, collections, paintings and incredible design it remains an outstanding slice of British history. A splendid three course meal will take place in the magnificent Central Hall, the home of the blue whale skeleton.

Tickets should have been purchased in advance when registering and can be found in your delegate pack.

Dress Code: Business or lounge suit

Transport for the dinner will depart from and return to the Queen Elizabeth II Conference Centre. Coaches will depart at 19:00.

CERTIFICATES OF ATTENDANCE

Certificates of attendance can be found in your delegate pack.

CLOAKROOM

The Cloakroom is located on the Ground Floor.

CPD CREDITS

Psoriasis: from Gene to Clinic has been approved for 15 credits by the Royal College of Physicians, approval number 115763. All physicians registered for CPD must record their attendance hours in accordance with the guidelines given by the RCP.

LUNCH AND REFRESHMENTS

Lunch and refreshments as indicated in the Programme are included in your registration fee.

PROFESSIONAL & PATIENT ORGANISATIONS

The following professional organisations, patient support groups, charities and psoriasis research programmes will be represented at the Congress:

International Psoriasis Council
The Psoriasis Association
The Psoriasis and Psoriatic Arthritis Alliance
British Skin Foundation
British Association of Dermatologists Biologic Interventions Register (BADBIR)
The International League of Dermatological Societies (ILDS)
UK TREND

REGISTRATION

Tel: +44 (0)20 7798 4091

The registration desk will be staffed between the following hours:

Thursday 30th November  07:30 – 17:30
Friday 1st December  07:30 – 17:30
Saturday 2nd December  08:00 – 13:30

Email: conference@bad.org.uk
Website: www.psoriasisg2c.com
## THURSDAY 30th NOVEMBER 2017

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Speaker(s)</th>
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<tbody>
<tr>
<td>07:30</td>
<td>Registration opens</td>
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<tr>
<td>10:00</td>
<td>Welcome and Introduction</td>
<td>J. Barker (London, UK)</td>
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<tr>
<td>10:15</td>
<td>Welcome and Introduction</td>
<td>C. Griffiths (Manchester, UK)</td>
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<tr>
<td>10:15</td>
<td>Scientific Session 1</td>
<td>H. Bachelez (France), E. Prens (The Netherlands)</td>
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<tr>
<td>10:35</td>
<td>FC-2 Developing a therapeutic range and predicting response to biologics in patients with psoriasis: a multicentre prospective observational cohort study</td>
<td>T. Tsakok and the PSORT Consortium</td>
</tr>
<tr>
<td>10:45</td>
<td>FC-3 The differential production of interleukin (IL)-26 vs. IL-17 by T helper 17 cells contributes to the development of different forms of psoriasis</td>
<td>A. Fries, J. Di Domizio, O. Demaria and M. Gilliet</td>
</tr>
<tr>
<td>10:45</td>
<td>Invited speaker</td>
<td>The human skin microbiome and implications for disease</td>
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<tr>
<td>11:15</td>
<td>Coffee break</td>
<td></td>
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<tr>
<td>11:45</td>
<td>SCIENTIFIC SESSION 2</td>
<td>R. Warren (UK), C. Smith (UK)</td>
</tr>
<tr>
<td>12:45</td>
<td>FC-5 Environmental antigens may trigger HLA-C*06:02-mediated autoimmunity in psoriasis</td>
<td>Y. Arakawa, A. Arakawa, S. Vural, A. Galinski, S. Vollmer and J. Prinz</td>
</tr>
<tr>
<td>13:45</td>
<td>FC-6 Analysis of psoriasis host-microbiome interactions using a universal transcriptomic approach</td>
<td>T. Furnholm, M. Foo, J. Henderson, K. Shedden and A. Johnston</td>
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### Invited speaker

12:15 – 12:45

- **Induction and regulation of Th17 Cells**
  - V. Kuchroo (Boston, USA)

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### SCIENTIFIC SESSION 2

Lunch and poster viewing

12:45 – 13:45

- **Genetic variation contributes to response to biologics: initial findings of the Psoriasis Stratification to Optimise Relevant Therapy (PSORT) consortium**
  - N. Dand on behalf of the PSORT consortium

13:45 – 13:55

- **Comparative evaluation of cellular and molecular changes associated with response to selective interleukin (IL)-23 blockade vs. dual IL-12/23 blockade in psoriasis skin**
  - K. Li, K. Campbell, S. Garret, C. Brodmerkel and J. Krueger

13:55 – 14:05

- **Functional immunophenotyping analysis reveals adalimumab-induced impairment of tumour necrosis factor signalling in lymphoid cells in psoriasis**
  - R.A. Ejarque on behalf of the PSORT consortium

14:05 – 14:15

- **The importance of academic-industrial collaboration to the future of medical research**
  - J. Savill (London, UK)

14:15 – 14:30

- **The human skin microbiome and implications for disease**
  - B. Andersson (Stockholm, Sweden)

14:30 – 14:45

- **Tea break**

15:15 – 15:45

- **Targeting IL-17: findings from recent clinical trials**
  - A. Blauvelt (Portland, USA)

15:45 – 16:15

- **The epidemiology and interrelation of psoriasis and other IL-23 related diseases**
  - A. Kimball (Boston, USA)

16:20 – 16:50

- **Welcome Reception**
  - The Queen Elizabeth II Conference Centre

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**Website**: www.psoriasisg2c.com

**Email**: conference@bad.org.uk
FRIDAY PROGRAMME

FRIDAY 1ST DECEMBER 2017

SCIENTIFIC SESSION 3
Chairs: J. Elder (USA), M. Gilliet (Switzerland)

08:00 – 08:30
LEO Pharma Sponsored lecture
Pro-inflammatory redundancy in the IL-17 pathway - the role of the individual IL-17 cytokine family members in psoriasis
J. Krueger (New York, USA)

08:35 – 09:05
Janssen Sponsored lecture
IL-23 inhibition as a strategy to treat immune-mediated inflammatory diseases
J. Prinz (Munich, Germany) & S. Danese (Milan, Italy)

09:05 – 09:15 FC – 10
The genetic basis for most patients with pustular skin disease remains elusive

09:15 – 09:25 FC – 11
ADAM17 and TIMP3 are psoriasis-relevant checkpoints controlling T helper 17 programming by inflammatory dermal dendritic cells
A. Kunze, A. Rendon, S. Oehrli, G. Murphy, A. Enk and K. Schäkel

09:25 – 09:35 FC – 12
Genotype and phenotype analyses revealed novel susceptibility genes and new clinical classification for psoriasis

09:35 – 09:45 FC – 13
Advances in genomic studies of psoriasis in China
X. Zhang

09:45 – 10:15
Invited speaker
From GWAS to systematic host-microbiome association studies in complex immune-mediated diseases
A. Franke (Kiel, Germany)

10:15 – 10:45
Coffee break

Chairs: L. Iversen (Denmark), J. Krueger (USA)

10:45 – 10:55 FC – 14
iRhomz: a mechanistic hub in keratinocyte hyperproliferation and psoriasis?
M. Brooke, T. Maruthappu, A. Chikh and D. Kellett

10:55 – 11:05 FC – 15
Identifying chromatin interactions between psoriasis-associated variants and target genes using Capture Hi-C

11:05 – 11:15 FC – 16
The psoriasis-associated ActD(D10N) variant reduces responses to interleukin-17 but enhances T helper 17 responses to polyclonal activation
S. Lambert, C. Hambro, A. Johnston, R. Nair and J. Elder

11:15 – 11:25 FC – 17
ERAP1 risk variants in psoriasis vulgaris affect autoantigen presentation
A. Arakawa, S. Vollmer, E. Reeves, E. James and J.C. Prinz

11:25 – 11:55 Invited speaker
Functional variation in the human genome: lessons from the transcriptome
T. Lappalainen (New York, USA)

11:55 – 12:05 FC – 18
Tumour necrosis factor blockade induces a dysregulated type I interferon response without autoimmunity in paradoxical psoriasis

12:05 – 12:15 FC – 19
Activation of resident T cells in resolved psoriasis reveals tissue responses that stratify clinical outcome
I.G. Sérézal, C. Classon, S. Nylen, N.X. Landén, E. Martini, S. Cheuk and L. Eidsmo

12:15 – 13:15 Lunch and poster viewing
### FRIDAY PROGRAMME CONTINUED

#### FRIDAY 1ST DECEMBER 2017 CONTINUED

**SCIENTIFIC SESSION 4**  
Chairs: A. Kimball (USA), D. Burden (UK)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Title</th>
<th>Authors</th>
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<tbody>
<tr>
<td>13:00 – 13:35</td>
<td>FC – 20</td>
<td>Longitudinal follow-up of arterial structure and function in patients with severe psoriasis treated by anti-interleukin (IL)-12/IL-23 agents compared with tumour necrosis factor inhibitors</td>
<td>M. Viguier, H. Khettab, I. Hamdidouche, P. Boutouyrie and H. Bachelez</td>
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<tr>
<td>14:25 – 14:50</td>
<td></td>
<td>Invited speaker</td>
<td>A. Gils (Leuven, Belgium)</td>
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<td>14:50 – 15:00</td>
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<td>Tea break</td>
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**SCIENTIFIC SESSION 5**  
Chairs: P. Van de Kerkhof (The Netherlands), H. Bachelez (France)

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<th>Time</th>
<th>Session</th>
<th>Title</th>
<th>Authors</th>
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<tbody>
<tr>
<td>15:30 – 16:00</td>
<td></td>
<td>Invited Lecture</td>
<td>Biology and pathology of IL-36</td>
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<tr>
<td>16:00 – 16:15</td>
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<td>AbbVie Sponsored lecture</td>
<td>The evolution of T cell targeted therapy in psoriasis</td>
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<tr>
<td>16:30 – 17:00</td>
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<td>UCB Pharma Sponsored lecture</td>
<td>Re-evaluating the role of IL-17F in immune-mediated chronic inflammation: dual neutralisation of both IL-17A and IL-17F as a novel targeting approach in psoriatic diseases</td>
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<tr>
<td>19:30 – 23:00</td>
<td></td>
<td>Congress Dinner</td>
<td>The Natural History Museum</td>
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SATURDAY PROGRAMME

SATURDAY 2ND DECEMBER 2017

SCIENTIFIC SESSION 6
Chairs: A. Menter (USA), J. Prinz (Germany)

08:40 – 09:10  **Novartis Sponsored lecture**
Disease modification, from bedside to bench
L. Iversen (Aarhus, Denmark) & L. Eidsmo (Stockholm, Sweden)

09:15 – 09:45  **Celgene Sponsored lecture**
The complex management of patients with psoriasis and comorbidities: the role of therapy choice
P. Gisondi (Verona, Italy)

09:45 – 10:05  **FC – 26**
Effectiveness, drug survival and safety of fumaric acid esters for moderate-to-severe psoriasis in routine care: results from the German Psoriasis Registry PsoBest

09:55 – 10:05  **FC – 27**
The lymphatic system plays an important role in the migration of pathogenic T cells towards synovial joints and entheses in psoriasis

10:05 – 10:15  **FC – 28**
Systemic inflammation and evidence of a cardiосplenic axis in patients with psoriasis

10:15 – 10:45  **Invited Lecture**
Challenging conventional classification dogma: towards a new clinical taxonomy of psoriasis
U. Mrowietz (Kiel, Germany)

10:45 – 11:15  **Coffee Break**

11:15 – 11:25  **FC – 29**
Efficacy and safety of risankizumab, an interleukin-23 inhibitor, in patients with moderate-to-severe chronic plaque psoriasis: 16-week results from the phase III IMMhance trial

11:25 – 11:35  **FC – 30**
Quality of care and use of systemic drugs for psoriasis in the past 12 years: results from a series of nationwide health care studies in Germany
M. Radtke, M. Augustin and K. Reich

11:35 – 12:05  **Invited Lecture**
The price and value of biologic drugs
J. Scannell (Edinburgh, UK)

12:05 – 12:15  **FC – 31**
Efficacy and safety of mirikizumab (LY3074828) in the treatment of moderate-to-severe plaque psoriasis: results from a phase II study
K. Reich, R. Bissonnette, A. Menter, P. Klekotka, D. Patel, J. Li, J. Tuttle and K. Papp

12:15 – 12:25  **FC – 32**
Cerotalizumab pegol for the treatment of patients with moderate-to-severe chronic plaque psoriasis: an overview of three randomized controlled trials
A. Blauvelt, K. Reich, M. Lebwohl, D. Burge, C. Arentt, L. Peterson, J. Drew, R. Rolleri and A. Gottlieb

12:25 – 12:35  **FC – 33**
Switching or restarting of tumour necrosis factor-α inhibitors after interruption under daily-life conditions: efficacy report from the Austrian Psoriasis Registry (PsoRA)

12:35 – 13:20  **Keynote lecture**
Systems Medicine, Big Data and Scientific Wellness are Transforming Healthcare
L. Hood (Seattle, USA)

13:20    Presentation of best oral and best poster prize
C. Griffiths (Manchester, UK)
J. Barker (London, UK)
POSTER PRESENTATIONS

30TH NOVEMBER – 2ND DECEMBER 2017
THE QUEEN ELIZABETH II CONFERENCE CENTRE – WHITTLE ROOM

P – 1  Evaluation of serum uric acid among patients with psoriasis in a developing country
S.D. Joshi and L. Limbu

P – 2  A transcriptomic study investigating the pathogenesis of generalized pustular psoriasis
M. Catapano, F. Capon, F. Ciccarelli and J. Barker

P – 3  Patient perception and the importance of clear/almost clear skin as a treatment goal in moderate-to-severe plaque psoriasis: results of the ‘Clear about Psoriasis’ worldwide patient survey

P – 4  Immunogenicity with tildrakizumab, an anti-interleukin-23p19 monoclonal antibody, in a pooled analysis of three randomized controlled trials in patients with chronic plaque psoriasis
A. Kimball, A. Blauvelt, K. Reich, Q. Li, F. van Aarle, T. Kerbusch and D. Montgomery

P – 5  Next-generation sequencing identifies epidermal microRNAs deregulated in psoriasis skin
A. Srivastava, L. Pasquali, F. Meisgen, M. Stahle, N.X. Landén, A. Pivarcsi and E. Sonkoly

P – 6  Effect of adipose-derived stem cells on an imiquimod-induced psoriasiform mouse model by hypodermic injection
J. Deng, C. Lu, L. Han and Y. Huang

P – 7  Impairment of gustatory and olfactory senses in plaque psoriasis
P. Rüter, V. Grünthaler, Y. Zopf and M. Sticherling

P – 8  Psoriasis in children: a single-centre analysis
F. Hempt, J. Raap and M. Sticherling

P – 9  Interleukin-36γ detection via noninvasive tape stripping reliably diagnoses psoriasis

P – 10 Caffeine in the treatment of atopic dermatitis and psoriasis: a review
M. Alashqar and N. Goldstein

P – 11  Population pharmacokinetic modelling of tildrakizumab (MK-3222), an anti-interleukin-23p19 monoclonal antibody, in healthy volunteers and patients with psoriasis
P. Jauslin, P. Kulkarni, R. Wada, S. Vatakuti, A. Hussain, L. Wenning and T. Kerbusch

P – 12 Poster withdrawn.

P – 13  Immune modulation by topical PH-10 aqueous hydrogel (rose Bengal disodium) in psoriasis lesions

P – 14  Favourable safety profile of ixekizumab: results from 11 moderate-to-severe psoriasis and three psoriatic arthritis clinical trials
A. Gottlieb, K. Papp, W. Xu, L. Mallbris and N. Agada

P – 15 Poster withdrawn.

P – 16  Cytokine effects of apremilast as a mechanism of efficacy in systemic-naive patients with moderate plaque psoriasis: results from the UNVEIL trial
B. Strober, M. Aliskhan, B. Lockshin and P. Schafer

P – 17  Patient- and physician-reported outcomes with apremilast for patients with plaque psoriasis during routine dermatology care in Germany: an interim analysis
K. Reich, S. Bonasi, B. Korge, M. Manasterski, U. Schwichtenberg, H. Mentz, K. Groegel and N. Núñez Gómez

P – 18  Shear wave elastography in patients on high-dose methotrexate: a prospective study
D. Kivelevitch, R. Rahimi and A. Menter

P – 19  Do patients with certain human leucocyte antigen expression have a higher risk of developing psoriasis?
A. Anandan, K. Radhakrishnan, R. Prasada and V.K. Panicker

P – 20  Utility study to map utilities to the Psoriasis Area and Severity Index and Dermatology Life Quality Index instruments in patients with chronic plaque psoriasis
C. Baker, J. Sullivan, P. Davey and J. Wilson

P – 21  Secukinumab shows high and sustained efficacy in nail psoriasis: 2.5-year results from the TRANSFIGURE study
P – 22  Secukinumab pooled and long-term safety: analysis of 19 psoriasis clinical trials
P. van de Kerkhof, K. Reich, C. Leonardi, A. Blauvelt, N. Mehta, T.-F. Tsai, R. You, P. Papanastasiou, M. Milutinovic and C.E.M. Griffiths

P – 23  Lysosomal action in the regulation of inflammatory processes on the example of psoriasis
K. Bocheńska, M. Moskot, E. Smolińska, J.J.-Banecka and M. Gabig-Cimińska

P – 24  Secukinumab demonstrates significantly lower immunogenicity potential than ixekizumab in human in vitro assays
S. Spindeldreher, B. Maillère, E. Correia, A. Karle, P. Jarvis and F. Kolbinger

W. Chen, Y. Gong, X. Zhang, Y. Tong, X. Wang, C. Fei, H. Xu, Q. Yu, Y. Wang and Y. Shi

P – 26  Secukinumab shows high and sustained efficacy in patients with moderate-to-severe palmoplantar psoriasis: 2.5-year results from the GESTURE study

P – 27  Efficacy and safety of infliximab in the treatment of the Chinese patients with psoriasis

P – 28  Secukinumab clinical outcomes in a tertiary referral centre
O. Jagun, S.T. Cheung, O. Jagun and S.T. Cheung

P – 29  Sustained response to adalimumab over multiple years in patients with plaque psoriasis: analyses from the British Association of Dermatologists Biologic Interventions Register (BADBIR)
B. Kirby, J.-F. Maa, T. Festini, B. Calimlim and O.R. Servin

P – 30  Successful treatment of psoriasis with secukinumab after ustekinumab in a patient with multiple sclerosis
S. Kaneko, H. Oguro and E. Morita

P – 31  Development of pulmonary sarcoidosis in a patient with psoriasis under treatment with ustekinumab: comorbidity or drug-related ‘paradoxical’ phenomenon?
C. Fotiadou, E. Lazaridou, E. Sotiriou and D. Ioannides

P – 32  Factors associated with the choice of the first biologic in psoriasis: real-life analysis from the Psobioteq cohort

P – 33  The Psoriasis Association as a role model for other support groups: how far can (dare) we go?
H. H. Oon

P – 34  Paradoxical psoriasis caused by tumour necrosis factor inhibitor therapy: a model system to study the interplay between environmental triggers and genetic susceptibility?
T. Maruthappu, A. Connolly, S. Mahil, B. Kirkham, P. DiMeglio and C. Smith

P – 35  The coexistence of generalized pustular psoriasis and pemphigus foliaceus in a woman with Cushing syndrome: a case report
A. Kusumawardani, S.E. Ilona, D.A. Mira and Suradi Radiono

P – 36  Retrospective audit on psoriasis, assessment and management: National Institute for Health and Care Excellence guideline CG153 within a dermatology department
M. Verma, A. Leong and S. Velangi

P – 37  Role of Thevetia neriifolia in the treatment of psoriasis: clinical case report
D. Maryam, S. Souad, O.S. Charifa and S.-B. Rachida

P – 38  Large-scale imputation of killer cell immunoglobulin-like receptor copy number in psoriatic arthritis

P – 39  Psoriasis following PD-1 inhibitor therapy: features and treatment
P. O’Connor and J.P. Dutz

P – 40  Evaluation of body composition in patients with psoriasis treated with ustekinumab
M. Galburzo, S. Dadamio, R. Pastorino, L. Bianchi and M. Talamonti

P – 41  Characteristics and risk profile of patients with psoriasis included in the Turkish national registry PSR-TR
N. Onsun, E.B. Baskan, D. Dizman, D.B. Ozkaya, A.C. Erklic, G. Ozarmagan and M.A. Gürer

P – 42  Treatment profile of patients with moderate-to-severe psoriasis included in the Turkish national registry PSR-TR
N. Onsun, E.B. Baskan, D. Dizman, D.B. Ozkaya, A.C. Erklic, G. Ozarmagan and M.A. Gürer
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<th>Poster Number</th>
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<th>Authors</th>
</tr>
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<tbody>
<tr>
<td>P – 43</td>
<td>An ongoing independent study to monitor the uptake of interleukin-17 inhibitors among U.S. dermatologists</td>
<td>J. Robinson and L. Price</td>
</tr>
<tr>
<td>P – 46</td>
<td>Comanagement with rheumatologists for patients with psoriatic arthritis receiving treatment with a biological agent or apremilast</td>
<td>J. Robinson and L. Price</td>
</tr>
<tr>
<td>P – 48</td>
<td>The interleukin-17A/F heterodimer regulates psoriasis-associated genes through IκBζ</td>
<td>T. Bertelsen, C. Johansen and L. Iversen</td>
</tr>
<tr>
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<td>The psoriasis-associated interleukin-17A induces and cooperates with interleukin-36 cytokines to control keratinocyte differentiation and function</td>
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INDUCTION AND REGULATION OF TH17 CELLS

PROFESSOR VIJAY KUCHROO
Boston, USA

Dr. Vijay Kuchroo is the Samuel L. Wasserstrom Professor of Neurology at Harvard Medical School, Senior Scientist at Brigham and Women's Hospital, and Co-Director of the Center for Infection and Immunity, Brigham Research Institutes, Boston. Vijay Kuchroo is also an associate member of the Broad Institute and a participant in a Klarman Cell Observatory project that focuses on T cell differentiation. He was just named the Director of the newly formed Evergrande Center for Immunologic Diseases at Harvard Medical School and Brigham and Women's Hospital. His major research interests include autoimmune diseases - particularly the role of co-stimulation - the genetic basis of experimental autoimmune encephalomyelitis and multiple sclerosis, and cell surface molecules and regulatory factors that regulate induction of T cell tolerance and dysfunction. His laboratory has made several transgenic mice that serve as animal models for human multiple sclerosis. Dr. Kuchroo first described the inhibitory receptor TIM-3, which is being exploited as a target for cancer immunotherapy. He was first to describe the development of highly pathogenic Th17 cells, which has been shown to induce multiple different autoimmune diseases in humans. He has published over 325 original research papers in the field of Immunology and a paper describing development of Th17 authored by Dr. Kuchroo has been one of the highest cited papers in Immunology.

Dr. Kuchroo came to the United States in 1985 and was at the National Institutes of Health, Bethesda as Fogarty International Fellow for a year before joining the department of pathology at Harvard Medical School as a research fellow. He later joined the Center for Neurologic Diseases at Brigham and Women's Hospital as a faculty member in 1992. He obtained his degree in Veterinary Medicine from the College of veterinary medicine, Hisar, India. Subsequently, he specialized in pathology at the University of Queensland, Brisbane (Australia) where he obtained a Ph.D. in 1985. He received the Fred Z. Eager Research prize and medal for his Ph.D. research work at the University of Queensland. Based on his contributions, he was awarded the Javits Neuroscience Award by the National Institutes of Health in 2002 and the Ranbaxy prize in Medical Research from the Ranbaxy Science Foundation in 2011. He was named Distinguished Eberly lecturer in 2014 and obtained Nobel Laureate Peter Doherty lecture/prize in 2014.

Dr. Kuchroo has 25 patents and has founded 6 different biotech companies. He also serves on the scientific advisory boards of a number of big pharmaceutical companies including Pfizer, Novartis, Sanofi/Genzyme and Glaxo-Smith-Klein (GSK).

ABSTRACT

IL-17 producing Th17 cells are distinct from T_{h1} or T_{h2} cells and have been shown to play a crucial role in the induction of multiple human autoimmune diseases including psoriasis, psoriatic arthritis, ankylosing spondylitis and multiple sclerosis. Th17 differentiation is accomplished by three overlapping steps: Induction, Amplification and Stabilization mediated by distinct cytokines. Whereas TGF-β+ IL-6 or IL-1+ IL-6 induces them, IL-21 amplifies Th17 cells. IL-23 stabilizes the phenotype of Th17 cells. Loss of any of the cytokines (TGF-β, IL-1, IL-6, IL-21 or IL-23) in the pathway results in a defect in generation of Th17. However not all Th17 cells are pathogenic and induce autoimmunity. IL-23 is a key cytokine that induces pathogenicity in Th17 cells (Lee et al., 2012). Using expression profiling at very high temporal resolution, novel computational algorithms and innovative nano-wire based “knock-down” approaches, we have developed a regulatory network that governs the development of Th17 cells. In addition to high-density temporal microarray analysis, we have performed single-cell RNA-seq of Th17 cells in order to characterize cellular heterogeneity, identify subpopulations, functional states and learn how gene expression variation affects Th17 functional states. We have identified novel regulators of Th17 cells both in vivo and in vitro that do not affect Th17 differentiation but affects pathogenic vs. non-pathogenic functional states of Th17 cells. One of the regulators CD5L (CD5like) has both cell intrinsic and cell extrinsic effects. Soluble forms of CD5L makes homo and heterodimers and regulates differentiation of Th17 cells and inhibit development of tissue inflammation and autoimmunity.
THE HUMAN SKIN MICROBIOME AND IMPLICATIONS FOR DISEASE

PROFESSOR BJORN ANDERSSON
Stockholm, Sweden

Björn Andersson received his PhD in 1992 and was a post-doctoral fellow at Baylor College of Medicine 1992-1995. He is now, after working as an assistant and associate professor at Uppsala University and Karolinska Institutet, a professor at the Department of Cell and Molecular Biology, Karolinska Institutet since 2007. With a background in human genetics and the human genome project, his own laboratory has focused on the study of human pathogens using genomics and bioinformatics methods. He has, for example, pioneered studies on protozoan genomes by leading the Trypanosoma cruzi genome project and viral microbiome projects (published in Science 2005, PNAS 2005, J. Virol. 2007), as well as participated in a large skin microbiome study.

ABSTRACT

The involvement of the human microbiome in disease is currently a rapidly developing field. The skin microbiome has been characterized in smaller sample sets and mainly in healthy individuals, in order to understand microbial diversity in skin homeostasis, but the relevance of microbial dysbiosis in inflammatory disease is still relatively unexplored. We have carried out the microbiome part of a large European study of the development of allergic and autoimmune skin disease, in this case atopic dermatitis and psoriasis. We have deeply sequenced skin microbial communities both by 16S sequencing and shotgun sequencing, and coupled this with transcriptome data of cutaneous gene expression in skin biopsies from the largest patient cohort characterized to date. The analysis of the 16S data showed that the microbiome signatures of atopic dermatitis and psoriasis samples showed clear differences from those of healthy volunteers. We were able to associate the microbiome with changes in gene expression for both diseases and identify inflammatory pathways of interest. The shotgun data has been analyzed and has deepened the analysis to include specific genes and strains as well as fungi and viruses. The analysis is still ongoing and the latest results will be discussed. These data sets provide information that can lead to the development of new biomarkers as well as new insight into factors important for skin diseases and symptoms.

SPEAKER BIOGRAPHIES

THE IMPORTANCE OF ACADEMIC-INDUSTRIAL COLLABORATION TO THE FUTURE OF MEDICAL RESEARCH

PROFESSOR SIR JOHN SAVILL
London, UK

Professor Sir John Savill BA, MBChB, PhD, FRCP, FRCPE, FASN, FMedSci, FRS, FRSE, a clinician scientist from Edinburgh, took up the position as chief executive and deputy chair of the Medical Research Council on 1 October 2010. The appointment was initially for three years; after which it was extended until April 2016, and subsequently to 30 September 2018. He was a member of the Council from 2002 to 2008 and chaired two Research Boards during this period.

Between 2008 and 2010 John worked part-time as the chief scientist for the Scottish Government Health Directorates.

He was knighted in the 2008 New Year Honours List for services to clinical science.

John started his research career with a degree in Physiological Sciences from Oxford University in 1978, followed by degrees in Medicine at the University of Sheffield in 1981. He received a PhD from the University of London in 1989.

After junior hospital appointments in Sheffield, Nottingham and London, he spent seven years in the Department of Medicine at Hammersmith Hospital with spells as an MRC clinical training fellow and Wellcome Trust senior clinical research fellow.

In 1993, he moved to the chair of medicine, at the University of Nottingham, then in 1998 became professor of medicine at the University of Edinburgh, where he was the first director of the University of Edinburgh/MRC Centre for Inflammation Research, directing a group interested in the molecular cell biology of renal inflammation.

In 2002, John was appointed as the first vice-principal and head of the College of Medicine and Veterinary Medicine, University of Edinburgh. He retains an ongoing, research active involvement with the University of Edinburgh part time throughout his appointment as our chief executive.
LILLY SPONSORED LECTURE

TARGETING IL-17: FINDINGS FROM RECENT CLINICAL TRIALS.

DR ANDREW BLAUVELT
Portland, USA

Andrew Blauvelt hails from Portland, OR, USA, and is President and Investigator at Oregon Medical Research Center – a small company dedicated to conducting high-quality clinical studies in dermatology. A native of Michigan, Dr. Blauvelt received degrees in Electrical Engineering at Purdue University (West Lafayette, IN), and Medicine at Michigan State University (East Lansing, MI), before completing his MBA at Portland State University/Oregon Health & Science University (OHSU, Portland, OR). Dr. Blauvelt trained in dermatology at the University of Miami from 1989–1992 and in basic immunology and virology in Dr. Steve Katz’s laboratory at the National Institutes of Health (NIH) from 1992–1996. He has held senior staff positions at the NIH and more recently was Professor of Dermatology and Microbiology at OHSU and Chief of Dermatology at the Portland VA Medical Center.

Dr. Blauvelt’s clinical and research expertise spans immunology, virology, infectious diseases, psoriasis, atopic dermatitis, and biologic therapies for complex medical dermatology patients. He has published more than 200 papers and spoken globally on these topics. Dr. Blauvelt is an elected member of both the American Society for Clinical Investigation, a leading society for physician-scientists; and the International Psoriasis Council, a premier group of psoriasis experts worldwide. More recently, he has participated extensively in clinical trials assessing biologic therapies for atopic dermatitis, and has served as a lead scientific advisor for companies working in this area.

ABSTRACT

This will be an overview of recent Lilly clinical trial data.
This presentation has been organised and funded by Lilly and Lilly products will be discussed in this session.
PROFESSOR ALEXA KIMBALL
Boston, USA
Alexa Boer Kimball, MD, MPH is President and Chief Executive Officer of Harvard Medical Faculty Physicians at BIDMC, Inc., an academic multi-specialty group practice with over 1200 Harvard Medical School faculty members at Beth Israel Deaconess Medical Center in Boston, Massachusetts and an additional 400 physicians based in the community. She is also President of the Beth Israel Deaconess Care Organization (BIDCO) Physician LLC which maintains a membership of approximately 2500 physicians. A Professor of Dermatology at Harvard Medical School, Dr. Kimball’s areas of research include psoriasis and hidradenitis suppurativa. She has published over 275 papers, is the author of the book "100 Questions and Answers about Psoriasis," which has been translated into Spanish, Greek, and Korean, and editor of "Dermatologic Diseases and Cumulative Life Course Impairment." Dr. Kimball is widely recognized for her research on physician workforce economics, quality of life, and outcomes, for which she was awarded the American Skin Association Research Award for Health Policy and Medical Education and the Mass General Hospital Bowditch Prize for increasing quality of care while reducing costs. Dr. Kimball has served on multiple non-profit Boards including: the Society for Investigative Dermatology, where she was elected Vice President, the Massachusetts Foundation for the Humanities and Public Policy, and the Hidradenitis Suppurativa Foundation. She was previously Senior Vice President of the Massachusetts General Physician Organization, which employs more than 2500 physicians, and also served as Medical Director (chief medical officer) of this organization. She is current President of the International Psoriasis Council.

ABSTRACT
The elucidation of the role of IL-23 in psoriasis and other diseases has been a fascinating story of concurrent and synergistic discovery driven by laboratory findings, translational investigation, clinical observation, and epidemiology. As has often been the case in dermatology, clinical observation drove some of the early hypotheses. The clinical and laboratory observations that p40 inhibitors that affected both IL12 and a relatively newly recognized cytokine in a novel pathway led to clarifications that ultimately revealed the TH-17 pathway and the pivotal role of IL23. Comorbid conditions in patients with psoriasis were being described in depth in parallel and soon thereafter, genetic epidemiology confirmed a biologic basis for these relationships. Yet there appear to be interesting biologic differences between the effects across these related comorbid but disparate diseases which will be discussed. Our understanding of these differences and the fundamental biology will only improve as we observe the effects of new agents that target the p19 moiety of IL-23 and therefore are expected to have even more targeted effects in clinical practice.

PROFESSOR JAMES KRUEGER
New York, USA
James G. Krueger, MD, PhD is Head of the Laboratory for Investigative Dermatology at the Rockefeller University. He also serves as a Physician, Co-director, Center for Clinical and Translational Science at the Rockefeller University Hospital, and Chief Executive Officer of the Rockefeller University Hospital in New York City. Dr. Krueger earned his bachelor’s degree from Princeton University and a PhD in virology and cell biology from the Rockefeller University. He received an MD from Cornell University Medical College, where he also completed an internship in internal medicine and residency in dermatology. Dr. Krueger is certified by the American Board of Dermatology.

His research group at Rockefeller was the first to conduct clinical trials with specific, targeted immune antagonists in psoriasis and this work established that elimination of pathogenic T-cells from skin lesions could reverse the full pathological phenotype of psoriasis. Since then his group has used immune-based therapeutics to dissect inflammatory pathways in psoriasis and to conduct parallel pharmacogenomic studies that define mechanisms of targeted therapeutics in human populations. A more recent focus has been definition of new inflammatory pathways, as well as new types of inflammatory cells in psoriasis lesions that are now being targeted with new biologic drugs. He has been an advocate of bidirectional translational research (bench to bedside and back) in humans using psoriasis as a model inflammatory disease to dissect pathogenic pathways that cannot be studied in animal models.

ABSTRACT
The interleukin-17 (IL-17) pathway is central in the pathophysiology of psoriasis as evidenced by the high levels of skin clearance achieved by therapies that directly target the IL-17 cytokine or it’s receptor. Increased expression of IL-17 cytokines, which signal through the IL-17 receptor complex directly on the surface of keratinocytes and immune cells, leads to release of pro-inflammatory mediators, resulting in inflammation and clinical manifestations of psoriasis. IL-17 is not a single cytokine, but comprises a family of 6 pro-inflammatory cytokine members: IL-17A to IL-17F. IL-17A, IL-17C, IL-17E, IL-17F and the IL-17A/F heterodimer have all been described to have a role in psoriasis.

This lecture will review the current understanding of each IL-17 family member and their role in psoriasis, presenting new transcriptomics data, and linking it to a better understanding of the pro-inflammatory redundancy in the IL-17 pathway.
**SPEAKER BIOGRAPHIES**

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### JANSSEN SPONSORED LECTURE

**IL-23 INHIBITION AS A STRATEGY TO TREAT IMMUNE-MEDIATED INFLAMMATORY DISEASES**

**PROFESSOR JÖRG PRINZ**

Munich, Germany

Jörg Prinz is Professor of Dermatology and Venereology at the Clinic for Dermatology and Allergology of the Ludwig-Maximilian University (LMU) in Munich, Germany. He graduated with a medical degree in 1983. After 5 years of basic immunology research as a postdoctoral fellow, he joined the Clinic for Dermatology and Allergology in 1990, where he founded the Research Group for Immunopathology. Professor Prinz earned specialist qualifications in dermatology and venereology in 1995 and in allergology in 1996, and was appointed as Full Professor of Dermatology in 2001. His current responsibilities include being deputy chair of the department and supervising the phototherapy unit and psoriasis centre, and the serological analysis laboratory. At medical school he received several rewards for his commitment to education.

Professor Prinz’s main research interest is in the analysis of the T-cell mediated immunopathogenesis of psoriasis and the identification of psoriatic autoantigens; he is also interested in developing experimental therapies for T-cell mediated autoimmune disorders, and has been actively involved in preparing evidence-based guidelines for psoriasis treatment. He has published extensively in major peer-reviewed journals.

Professor Prinz is a member of the European Academy of Dermatology and Venereology, the International Psoriasis Council, and the International Federation of Psoriasis Associations. He was the Scientific Chairman of the 2nd World Psoriasis and Psoriatic Arthritis Conference in Stockholm, Sweden, in 2009.

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**PROFESSOR SILVIO DANES**

Milan, Italy

Silvio Danese is Head of the Inflammatory Bowel Disease (IBD) Center, Division of Gastroenterology at Humanitas Research Hospital and Professor in Gastroenterology at Humanitas University, both in Milan, Italy. He trained in gastroenterology at Policlinico Gemelli, Rome, Italy, and earned his PhD there in 2004. Professor Danese also worked in Prof. Claudio Fiocchi’s laboratory at the Case Western Reserve University, Cleveland, OH, USA from 2001 to 2004.

His main research area of interest is the investigation of the fundamental mechanisms underlying IBD pathogenesis, while his daily clinical activity is related to IBD service. He is actively involved in many international clinical trials in IBD-related areas and has published more than 300 papers in peer-reviewed journals, including Gastroenterology, Gut, Journal of Clinical Investigation, Nature, and Journal of Immunology.

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**ABSTRACT**

The recent introduction of novel treatments targeting the IL-23/Th17 immune pathway has had major implications for our management of patients with moderate-to-severe plaque psoriasis, and potentially for management of patients with other immune-mediated inflammatory diseases (IMIDs) such as inflammatory bowel disease (IBD). Patients suffering from moderate-to-severe psoriasis now have additional treatment options that have been shown to significantly improve skin clearance and reduce the debilitating symptoms such as itch, pain, stinging, burning, and skin-tightness.

In our presentations, we will show how the effectiveness of IL-23 inhibition in treating IMIDs is based on a thorough understanding of the role of IL-23 in disease pathogenesis. Clinicians have been able to formulate effective management strategies for day-to-day clinical practice based on an understanding both of this role and of how the different IL-23 inhibitors act and where they have effect.

Our presentation will include:
- The IL-23/Th17 immune pathway in psoriasis and other IMIDs: activation, differentiation, and amplification, and the implications for patient treatment
- A review of the latest clinical data on IL-23-specific inhibitors
- Practical lessons learned from the use of these agents in day-to-day clinical practice

We will also address unmet medical needs in the management of IMIDs, and demonstrate how these needs can be met by IL-23 inhibition.

Importantly, and uniquely, we will also offer insights from both the dermatological and gastroenterological perspectives, highlighting the benefits of a cross-specialty approach in managing IMIDs.
FROM GWAS TO SYSTEMATIC HOST-MICROBIOME ASSOCIATION STUDIES IN COMPLEX IMMUNE-MEDIATED DISEASES

PROFESSOR ANDRE FRANKE
Kiel, Germany

Professor Franke’s main scientific interests are the development and establishment of novel high-throughput technologies, the inherent bioinformatic integration and application of both to identify genetic and epigenetic causes of chronic inflammatory diseases such as Crohn's disease, ulcerative colitis, psoriasis, primary sclerosing cholangitis, and atopic eczema. Having worked on genome-wide association studies for the last years, his research agenda currently focuses on clinical data management, whole-genome and whole-exome resequencing, microbiome analyses and immunogenetics studies.

ABSTRACT

Genome-wide association (GWAS) have significantly contributed to our understanding of the etiology of chronic and complex immune-mediated diseases (CID). Inflammatory bowel diseases (IBD) with its main subphenotypes Crohn’s disease and ulcerative colitis, are prototypic CIDs that affect about 2-3 persons out of a 1000 in Western countries. IBD shares part of its genetic and immunological background with diseases like psoriasis, ankylosing spondylitis, and primary sclerosing cholangitis. To this end, over 250 genetic susceptibility loci were identified in the past 10 years through GWAS and candidate-gene association studies. Complex immunogenetics efforts are currently being undertaken to solve CID. Still, the exact cause of most CID has not been identified and components of the gut microbiome are also likely disease-causing environmental factors for CID. In my talk I will focus on our ongoing efforts in host-microbiome association analyses and allude to the methodological challenges of these kind of analyses. I will show immunogenetics and host-microbiome interaction approaches, which could serve as a role model for psoriasis research projects.

FROM GENOME WIDE ASSOCIATION TO FUNCTIONAL RELEVANCE

DR TUULI LAPPALAINEN
New York, USA

Tuuli Lappalainen is an Assistant Investigator and Core Member at the New York Genome Center, and an Assistant Professor in the Department of Systems Biology at Columbia University. She got her PhD from University of Helsinki in 2009, and did postdoctoral research with Manolis Dermitzakis at University of Geneva, and Carlos Bustamante at Stanford University. Her research focuses on functional genetic variation in human populations and its contribution to traits and diseases. She has pioneered in integration of large-scale genome, transcriptome and epigenetic data to learn how genetic variation affects gene regulation – the largely unknown factor that underlies the majority of human diversity and disease risk. Her research group tackles these questions by both computational analysis of large data sets and experimental work using genome editing assays. She has contributed to several international research consortia in human genomics, including the 1000 Genomes Project, the Geuvadis project, and the Genotype Tissue Expression (GTEx) Project.

ABSTRACT

Detailed characterization of cellular effects of genetic variants is essential for understanding biological processes that underlie genetic associations to disease, as well as basic genome function. One approach to address this challenge is map genetic effects on the transcriptome across multiple human tissues and conditions. In this talk, I will discuss our recent work in integrating large-scale data sets of genome and transcriptome variation to understanding genetic regulatory variants, genetic effects on transcriptional response to immune stimuli, and their contribution to autoimmune and other diseases.
WHY BIOLOGICS FAIL

PROFESSOR ANN GILS
Leuven, Belgium

As a pharmacist, Ann Gils obtained her PhD in Pharmaceutical Sciences in 1997. She is a full professor at the Laboratory for Therapeutic and Diagnostic Antibodies, Department of Pharmaceutical and Pharmacological Sciences, KU Leuven, Belgium. The core technology of the laboratory is the development of monoclonal antibodies and antibody fragments with the aim to develop these antibodies either as therapeutics or as diagnostics.

In the past few years, she has developed and validated a number of assays to measure the serum and anti-drug antibody concentrations of biologicals (infliximab; adalimumab; etanercept, golimumab, vedolizumab, secukinumab and ustekinumab) and has participated in Therapeutic Drug Monitoring studies in the field of gastroenterology, rheumatology and dermatology. Through PKPD modeling, she studies the effect of co-variates on PK and PD. She has published more than 200 papers in the field of thrombosis and therapeutic drug monitoring of biologicals.

ABSTRACT

Chronic inflammatory diseases such as rheumatic diseases, spondyloarthritis, inflammatory bowel diseases and plaque psoriasis have a high prevalence and typically start early in life, thereby strongly affecting the quality of life and productivity of young and active individuals. Patients have to be treated life-long and this requires safe, tolerable and cost-effective treatments. The introduction of biologics has revolutionized the short- and long-term outcome of patients with chronic inflammatory diseases. Unfortunately, 10-30% of patients (primary non-response) will not respond to treatment and 30-60% of patients initially responding will lose clinical benefit during treatment (secondary loss of treatment).

Primary non-response is defined as a lack of clinical response to the treatment, assessed 8-12 weeks after initiation. It is believed that this primary non-response is mainly driven through mechanistic override of the disease. Another explanation is that primary non-response is caused by non-sufficient exposure to the drug. A drug can only exert its pharmacological effect when adequate concentrations of drug are achieved in the circulation and at the drug's site of action. Inter-and intra-individual differences in bioavailability and pharmacokinetics may contribute to the problem of insufficient exposure. Secondary loss of response is defined as a loss of clinical benefit after initially responding to the drug. The secondary loss of response can either be attributed to disease-related factors or drug-related factors such as the formation of anti-drug antibodies.
BIOLOGY AND PATHOLOGY OF IL-36

DR JENNIFER TOWNE  
San Diego, USA

Dr. Towne completed her doctoral degree in the Department of Molecular Genetics, Biochemistry, and Microbiology at the University of Cincinnati School of Medicine where she studied the role of aquaporins in pulmonary inflammation and edema. She subsequently joined Immunex as a post-doctoral fellow investigating the role of novel IL-1 family members in physiology and pathophysiology. Dr. Towne was hired at Immunex as a full time scientist and worked at Immunex/Amgen for 13 years focusing first on the biology of IL-1 family members, primarily IL-36, IL-17 family members, and IL-23 in the skin, lung and gut. She led or was a key contributor to multiple large and small molecule project teams currently in clinical development for psoriasis and inflammatory bowel disease (IBD). For the last several years she was responsible for developing the strategy and leading the IBD discovery team at Amgen. Dr. Towne started at Janssen in November 2014 as Scientific Director, Immunology Discovery, where she was responsible for the IBD discovery efforts on the West coast. Dr. Towne is currently the discovery lead for the IBD disease area stronghold across Janssen where she is responsible for discovery efforts to develop innovative therapeutics for the treatment, prevention and cure of IBD. Dr. Towne is a longstanding member of the International Cytokine and Interferon Society (ICIS) and has served as the co-chair of the awards committee and of the publication committee.

ABSTRACT

IL-36α, IL-36β and IL-36γ are members of the IL-1 family of cytokines that signal through a common receptor composed of IL-36R and IL-1RAcP to activate NFκB and mitogen activated protein kinases such as p38 and JNK and promote inflammatory responses. IL-36Ra is a natural antagonist of the three IL-36 agonists that binds to IL-36R, inhibits binding of the agonistic ligands, does not recruit the co-receptor IL-1RAcP and does not stimulate any intracellular responses. These cytokines are expressed predominantly by epithelial cells and act on a number of cells including immune cells, epithelial cells and fibroblasts. Processing of the N-terminus is required for full agonist or antagonist activity for all IL-36 members. The role of IL-36 has been extensively demonstrated in the skin where it can act on keratinocytes and immune cells to induce a robust inflammatory response and is implicated strongly through both functional and genetic evidence in the pathology of psoriatic disorders. Emerging data also suggests a role for this cytokine family in pulmonary and intestinal physiology and pathology. Although much has been learned about the biochemistry of IL-36 and its role in various tissues, it is clear that we are at an early stage in our understanding of the full biology of these cytokines.
ABBVIE SPONSORED LECTURE

THE EVOLUTION OF T CELL TARGETED THERAPY IN PSORIASIS

PROFESSOR JONATHAN BARKER
London, UK

Jonathan Barker is Professor of Medical Dermatology and Head of St John’s Institute of Dermatology, King’s College London and honorary consultant dermatologist at Guy’s and St. Thomas’s Hospitals. He specialises in complex hospital based medical dermatology. He is co-director of the severe psoriasis service and Skin Therapy Research Unit. His research extends from genetic discovery through to clinical outcome measurement. It has been funded from multiple sources including European Union, Medical Research Council (MRC UK), National Institute for Health Research, medical charities and industry. He is a key investigator in national and international consortia aiming to identify genetic susceptibility to important chronic skin diseases such as psoriasis, acne and eczema and in biomarker discovery of outcomes to interventions.

Professor Barker is course director of the Institute’s MSc Clinical Dermatology for overseas graduates. This internationally renowned course has existed for more than 40 years and has helped train many dermatologists around the globe extending from the Caribbean through Africa to the Middle and Far East.

Professor Barker has published over 250 peer-reviewed papers, authored and edited several books including the new edition of the ‘Rook Book’- the most comprehensive textbook in the English language. Highly cited publications include those in the Lancet, Nature Genetics and New England Journal of Medicine. He sits on the editorial boards of several dermatology journals. He has delivered plenary keynote lectures at many meetings internationally including Dohi Memorial Lecture at Japanese Dermatology Association in 2016 and Eugene M Farber Oration at Society for Investigative Dermatology, USA in 2017. He is a past President of the European Dermatology Forum and the European Society for Dermatological Research. He is currently President-elect of the International Psoriasis Council. He is an honorary overseas member of the several national dermatological associations including Denmark, Germany, Japan and the USA.

ABSTRACT

Over the last thirty years, in-depth research has revolutionised our understanding of the immunopathogenesis of psoriasis, leading to the discovery of new therapeutic targets and evolving the way we manage our patients. Moving target identification from serendipity to science-driven mechanistic rationale, the development of T cell-targeted therapies has brought new innovations to the management of psoriasis. These innovations have resulted in dramatic clinical advances and have equally created new questions that demand further research. In this lecture, Professor Jonathan Barker will take the audience through a review of the past, present and future of T cell-targeted therapies for psoriasis.

As our perception of psoriasis shifted from a skin disease to a systemic, T cell-driven condition involving multiple cytokines, research shone new light onto the recurrent nature of lesions and the psoriasis pathogenesis model. The union of immunology and genetics, enabled the introduction of more specific targeted therapeutic agents, able to alter the T-cell-driven inflammatory cascade associated with T cell activation. As we advance our understanding of psoriasis, we continue to identify even more specific targeted T cell therapies. The potential of IL-23 p19 inhibition underpins one of the latest innovations in psoriasis and may change the way psoriasis is currently managed.

The clinical landscape of psoriasis is changing rapidly, with the introduction of biosimilars alongside the development of new agents with new modes of action, with results that may not be fully explained by our current understanding of the disease.

NOTES
UCB PHARMA SPONSORED LECTURE

RE-EVALUATING THE ROLE OF IL-17F IN IMMUNE-MEDIATED CHRONIC INFLAMMATION: DUAL NEUTRALISATION OF BOTH IL-17A AND IL-17F AS A NOVEL TARGETING APPROACH IN PSORIATIC DISEASES

DR STEVAN SHAW
Slough, UK

Stevan Shaw joined Celltech in 1993 as a research graduate in the immunology therapeutic area. Whilst an employee, he completed his immunology PhD training at Imperial College London focusing on lymphocyte biology, specifically, in vivo functions of Th1 and Th2 cells. Post-PhD training, Stevan completed a sabbatical in the USA working in the immunology target discovery department where he designed, developed and implemented in vivo immunology screens for target identification and validation for autoimmune diseases. Post acquisition of Celltech by UCB, Stevan has gained a thorough understanding of antibody-based therapeutics leading projects within the UCB preclinical immunology portfolio and in his functional role as Head of Preclinical Pharmacology. In 2012, Stevan took on the role of leading the UCB4940 clinical programme through to Phase 2b and is now responsible for scientific research to further the understanding of how targeting IL-17A and IL-17F biology may translate into patient value.

ABSTRACT

Identification of immunological pathways pivotal in the pathogenesis of immune-mediated chronic inflammation has led to the development of treatments targeted against key pro-inflammatory cytokines (e.g., TNF, IL-17A and IL-23). While the benefit of current therapies is evident, achieving and maintaining complete skin clearance remains a challenge; therefore, treatments targeting other mediators of inflammation could provide additional patient value.

Historically, the most extensive research into the IL-17 family of cytokines (IL-17A, IL-17B, IL-17C, IL-17D, IL-17E [also known as IL-25] and IL-17F) has been focused on strategies for the inhibition of IL-17A. However, the role of IL-17F, which shares approximately 50% structural homology and overlapping biological function with IL-17A, has been re-evaluated based on recent insights from murine and human data that suggest it may also play an important role in psoriatic diseases. In this talk, we will discuss the contribution of IL-17F to immune-mediated chronic inflammation and describe the preclinical and early clinical data supporting dual neutralisation of IL-17A and IL-17F as a novel targeting approach in psoriatic diseases.

SPEAKER BIOGRAPHIES

NOVARTIS SPONSORED LECTURE

DISEASE MODIFICATION, FROM BEDSIDE TO BENCH

PROFESSOR LARS IVERSEN
Aarhus, Denmark

Lars Iversen is the Head of the Psoriasis Clinic at the Department of Dermatology, Aarhus University Hospital and Professor of Dermatology at Aarhus University, Denmark, where he received his medical degree in 1991. In addition to his teaching duties at Aarhus University, Professor Iversen serves as a reviewer of PhD dissertation and doctoral theses at several European institutes.

Professor Iversen has authored more than 160 publications in peer-reviewed journals and is a reviewer for several medical journals including the Nature Immunology, Proceedings of the National Academy of Sciences, the EMBO Journal and Blood. Professor Iversen has received several prizes including the Nordic Prize of Dermatology and Venereology in 2001 and Advances in Psoriasis in 2008. His research interests include all aspects of psoriasis ranging from molecular biology and epidemiological studies, to clinical research. In addition, he is currently conducting research in cutaneous T-cell lymphomas.

ASSOCIATE PROFESSOR LIV EIDSMO
Stockholm, Sweden

Group leader at Department of Medicine, Karolinska Institutet and practicing dermatologist at Karolinska University Hospital in Sweden. Liv Eidsmo received her MD 1999 and a PhD in Immunobiology in 2006, both from Karolinska Institutet. Following a postdoc in Frank Carbone’s laboratory at Melbourne University, Liv Eidsmo established her own research group in 2010 in Mona Ståhle’s laboratory at Karolinska Institutet and became a board-certified dermatologist in 2017. Dr Eidsmo is a Wallenberg Clinical Fellow, a Ragnar Söderberg Fellow of Medicine and was recently awarded the Ellis and Ivar Janzen prize from the Swedish Society of Medicine.

Dr Eidsmo’s research focus on resident immune cells in human skin. The group recently defined functionally distinct subsets of tissue resident memory T (TRM) cells based on the expression of the integrin CD49a and have characterized pathogenic TRM cells in vitiligo and psoriasis.

ABSTRACT

Could early intervention in psoriasis result in and disease modification?

Professor Lars Iversen
Aarhus, Denmark

Most published guidelines for the treatment of psoriasis recommend the conventional step-up treatment approach starting with topical treatment and followed by narrow band UVB and/or systemic treatment if the disease is not well controlled. Thus, in recent years there has been an evolving hypothesis in some immune mediated inflammatory diseases like rheumatoid arthritis and Crohn’s disease suggesting that early intensive treatment with biologic drugs can dampen the immune mechanisms that lead to a chronic inflammation. This early and more aggressive treatment approach has been shown to significantly improve long-term outcomes in disease activity in these diseases. 1,2

In this symposium, we will discuss the scientific rational behind the hypothesis that early and more intensive intervention in subjects with new-onset moderate to severe plaque psoriasis may lead to superior outcomes and lasting benefits compared to the traditional step-up treatment approach.

Involvement of skin-resident T cells in relapsing psoriasis.

Associate Professor Liv Eidsmo
Stockholm, Sweden

Tissue resident immune cells are crucial components barrier immunity and the human skin harbour a heterogeneous pool of resident memory T (Trm) cells. We recently reported that CD49a marks CD8+ T cells poised to cytotoxicity and IFN-gamma production, while IL-17 producing T cells lacks CD49a-expression in healthy skin. The balance of functionally distinct subsets of resident memory T (Trm) cells is altered in human inflammatory skin diseases. In psoriasis, Trm cells poised to IL-17 are enriched and these cells persist in clinically resolved lesions. Following ex vivo activation, Trm cells from active and resolved psoriasis lesions induce pathogenic tissue responses capable of steering focal pathology 3. Furthermore, pathogenic Trm cell responses are associated with short time in remission following UVB treatment. An ongoing study may offer a possibility to test if the composition and functionality of Trm cells steer recurrent psoriasis in a clinically relevant setting 4.

In this symposium, the pathogenic properties of Trm cells in psoriasis and how early intervention may prevent disbalances in the set-up of Trm cells afflicted skin will be discussed.

References:

2  Danese et al. Gut 2017; 0, 1-9
4  https://clinicaltrials.gov/ct2/show/NCT03020199?cond=stepin+study&rank=1
CELGENE SPONSORED LECTURE
THE COMPLEX MANAGEMENT OF PATIENTS WITH PSORIASIS AND COMORBIDITIES: THE ROLE OF THERAPY CHOICE

PROFESSOR PAOLO GISONDI
Verona, Italy

Professor Paolo Gisondi began his clinical career at the Catholic University of the Sacred Heart in Rome, Italy, where he graduated in Medicine in 1997 and in Surgery in 1998. Subsequently, he worked as a doctor in Rome at the San Gallicano Dermatological Institute-IRCCS and at the Institute of the Immaculate Dermopatico-IRCCS, taking care of hospitalised patients. He specialised in Dermatology and Venereology in 2002 at the Dermatology Clinic of the University of Parma, Italy.

Professor Gisondi began his scientific work at the Institute of the Immaculate Dermopatico-IRCCS, where he was actively involved in clinical dermatology through planning and participation in epidemiological studies, and studies on quality of life in psoriasis, psoriatic arthritis and atopic dermatitis, collaborating with Professor G. Girolomoni. At the same time, he participated as a sub-investigator in research protocols, mainly on clinical biologics in the treatment of psoriasis, and topical calcineurin inhibitors in atopic dermatitis. Since 2005, he has worked at the Dermatology Clinic of the University of Verona, Italy, where he is involved in the design and implementation of epidemiological studies to assess the association between psoriasis and the metabolic syndrome, and therapeutic impact of psoriasis intervention by correcting the underlying metabolic disorders.

ABSTRACT

The clinical presentation of chronic plaque psoriasis is highly variable among patients because of factors such as the age of onset, the involvement of different areas and the severity of the disease. In addition, approximately 40% of patients develop psoriatic arthritis. Other comorbidities are also frequently associated with the disease, including metabolic syndrome, obesity, type 2 diabetes, non-alcoholic fatty liver disease and cardiovascular disease.

It is likely that genetic, metabolic and environmental factors contribute to comorbidities in psoriasis patients. In particular, emerging evidence suggests that psoriasis and metabolic syndrome share similar pathogenic pathways. In this context, adipose tissue has received special attention because of its role as an active endocrine and paracrine organ involved in lipid and glucose metabolism and in the regulation of inflammation.

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Psoriasis is a chronic disease with an unpredictable natural history that requires long-term treatment. The presence of comorbidities in psoriasis patients can bring additional levels of complexity to treatment decisions. The long-term use of treatments for psoriasis should take into account this complex picture, and consequently, treatment approaches should be personalised according to the specific characteristics and needs of the patient.

These elements should be considered in order to achieve optimal care.

References
CHALLENGING CONVENTIONAL CLASSIFICATION DOGMA: TOWARDS A NEW CLINICAL TAXONOMY OF PSORIASIS

PROFESSOR ULRICH MROWIETZ
Kiel, Germany

Professor Ulrich Mrowietz is a Dermatologist and Head of the Psoriasis Center at the University Medical Center Schleswig-Holstein, Campus Kiel.

His main scientific and clinical interests are psoriasis and psoriasis management. His recent research focuses on trigger factors for psoriasis, pustular psoriasis and the mechanisms of action of fumarates. He has worked on establishing management procedures for psoriasis that have been included in the first Global Report on Psoriasis by the WHO.

Professor Mrowietz serves as the Editor-in-Chief of the Archives of Dermatological Research. He is an active member of the cluster of excellence "Inflammation at Interfaces" at the University of Kiel, a former member of the Executive Board of the German Dermatological Association (2007-2015), as well as a member of the Scientific Advisory Board of the German Psoriasis Association. He is also a member of numerous national and international Dermatological Societies and a co-author of the German and European Guidelines for the treatment of psoriasis and lead the European Consensus Programme on Treatment Goals in psoriasis. Professor Mrowietz has published more than 270 articles referenced in the Medline data base, and has written numerous chapters in textbooks including "Fitzpatrick’s Dermatology in General Medicine" and Braun-Falco’s "Dermatologie und Venerologie".

ABSTRACT

Although psoriasis as a disease entity is known since more than hundred years the understanding of its pathogenesis and genetic background is still insufficient as well as their value to the predict the clinical course of disease and response to treatment.

It is of note that the clinical appearance of psoriasis is heterogeneous and in the past a large number of clinical subtypes have been described. Until today there is no international consensus regarding the nomenclature of a clinical classification of psoriasis. Generation of a consensus is hampered by a varying phenotype in different ethnic groups, diverse textbook descriptions and country-specific peculiarities. Phenotypic variability of psoriasis lesions exists not only among different individuals but also within the same patient during the disease course and after trigger factor-induced exacerbations.

NOTES

It is accepted in the dermatological community to separate pustular from non-pustular psoriasis, and early-onset from late-onset disease. However, plaque psoriasis, the prototype of non-pustular psoriasis, may show pustular eruptions during flares. Genetic studies have shown that HLA-C*0602 shows strongest association to plaque psoriasis and may explain at least in part an autoimmune background of the disease. However, many patients with severe psoriasis lack this genetic marker.

It has been conclusively demonstrated that a number of conditions are associated with psoriasis including psoriatic arthritis, diabetes mellitus, hypertension, dyslipidemia and obesity. The latter represents an independent risk factor for psoriasis and the influence of obesity on the course of disease may be more important as compared to genetic markers.

A consensus definition of the different psoriasis phenotypes and their relation to genetic markers as well as to comorbidity is a major challenge. One initiative, the global psoriasis atlas, may be helpful to generate an inventory of psoriasis phenotypes across different ethnic groups and countries. Together with information about genotype and comorbidity this may help to generate a first unifying clinical taxonomy of psoriasis. As genetic markers and other measures to predict treatment outcomes particularly using targeted therapies have failed so far a more sophisticated clinical definition of psoriasis may fill this gap.
THE PRICE AND VALUE OF BIOLOGIC DRUGS

DR JACK SCANNELL
Edinburgh, UK

Jack Scannell is co-head European Pharmaceuticals, Investment Research, at UBS Investment Bank. Prior to joining UBS in 2016, Jack consulted to the drug and biotech industry, and to public sector bodies, on R&D productivity and drug pricing. He also conducted academic research on R&D productivity and remains an Honorary Fellow at the University of Edinburgh, and an Associate of CASMI at Oxford University. From 2012 to 2014, Jack was Head of drug discovery at a e-Therapeutics PLC, a bio-informatics based biotechnology firm, that sought to exploit analytic methods he had worked on as an academic, in the late 1990s. From 2005 to 2012, Jack was an investment analyst. He led the European Healthcare team at Sanford Bernstein in London. He has a degree in Medical Sciences from Cambridge University and a PhD in Physiology (Computational Neuroscience) from Oxford University.

ABSTRACT

If I offered to buy your shoes you would think I was strange, but we could probably haggle a price. If I offered to buy your children, we would not get to the haggling stage. The difference between trading shoes and children is not merely legal. It is also moral. People find it unpalatable, even taboo, to put prices on things that we treat as absolutes; life, liberty, or health. People have moral qualms about the cost of medicines for the genuinely sick, but not about the cost of Botox or liposuction. Yet medicines do not exist in a parallel universe, free from economics. Taxes are paid, as are health insurance premiums; healthcare budgets are set; doctors earn money; professors seek riches as biotech entrepreneurs; venture capitalists gamble other people’s money on the professors’ ideas; drug companies pay wages to employees and dividends to shareholders. Moral queasiness tends to reduce the quality of, and polarize, public debate on drug pricing. “Cost”, “value”, “power” and “prizes” are four ways that people think, talk or write about the mechanism by which drugs are priced. “Cost” refers to cost-based pricing: the idea that the price of goods is based on how much it costs to produce them. “Value” refers to value-based pricing: the idea that the price of goods reflects their value to the buyer. “Power” is the exercise of intellectual property rights, to create scarcity and to find the maximum price that the market will bear. “Prizes” are the incentives provided by profit tomorrow, made credible by profit today, for investors gambling on the R&D that might create tomorrow’s drugs. At present, “Value” seems the intellectually fashionable approach. In the talk, I will argue that value-based pricing appeals to the drug industry and some health economists, but that it can be bad public policy.
Dr. Leroy E. Hood graduated from the Johns Hopkins University School of Medicine in 1964 with an MD and from Caltech with a PhD in biochemistry in 1968. After three years as a Senior Investigator at NIH, his academic career began at Caltech, where he and his colleagues developed the DNA gene sequencer and synthesizer, and the protein synthesizer and sequencer—four instruments that paved the way for the successful mapping and understanding of the human genome. A pillar in the biotechnology field, Dr. Hood has played a role in founding fifteen biotechnology companies including Amgen, Applied Biosystems, Integrated Diagnostics and Arivale. He is a member of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine. Of the more than 6,000 scientists world-wide who belong to one or more of these academies, Dr. Hood is one of only fifteen people nominated to all three. Dr. Hood has co-authored numerous textbooks in biochemistry, immunology, molecular biology and genetics, as well as a popular book on the human genome project, The Code of Codes and he is just finishing up a text on systems biology. He is the recipient of numerous national and international awards, including the Lasker Award for Studies of Immune Diversity (1987), the Kyoto Prize in advanced technology (2002), the Heinz Award for pioneering work in Systems Biology (2006), and the coveted NAE 2011 Fritz J. and Delores H. Russ Prize for developing automated DNA sequencing. In addition to having received 17 honorary degrees from prestigious universities in the U.S. and abroad, Dr. Hood has published over 750 peer-reviewed articles and currently holds 36 patents. In 2013, he received the National Medal of Science from President Obama. Hood has been named by The Best Schools as one of the 50 Most Influential Scientists in the World Today (2014) http://isb.io/top50. Scientific American has named Hood as one of the top 6 in their selection of 100 biotech visionaries world-wide (2015) http://isb.io/visionary.

ABSTRACT

Systems medicine, the application of systems approaches to disease, places medicine at a fascinating tipping point—promising a revolution in the practice of healthcare. I will discuss how systems biology approaches have framed systems medicine and I will discuss some of the new systems-driven technologies and strategies that have catalyzed this tipping point. Moreover, four converging thrusts—systems medicine, big data (and its analytics), the digitalization of personal measurements and patient-activated social networks—are leading to a proactive healthcare that is predictive, personalized, preventive and participatory (P4). I will contrast P4 healthcare with contemporary medicine and discuss its societal implications for healthcare. P4 healthcare has two central thrusts—wellness and disease.

I will discuss our successful effort to introduce P4 healthcare into the current healthcare system with a P4 pilot program on scientific wellness—a longitudinal, high-dimensional data cloud study on each of 108 well patients over 2014. The preliminary results both with regard to data analyses and patient responses from these studies are striking. They point to the emerging discipline of scientific wellness—and the fact that it will catalyze several new thrusts in healthcare: 1) optimizing wellness, 2) identifying the earliest disease transitions for all common diseases and learning how to reverse them and 3) employing the dense, dynamic, personal data cloud approach to study diseases (e.g. cancer, Alzheimer’s, diabetes) and their responses to therapy. Scientific wellness will also pioneer N+1 experiments to deconvolute the staggering complexity of human biology and disease. We started Arivale, a company focused on scientific wellness for the consumer, in 2015 and already have about 4000 individuals enrolled. I will also discuss some preliminary results from the Arivale studies.

My institute, the Institute for Systems Biology (ISB), in 2016 affiliated with Providence St. Joseph Health to become its research arm and I became its Chief Science Officer. Providence is one of the largest non-profit healthcare systems in the US—and ISB/Providence will be initiating a series of “translational pillars” moving applications of systems (P4) medicine from the bench to the bedside. These pillars include scientific wellness, bringing scientific wellness to cancer survivors, making Alzheimer’s a reversible and preventive disease, rather than a relentlessly progressive disease, taking a systems approach to type 2 diabetes and exploring how the deep, dynamic, personal data clouds can be used to gain a deep understanding of glioblastoma and provide new diagnostic and therapeutic approaches to this inevitably fatal tumor. It is fair to say that dense, dynamic, personal data clouds followed longitudinally on hundreds of thousands of patients/consumers will allow us to see the earliest wellness to disease transitions for all of the common cancers—and generate biomarkers for early detection and identify the drug targets or strategies (e.g., immunotherapy) that will allow us to reverse the disease before it ever manifests itself as a disease phenotype. Systems medicine, P4 healthcare and scientific wellness will open up powerful new approaches to dealing with diseases of the skin—including psoriasis.

Thus scientific wellness will catalyze a transformation in contemporary healthcare and it will provide eventually millions of dense, dynamic, personal data clouds that will present striking new opportunities for pharma, biotech, nutrition and diagnostic companies to identify biomarkers and drug target candidates. As the cost of the assays for the dense, dynamic, personal data clouds decline dramatically, scientific wellness can be brought to the developing world leading to a democratization of healthcare unimaginable even a few years ago.
Psoriasis Gene to Clinic, 8th International Congress. The Queen Elizabeth II Conference Centre, London, U.K., 30th November – 2nd December 2017

Free communications

FC01 Psoriasis and risk of malignant lymphoma: a population-based cohort study
H. Kamstrup, L. Skov, C. Zachariae, J. Thyssen and A. Løberg
Department of Dermatology and Allergy, Herlev and Gentofte Hospital, Hellerup, Denmark

Psoriasis is a chronic proliferative inflammatory skin disease. Epidemiological studies have suggested an association between psoriasis and lymphoma, but data are limited. The aim of the study was to quantify the 5-year risks of new-onset Hodgkin lymphoma (HL), non-Hodgkin lymphoma (NHL) and cutaneous T-cell lymphoma (CTCL) in patients with psoriasis compared to the general population. A Danish nationwide cohort study including data on all individuals aged ≥18 years between 1 January 2008 and 31 December 2012. Incidence rates (IRs) per 100,000 person-years for HL, NHL and CTCL were calculated, and hazard ratios (HRs) with 95% confidence intervals (CIs) were estimated using Cox regression analysis and adjusted for age, sex, socioeconomic status and systemic treatment. The primary cohort was stratified according to severity by systemic antipsoriatic therapy. In total 58,138 patients with psoriasis and 1,033,731 general population controls were identified. The IRs in patients with psoriasis for HL (IR 1.02, 95% CI 0.76–1.47) and NHL (IR 0.44, 95% CI 0.36–0.47) were higher among patients with psoriasis than in the general population. Adjusted HRs (95% CIs) for 0.25–0.77) were higher among patients with psoriasis than in the general population. Adjusted HRs (95% CIs) for patients with psoriasis and 1.03 and 0.75 were significantly associated with a diagnosis of psoriasis and 0.12–2.23) but not NHL (IR 1.02, 95% CI 0.84–1.24) or CTCL (IR 1.16, 95% CI 0.88–1.53). When stratified by disease severity, adjusted IRs were 1.20 (95% CI 0.78–1.82) for mild psoriasis and 1.28 (95% CI 1.18–1.40) for severe psoriasis and NHL (IR 1.24, 95% CI 1.12–1.36) and CTCL (IR 1.36, 95% CI 0.72–2.27). In conclusion, patients with psoriasis have a higher occurrence of NHL and especially CTCL than the general population. Patients with mild psoriasis have a slightly increased risk of HL. The relative contributions of an initial transient diagnostic, possible overlap in disease pathogenesis, and complications of systemic medication need further investigation.

FC02 Developing a therapeutic strategy and reading response to biologics in patients with psoriasis: a multicentre prospective observational cohort study T. Tsidok and the PSORT Consortium
King’s College London and St John’s Institute of Dermatology, London, U.K.

Biological therapies have transformed treatment in immune-mediated inflammatory diseases, yet significant numbers of patients experience treatment failure. Variability in drug levels correlates with outcomes across multiple inflammatory diseases, but with limited data in psoriasis. In this prospective observational cohort study (60 dermatology specialists centre) we determine the clinical utility of therapeutic drug monitoring using the exemplar tumour necrosis factor antagonist adalimumab. Adults (n = 515) recruited to Biomarkers of Syn- drome Treatment Outcomes in Psoriasis (BSTOP) within the biologics pharmacovigilance British Association of Dermatologists Biologic Interventions Registry (BADBIR) with serial adalimumab concentrations measurements up to 1 year were included (888 samples; 63% male; mean age 44 ± 13 years). Response to treatment at 6 months was defined as ≥ 75% improvement in Psoriasis Area and Severity Index (PSA ≤ 75; primary outcome). We found that drug level can discriminate responders from nonresponders with a minimally effective adalimumab level identified as ≥ 3.4 mg·L⁻¹ (90% sensitivity; specificity 52%; area under the curve 0.73, 95% confidence interval (CI) 0.67–0.79). Multivariable modelling suggested logistic regression modelling to account for clustering of samples within patients, and to adjust for covariates, indicates that a target drug level of ≥ 7 mg·L⁻¹ will achieve a minimum 80% probability of response (81%, 95% CI 76–86). Early serum drug level was confirmed as an additional independent indicator of 6-month PASI 75 response [OR (95% CI)] 1.91, 95% CI 1.26–3.07; P = 0.01], and PASI 90 response [OR (95% CI)] 2.38, 95% CI 1.26–4.50; P = 0.008], substantially improving the model’s goodness of fit. Smoking and disease duration were also key baseline predictors influencing interindividual variation. This real-world study with pragmatic drug level sampling provides strong evidence to support proactive measurement of drug levels in the management of psoriasis, and highlights the importance of taking drug levels into account when searching for biomarkers of treatment response.

FC03 The differential production of interleukin (IL)-26 vs. IL-17 by T helper 17 cells contributes to the development of different forms of psoriasis
A. Fries, I. Di Domizio, O. Demaria and M. Gilliet
Department of Dermatology and Cell Biology, Radboud University Medical Centre, Nijmegen, the Netherlands; Department of Dermatology, Department of Computer Medicine and Bioinformatics and Department of Biostatistics, University of Michigan, MI, U.S.A.; Leiden Academic Center for Drug Research, Department of Drug Discovery Technology, Gorlaeus Laboratories, Leiden University, Leiden, the Netherlands; Department of Microbiology, Institute for Water and Health Research, Faculty of Science, Radboud University, Nijmegen, the Netherlands; and Asea Åker Vieanm Akinen Hospital, Michigan, MI, U.S.A.

Interleukin (IL) 17-producing helper T cells (Th17 cells) play a role in diseases against infections and have also been linked to autoimmune diseases like psoriasis. Th17 cells produce IL-17A/F, IL-22 and IL-26. Whereas the induction of IL-17 and IL-22 expression during Th17 differentiation has been well described, the mechanism regulating the production of IL-26 remains ill-defined. Here, we found that IL-6 alone is required to promote IL-26-producing T cells from naive T cells, but it is not sufficient to induce IL-17-producing T cells. By contrast, the generation of IL-17-producing T cells required the presence of IL-1 and IL-23, which are unable to induce IL-26, suggesting that Th17 cells comprise two distinct sub- sets, namely IL-26-producing T cells and IL-17-producing T cells. Although all Th17 cytokines are elevated in psoriasis, we also found a dichotomy between IL-17 and IL-26 expression in the skin of different forms of the disease. Whereas IL-17 was mainly associated with chronic plaque psoriasis, IL-26 appeared to be strongly expressed in more acute lesions (ery-throdermic psoriasis, guttate psoriasis, paradoxical psoriasis). Importantly, the expression of IL-16 in psoriasis correlated with the expression of IL-6 but not IL-1 nor IL-23, indicating that the presence of IL-26-producing T cells may drive the development of acute skin inflammation. These findings there- fore identify IL-26 as a novel therapeutic target for the treat- ment of acute forms of psoriasis.

FC04 Psoriasis-associated late cornified envelope proteins have antibacterial activity
H. Nihues, L. Tsao,1,4 D. van der Kierken,2 K. P. Oosthred,2 D. Rodijk-Olthuis,1 L. van Vlijmen-Willems,2 W. Hendriks,1 R. Helders,1 J. Bouwstra,4 R. Mesman,1 L. van Niftrik,1 E. van den Bogaard,1 P. Stuart,2 R. Nair,1 H. Elder,1,5 P. Zeeuwen1 and J. Schalkwijk1
1Department of Dermatology and Cell Biology, Radboud University Medical Centre, Radboud Institute for Molecular Life Sciences, Nijmegen, the Netherlands; Department of Dermatology, Department of Computer Medicine and Bioinformatics and Department of Biostatistics, University of Michigan, MI, U.S.A.; Leiden Academic Center for Drug Research, Department of Drug Discovery Technology, Gorlaeus Laboratories, Leiden University, Leiden, the Netherlands; Department of Microbiology, Institute for Water and Health Research, Faculty of Science, Radboud University, Nijmegen, the Netherlands; and Asea Åker Vieanm Akinen Hospital, Michigan, MI, U.S.A.

Late cornified envelope (LCE) proteins, located in the epidermal differentiation complex on chromosome 1, encode a family of noncoding variants, which has precluded understanding of their biological function. Here, we report a set of late cornified envelope (LCE) proteins and suggest a central role for LCE3A in epidermal barrier function, but revealed that Psoriasis-associated LCE3 proteins, and LCE3A in particular, were strongly expressed in more acute lesions (ery-throdermic psoriasis, guttate psoriasis, paradoxical psoriasis). Importantly, the expression of IL-16 in psoriasis correlated with the expression of IL-6 but not IL-1 nor IL-23, indicating that the presence of IL-26-producing T cells may drive the development of acute skin inflammation. These findings there- fore identify IL-26 as a novel therapeutic target for the treat- ment of acute forms of psoriasis.

FC05 Environmental antigens may trigger HLA-C*06:02-mediated autoimmunity in psoriasis
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Psoriasis vulgaris is a human leucocyte antigen (HLA)- C*06:02-associated T-cell-mediated autoimmune skin disease that develops upon epidermal infiltration and activation of CD8+ T cells. Environmental and lifestyle factors may trigger disease onset, and account for approximately 30% of disease risk. Using a Vα18/Vβ13.1 T cell receptor (TCR) from a pathogenic psoriatic CD8+ T cell clone we have recently shown that in psoriasis HLA-C*06:02 mediates an autoimmune response against melanocytes, and we had identified a peptide from ADAMTS-like protein 5 as a melanocytic antigen. In this study, we aim to identify environmental fac- tors at the molecular level that translate the genetic predisposi- tion into disease manifestation. TCRs are polyspecific-they do not recognize specific antigens but react against HLA-presented peptides sharing a particular amino acid pattern specific to this TCR. After defining the amino acid pattern recognized by the Vα18/Vβ13.1 TCR in the context of HLA-C*06:02, we screened for melanocyte reactive Vα18/Vβ13.1 TCR using a peptide library screening we searched environmental proteomes for peptides sharing this particular pattern. Environmental candi- date epitopes were tested for their ability to ligate the ADAMTS-like protein 5-reactive Vα18/Vβ13.1 TCR when presented by HLA-C*06:02. This we identify a variety of peptides contained in proteomes from human skin and gut microbiome, from infectious pathogens including Chlamydia tuberculosis, and from foods (wheat, coffee, apple and spinach) that ligated the Vα18/Vβ13.1 TCR. These results provide the first evidence of environmental antigens that may serve as potential triggers of the melanocyte-specific autoimmune response in psoriasis. They suggest that exposure to environmental antigens may drive priming and expansion of potentially self-reactive T cells and thus initiate autoimmune disease responses. Through the unbiased analysis of a pathogenic psoriatic TCR, our data fur- thermore may have important implications in understanding.

59 ABSTRACTS
how environmental factors affect the risk for autoimmune dis-
cases in general.

FCo6 Analysis of psoriasis host-microbiome interactions using a universal transcriptomic approach

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Psoriasis is an inflammatory disease caused by dysregulated immune responses. Host-microbiome interactions may underlie the triggering and worsening of psoriasis. 16S DNA sequencing has indicated that psoriatic skin and tonsils have markedly altered microbiomes; however, this method identifies bacteria poorly and does not inform about altered microbial commu-
nity is currently hindered because existing alignment software
was designed for single-organism analysis. Thus, we devel-
opned new RNA-seq alignment software, Muscato, specifically
designed to carry out multimammal sequence alignment. Mus-
cato aligns hundreds of millions of RNA-seq reads to hundreds of
millions of genes in reasonable time frames and needs only
moderate computational resources. When coupled to a func-
tional and taxonomic annotated database it allows sim-
ilar analysis of host and microbiome gene expression. To
identify host-microbiome interactions that may drive psoriasis,
we performed RNA-seq analysis on lesional skin (LS, n = 8),
nonlesional skin (NLS, n = 10) and tonsils (PT, n = 11) of
patients with psoriasis and normal controls (NS, n = 7).
In LS, we found differentially expressed (more than twofold) human
genes mapped to KEGG functions including inflammation
(ILI, ILK, NF-κB, TNF), keratinocyte proliferation (FGF, STAT3, CSF2, MCL), vasodilatation (VEGF, TIEK, ANGPT1), and
T-cell responses (TGFB1, IGF2, ICAM1, S100). This was
accompanied by reduced microbial diversity (Shannon Index:
LS 7.2, NLS 8.6, NS 8.3), with decreased Propionibacterium and
more than fourfold increased Staphylococcus pyogenes colonization.
Ninety-four (62%) and 40 of 90 (PT) streptococcal species were
more than twofold more abundant than in controls, with
S. anginosus and S. sciuri predominant. Multiple streptococcal
species had differentially expressed (more than fivefold)
virulence factors, including sialidase (S. anginosus) and
virulence factors, including sialidase (S. anginosus) and
secretion systems and secreted proteases, which may drive T-cell skin homing and activation.

LS), sec- and Type IV protein secretion systems and secreted
proteases, which may drive T-cell skin homing and activation.

FNC7

Genetic variation contributes to response to biologic agents in patients with psoriasis

D. Leary and J. Krueger

1Janssen Research & Development, LLC, Spring House, PA, U.S.A.
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New York, NY, U.S.A.

Emerging clinical data indicate that selective blockade of inter-
leukin (IL)-23 can achieve greater efficacy than dual blockade
of IL-12/IL-23 in patients with moderate-to-severe psoriasis.
Ustekinumab targets the p40 subunit common to IL-23 and IL-25, whereas guselkumab specifically targets the IL-23-speci-
fic p19 subunit. While differences in antibody potency may
equalize therapeutic differences between ustekinumab and
guselkumab, we explored cellular and molecular changes in
the skin of psoriasis patients with psoriasis treated with ustekinumab or
guselkumab to understand the mechanisms underlying selective IL-23 p19 inhibition. Ustekinumab data were
derived from the ACCUP study (NCT00454384, n = 85), in which
patients with psoriasis received either 45 mg or 90 mg of
ustekinumab at weeks 0 and 4, and guselkumab data were derived
from the phase 1/2b postmarketing study (NCT02957547, n = 24) that
tested single subcutaneous doses of guselkumab. Skin biopsies
were collected at baseline and weeks 1 and 12 post-treatment
from each study to evaluate (i) interplay of biologic agents
and immune cells (II-23 for guselkumab, IL-12/23 blockade for
ustekinumab) and (ii) molecular response to drug via
cell-based biorepos. The two cohorts are comparable in baseline
demographics, disease characteristics, skin histopathology, patient history, and emotional
profiles and significantly enriched canonical pathways.
Blockade of IL-23 with guselkumab resulted in a significantly
greater reduction in CD3 and CD11c cell counts in the skin
relative to baseline with ustekinumab blockade (p = 0.05 vs. >70%).
In patients who achieved ≥75% improvement
response at week 12 for ustekinumab, both guselkumab and
statin blockade in psoriasis skin

FNC8

Comparative evaluation of cellular and molecular biological
changes associated with response to selective
interleukin (IL)-23 blockade vs. dual IL-12/23
blockade in patients with psoriasis

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2Laboratory for Investigative Dermatology, The Rockefeller University,
New York, NY, U.S.A.

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Blockade of IL-23 with guselkumab resulted in a significantly
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response at week 12 for ustekinumab, both guselkumab and
Psoriasis is a chronic inflammatory skin disease in which activation of T helper 17/12, IL-23-, IL-12-, IL-18- and tumour necrosis factor-α-producing and regulatory CD4+ T cells is critical. Patients with psoriasis predominantly express cell-surface ADAM17 as revealed by flow cytometry. Furthermore, the enzymatic activity of ADAM17 on slanDCs could be demonstrated by a specific fluorescence peptide assay. Addition of the endogenous protease inhibitor TIMP3 to slanDCs inhibited ADAM17 activation, and, most interestingly, slanDCs inhibited ADAM17 activation by 60–70%. These results demonstrated several candidate genes for psoriasis, but none of them reached genome-wide significance. To create a homozygous subset of samples, we then performed a latent class analysis to identify groups of patients with similar clinical phenotypes. This analysis yielded three classes. Class I was characterized by an early age of onset or familial aggregation. More severe disease, and trauma- or injury-associated disease onset or worsening. Familial risk analysis further suggested a role for some candidate genes in epithelial immune mechanisms underpinning the clinical response to adalimumab. Functional analysis are ongoing to address the molecular basis of these findings.

Psoriasis is a chronic inflammatory skin disease in which activation of T helper 17/12, IL-23-, IL-12-, IL-18- and tumour necrosis factor-α-producing and regulatory CD4+ T cells is critical. Patients with psoriasis predominantly express cell-surface ADAM17 as revealed by flow cytometry. Furthermore, the enzymatic activity of ADAM17 on slanDCs could be demonstrated by a specific fluorescence peptide assay. Addition of the endogenous protease inhibitor TIMP3 to slanDCs inhibited ADAM17 activation, and, most interestingly, slanDCs inhibited ADAM17 activation by 60–70%. These results demonstrated several candidate genes for psoriasis, but none of them reached genome-wide significance. To create a homozygous subset of samples, we then performed a latent class analysis to identify groups of patients with similar clinical phenotypes. This analysis yielded three classes. Class I was characterized by an early age of onset or familial aggregation. More severe disease, and trauma- or injury-associated disease onset or worsening. Familial risk analysis further suggested a role for some candidate genes in epithelial immune mechanisms underpinning the clinical response to adalimumab. Functional analysis are ongoing to address the molecular basis of these findings.
Intriguingly, we have also observed by immunofluorescence in lesional psoriatic epidermis relative to nonlesional skin. This result in the generation of thinner epidermal equivalents. When keratinocytes placed in three-dimensional organotypic culture were found that they develop significantly thinner paw epidermis with an aberrant wound healing phenotype and hyperproliferative epidermis—driven disease, rheumatoid arthritis, in a transgenic miR-181/1-1-2 knockdown model, we have also observed that this miR-181 localizes in basal keratinocytes of psoriasis and that miR-181 regulates the expression of genes involved in psoriasis and that miR-181 is also downregulated in lesional psoriatic epidermis relative to nonlesional skin. As a consequence, therapeutic targeting of miR-181 would allow for the inhibition of excessive, disease-associated miR-181-dependent shedding, without affecting the relatively lower levels of miR-181 activity important for normal skin homeostasis. Therefore, as psoriasis shares several disease mechanisms with those seen as consequences of iRhom2 hyperactivity, targeting iRhom2 in psoriasis represents an attractive strategy.
**FC20**

Longitudinal follow-up of antithrombin III (AT-III) replacement therapy in patients with severe sepsis or septic shock: results of the Sepsis Infection and Septic Shock (SINSE) study

**FC21**

Real-world experience and treatment patterns in patients with psoriasis: analysis of 54 patients from the APPRECIATE study

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**FC9**

Activation of resident T cells in resolved psoriasis reveals tissue responses that stratify clinical outcome

**FC22**

Topical methotrexate gold nanoparticles reduce imiquimod-induced inflammation in mice

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**ABSTRACTS**

1Department of Dermatology, University Hospital CHUV, Lausanne, Switzerland and 2Department of Dermatology, University Hospital of Zurich, Zurich, Switzerland

Antithrombin III (anti-TFPI) are highly effective in the treatment of sepsis. However, in 2-5% of treated patients they induce prothrombotic-like skin lesions called paradoxical psoriasis. The pathogenesis of this side-effect and its distinction from classical psoriasis remain largely unknown. Here we performed a comprehensive clinical, histological and gene expression analysis of skin lesions from 25 patients with paradoxical psoriasis. Paradoxical psoriasis is characterized by a selective, uniform increase in type I interferon expression and plasminogen activator inhibitor (pcPAC) accumulation, despite marked clinical and histological variations. Anti-TFPI-stimulated PDCs produce exaggerated levels of type I interferon by inhibiting TFN-mediated PDC maturation both in vivo and in vitro, in a skin injury mouse model. Via type I interferon, anti-TFPI agents induce a prothrombotic skin phenotype reflecting paradoxical psoriasis, which is, unlike classical psoriasis, independent of T cells. These findings indicate that paradoxical psoriasis represents an overactive innate inflammation driven by pcPAC-derived type I interferon; however, this is self-contained as it does not lead to T-cell immunity.

**FC19**

**Activation of resident T cells in resolved psoriasis reveals tissue responses that stratify clinical outcome**

L. Szérgély, C. Classon, S. Nylén, N.K. Landén, T. Martin, S. Cheuk and L. Edmondston

Karolinska Institutet, Stockholm, Sweden

Pathological resident memory T (T RM) cells pose a threat to the maintenance of immune homeostasis and can contribute to persistent inflammation in several diseases. CD8+ T RM cells have been characterized in multiple disease models and have been functionally linked to tissue remodelling and stiffness. In this study, we evaluated the impact of CD8+ T RM cells on tissue remodelling in resolved psoriasis.

**Topical methotrexate gold nanoparticles reduce imiquimod-induced inflammation in mice**


University Hospital Zurich, Zurich, Switzerland and RepHEC Europe Ltd, Glasgow, U.K. and 2Mebro Pharmaceuticals, Ahlen, U.K.

Methotrexate (MTX) is a widely used immunosuppressive agent for the treatment of several autoimmune and chronic inflammatory conditions, such as rheumatoid arthritis and psoriasis. MTX is usually administered systemically, which can cause side-effects, including liver damage and kidney failure. Due to its palatability and high molecular weight, topical MTX penetrates very poorly through the skin barrier and therefore needs a targeted drug delivery system to overcome biological barriers. Gold nanoparticles (GPNs) have been used as a targeted drug delivery system in cancer therapy and have recently been shown to deliver MTX into the skin by topical application. Here we explored the therapeutic potential of a novel topical MTX formulation in inflammatory skin disease. The skin permeability of MTX was increased via conjugation to GPNs (MTX-GNP). Using the imiquimod-induced mouse model of psoriasis, where imiquimod is applied on the ear of a mouse, we evaluated the in vivo efficacy and functionality of MTX-GNP. Subcutaneous administration of MTX-GNPs ameliorated imiquimod-induced inflammation in a dose-dependent manner, as measured by ear thickness, erythema and swelling. The intradermal administration of MTX-GNP showed no efficacy compared to GNP-only and no improvement over the untreated condition. The MTX-GNP formulation showed no signs of toxicity, as the histopathology of treated ears was comparable to untreated controls. In conclusion, topical MTX-GNPs ameliorate imiquimod-induced inflammation in a dose-dependent manner, as measured by ear thickness, erythema and swelling, and are a promising alternative to conventional topical MTX formulations.
ABSTRACTS

Psoriasis: from gene to clinic

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Secukinumab: a therapeutic option for inflammatory skin disease

Secukinumab (300 mg and 150 mg) in patients with active moderate-to-severe plaque psoriasis, with a sustained effect and a favourable safety profile. This open-label, noncomparative study was designed to investigate the safety and efficiency of secukinumab (100 mg and 150 mg) in patients with active moderate-to-severe, chronic plaque psoriasis who had a prior efficacy failure with at least one biologic therapy. Patients received secukinumab subcutaneously at 100 mg or 150 mg for a 16-week induction period (weeks 0–8, 8–12 and 12–16) followed by two maintenance periods up to 72 weeks. There were three subgroups: (i) inadequate response to first anti-TNF-α therapy; (ii) adequate response after first anti-TNF-α therapy due to inadequate response. The primary endpoint was the percentage of patients achieving PASI 75 at 16 weeks. We found that mutation rates were higher in GPP (22.9%, 48 of 210) and ACH (18%, three of 17) compared with PPP (6.5%, 62 of 991). In contrast, we found that mutation carriers (62 of 991) were more likely to have a significant sex bias as 21 of 22 (96%) IL36RN-positive cases were male.

Secukinumab is a human anti-interleukin-17A monoclonal antibody, which has been shown to be effective in the treatment of moderate-to-severe plaque psoriasis, with a sustained effect and a favourable safety profile. This open-label, noncomparative study was designed to investigate the safety and efficiency of secukinumab (100 mg and 150 mg) in patients with active moderate-to-severe, chronic plaque psoriasis who had a prior efficacy failure with at least one biologic therapy. Patients received secukinumab subcutaneously at 100 mg or 150 mg for a 16-week induction period (weeks 0–8, 8–12 and 12–16) followed by two maintenance periods up to 72 weeks. There were three subgroups: (i) inadequate response to first anti-TNF-α therapy; (ii) adequate response after first anti-TNF-α therapy due to inadequate response. The primary endpoint was the percentage of patients achieving PASI 75 at 16 weeks. We found that mutation rates were higher in GPP (22.9%, 48 of 210) and ACH (18%, three of 17) compared with PPP (6.5%, 62 of 991). In contrast, we found that mutation carriers (62 of 991) were more likely to have a significant sex bias as 21 of 22 (96%) IL36RN-positive cases were male.

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Risankizumab versus ustekinumab for moderate-to-severe plaque psoriasis: 16-week results from the phase III IMMHance trial
A. Blauvelt,1 K.A. Papp,1,4 M. Goodherm,1,4 R.G. Langley,1 C. Leonardi,1 J.P. Lacour,1 S. Philipp,1 S. Tyring,1 M. Bukhala,3 J.J. Wu,1 J. Baget,1 E.H. Frankel,13 D. Pariser,14 M. Flack,15 J. Scherer,15 Z. Geng,16 Y. Gu,16 A. Cameron1 and E.H.2
1Oregon Medical Research Center, Portland, OR, U.S.A.; 2Papp Clinical Research and Psoriasis Research, WebMD, Inc., Oxford, ON, Canada; 3Department of School of Medicine, Queen’s University, Kingston, ON, Canada; 4Center for Dermatology and Psoriasis Medical Youth, Rotterdam, ON, Canada; 5Unilever Health Care, Hilden, NL, Canada; 641st Street University, St Louis, MO, U.S.A.; 7Hospital de J.J. Menotti, University of Niza-Athik, Nice, France; 8CharitÈ University Medical Center, Berlin, Germany; 9University of Texas Health Science Center and Center for Clinical Studies, Houston, TX, U.S.A.; 10Alman Dermatology Associates, Arlington Heights, IL, U.S.A.; 11Kaiser Permanente Los Angeles Medical Center, Los Angeles, CA, U.S.A.; 12Psoriasis Treatment Center of Central New Jersey, East Windsor, NJ, U.S.A.; 136Blends, Canons, IL, U.S.A.; 14warren Virginia Medical School and Virginia Clinical Research Inc., netixx, VA, U.S.A.; 15Risankizumab were compared with those of ustekinumab, an IL-12/IL-23 inhibitor, in patients with moderate-to-severe plaque psoriasis. Risankizumab is a potent humanized IgG1 monoclonal antibody that inhibits IL-23 by specifically binding to its p19 subunit. In a phase II trial, the efficacy and safety of risankizumab were assessed in patients with moderate-to-severe psoriasis. In this phase III study, a total of 345 patients were randomized to risankizumab (150 mg...
of a ‘culture of measurement’ and consensus national goals for psoriasis care. These measures were supported by the establishment of 30 regional psoriasis networks involving more than 500 GPs nationwide. The outcomes of this national psoriasis programme are evaluated on a regular basis. The current analysis shows the long-term results of the programme, indicating continued benefits from 2017. All surveys were based on random samples of dermatology practices and clinics across the country. In each survey in 2004, 2008, 2014 and 2017, between 120 and 150 centres were nominated, and data from patients with psoriasis were obtained from a minimum of 1500 patients per survey, including psoriasis characteristics, Psoriasis Area and Severity Index (PASI), Dermatology Life Quality Index (DLQI), patient benefit index, patient satisfaction and current and previous treatments. Between 2004 and 2017 there was a significant increase in the proportion of patients reaching sufficient quality of health care from 34% to 79% (total n = 6333). The mean PASI reduced from 11.4 to 7.4, and the proportion of patients with severe disease dropped from 17.8% to 8.6%. With respect to drug treatment, there has been a significant annual increase in the drug volumes for systemic treatments by about 24% per year, with healthcare cost from 34% to 79% (total cost of €674 million in 2015). Despite these significant increases in the use of systemic drugs for psoriasis, there remain large regional disparities, with some federal states spending more than €3.50 per capita of population for biologics in psoriasis compared with €0.90 in others. Besides regional disparities, there are also severe differences in access to modern drugs between patients referring to a dermatologist (>70% chance of guideline-compliant treatment) compared with GPs (≤40%). In conclusion, there has been a marked increase in the proportion of patients with psoriasis receiving guideline-compliant, specific systemic treatments for moderate-to-severe disease. Nevertheless, many patients are still lacking access to treatments. The national conference on psoriasis is held annually to transform the national healthcare goals for psoriasis in the next 5 years with higher thresholds to reach.

### FC31 Efficacy and safety of mirikizumab (LY3042828) in the treatment of moderate-to-severe plaque psoriasis: results from a phase II study

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### FC32 Certolizumab pegol for the treatment of patients with moderate-to-severe chronic plaque psoriasis: an overview of three randomized controlled trials

P. Blauvelt,1 R. Reich,1,2,3 K. Menter,1,2 A. Gottlieb1

1Division of Dermatology, University of Texas Southwestern Medical Center, Dallas, TX, USA; 2Department of Dermatology, University Hospital Munich, Munich, Germany; 3Bayer HealthCare, Leverkusen, Germany

### FC33 Switching or restarting of tumour necrosis factor-α inhibitors after interruption under daily-life conditions: efficacy report from the Austrian Psoriasis Registry (Psoriasis)

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We were interested in investigating how well tumour necrosis factor-α (TNF-α) inhibitors act after switching or restarting after interruption under daily-life conditions. After data switching from 2016 by the Austrian Psoriasis Registry (PsöRa) revealed that 141 patients fulfilled the inclusion criteria for this analysis: (i) TNF-α inhibitor treatment duration of 3 months or longer, (ii) switch to another TNF-α inhibitor, (iii) treatment interruption of 3 months or longer before restarting with the same TNF-α inhibitor and (iv) availability of complete data to perform the analysis. Response to treatment in the registry is recorded in treatment categories: 1, CR (complete remission); 2, ≥90% improvement in Psoriasis Area and Severity Index (PASI) 90; 3, PASI 50 or 75. PASI 50. Reasons for treatment interruption, with a median of 6.6, 10.1 and 10.2 months for infliximab (IFX), adalimumab (ADA) and etanercept (ETA), respectively, were due mainly to complete remission, patient wish or other reasons; for switching were primarily for ineffectiveness or loss of initial response. The main switch/restart combinations were ETA/ADA, n = 72, ADA/ETA, n = 20, ADA/ADA, n = 12 and ETA/ETA, n = 12. These data generated under daily-life conditions are consistent with the data also support the concern that after treatment interruption, patient wish or other reasons; for switching were primarily for ineffectiveness or loss of initial response. The main switch/restart combinations were ETA/ADA, n = 72, ADA/ETA, n = 20, ADA/ADA, n = 12 and ETA/ETA, n = 12. These data generated under daily-life conditions are consistent with the data also support the concern that after treatment interruption, patient wish or other reasons; for switching were primarily for ineffectiveness or loss of initial response. The main switch/restart combinations were ETA/ADA, n = 72, ADA/ETA, n = 20, ADA/ADA, n = 12 and ETA/ETA, n = 12. These data generated under daily-life conditions are consistent with
Psoriasis is a common chronic inflammatory skin disorder. Studies have shown that psoriasis can progress to systemic involvement such as in psoriatic arthritis, metabolic syndrome, and uric acid and lipid metabolism derangement. The aim of this study was to evaluate serum uric acid levels among patients with psoriasis. The departments of dermatology of two tertiary-care hospitals in Kathmandu, 138 patients were enrolled in the study. Among them were 69 patients with psoriasis selected as cases, 36 male and 33 female; 69 patients with other dermatological diseases after matching age and sex were selected as controls. After informed written consent was received, a full detailed history and physical examination was conducted, and determination of the body mass index (BMI) and Psoriasis Area and Severity Index (PASI) of cases was calculated. Serum samples of both cases and controls were sent for uric acid investigation. Chronic plaque psoriasis was the most common variant (60%, 87%), and there was no significant association between psoriasis type and sex. The male-to-female ratio was 1:1.1. The majority of the patients with psoriasis (76%) were among the younger population, and most of them (91%) had a normal serum uric acid level. Most of the patients (55%, 80%) did not have a family history of psoriasis. Most patients (58%, 84%) had history of a flare-up in the winter season. BMI was found to be in the normal range in most of the patients (65%, 94%). Among the control group, eczema was the most common diagnosis (16%, 33%). Among the patients (91%) had normal serum uric acid levels. No significant association between PASI score and serum uric acid level was found in the study (P = 0.81). Most of the patients had aggravation of psoriasis in winter. However, serum uric acid was not significantly associated with PsA, which may be due to psoriasis lesions being predominant on the body surface area involvement and no systemic complication issues. However, we have to rule out other systemic complications due to psoriasis in a long-term follow-up.

For a transcriptional study investigating the pathogenesis of generalized pustular psoriasis, M. Catapano,1,2 F. Capon,1 F. Ciccarelli2 and J. Barker1
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PSORIASIS
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Immunoogeneity with tildrakizumab, an anti-interleukin-23p19 monoclonal antibody, in a pooled analysis of three randomized controlled trials in patients with chronic plaque psoriasis A. Kimball1, A. Blauvelt2, K. Reich3, Q. Li4, L. F. van Alten5, T. Kerbschul6 and D. Montgomery7
1Harvard Medical School, Boston, MA, U.S.A.; 2Oregon Medical Research Center, Portland, OR, U.S.A.; 3MClinic Brussels Institute and Demeesterhuis Henegouwen, Gent, Belgium; 4Ernst & Söhne, Freiburg, Germany; 5Ernst & Söhne, Inc., Kenilworth, NJ, U.S.A.; 6Centres Universitaires de Strasbourg, France; 7Novartis Pharmaceutical Corporation, East Hanover, NJ, U.S.A.

We evaluated anti-tumor necrosis factor (TNF) therapy in patients with psoriasis who have failed to respond to two or more prior systemic therapies and who were candidates for consideration of biologic therapy. A total of 304 patients were enrolled in a randomized, double-blind, placebo-controlled, parallel-group study (P004). Patients were randomized to receive tildrakizumab (200 mg every 4 weeks or every 2 weeks) or placebo. Overall, 86% of patients treated with tildrakizumab achieved PASI 75 at week 12 (94% in the group receiving tildrakizumab every 2 weeks). In an analysis of patients with an absolute change of ≥10 in PASI score, 50% (95% CI 37–63) of patients treated with tildrakizumab achieved an improvement of ≥75% in PASI score compared with placebo (odds ratio 2.2, 95% CI 1.3–3.5; p = 0.002).

ADJUSTMENTS
**Poop**  
Next-generation sequencing identifies epidermal microRNAs deregulated in psoriasis skin  
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Karolinska Institutet, Stockholm, Sweden and  
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Psoriasis is a common chronic inflammatory skin disease, which is thought to be a result of aberrant interaction between keratinocytes and the immune system. MicroRNAs (miRNAs) are small noncoding RNAs that regulate the expression of the majority of protein-coding genes. We and others have previously identified miRNAs deregulated in psoriatic skin, which regulate keratinocyte and/or immune cell functions. Most of the previous studies utilized full-depth skin biopsies, which contain a mixture of cells, and thus cell-specific transcriptomic changes may have been masked by expression in other cell types. Here we analyzed the miRNome of CD45 (nonimmune) sorted epidermal cells from lesional and nonlesional skin of patients with psoriasis, and from healthy skin of control patients by next-generation sequencing (RNA-seq) of small RNAs. Our results revealed differential expression of 104 miRNAs in the epidermal cells in psoriasis lesions, including several known and novel miRNAs that have not been previously associated with psoriasis. MiR-149 was identified as one of the significantly downregulated miRNAs, and quantitative polymerase chain reaction analysis confirmed its downregulation in psoriatic epidermal cells compared with those from nonlesional or normal skin. In primary human keratinocytes and in three-dimensional epidermal equivalents, miR-149 was significantly downregulated by interferon (IFN)-γ overexpression of miR-149 suppressed the IFN-γ-induced expression of several inflammatory cytokines, as well as T-cell-attracting chemokines, while inhibition of endogenous miR-149 led to increased induction of these mediators. Taken together, we have characterized the cell-specific miRNome in psoriatic nonimmune cells in psoriatic skin, and identified epidermal miRNAs previously not associated with psoriasis. MiR-149 has been identified as a miRNome regulating the response of keratinocytes to IFN-γ. Our results can provide a basis for further functional studies of miRNAs in keratinocytes, which might lead to identification of potential targets for topical therapy in psoriasis.

**Poop**  
Effect of adipose-derived stem cells on an imiquimod-induced psoriasis mouse model by hypodermic injection.  
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We compared the effects of adipose-derived stem cells (ADSC) on psoriasis mouse model induced by hypodermic injection of imiquimod. ADSC were topically painted with imiquimod for seven consecutive days to develop psoriatic skin lesions. Mice in the experimental group had hypodermic injection of ADSC at day 3 and day 6, while the control group were given a hypodermic injection of normal saline. Various analyses related to lesion severity, inguinal lymph node changes and cytokines expression of lesions were carried out. Compared with the control group, the Psoriasis Area and Severity Index score was reduced in the experimental group. Inguinal lymph nodes as the immune organ were enlarged due to imiquimod painting in the control group. However, the size of inguinal lymph nodes trended to normal after ADSC injection. T helper (Th)1/Th2/Th17 cytokines in lesions were decreased by flow cytometry. Compared with the control group, the expressions of interferon-γ and tumor necrosis factor-α were decreased after ADSC injection. ADSC hypodermic injection can alleviate imiquimod-induced psoriasis lesion, and may exert immune regulation effects by direct cell-to-cell contact.

**Poop**  
Impairment of gustatory and olfactory senses in plaque psoriasis.  
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1University of Leeds, Leeds Institute of Rheumatic and Musculoskeletal Medicine, Leeds, UK  
2School of Cellular and Molecular Biology, Leeds, UK  
3University of Leeds, School of Dermatology, Leeds, UK  
4University of Bonn, Department of Dermatology, Bonn, Germany

Psoriasis is an autoimmune inflammatory skin disease. The various aspects of nutrition are a major issue in patient care for psoriasis. Subclinical disorders and increased body mass index are frequently encountered in this patient group and may result from systemic inflammation characteristic for the disease and/or unbalanced intake of food calories by the patients. Interestingly, in chronic inflammatory diseases relevant gustatory and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a disturbed food intake and are normalized again upon successful treatment of the disease. Here, patients with psoriasis were tested before any systemic treatment for the gustatory qualities and olfactory changes have been detected. These result in a
T-cell activation markers including ICOS and CTLA4, changes that were paralleled by decreases in myeloid (CD11c+)-dendritic cells and T cells using immunofluorescent measures. The results of this study establish that PH-10 has highly significant ability to modulate psoriatic inflammation, including key cytokine drivers of this disease, but only a subset of patients showed detectable plasma targets to that of non-lesional skin. This type of ‘mixed’ response outcome occurs with other topical or systemic drugs now approved for psoriasis, highlighting a need to personalize treatments and potentially produce specific responder biomarkers for individual drugs.

**PsA**

**Poster withdrawn.**

**PsA**

Immune modulation by topical PH-10 aqueous hydrogel (rose Bengal diosodium) in psoriasis lesions. J.G. Krueger,1 S. Garcez,1 J. Fuentes-Duculan,2 N. Kurkinen,3 L. Cueto,4 L. Li,5 J.M. Singer5 and E.A. Wachtel5


PH-10 is topical hydrogel formulation that yields selective delivery of rose Bengal diosodium (RB) to epithelial tissue. RB is a fluorescein derivative capable of producing singlet oxygen upon photoactivation, but its therapeutic mechanism in psoriasis is not established. We thus conducted a mechanically focused study of PH-10 in 10 patients with psoriasis lesions using sequential vehicle and active drug treatments for 4 weeks each (registered clinical trial NCT03320846). Skin biopsies were collected before treatment and at the end of each treatment period. Effects of placebo vs. active treatment were assessed on cellular immune infiltrates, driver cytokines of psoriasis and the overall disease transcriptome using immunohistochemistry and gene-expression profiling with Affymetrix U133 3.0 arrays and reverse-transcriptase polymerase chain reaction (RT-PCR). Vehicle treatment for 4 weeks did not significantly alter expression of core inter-leukin (IL)-23/IL-17-mediated genes or the overall disease transcriptome (using a principle component analysis, PCA). However, 4 weeks of treatment with PH-10 significantly (fold change $>1.5$, $P<0.05$) increased transcripts encoding IL-23, IL-26, IL-16, and keratin 16 mRNA as assessed by RT-PCR, while a PCA analysis of the gene array results showed a shift towards non-lesional skin with some post-treatment biopsies clustering within the non-lesional skin profile. Pathways that were significantly improved by PH-10 included published psoriasis transcripts and cellular responses mediated by IL-17, IL-22 and IL-26. To strengthen the analysis of immune- and psoriasis-related gene modulation by PH-10, we divided patients into responders vs. nonresponders based on the PCA analysis after 4 weeks of treatment (comparing to non-lesional skin at baseline). Using this approach, more than 500 disease-related genes were downregulated during 4 weeks of treatment with PH-10, and expressions of a wide range of central ‘psoriasis related’ genes including IL-23, IL-17, IL-22, S100A8, IL-19, IL-36 and CCL2 were effectively normalized; treated non-lesional skin had values in the same range as baseline non-lesional skin. We also measured decreased expression of monocyte/macrophage. Based on PK data only, there is no need for dosage adjustment for these intrinsic and extrinsic factors. Nonetheless, bodyweight was influential and was subsequently evaluated in a PK-pharmacodynamics model.

**PsA2**

**Poster withdrawn.**

**PsA**

Favourable safety profile of ixekizumab: results from 11 moderate-to-severe psoriasis and three psoriatic arthritis clinical trials. A. Gottlieb,1 K. Papp,2 W. Xu,3 L. Malbris3 and N. Agda4

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IxE psoriasis safety data were evaluated in a PK-pharmacodynamics model. Evidence for an acceptable safety profile of ixekizumab is derived from 11 controlled and uncontrolled clinical trials in patients with moderate-to-severe plaque psoriasis, including the phase III trials UNCOVER-I, -2 and -3. Integrated DEE PH data safety data were from three controlled and uncontrolled clinical trials in patients with active PsA, including the phase III trials SPURT-Pi and -Pi2. Frequencies of treatment-emergent adverse events (AEs) and adverse events of special interest (AESIs) are provided here. Exposure-adjusted incidence rates (IRs) of AESIs were summarized. The IR was expressed as the number of patients with a particular AEFI, mostly per patient-years (PYs), using the entire duration of exposure during treatment period. The 95% confidence interval of the IR was calculated based on the Poisson regression model with treatment as the explanatory variable. Major adverse cerebro-cardiovascular events were adjudicated by an external adjudication committee. A total of 5689 patients (4210 IxE and 1479 Placebo) with psoriasis and 1118 patients (1373 PU) with PsA were exposed to DEE. AEs, mostly mild or moderate, were reported by 475 (8.3%) patients with psoriasis and by 688 (7.6%) patients with PsA. Injection-site reactions, mostly minor or moderate, were reported by 840 (14.8%) patients with psoriasis and by 227 (20.3%) patients with PsA, and rarely resulted in study drug discontinuation. The IRs of injection-site reactions decreased substantially over time in both patients with psoriasis and those with PsA. Setons adverse events occurred among 470 (11.8%) and 91 (8.1%) patients with psoriasis and PsA, respectively. Adverse events leading to discontinuation of study drug occurred among 379 (6.7%) patients with psoriasis and 80 (7.7%) patients with PsA. There were 23 (0.4%) deaths among patients with psoriasis and two (0.2%) deaths among patients with PsA. Most deaths resulted from cardiovascular events in those with a history of risk factors, and none was attributed to the study drug. The present report supports a favourable safety profile of ixekizumab in different patient populations, those with moderate-to-severe psoriasis and those with active PsA. This study was funded by Eli Lilly and Company, Indianapolis, IN, U.S.A.

**PsA3**

**Poster withdrawn.**

**PsA**

Cytokine effects of apremilast as a mechanism of efficacy in systemic-naive patients with moderate plaque psoriasis: results from the UV-EIL trial. B. Strayer,1 M. Alkibah,2 B. Leckshin2 and P. Schaller3

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Previous pharmacodynamic (PD) subanalyses of clinical trials have demonstrated that the effects of apremilast on key cytokines involved in the pathogenesis of plaque psoriasis play a role in determining clinical efficacy. It was therefore of interest to perform a more detailed PD substudy of a phase IV randomized, controlled trial (UV-EIL), which evaluated the efficacy and safety of apremilast 30 mg twice daily (APB) in the treatment of systemic-naive patients with moderate plaque psoriasis (psoriasis-involved body surface area (BSA) 5%-10%; state Physician’s Global Assessment (sGA) <3 moderate). Patients were randomized (2 : 1) to APB or placebo (PBO) for 16 weeks. From the PD patient subset, blood samples obtained at weeks 0 (baseline) and 16 were analysed for interleukin (IL)-17A, -17F, -22, IL-23, IL-23, IL-26; adiponectin; apolipoproteins A-I, A-II, B and E; and numbers of circulating T helper 17 (Th17) cells, regulatory T cells (Treg) and total T-cell numbers. Correlations were examined between percent-change from baseline in key inflammatory biomarkers and clinical efficacy, based on assessments using the product of the sGA and psoriasis-involved BSA (P<0.05). Of the total 321 patients randomized into the phase of treatment, the PD sub-population included 38 patients (APB, n = 26; PBO, n = 12). At week 4, the median percentage changes from baseline in IL-17A, IL-17F, IL-22 and IL-23 with APB vs. PBO were P<0.05 (IL-17A, -17F, -22 and -23 with APB vs. PBO were P<0.05). At week 16, percentage change in IL-17A was significantly correlated with percentage change (improvement) in P<0.05). At weeks 4 and 16, levels were $<0.2$% and $<0.2$% different among patients with psoriasis and those with PsA. Most deaths resulted from cardiovascular events in those with a history of risk factors, and none was attributed to the study drug. The present report supports a favourable safety profile of ixekizumab in different patient populations, those with moderate-to-severe psoriasis and those with active PsA. This study was funded by Eli Lilly and Company, Indianapolis, IN, U.S.A.
Patients with certain human leukocyte antigen expression have a higher risk of developing psoriasis1
A. Anandan, K. Radhasrinivas, R. Prasada and T.K. Panicker

Severe atopy in patients with psoriasis: A prospective study
D. Kivelietvicius,1 K. Rahimi1 and A. Menter1

Apremilast: a novel oral drug for the treatment of plaque psoriasis
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Nail psoriasis is associated with decreased finger mobility, functional impairment, pain and reduced quality of life and is often difficult to treat. It correlates with more severe psoriatic disease and is an important predictor of psoriatic arthritis (PsA). Nails are affected in up to 50% of patients with

ABSTRACTS

Psoriasis is an immune-mediated genetically determined skin disorder affecting 1–3% of people worldwide. Psoriasis is a multifactorial disease and has been associated with certain human leukocyte antigen (HLA) expressions. This review is focused on HLA association in psoriasis by screening patients with psoriasis for HLA class I and determining its odds ratio (OR). The study was conducted with the help of the Department of Transfusion Medicine at a tertiary-care hospital. In total 100 patients with psoriasis (cases) and 100 controls (healthy blood donors) were enrolled in the study. Samples from the patients with psoriasis were collected from dermatology outpatient department and the samples from the controls were taken from voluntary blood donors at the department of transfusion medicine after obtaining consent. HLA typing for class I was done using the single-specific primer polymerase chain reaction method. The HLA results were statistically analyzed and the association with psoriasis was determined. The alleles that were found in higher frequency among the cases were HLA-A*02 (45%, OR 2.15); HLA-A*01 (35%, OR 2.00) and HLA-B*07 (36%, OR 2.40). The study was conducted at the department of transfusion medicine. In this study, 91 patients on high-dose MTX were evaluated using SWE. Fibrosis stage was defined as: 6–7.9 kPa (stage 1); 8–9.9 kPa (stage 2); 10–11.9 kPa (stage 3) or ≥ 12 kPa (stage 4 or early cirrhosis). A subtest, liver stiffness measurement was performed based on the results of SWE and risk factors for metabolic syndrome. Between January 2014 and May 2016, 91 patients were enrolled: mean age 61 years, 76% female; 59% with psoriasis (52 patients), 30% rheumatoid arthritis (26 patients) and 11% other (seven patients); mean body mass index (BMI) 32.1 ± 7.9 kg m−2; on a mean high cumulative MTX dose of 1374.4 ± 1391 mg. Nonalcoholic fatty liver disease (NAFLD) was diagnosed on imaging or history and diabetes were seen in 38 (42%) and 19 (21%) patients, respectively. Six patients were excluded due to missing data. Higher liver stiffness was associated with NAFLD (32.1 ± 7.9 kg m−2) compared to DAH patients (20.7 ± 6.8 kg m−2) (p = 0.001). NAFLD patients had a significantly higher body mass index (BMI) than DAH patients (p = 0.001). NAFLD patients also had a significantly higher BMI than patients with diabetes (p = 0.04). Multivariate logistic regression analysis showed that the association with psoriasis was independent of BMI (35%, OR 2.15). HLA-A*02 (36%, OR 2.40) was observed at a higher frequency among the cases. The alleles that were found in higher frequency among the controls were HLA-A*02 (45%) and HLA-A*03 (36%). AEs were consistent with the known safety profile of apremilast. In clinical routine care in Germany, outcomes of apremilast treatment for patients with psoriasis without comorbidities were comparable with results from apremilast clinical trials. This study was funded by Celgene Corporation.

Psoriasis is one of the most common chronic inflammatory skin diseases affecting about 3% of people worldwide. The condition is characterized by the development of plaques of thick, scaly skin, which is itchy and may cause discomfort. The disease affects people of all ages and can be triggered by a combination of genetic and environmental factors. Although the exact cause of psoriasis is not fully understood, it is believed to involve an imbalance in the immune system, leading to an overproduction of skin cells. This results in the formation of plaques on the skin, which can appear anywhere on the body but are most commonly found on the elbows, knees, and scalp. Psoriasis is a lifelong condition that cannot be cured, but treatments are available to help manage symptoms and control the disease.

Apremilast is a novel oral drug that works by inhibiting a protein called Janus kinases (JAKs). JAKs are enzymes that play a role in the immune system, and their activity is thought to be increased in people with psoriasis. By blocking JAKs, apremilast can help reduce the immune system’s response to trigger the psoriasis plaques. This mechanism of action is similar to other drugs used to treat psoriasis, such as methotrexate, but apremilast offers a different target for treatment.

The studies reviewed in this review focused on the use of apremilast in treating psoriasis, with a particular emphasis on its efficacy and safety profile. The review highlights the results of clinical trials that have evaluated apremilast’s effectiveness in reducing the symptoms of psoriasis, such as plaque size, itchiness, and redness. It also discusses the drug’s safety, including the incidence of adverse events and the impact of these events on patient quality of life. The review concludes with an analysis of the costs associated with apremilast treatment and a consideration of the drug’s overall value in the context of the current management of psoriasis.

The use of apremilast in clinical practice is supported by the clinical trial data, which demonstrate its efficacy and safety. However, further research is needed to fully understand the drug’s long-term effects and to explore its potential as a treatment for other indications, such as psoriatic arthritis. Overall, apremilast offers a promising new therapy for people with psoriasis, providing an additional option in the management of this chronic and challenging disease.
carried forward). Patients showed considerable improvements from baseline to 2.5 years by ment in Psoriasis and Psoriatic Arthritis quality-of-life scores (150 mg), and 70.2% (300 mg) and 71.0% (150 mg) of this study were met, demonstrating superiority of secukinumab 150 and 300 mg subcutaneous in moderate-to-severe nail psoriasis treated with secukinumab. TRANSFIGURE is a study of clinically meaningful efficacy, large quality-of-life improvement and a favourable safety profile up to 2.5 years in difficult-to-treat nail psoriasis. This investigation was sponsored by Novartis Pharma AG, Basel, Switzerland.

Ps2
Secukinumab pooled and long-term safety: analysis of 29 psoriasis clinical trials
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We report exposure adjusted incidence rates (IRs) for treatment emergent adverse events per year from a pooled analysis of all secukinumab psoriasis trials to date (19 studies, 4647 patients, 10 061 patient-years of exposure). Adverse event (AE) IRs (per 100 patient-years) were examined per year for patients who received either secukinumab 500 mg or any dose of etanercept, infliximab, and, for 1 year only, for patients receiving placebo (PBO), etanercept (ETN) 50 mg or ustekinumab (UST) 45/75 mg. The pooled safety of secukinumab remained favourable over the 5 years of treatment with no new safety signals over time. Additionally, secukinumab demonstrated a comparable pooled safety profile to that of PBO, ETN and UST over the course of 1 year (see Table; MACE, major adverse cardiovascular event; NMSC, non-melanoma skin cancer; USTI, upper respiratory tract infection). This comprehensive pooled analysis supports the favourable long-term safety profile of secukinumab in patients with psoriasis. This investigation was sponsored by Novartis Pharma AG, Basel, Switzerland.

Ps3
Lymphoma action in the regulation of inflammatory processes on the example of psoriasis
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Chronic inflammation is strongly associated with the pathogenesis of many chronic inflammatory diseases, including psoriasis. Despite the ever-growing knowledge of the causes and development of abnormal immune responses, factors involved in these processes are still being sought. The main goal of the following research is clarification of the role of lysosomes in the development of inflammation and results obtained regarding correlation of lysosomal calcium ion levels and calcium activity in vitro at later stages. Studies are carried out on three models: (i) in vitro ‘psoriasis-like’ activated HaCaT cells, (ii) in vitro keratinocytes derived from patient tissue (from lesional and nonlesional psoriatic skin as a control), and (iii) in vitro directly on skin tissue biopsies (from lesional and nonlesional psoriatic skin and normal skin as a control). Preliminary analyses were designed to examine changes in the amount of lysosomes in the HaCat cell line. Keratinocytes were treated with proinflammatory cytokines [interleukin (IL)-1A, IL-17A, IL-22, oncostatin M and tumour necrosis factor α] to achieve the ‘psoriasis-like’ phenotype, and then stained for the fluorescence microscopy images. In the course of this work we concluded that the number of lysosomes is reduced in stimulated keratinocytes compared with controls (nonactivated cells). To verify the validity of the obtained data, the expression of genes responsible for lysosomal biogenesis (i.e., Mit family genes: THG, TFB, TFC and MTII) by real-time quantitative reverse-transcriptase polymerase chain reaction in patients’ skin biopsies was examined. The results showed downregulation of THG, TFB and MTII in both lesional and nonlesional psoriatic skin compared with the normal skin from healthy individuals. In turn, the level of the THG gene was elevated in both cases, or unchanged in lesional vs. nonlesional tissue. The above data indicate potential lysosomal biogenic disturbances, in the direction of lowering the efficiency of this process in skin of patients with psoriasis. The obtained results were compared with the activity of genes encoded by calcium-binding subunits PPP6C and PPP1C. We observed reduced expression of both genes in psoriatic skin tissue, which may indicate that dysfunction of factors in the Mit family can be aggravated by impaired calcium homeostasis and their regulation by calcinurin. These data may complement the lack of knowledge about the role of lysosomes in the development of inflammation on the example of psoriasis and to the development of new therapeutic targets and strategies.

Ps4
Secukinumab demonstrates significantly lower immunogenicity potential than ixekizumab in human in vitro assays
S. Spindlerdrefhy1, B. Mailler2, E. Correa3, M. Tenon4, A. Kärle5, P. Jarvis6 and F. Kolbinger7
1Novartis Pharma AG, Basel, Switzerland and Vita-Sedex, Institute Frédéric Joliot, Gif sur Yvette, France. Secukinumab, a fully human monoclonal antibody (mAb) that selectively neutralizes interleukin-17A, has significant efficacy in the treatment of moderate-to-severe plaque psoriasis and psoriatic arthritis (PsA). It demonstrates a rapid onset of action and sustained responses with a favourable safety profile and 1% immunogenicity rate in nonclinical or clinical studies. Secukinumab has previously demonstrated lower potential for immunogenicity than other biotechnologies used to treat psori- nisms and PsA in in vivo assays. Here we extend the analysis and compare the T-cell precursor frequencies against 4 mAbs (secukinumab, ixekizumab, adalimumab and ustekinumab). Two sets of 16 healthy donors were analysed (study 1 and study 2). Immunogenic potential was evaluated using an in vitro T-cell amplification assay to measure the frequency of mAb-specific pre-existing T cells from those donors. Monocytederived dendritic cells (DCs) were generated from peripheral blood mononuclear cells and exposed in vitro to mAbs or a positive control (keyhole limpet haemocyanin) and matured. CD4 T cells were stimulated by matured protein-loaded DCs and cultured for 21 days. An Elispot assay was used to assess the antigen specificity of T-cell lines. The frequencies of mAb-specific pre-existing T cells were calculated relative to the proportion of culture wells that reacted to the protein. The data were analysed using a Wilcoxon rank test. In study 1, one of 16 donors responded to secukinumab, generating one T-cell line (mean frequency 0.02 cells per million T cells; low immunogenicity potential). In contrast, nine of 16 donors responded to ixekizumab (15 T-cell lines, frequency 0.54), nine of 16 responded to adalimumab (14 T-cell lines, frequency 0.31) and six of 15 responded to UST (14 T-cell lines, 0.19), show- ing moderate immunogenic potential. In study 2, one of 15 generated T-cell lines was specific for secukinumab, with a mean frequency of 0.03 cells per million CD4 T cells (low immunogenic potential), while seven donors responded to ixekizumab, with a frequency of 0.31–0.87 specific CD4 T
P025 Decreased expression of interleukin-27 in moderate-to-severe psoriasis and its anti-inflammatory role in an iniquin-induced psoriasis-like mouse model

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Psoriasis is a T-cell-mediated chronic inflammatory skin disease characterized by aberrant keratinocyte hyperproliferation. A recent study reported that interleukin (IL)-27, which plays a versatile role in inflammatory skin disorders, could induce the function of T helper (Th)1 cells in psoriasis. Further studies found a suppression role of IL-27 in Th17. Here we investigated and compared the expression of IL-27 and its receptor in patients with moderate-to-severe psoriasis vs. healthy controls. Afterward, we identified the role of IL-27 in an iniquin-induced mouse model by injection with IL-27 or IL-27p28 antagonist. The results showed that the expression and distribution of IL-27 and its receptor were decreased in the skin and peripheral blood of patients with moderate-to-severe psoriasis compared with healthy controls, and IL-27 concentration was significantly decreased in psoriasis serum. Moreover, mRNA levels of IL-27p28, WSC1 and gpi10 in patients with moderate-to-severe psoriasis were significantly lower than those of healthy controls. IL-27 concentration and mRNA level of BRII mRNAs did not vary remarkably. Iniquin-induced mice treated with IL-27 showed a relatively mild clinical manifestation. None of the IL-27p28 mice in iniquin-induced mice significantly changed disease activity, and mRNA levels of IL-27p28, WSC1 and gpi10 in patients with moderate-to-severe psoriasis were significantly lower than those of healthy controls.

P026 Secukinumab shows high and sustained efficacy in patients with moderate-to-severe palmoplantar psoriasis: 2.5-year results from the GESTURE study

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To analyse the clinical efficacy and safety in Chinese patients with psoriasis treated with infliximab, all treated patients in the Dermatology Department of Peking University Third Hospital were reviewed. The types of psoriasis, Psoriasis Area and Severity Index (PASI) score and clinical response were calculated. Infliximab was given at a dose of 5 mg kg⁻¹ by intravenous infusion at weeks 0, 2, 6, 12 and 16, followed by maintenance infusion every 8 weeks. From 2015 to 2017, 27 of 98 infliximab patients in our department were treated with infliximab: 18 male, nine females, mean age 40.6 ± 13.7 years (range 21–71), and the mean duration of psoriasis was 7.6 ± 9.9 years (range 0.4–49). Twenty-two cases were of plaque psoriasis, including 14 male and eight female patients. The mean baseline PASI was 19.0 ± 11.4 (range 2.4–46.7). The mean plaque psoriasis baseline PASI was 17.8 ± 10.7 (range 2.4–46.7). Most patients received three to seven infusions. Fourteen patients, including 11 with plaque psoriasis, strictly adhered to the therapeutic schedule before 14 weeks. In all 14 with at least 14 weeks of strict adherence to therapy, the average PASI score decreased from 18.7 to 2.7 at the 14th week. The percentage change from baseline in PASI reached 81% at the week 14. In 11 patients with plaque psoriasis, 81% of patients reached 81% at least 14 weeks of strict adherence to treatment, the average PASI score decreased from 16.7 to 3.7 at the week 14. The percentage change from baseline in PASI reached 81% at the week 14. In total 37% of patients reached PASI 90 (>90% improvement from baseline), 18% of patients reached PASI 75 (75% of patients reached PASI 50). One case aggravated and one did not adhere to the therapeutic schedule before 14 weeks. Two patients maintained PASI 90 for 10 months and 17 months, respectively. Five patients maintained PASI 90 for seven patients, including four with plaque psoriasis, maintained PASI 50. One patient showed rebounded after 6 months without any treatment. Six cases relapsed, in six cases, where the improvement in PASI score fell below 50% from baseline. None rebound in 3 months. Six of 27 (22%) patients had adverse reactions such as pruritus, induration, shortness of breath, cold sweat, blood pressure decrease, mild distending head pain, hoarses zoster, slight chest distress, shortness of breath and low-grade fever. There were no severe adverse reactions. Infliximab has a good clinical outcome and is relatively safe in the treatment of the Chinese patients with psoriasis.

P027 Secukinumab clinical outcomes in a tertiary referral centre

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Secukinumab is a fully human monoclonal antibody against interleukin-17A. Long-term efficacy has been demonstrated in clinical trials, with up to 26% of patients achieving ≥ 90% improvement in Psoriasis Area and Severity Index (PASI) 90 at week 52 (Blauvelt A, Reich K, Tsai TF et al. Secukinumab is superior to ustekinumab in the treatment of patients with moderate-to-severe plaque psoriasis up to 1 year: results from the CLEAR study. J Am Acad Dermatol 2016;74: 60–9.). The National Institute for Health and Care Excellence (NICE) recommends secukinumab in patients with severe psoriasis [PASI and Dermatology Life Quality Index (DLQI) > 10] and in those with contraindications, intolerance or failed responses to other systemic therapies. There is therefore a need to evaluate the appropriateness and efficacy of secukinumab in all the patients treated in our tertiary referral centre. Patients were identified from our pharmacy database and data were collected from electronic patient record systems and research trial documentation between December 2013 and March 2017. The PASI and DLQI scores were extracted at baseline and weeks 12 and week 52. Sixteen patients were identified (n = 14) of whom met the criteria for severe psoriasis. Thirteen (81%) had been on previous systemic, 13% (n = 3) had contraindications and 13% (n = 5) were intolerant to other systemic agents. Seven were clinical trial patients, while nine were nontrial patients; nine patients were biologic naïve while seven had previous biologics. An average PASI reduction of 80.6% was observed after 12 weeks and 73.2% after 52 weeks. Of the data available, 10% of patients achieved PASI 100, 40% PASI 90 and 30% PASI 75 at week 52. Overall, 90% (n = 10) and 80% (n = 6) of patients achieved the NICE criteria for response assessment at weeks 12 and week 52, respectively, of the data available. Our data demonstrate that secukinumab, despite being more efficacious in biologic-naïve patients, is also effective in treatment-resistant patients who have failed previous biologics at 1-year. More real-life data are required to assess longer-term safety and efficacy.
Successful treatment of psoriasis with secukinumab, a novel interleukin-17A inhibitor: a real-world experience

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Objectives: To evaluate the clinical performance of secukinumab, a novel interleukin-17A inhibitor, in the real world.

Methods: We retrospectively reviewed the clinical records of all patients who were treated with secukinumab at multiple sites in Japan from January 2014 to September 2016. The records were analyzed regarding efficacy, safety, and patient characteristics.

Results: A total of 3311 patients were treated with secukinumab. Most patients were male (57.3%), and the mean age at treatment initiation was 46.6 years. The mean PASI and DLQI at baseline were 14.9 and 13.2, respectively. Of these patients, 1040 patients (31.2%) were treated with secukinumab for more than 1 year. The mean PASI and DLQI at the end of treatment were 3.8 and 13.7, respectively. The proportion of patients who achieved PASI 75, 90, and 100 responses were 67.4%, 34.3%, and 20.3%, respectively. The proportion of patients who achieved DLQI ≤ 5 at 12 weeks was 56.2%. The proportion of patients who achieved PASI 75 or 90 with a complete improvement in their PGA score was 61.7%. The proportion of patients who achieved PASI 75, 90, and 100 responses were 37.2%, 23.4%, and 12.3%, respectively. The proportion of patients who achieved DLQI ≤ 5 at 12 weeks was 56.2%.

Conclusion: Secukinumab is a novel interleukin-17A inhibitor that has been shown to be effective and safe in the real world. It is a promising treatment option for patients with psoriasis.
loss. Two patients developed de novo paradoxical palmoplantar pustular psoriasis affecting both upper extremities, posterior trunk and gluteus. One patient had a dose of 8 mg per day and ciclosporin 125 mg per day, and was switched to an alternative TNFi, resulting in a flaccid bullous. The patient had had PXP for 1 year and since then she has received methyl prednisolone (MP) and methotrexate. There was no history of similar illness in her family. Physical examination demonstrates signs of Cushing syndrome. Dermatological examination revealed multiple erythematous plaques, vesicles, pustules and erosions in an annular shape with scale accompanied by multiple striae on the anterior and posterior trunk and abdomen. On the upper extremities, posterior trunk and gluteus were vesicles and flaccid bullae on erethenous skin. There was no mucosal involvement or nail abnormalities. Histopathological examinations using hematoxylin and eosin staining taken from two different places were both consistent GPP and MP. Examinations of direct immunoflorescence supported MP. The patient is being treated with MP and ciclosporin. In the first 2 months of therapy the patient was tapering the dose MP. The patient was maintained with MP dose of 8 mg per day and ciclosporin 125 mg per day, and his condition has been controlled for 9 months. GPP and PXP are two very different skin diseases, both clinically and pathogenetically. Their consistent clinical expression possibly happened because of T-cell-dependent chronic immunostimulation and phototherapy. In PXP and psoriasis vulgaris, their specific human leucocyte antigen allele, namely HLA-DR3, has been identified, which is also a specific gene in psoriasis. Systemic corticosteroids are the mainstay of therapy for PXP, but not in the routine management of patients, especially for PXP. Currently there are no therapy guidelines for PXP. Therefore, a strategy for management of corticosteroid administration is needed.

A retrospective clinical audit was conducted to evaluate the assessment and management of patients with psoriasis within a tertiary centre. The data were compared against the U.K. National Institute for Health and Care Excellence (NICE) Psoriasis: Assessment and Management (CG153). The parameters reviewed included use of validated tools: Psoriasis Area and Severity Index (PASI), Physician Global Assessment, Dermatology Life Quality Index (DLQI) and PEST (Psoriasis Epidemiological Screening Tool). The arms were to assess current clinical practice as measured against the NICE standards with review of services including rheumatology referral, ultraviolet (UVB) phototherapy and use of systemic biologic treatments. UVA phototherapy and systemic biologic treatments were not assessed. Data were collected from 50 patients via random selection using data informatics. One patient was excluded (no psoriasis). A hospital electronic database was used to collate patient data, which were entered into a Microsoft Excel spreadsheet using the Clinical Audit Toolkit (NICI CG151) for generation of results. Use of UVB phototherapies had 53% compliance and biologic therapies 62% compliance. PASI and DLQI assessment completion were both 82%, and Physician’s Global Assessment compliance was 55%. PASI is the most utilised and validated clinical tool to assess severity in patients with psoriasis. Documentation of body surface area compliance was 87%, with difficult-to-treat sites at 64% and recording of systemic upset at 60%. Studies have shown that use of both PASI and Physician’s Global Assessment simultaneously is redundant. Result analysis and interpretation were processed automatically by the national toolkit database; however, raw data interpretation led to plausible rationales for some discrepancies. To increase PASI, DLQI and PASI compliance with the NICE guidance, we have designed a tool with a set of electronic pro forma that saves data collection and provides prompts for essential information for completion within consultations. Outcomes for the project include a ‘how to assess patients with psoriasis’ template for consultations, departmental audit presentation and resultant 1 year after the alliance NPC was set up. The British Association of Dermatologists in General Practice recommends a central resource for scoring tools and disease-specific pro formas to assess patients, but currently there are none. Clinicians will use standard scoring systems that will allow appropriate classification of patients and individualized treatment plans. These are crucial in the management of patients with psoriasis.

A large-scale imputation of killer cell immunoglobulin-like receptor copy number in psoriatic arthritis

K. Amr,1, D. Valcupe,1,2, A. Motey,1,3 A. Ellingshaus,4 L.C. Tsoi,5 R.P. Nair,5 C. Palmer,6 J. Oksenberg,1 D. T. Neriifolia belongs to the family Apocynaceae and is a tropical shrub that can be found in Indonesia, Malaysia, the Philippines, and Australia. The plant contains a milky sap that can be extracted from either the leaves or seeds. This sap is full of active substances and has antihyperlipidemic and anti-inflammatory properties. In addition, it may prevent the cell damage caused by psoriasis. We describe the case of a male patient, 31 years old, who lost his parents and was very affected by their death. Firstly, he developed erythrodermic psoriasis lesions (psoriasis lesions) in the inner side of both feet. The plaque lesions went on to cover most of the body without fever. His emotional stress was implicated in the induction of the pustules. He has been using topical steroids since July 2016, with no improvement of the condition (Arena C, Morti AS, Blanchet T et al. Impact of glucocorticoid-induced adverse events on adherence in patients receiving long-term systemic glucocorticoid therapy. J Derr 2010; 143: 812-7). The patient used the milky sap of T. neriifolia for 1 month. During this period we noticed a favourable evolution of his disease. The erythrodermic psoriasis lesions started to disappear gradually after 2 weeks of daily application of T. neriifolia on his skin. The patient is still under treatment. Through this case, T. neriifolia could be considered a potential source in the manufacture of drugs against psoriasis.
There is evidence that patients with psoriasis have increased visceral adiposity, which represents not only an energetic deposit but also an endocrine organ secreting adipokines. The main goal of psoriatic therapies is to control the disease and its clinical manifestations. The use of biological drugs, such as antitumour necrosis factor (anti-TNF) agents, seems to be associated with an increase in adiposity. In the literature it is reported that ustekinumab does not increase body mass index (BMI) in patients with chronic plaque psoriasis, but there are no studies on changes of the body composition in these patients. Unfortunately, BMI fails to distinguish body components such as fat mass and free fat mass in single individuals. On the other hand, obesity may influence the therapeutic approach to psoriasis and the clinical response to treatment. In adipose tissue may alter the volume of distribution, limiting the efficacy of the drug. The purpose of this study was to investigate the body composition in patients with psoriasis treated with ustekinumab using bioelectrical impedance analysis and to correlate the bioelectrical impedance data with clinical response to treatment. The study population consisted of 310 patients affected by moderate-to-severe chronic plaque psoriasis, naive to biological treatment and treated with ustekinumab for at least 1 year. All anthropometric measurements, including weight, height and waist circumference, were collected at baseline, after 6 months of treatment and then after 1 year. For each patient, we evaluated body composition using bioelectrical impedance analysis and Bivar/Vector analysis. Linear mixed-effect models for repeated measures were applied in order to assess the differences in the body composition from baseline to 6 and 12 months. For each variable of the body composition, we fit a linear mixed-effect models where time (baseline, 6 months and 12 months) and confounding factors were the explanatory variables. Significant decrease was found at month 6 for weight (–0.94 kg, P = 0.03), BMI (–0.30 kg m⁻², F = 0.005) and fat mass (–1.06, P = 0.03). Combinatorially, free fat mass and total body water showed increases of 1.06 (F = 0.003) and 0.78 (P = 0.01), respectively. The significant changes from baseline were not confirmed at month 12. A significant increase was found at month 12 for intracellular water (2.41, P = 0.001) and a significant decrease for extracellular water (–2.37, F = 0.001). In conclusion, the response to ustekinumab treatment, we did not find a difference between the rate of responders and the body composition. This is the first study to analyse body composition changes in patients treated with ustekinumab, and in contrast with data reported in studies on anti-TNF-α drugs, we can say that ustekinumab does not increase adipose tissue, and it appears to improve intracellular hydration. In addition, the efficacy of the drug does not appear to be correlated with the patient’s body composition.
perceive a difference between the two agents, 78% believe ustekinumab is superior when asked to compare the two for ‘efficacy in skin clearance’. Twice as many dermatologists also believed that ustekinumab was faster than adalimumab, an attribute important to patients. Secukinumab holds a better perceived advantage over ustekinumab for effectiveness in psoriatic arthritis, which is currently the only FDA-approved IL-17 agent available. Future expectations for the IL-17 class are favourable, with half of the surveyed dermatologists anticipating an increase in their use of both ustekinumab and secu- kinumab in the second half of 2017. To what extent the IL-17 inhibitors can grow will depend on two leading factors: competition from recently approved guselkumab, which will compete for a similar patient type, and restrictions by man- aged care, which dermatologists note are the greatest barriers to increased use of these agents.

**Po44**

Electronic monitoring of psoriasis outcomes and goals in practice: development and introduction of a standard dataset and digital management system

M. A. Radtke and A. Navarini

A retrospective patient chart review of over 1000 patients with psoriasis was conducted in August 2017 by an independent healthcare market research firm that specializes in tracking biological uptake in various autoimmune conditions including psoriasis and psoriatic arthritis. Over 200 U.S. dermatologists in clinical practice conducted an in-depth review of patients they had recently switched from one biological or apremilast to a different agent. To qualify, patients had to have been switched to another biological or apremilast within the past 3 months. The audit was compliant with the Health Insurance Portability and Accountability Act and conformed to requirements under the Safe Harbor Act. Additionally, participating dermatologists completed a questionnaire about their practice and their opinions about the use of biologics and small molecule agents in practice. Those who completed a Delphi process, followed by a second consensus round on critical clinical thresholds and end points. The final set of outcomes was tested in selected centres for feasibility, patient acceptance and technical performance. The technical solution was provided by Swiss- mediated, a technology company for digital solutions in dermatology. All development steps were cross- validated with conventional clinical trial data. The electronic outcomes set included Psoriasis Area and Severity Index, body surface area, Dermatology Life Quality Index and Patient Benef- it Index, as well as patient treatment satisfaction and, optionally, a grid body surface permitting patients to mark the body areas affected. Other facultative instruments were included. For the identification of disease-controlled days, an electronic diary was developed and validated, which measured a continuous patient-reported data. For each instrument, a series of electronic documentation systems was developed. Finally, a series of goals of outcomes was achieved by monitoring relevant patient outcomes in an electronic switch- board, enabling continuous recording of the patient outcomes and controlling the achievement of out- comes. In the first testing, the electronic documentation and management system showed good technical properties and was well accepted by patients. The patient education tool markedly increased patient satisfaction and adherence. In con- clusion, the electronic documentation system and the resulting digital toolboxes are feasible and beneficial technologies for the improved management of psoriasis in practice. Areas of use included routine patient record reports, patient regestries and clinical trials.

**Po45**

Real-world data identify reasons for biological switching in patients with psoriasis

J. Robinson and L. Price

Jubilant GS Services Ltd, PA, U.S.A.

A retrospective patient chart review of over 1000 patients with psoriasis was conducted in August 2017 by an independent health care market research firm. The main objective of the study was to identify the factors that contribute to switching between biological agents in psoriasis. The audit was compliant with the Health Insurance Portability and Accountability Act and conformed to requirements under the Safe Harbor Act. Additionally, participating dermatologists completed a questionnaire about their practice and their opinions about the use of biologics and small molecule agents in practice. Those who completed a Delphi process, followed by a second consensus round on critical clinical thresholds and end points. The final set of outcomes was tested in selected centres for feasibility, patient acceptance and technical performance. The technical solution was provided by Swiss- mediated, a technology company for digital solutions in dermatology. All development steps were cross- validated with conventional clinical trial data. The electronic outcomes set included Psoriasis Area and Severity Index, body surface area, Dermatology Life Quality Index and Patient Benef- it Index, as well as patient treatment satisfaction and, optionally, a grid body surface permitting patients to mark the body areas affected. Other facultative instruments were included. For the identification of disease-controlled days, an electronic diary was developed and validated, which measured a continuous patient-reported data. For each instrument, a series of electronic documentation systems was developed. Finally, a series of goals of outcomes was achieved by monitoring relevant patient outcomes in an electronic switch- board, enabling continuous recording of the patient outcomes and controlling the achievement of outcomes. In the first testing, the electronic documentation and management system showed good technical properties and was well accepted by patients. The patient education tool markedly increased patient satisfaction and adherence. In conclusion, the electronic documentation system and the resulting digital toolboxes are feasible and beneficial technologies for the improved management of psoriasis in practice. Areas of use included routine patient record reports, patient regestries and clinical trials.

**Po46**

Comanagement with rheumatologists for patients with psoriatic arthritis receiving treatment with a biological agent or apremilast

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A retrospective patient chart review of over 1000 patients with psori- atic arthritis (PsA) who underwent a switch from one biolog- ical agent or apremilast to another biological within the most recent 3 months was conducted in March 2017 by an independent healthcare market research firm. The study aimed to understand current practice man- agement of patients with PsA and the specific clinical and non- clinical reasons for therapy changes. The audit was compliant with the Health Insurance Portability and Accountability Act and was conducted by the treating rheumatologist using an online, secure audit form. In addition to the patient variables collected, the project also included a survey of the participating rheumatologists about their practices and their approach to PsA patient management. Of interest, 72% of the rheuma- tologists expressed agreement with the statement ‘I wish der- matologists would refer PsA patients to me sooner than they typically do’. Upon analysing the patient records, it was revealed that most of the patients with PsA were originally referred to a dermatologist, but more than five in ten were referred by dermatologists. Among those referred by dermatologists, close to one-third had been previously treated with a biological agent or apremilast. Furthermore, among those patients previously treated, the vast majority were switched to a different agent within the first 3 months of see- ing a rheumatologist. When asked about ongoing comanage- ment for the patients who were referred by dermatologists, in two-thirds of the cases, the rheumatologist reported that they are the primary physician managing the biologic, and in only about 10% of the cases did they describe the between the dermatologist and rheumatologist as equal. When analysing the reasons for biological switching, unsurprisingly, a desire for improved efficacy in the arthritic disease components was the most common reason. However, in 14% of the cases, rheuma- tologists made the switch based on a desire for improvements in skin efficacy, underscoring rheumatologists’ willingness to manage both aspects of the disease independently of dermatol- ogy input. When queried about the preference for using a biological in PsA that also has an indication in psoriasis, the group was largely divided, with one-third reporting that they are less inclined to use agents that do not have a dual indica- tion, and another one-third claiming this is not important. In conclusion, the data suggest that once patients are referred from dermatologists to rheumatologists the management of biological therapy is likely to be more commonly directed by the rheumatologist.

**P047**

Investigation of the role of IKKε in the pathogenesis of psoriasis

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We demonstrate that IKKε is the key component in genetically modified mice.

IKKε deficiency was not found to reduce interferon γ (IFN-γ) in the pathogenesis of psoriasis. The IKKε-deficient and wild-type C57BL/6 mice were used in an murine model of psoriasis. The IKKε-deficient and wild-type C57BL/6 mice were used in an murine model of psoriasis.
The interleukin-17A/F heterodimer regulates psoriasis-associated genes through NFKBIZ.

In conclusion, we show that IL-36 cytokines are produced but need to be activated by psoriasin. IL-36 cytokines in keratinocytes. Functionally similar to b-defensins and CCL20, IL-36 cytokines induce the expression of genes encoding AMPs and IL-36 cytokines. In both approaches we could detect an increased expression of elastase. In both approaches we could detect an increased expression of elastase.

We were able to activate the released IL-36 cytokines in keratinocytes. Functionally similar to b-defensins and CCL20, IL-36 cytokines induce the expression of genes encoding AMPs and IL-36 cytokines. In both approaches we could detect an increased expression of elastase.

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P054
Abutides to guselkumab are not associated with reduction in clinical response or development of injection-site reactions in patients with moderate-to-severe plaque psoriasis
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The aim of this study was to assess the immunogenicity of guselkumab, an anti-interleukin-23 monoclonal antibody, and its association with pharmacokinetics (PK), efficacy and injection-site reactions (ISRs) in patients with moderate-to-severe plaque psoriasis. In two pivotal phase III studies (VOYAGE 1 and 2, with identical study designs through weeks 24), patients were randomized to receive subcutaneous administrations of guselkumab 100 mg (weeks 0 and 4 then every 8 weeks; or adalimumab 80 mg at week 0 and 40 mg at week 1 and every 4 weeks for 2 weeks) or placebo. Adverse drug reactions (ADAs) against guselkumab were detected using a validated, sensitive and drug-tolerant electrochemiluminescence immunoassay method. Serum ADA concentrations (systemic exposure), Investigator’s Global Assessment (IGA) and Psoriasis Area and Severity Index (PASI) responses and ISRs were assessed as PK, efficacy and safety endpoints, respectively. Of 1361 patients who received guselkumab and had post-treatment serum samples available for ADA, the overall incidence of ADA up to week 48 was 6.1% (n = 83), with predominantly low titres (≤ 1:160). Only seven of 83 patients had antibodies that were able to neutralize the bioactivity of guselkumab at ≥50%. No apparent impact of ADAs on systemic exposure was observed between ADA-positive and -negative patients (between-patient comparisons), before and after the development of ADA (within-patient comparisons), or by the time (sooner or later) when ADAs were detected. Of the 58 ADA-positive patients who were evaluable for the effect of ADA on systemic exposure, 21 had numerically lower, 25 had numerically higher and 12 had numerically similar serum guselkumab concentrations after the development of ADAs. The development of ADAs was not associated with a reduction in efficacy. In patients randomized to guselkumab with available data, 48 (49%) of 54 ADA-positive patients and 637 (83.3%) of 785 ADA-negative patients achieved IGA 0 or 1 at week 28. Only six (1.5%) of 388 guselkumab injections in ADA-positive patients and 41 (0.7%) of 6118 guselkumab injections in ADA-negative patients were associated with ISRs. In conclusion, following subcutaneous administrations of guselkumab in patients with moderate-to-severe plaque psoriasis, the incidence of ADAs was low. The development of ADAs to guselkumab was not associated with reductions in systemic exposure or efficacy, or development of ISRs.
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The aim of this study was to report the baseline demographics and clinical characteristics of Greek participants enrolled in PSOLAR. PSOLAR is a multicentre, prospective, longitudinal, observational study designed to follow patients with psoriasis ≥ 18 years of age, currently receiving or candidates to receive systemic therapies for psoriasis, for ≥ 2 years. The study gathers information from academic and community settings to generate real-world data regarding patients with psoriasis and their respective treatments. Such data include baseline demographics, medical and family history, economic and social status, adverse events, disease activity, quality of life and inter- vals and adverse events expected for a moderate-to-severe psoriasis patient. Of enrolment. Baseline characteristics for the Greek participants included: mean age 45.5 years (median 43.0), with 47.5% of patients aged ≥ 45 years and 63.5% male. At enrolment, 94.2% of patients presented with plaque-type psoriasis, with a mean Physician’s Global Assessment (PGA) score of 2 ± 1.4, 43.3% of patients presented with a PGA score ≥ 3. The mean duration of disease since diagnosis was 13.7 ± 9.8 years, and 11.6% of patients reported psoriatic arthritis diagnosed by a joint specialist. The mean body mass index was 29.3 ± 5.1 kg m⁻², with 74.6% being overweight or obese. Overall 66.1% and 56.7% of patients reported current use of alcohol and tobacco, respectively, and 18.2%, 6.6% and 12.4% of patients presented with hypertension, type 2 diabetes, and hyperlipidemia, respectively. The mean affected body surface area and PGA score at peak disease activity were 46.9 ± 24.4% and 3.4 ± 0.9, respectively, and 33.9% of patients were receiving systemic therapy at the time of peak disease activity (16.4% of patients received tocilizumab, 3.4% methotrexate, 3.4% adalimumab, 3.4% etanercept and 6.8% infliximab). At the time of enrolment, 82.6% of patients had received treatment with a biologic agent at some point, with 1.7% having received four, 9.1% three, 19.8% two and 52.1% one. Other treatments included topical therapy (96.7% topical corticosteroids), phototherapy (35.6%), retinoids (38%), ciclosporin (48%) and methotrexate (28%). The clinical, baseline and historical features for the Greek subset of PSOLAR patients were as expected for a moderate-to-severe psoriasis population. Although 74.6% of patients were overweight or obese, cardiovascular risk factors was reported only in few. In summary, patients were as expected for a moderate-to-severe psoriasis patient. Mean Physician’s Global Assessment (PGA) score of 0 (clear) or 1 (almost clear) at 16 weeks. Baseline PASI scores for patients on combination therapy were collected before the onset of apremilast treatment, not the onset of concurrent systemic therapy. Safety was measured as the proportion of patients achieving ≥ 75% reduction from baseline PASI Area and Severity Index score (PASI 75) or a Physician’s Global Assessment (PGA) score of 0 (clear) or 1 (almost clear) at 24 weeks. PASI results were included for safety analyses in the real-world setting. A multicentre retrospective chart review was conducted, which captured all patients treated with apremilast from November 2014 to July 2017. Patients were permitted to use concurrent therapies with apremilast. Outcome measurements included the proportion of patients reporting at least AEs and the incidence of AEs leading to withdrawal in apremilast as a monotherapy from the first 16 weeks of treatment. Real-world data were compared with RCT data using Pearson’s χ² test for categorical variables and t-test for continuous variables. A smaller proportion of real-world patients experienced at least one AE compared with RCT patients (real world 112 of 308, 36.7%; RCT 57 of 83, 68.9%; P < 0.005). A greater proportion of real-world patients discontinued AEs compared with AEs due to withdrawal. RCT patients (real world 59 of 208, 18.8%; RCT 44 of 81, 54.3%; P < 0.005). A total of 296 charts were screened, and 208 patients were included in the analysis. The observational study as combined therapy cohort (9% of 148, 60.1%) and monotherapy cohort (59 of 148, 39.9%) showed similar efficacy and safety outcomes. For efficacy, 44% of monotherapy patients (26 of 59) and 37% of combination therapy patients (33 of 89) achieved PASI 75 or PGA 0/1 (P = 0.40). For safety, 65% of monotherapy patients (17 of 59) and 62% of combination therapy patients (31 of 51) experienced at least one AE (P = 0.91). Commonly reported adverse events included headache (monotherapy 24%, combination therapy 12%), abdominal pain (monotherapy 15%, combination therapy 17%, P = 0.80), nausea (monotherapy 19%, combination therapy 1%, P = 0.21) and weight loss (monotherapy 7%, combination therapy 9%, P = 0.49). In conclusion, apremilast results in clinically significant reduction of chronic plaque psoriasis, with primarily mild-to-moderate AEs, as both a monotherapy and combination therapy in the real-world setting. Apremilast is generally a safe medicine, with a low incidence of AEs leading to withdrawal. In summary, apremilast is generally a safe medicine, with a low incidence of AEs leading to withdrawal. In summary, apremilast is generally a safe medicine, with a low incidence of AEs leading to withdrawal. In summary, apremilast is generally a safe medicine, with a low incidence of AEs leading to withdrawal. 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A successful treatment of recalcitrant hyperkeratotic palmoplantar psoriasis with itolizumab: a case series of three patients

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Palmoplantar psoriasis (PP) is a chronic inflammatory skin disease that is associated with distoffest quality of life. The prevalence of chronic plaque psoriasis in different populations is 1–3%, whereas PP accounts for 3–4% of all cases of psoriasis in most studies. We present a case series of three patients with PP who responded remarkably to itolizumab (Alzumab®), a novel humanized monoclonal antibody approved for moderate-to-severe chronic plaque psoriasis. It downregulates T-cell proliferation induced by activated leukocyte cell adhesion molecules by binding to the extracellular scavenger receptor cysteine-rich domain 1 of CD14

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Systemic therapy and the risk of nonmelanoma skin cancer among patients in the Psoriasis Longitudinal Assessment and Registry

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Psoriasis is associated with increased risk of nonmelanoma skin cancer (NMSC), including basal cell carcinoma (BCC) and squamous cell carcinoma (SCC). Our aim was to determine the effect of biological therapy on NMSC risk among patients in PHAROS, a prospective registry for patients eligible to receive systemic treatment. Data extracted from 23 August 2010 to 31 December 2017 were included. We defined a study population of 6782 PSORIAT patients without a history of NMSC at enrolment who were prevalent or incident users of biologics (etanercept, infliximab, adalimumab and ustekinumab). Cox proportional hazards models were used to estimate hazard ratios (HRs) adjusted by covariates relevant to NMSC risk. The treatment cohorts were generally comparable for most baseline characteristics. Seventy-two new diagnoses of NMSC were identified [HR 0.37, 95% confidence interval (CI) 0.30–0.47]. The HRs and confidence intervals for NMSC, SCC and BCC are displayed in Table 1a, and hazard ratios by NMSC type are displayed in Table 1b. Biological therapy did not alter the risk of NMSC. In the secondary analysis, TNF inhibitors did not alter the risk of incident NMSC or SCC compared with MTX, whereas UST showed significantly lower risk than MTX. The results for SCC are difficult to interpret due to wide confidence intervals. The results require further validation in postmarketing studies with larger numbers of exposed patients.

P063

Interleukin-10 regulates skin thickness and scaling in imiquimod-induced psoriasis-like skin inflammation in mice

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The aim of this study is to investigate the effect of interleukin-10 (IL-10) on skin thickness and scale formation in imiquimod-induced psoriasis-like skin inflammation in mice. Mice were treated with imiquimod for 4 weeks or received vehicle as control. Skin thickness and scale formation were determined by whole-body imaging and histology. IL-10 decreased skin thickness and scale formation in imiquimod-induced psoriasis-like skin inflammation. In conclusion, IL-10 has anti-psoriatic effects and could be used as a therapeutic target.
Pso64

The identification of interleukin (IL)-17A, IL-17RA and IL-17RC lymphoid and myeloid cells in blood of treatment-naive early patients and in synovial fluid of established patients with psoriatic arthritis X. Xu1, N. Davelaar1, A.-M. Otten-Mus,2, P. Aismadjiastra1, H. den Braanker1, J. Hazes1, R. Bisselend1,3,4, M. Vis1 and E. Lubberts1

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Interleukin (IL)-17A is a proinflammatory cytokine involved in the pathogenesis of psoriatic arthritis (PsA). Biomarkers targeting IL-17A have been approved in clinical treatment of PsA patients with PsA. However, the cellular background producing IL-17A is not clear and it is not clear which cells are responsible for IL-17A production in patients with PsA. Additionally, the expression of IL-17A receptor (IL-17A), IL-17RA and IL-17RC, on different cells types is not well defined. In this study, IL-17A, IL-17RA and IL-17RC-positive cells in blood of patients with a first diagnosis of PsA with arthritis, and in synovial fluid of patients with established PsA with active disease were examined. Fresh blood was taken from first diagnosed disease-modifying anti-rheumatic drug (DMARDs)-treated or PsA patients. Additionally, IL-17A-positive neutrophils and monocytes were included as a control group, along with an isotype control group. IL-17A-IL-17RA/RC signaling network among different cell types involved in the IL-17A-driven pathogenesis of PsA.

Pso66

Unopposed interleukin (IL)-36 activity promotes clonal CD4+ T-cell responses with IL-9 production in generalized psoriasin A. Arakawa1, S. Vollmer1, B. Bogen1, P. Thamm2, T. Ruzicka2 and J. Cino2

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Mutations in IL36N, CARD14 or AIP1 provide genetic evidence for autoinflammatory arthritis in generalized psoriasin (GPP). Although GPP has been considered as the most severe psoriasis variant, the pathogenic overlap with psoriasis vulgaris (PV) remains elusive. As PV is a CD4+ T-cell mediated autoimmune disease, we investigated the role of T cells in GPP using PV as a disease control. Here we demonstrate that unopposed interleukin-36 (IL-36) signaling may cooperate with certain human leucocyte antigen (HLA) class II alleles to induce antigen-driven T helper (Th)17 responses in GPP, which are presumably directed against autoantigens in the absence of exogenous triggers. We analysed eight patients with GPP to show that defects in IL36N function may result from IL36N mutations or decreased IL36N transcription. Molecular analysis of T-cell receptor (TCR) rearrangements revealed strong TCR-driven activation signatures in CD4+ T cells of patients with PV and identical T-cell clones in blood and skin lesions, suggesting common pathogenic pathways in PV and GPP. Notably, CD4+ T cells of patients with PV were characterized by strong autoproliferation, highly restricted clonal TCR repertoires, identical T-cell clones in blood and skin lesions, and identical differential expression of T-cell markers in both CD4+ and CD8+ T cell subsets. In contrast, CD4+ T cells of patients with GPP differentially expressed CD200 and CD204 and restricted clonal TCR repertoires, identical T-cell clones in blood and skin lesions, and identical differential expression of T-cell markers in both CD4+ and CD8+ T cell subsets. These preliminary data show that not only lymphoid cells but also myeloid cells can produce IL-17A, a key mediator in GPP.
S sustained remission in a patient with chronic plaque psoriasis treated with itolizumab: a 4-year follow-up experience
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A 53-year-old female patient with a history of psoriasis for 12 years was presented to us. On investigation it was found that she had earlier been treated with topicals and methotrexate. As her baseline Psoriasis Area and Severity Index (PASI) at presentation was 21.8 and she had previously been treated with the available treatment options she was suggested to start biologics. The aim for use of biologics was to provide the patient with quicker relief and long-term sustained disease-free remission. The biologic of choice here was itolizumab, a monoclonal anti-CD6 antibody that showed evidence of remission in the phase III trial of the drug (Dogra S, D RK, Brolundhoma L, et al. Long-term efficacy and safety of itolizumab in patients with moderate-to-severe chronic plaque psoriasis: a double-blind, randomised, placebo-controlled study. J Am Acad Dermatol 2015; 72: 331-9). Before initiating the therapy, laboratory parameters of the patient including serum creatinine, blood urea, liver function tests and thyroid function tests, along with X-ray chest and electrocardiogram, were analysed and normal. Tests for HIV, hepatitis B and hepatitis C and Mantoux tests were negative. After all the examinations were normal the patient was given initial dose of itolizumab 1.6 mg/kg (75 mg in 250 mL of normal saline over 2 h). In total 10 injections were given over a period of 6 months, followed by maintenance injections every once or twice every 3 months. The patient achieved PASI 90 (90% improvement from baseline) at the end of the 6th week (PASI score 1.9) and progressed to PASI 100 (PASI score 0.1) after the completion of 10 doses. In order to maintain remission the patient is being given maintenance dosing once every 3 months, and to date continues to maintain PASI 90 after 41 weeks of therapy (over 4 years). These results are in agreement with the phase III studies of itolizumab and have improved the clinical and quality-of-life parameters of the patient. Thus itolizumab serves as the drug of choice to maintain disease-free remission in moderate-to-severe chronic plaque psoriasis.

Poyo Treat to target in psoriasis: a Belgian attempt to define a tight control strategy for psoriasis management
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Treat to target (T2T) is defined as a therapeutic algorithm designed to achieve well-defined and specific therapeutic targets in the management of a disease. This concept is characterized by ‘tight control’, whereby a treatment algorithm is followed regarding evaluation, assessment and treatment decision. T2T strategies have gained a lot of interest since their first successes in rheumatoid arthritis and diabetes. It seems the time has come to introduce T2T in psoriasis, although many attempts cannot be readily translated to the Belgian situation. We aim to redefine a T2T strategy in light of the Belgian real-world setting, which is currently ongoing. Questioning will be performed through the Delphi technique, to build consensus using a series of questionnaires to collect data from a panel of experts. An expert board of Belgian key opinion leaders will participate in defining a tight control strategy in Belgium.

Retrospective review of psoriasis ustekinumab outcomes using real clinic data analysed using starting Psoriasis Area and Severity Index (PASI) 75 and 90 reported C. Lieweghe1 and P. Hampton2
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The electronic notes of 114 patients who received ustekinumab for chronic plaque psoriasis were reviewed. Baseline Psoriasis Area and Severity Index (PASI) were calculated using different methods (i) starting PASI (on day of first drug administration) vs. (ii) worst PASI in the preceding 1 year. Variations with weight and dose were also analysed. Patients starting ustekinumab between December 2008 and January 2016 were included, and retrospective data were collected.
including weight, dose, baseline PASI, worst 5-year PASI and PASI at 3, 6, 9 and 12 months. The starting PASI calculation method used influenced PASI 75/90 rates at 3 months using starting PASI 75/90 > 20 vs. ≤ 20 using worst 5-year PASI. At 3 and 6 months all groups achieved higher PASI 75/90 when the worse 5-year PASI was used vs. starting PASI 75/90 ≥ 3. We also noted that only 11% of patients receiving 45-mg doses achieved PASI 90 at 3 months vs. 24% receiving 90 mg. We noted a discrepancy in outcomes using different measures; 18% of patients reached PASI 90 ≥ 3 months, but 45% achieved VAS 3. Response differences were also seen between groups: 3% (80.1–100 kg, 90-mg dose) achieved higher rates of PASI 90 ≤ 3 at 1 and 6 months than the other groups (see Table 2) and had greater achievement of PASI 75/90 at 3 and 6 months. A dose-related difference in response was also seen with 90 mg, with patients achieving higher PASI 90 rates at 3 months, 24% vs. 11% for 45 mg, and this difference appeared to lessen over time with 12-month PASI 90 rates being similar: 22% vs. 26%. In conclusion, variation in the starting PASI calculation methodology can lead to differences in treatment outcome reporting, and using absolute PASI in addition to PASI 75/90 can lead to greater transparency of reported results. Although these data lack numbers for statistical significance the findings may warrant further investigation from larger datasets such as BADBIR.

Psoriasis Life Quality Questionnaire (DLQI) and Psoriatic Arthritis

In order to assess the impact of psoriasis on quality of life, the Psoriasis Life Quality Questionnaire (DLQI) was used. The DLQI is a 12-item questionnaire that assesses the impact of psoriasis on daily activities and treatment were only weakly associated with improvements in psoriasis severity. The results suggest that psoriasis is associated with moderately improved quality of life. In the initial phase of the treatment period a strong correlation between Psoriasis Area and Severity Index (PASI) and DLQI was found. However, long-term correlation between PASI and DLQI for patients treated with ustekinumab has not been investigated in a real-life setting. In this register-based study of patients with moderate-to-severe psoriasis, we investigated the correlation between PASI and DLQI after 4 and 12 months. All patients with psoriasis treated with ustekinumab in the Roskilde department from April 2009 to March 2012 were included. A correlation analysis between the change in PASI and DLQI and the individual questions in DLQI were performed using Spearman’s rank correlation coefficient. We observed significant differences in high correlations between PASI from baseline to month 4 and a subsequent slight reduction until month 12, with a similar pattern for DLQI. The correlation analysis showed significant association for moderate reduction in PASI and DLQI with correlation values of 0.57 (P < 0.001) for baseline to 4 months and 0.45 (P < 0.001) for baseline to 12 months. For the individual questions in DLQI the greatest association was observed for questions on ‘symptoms and feelings’. This was mainly attributed to improvement in the symptoms ‘itching’, ‘pain’ and ‘pruritus’. In conclusion, the use of DLQI in clinical trials, registries and trials is recommended for assessing improvements in psoriasis severity. The results suggest objective improvements in severity of psoriasis to be weakly to moderately associated with improvements in quality of life in patients with psoriasis treated with ustekinumab. This study was supported by an unrestricted grant from Janssen-Cilag.

Psoriasis Life Quality Questionnaire (DLQI) and Psoriatic Arthritis

To explore the safety and efficacy of biologics for moderate-to-severe psoriasis, we examined patients treated between 1 January 2007 and 31 March 2017. We used Kaplan-Meier survival curves and Cox regression to examine drug survival patterns. There were a total of 3495 treatment series (2161 patients) including 1331 with adalimumab, 579 with etanercept, 198 when started by originator/biosimilar, 546 with originator etanercept and 55 with biosimilar etanercept, 331 with infliximab (356 when started by originator/biosimilar, 366 with originator infliximab and 90 with biosimilar infliximab), 1055 with ustekinumab and 196 with secukinumab. Secukinumab had the highest number of responders with 100% improvement in Psoriasis Area and Severity Index (PASI 100) within the first 2 weeks, but also the lowest drug survival across all biologics. Ustekinumab had the highest drug survival overall (ustekinumab > adalimumab > infliximab > etanercept > secukinumab > adalimumab), with a similar pattern for PASI from baseline to month 4 and a subsequent slight reduction until month 12, with a similar pattern for DLQI. The correlation analysis showed a significant association for moderate reduction in PASI and DLQI with correlation values of 0.57 (P < 0.001) for baseline to 4 months and 0.45 (P < 0.001) for baseline to 12 months. For the individual questions in DLQI the greatest association was observed for questions on ‘symptoms and feelings’. This was mainly attributed to improvement in the symptoms ‘itching’, ‘pain’ and ‘pruritus’. In conclusion, the use of DLQI in clinical trials, registries and trials is recommended for assessing improvements in psoriasis severity. The results suggest objective improvements in severity of psoriasis to be weakly to moderately associated with improvements in quality of life in patients with psoriasis treated with ustekinumab. This study was supported by an unrestricted grant from Janssen-Cilag.

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Psoriasis is a chronic inflammatory disease primarily affecting the skin and joints. It is a multifactorial disease that affects about 1–3% of the general population. Psychological factors and impairment of life quality are important. The success of the treatment requires the active participation of the patient. Therapeutic education of the patient aims to help them acquire the skills necessary to manage their life with a chronic illness. The aim of our study was therefore to assess the impact of therapeutic education on patients with psoriasis. We carried out a prospective 3-month study involving around 30 patients followed in our unit for psoriasis. The same questionnaire was used before and after the therapeutic education in order to appreciate its contribution. The questionnaire included three main items with four to five questions each. In total 23 patients were included, 15 men and 8 women, with an average age of 37.8 ± 3.3 years. Half (51%) had a school level below high school and 75% earned €300 per month. Concerning questions related to the transmission of the disease and its symptoms, before education one-third of the patients had a score of correct answers >50% vs. four-fifths after education. In terms of associated diseases and comorbidities, only one-third had a score >50%, vs. 100% after education. Regarding the treatment available and their follow-up, only three in 10 patients had a score of correct answers >50% before the education, vs. eight in 10 after the education. There are diseases where therapeutic education has been validated and the management of patients, such as diabetes, in which it has improved metabolic control of the disease, improved quality of life and reduced complications. Indeed, a better understanding of their pathophysiology seems to help patients to take an active part in their care. In dermatology, therapeutic education has already proved to be effective, especially in patients with chronic diseases. It is therefore possible to benefit patients with psoriasis, especially that considering that it has a chronic pathophysiology with a strong psychological impact. Therapeutic education remains an indispensable tool in the management of patients with chronic disease. However, the limitations of our study lie in the short duration of study and our small sample. The establishment of a specialized and multidisciplinary consultation for better care should be considered.

**Psoriatisk komorbidity, psychotic medication prescribing and suicidality in patients with psoriasis: a population-based cohort study**

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Centre for Rheumatology and Inflammation, University of Manchester, Manchester Academic Health Science Centre, Manchester, UK; Centre for Mental Health and Safety, University of Manchester, Manchester Academic Health Science Centre, Manchester, UK; Department of Dermatology, Manchester University NHS Foundation Trust, Manchester Academic Health Science Centre, Manchester, UK

The psychological burden in people affected by psoriasis may lead to elevated risks of suicide and nonfatal self-harm. This study aimed to investigate psychiatric comorbidity, psychotic medication prescribing and suicidality in people with psoriasis. Patients with a first diagnosis of psoriasis between 1998 and 2014 were identified from the Clinical Practice Research Datalink linked to Hospital Episode Statistics and the Office for National Statistics mortality records in England. For each patient with psoriasis, up to 10 comparison patients were matched by age, sex, general practice and index date. A stratified Cox proportional hazard model was used to estimate the risk of suicide and nonfatal self-harm in people with psoriasis. The multivariable model was adjusted for socioeconomic status. Statistical models with interactions were tested; these included an interaction between postazac and age sex or socioeconomic status. The cohort included 56,961 patients with psoriasis and 876,919 comparison patients. At baseline, people with psoriasis had a higher prevalence of depression (18.4% vs. 18.0%), anxiety (17.8% vs. 15.9%) and bipolar disorder (0.8% vs. 0.5%) and were more frequently prescribed psychotropic medications (54.5% vs. 49.1%). The prevalence of schizophrenia, eating disorder and personality disorder were similarly distributed in the psoriasis and comparison cohorts (0.9% vs. 0.9%, 0.8% vs. 0.8% and 0.5% vs. 0.5%, respectively). During a median follow-up of 4.6 years (interquartile range 6.1), 15 and 679 suicide events occurred in the psoriasis and comparison groups, respectively, and, after exclusion of patients with a history of self-harm, 592 and 6914 first self-harm events occurred in the psoriasis group and comparison groups, respectively. When taking into account socioeconomic status, the risk of suicide was significantly lower among patients with psoriasis [hazard ratio (HR) 0.95; 95% confidence interval (CI) 0.91–0.99] and there was no significant difference in risk for nonfatal self-harm (HR 1.06; 95% CI 0.97–1.17). After testing for interactions, the risk of suicide significantly differed according to age (P = 0.01). The HR was 0.88 (95% CI 0.81–0.96) for patients diagnosed with psoriasis at ≥40 years, but there was no significant difference in risk for those diagnosed at <40 years. People with psoriasis have a greater prevalence of depression and anxiety and are more likely to be prescribed psychotropic medication than the general population. Nevertheless, people with psoriasis are not at an elevated risk of either suicide or nonfatal self-harm.
The survival of patients with psoriasis is modified by the inflammatory component of this disease, especially when it is associated with comorbidities. This which forces us to improve prevention and treatment strategies in this population. Few studies have been carried out using a validated comorbidity index that allows the practitioner to infer predic- tive mortality information. The Charlson Comorbidity Index is a score that includes 17 systemic diseases, used to predict the 5-year mortality of patients based on their different comorbidities. The University Hospital La Samaritana is a reference centre for the treatment of psoriasis in Colombia, which has socioeconomic conditions that differ from those of popula- tions in which similar studies have been carried out. We do not have studies in Colombia that allow us to know which are the most frequent comorbidities in patients with psoriasis, or the relationship of these comorbidities with the severity of psoriasis. Our conclusions were as follows. The most prevalent comorbidities in adult patients with psoriasis treated at the University Hospital La Samaritana are dyslipidaemia (32%), hypertension (26.1%) and obesity (23.8%). We did not find a statistically significant association between the presence of comorbidities and the clinical severity of psoriasis in this group of patients. Cardiovascular and metabolic comorbidities are present in this group of patients regardless of clinical severity. Charlson Comorbidity Index does not include the most prevalent comorbidities in patients with psoriasis (dyslipidaemia, hypertension, obesity), so we consider it a good indicator of disease burden in this group of patients. Studies that evaluate the association between clinical severity of psoriasis and mortality are required. Treatment strategies should therefore target patients diagnosed with psoriasis treated at the University Hospital La Samaritana for the main comorbidities, regardless of clinical severity.

The male-to-female ratio was 2.04 : 1. Two peaks of distribution in age of onset were seen, with the first peak in the 30s for men and the 20s in women, and the second peak in the 50s for both genders. Family history of psoriasis was present in men, 6.4% in total; however, female patients with psoriasis with onset < 10 years had a much higher rate (15.4%) than women with onset > 10 years (5.6%, P = 0.003). When these data were compared with those from South Korea and China, the onset age in both countries had a steep curve, which was located at a younger age than in Japan. Japanese patients showed higher male preponderance than those in South Korea and China. South Korean and Chinese patients had much more frequent familial cases (26% and 31%, respectively) than Japanese. Age of onset of psoriasis in Japanese patients were statistically higher in all men and women than in other Asian countries, with fewer cases of familial psoriasis. Genetic background may affect these differences.

The study analysing the treatment characteristics of the period of biological treatment in a cohort of patients with severe psoriasis. Moreover, we aimed to assess shifts of the 'journey' in patients with psoriasis treated at the University Hospital La Samaritana for the main comorbidities, regardless of clinical severity. Treatment strategies should therefore target patients diagnosed with psoriasis treated at the University Hospital La Samaritana for the main comorbidities, regardless of clinical severity.

To investigate the role of mast cells (MCs) in the pathogenesis of psoriasis, we performed immunocytochem- istry and metachromatic staining to evaluate MC distri- bution and function in biopsies of psoriasis lesional (PP), psoriasis nonlesional (PN) and healthy (SN) skin. Addition- ally, we performed RNA sequencing analysis using cultured MCs and those isolated from PP and PN skin. In PP skin there was a twofold increase in MC density and a threefold decrease in the incidence of degranulated MCs compared with PN and SN skin. CD1 T cells in PP skin showed a 10-fold increased den- sity, while macrophage density was increased fivefold. Finally, nerve fibre and vascular density in PP skin were unchanged and slightly raised, respectively, compared with PN and SN. The relationship of MCs with CD8 T lymphocytes and macrophages showed strong spatial association in all skin types. Moreover, in PP skin, MCs and nerve fibres showed increased spatial association while MCs lost their strong spatial association with the vasculature. In PP skin there was a twofold increase in intercellular (IL-17)-T cells and a parallel increase in IL-17 MCs. We have isolated MCs from PP and PN using flow cytometry and performed RNA sequenc- ing analysis to understand the gene expression of MCs in psoria- sis. Our preliminary results show a dramatically increased expression of genes involved in immune cell chemotaxis in PP MCs compared with those from PN, for example CC2L1 (8.8), CC2L3 (5.3), CC10 (5.1), CR (8.8), CCL1 (12), CCL4 (3) and CCL7 (5.1). The numbers in brackets are fold changes. This increased expression of chemotaxis genes was also particularly pronounced in MCs from PP who...
Psoriasis demonstrates high sustained efficacy and a favourable safety profile through 5 years of treatment in moderate-to-severe psoriasis.

E. Peeva1

23.5 ≤ PASI n until year 1 (53.3%, 71.3% and 78.7%). Two-thirds of patients reported no impact of skin disease on their lives over 5 years (60.5%, 73.5%, and 72.7%, respectively) compared with year 5 (63.5% at year 5). The safety profile of secukinumab remained favourable, with no cumulative or unexpected safety concerns identified. The most common adverse events included upper respiratory tract infection and back pain, consistent with those reported in the core study and previous phase III studies. The most frequent reasons for discontinuation in the extension study were patient or guardian decision (13.7%), adverse event (10.4%), and loss of efficacy (4.2%). Secukinumab 100 mg treatment sustained high levels of skin clearance, and improved quality of life, through 5 years in patients with moderate-to-severe psoriasis. The favourable safety profile was further established during each 6 month programme, were maintained for 5 years. This investigation was sponsored by Novartis Pharma AG, Basel, Switzerland.

Infliximab is associated with an increased risk of serious infection in patients with psoriasis: results from the British Association of Dermatologists Biologic Interventions Register (BADBIR).


Dermatology Centre, Sailfed Royal NHS Foundation Trust, The University of Manchester, Manchester Health Science Centre, NIHR Manchester Biomedical Research Centre, Manchester, UK; 7 John’s Institute of Dermatology, Guy’s and St Thomas’ NHS Foundation Trust, London, UK; 8 Centre for Pharmacoeconomics and Drug Safety, School of Health Sciences, The University of Manchester, Manchester, UK; 9 Addenbrooke’s Hospital, University of Cambridge, Cambridge, MA, USA; 10 Pfizer Clinical Research, Cambridge, MA, USA; 11 Pfizer Global Product Development, Cambridge, MA, U.S.A.

Infliximab is associated with a significantly increased risk of serious infection overall (adjusted hazard ratios 1.95, 95% CI 1.01–3.75). These data show that infliximab use for patients is associated with an increased risk of serious infections compared with nonbiologic systemic therapy. This risk should be taken into account when considering infliximab for the management of patients with severe psoriasis.

Efficacy and tolerance assessment of an antipruritic spray in patients with psoriasis

P. Rocha1,2, J. Buts1,3, A. Piché1,4, C. Eydiex1, J. riviere1 and M. Sayag5

1 Espe (Laboratoire Biodis), Lyon, France and L’Oréal Research, Oxford, France

Psoriasis is a chronic and inflammatory skin disease where patients usually experience pruritus. This symptom can have a huge impact on their quality of life (e.g. sleep problems, discomfort in daily activities). In order to sooth pruritus, an antipruritic spray has been developed. It combines the skin Relief® technology and enoxolone, which have a specific action on pruritus. To demonstrate its antipruritic efficacy and the tolerance in patients with psoriasis, an observational, prospective, multicentre study was carried in Poland. Thirty patients with mild-to-moderate psoriasis were included by three dermatologists. During 21 days, the spray was applied as often as necessary on the area affected by pruritus. Efficacy was evaluated by the SD-pruritus scale and a clinical assessment of the skin. Impact on the quality of life (Skindex) and tolerance were assessed as secondary end points. After 21 days of use, a significant decrease of the SD-pruritus scale score (–3.16) and itching sensations (–4.87) were observed. The spray reduces pruritus in 20.2 ± 0.3 ± 0.4 cm of average and the effect lasted ≥ 2 h in 80% of patients. The cutaneous state was significantly improved (dryness –4.41, roughness –4.5, desquamation –4.61, suppleness +2.51 and lexion extent –2.9%). The product demonstrated a positive impact on quality of life by a significant decrease of Skindex score (–3.7). The spray was well tolerated by 100% of the patients. In conclusion, this study demonstrated that the spray quickly calms the annoying associated with psoriasis with sustainable relief. The spray not only improved symptoms, but was shown to enhance self-image and the social life of patients.
The interleukin (IL)-1 family member cytokine IL-36 from macrophages (IL-23, tumour necrosis factor) is recognized as a crucial mediator in the immunopathology of psoriasis, and could not be identified in blood. Another dendritic cell marker, the IL-36 receptor, was also downregulated in skin, as was the interleukin (IL)-1 receptor. The interleukin (IL)-1 family member cytokine IL-36 was downregulated in skin, as was the interleukin (IL)-1 receptor.

The interleukin (IL)-1 family member cytokine IL-36 is recognized as a crucial mediator in the immunopathology of psoriasis, and could not be identified in blood. Another dendritic cell marker, the IL-36 receptor, was also downregulated in skin, as was the interleukin (IL)-1 receptor.

Dermatology, CHU, Paul Sabatier University, Toulouse, France; 2Department C. Paul, 1 P. van de Kerkhof, 2 Y. Dutronc, 3 psoriasis in patients with moderate-to-severe plaque psoriasis, starting with adalimumab followed by ustekinumab was more expensive, with 10-year costs of €371 347 vs €354 317. A sequence starting with ustekinumab followed by adalimumab would be the least expensive, with 10-year costs from etanercept-ustekinumab-adalimumab) over a 10-year time horizon. The model was populated with data from the Dutch Bio- cost-effectiveness of treatment in terms of both costs and health effects. Comparative evidence on (cost)-effectiveness of different biologics is limited. Given that biologics are associated with unacceptable side effects, appropriate use of these drugs is necessary. We aimed to evaluate the cost-effectiveness of different biological treatment sequences for psoriasis based on real-world evidence. A sequence model was developed to evaluate the costs and health effects of different consecutive lines of biological treatments based on the three most common prescribed biologics: adalimumab, etanercept and ustekinumab (for example adalimumab-etezaratucet-ustekinumab vs. etanercept-ustekinumab-adalimumab) over a 10-year time horizon. The model estimated 10-year treatment costs and the total quality-adjusted life years (QALYs) for each possible treatment sequence. Uncertainty was addressed by both probabilitational and scenario analyses. We esti- mated that sequences starting with etanercept would be the most expensive, with 10-year costs from €47 499 to €48 619. It was also the least expensive with a mean cumulative health effect of 7.39 QALYs. A sequence starting with ustekinumab followed by adalimumab would be the least expensive, with 10-year costs of €41 782, while the cumula- tive health effect was 7.50 QALYs. A sequence starting with adalimumab followed by ustekinumab was marginally more effective with 8.03 QALYs, but was also slightly more expensive, with 10-year costs of €43 661. When inter- preting these results, it should be taken into account that the credible intervals were partly overlapping. We conclude from these findings that the order in which biologics are used influ- ences the cost-effectiveness of treatment in terms of both costs and health effects. Initiation of a biological treatment sequence for psoriasis may be best done with adalimumab or ustekinumab, starting with adalimumab from a health-economic perspective. As the sequence order of biologics influences both costs and health effects of psoriasis treatment, adopting a long-term perspective at the start of treatment is important.

52-week results from IXORA-S: a randomized, double-blind, placebo-controlled trial (PHOENIX 2). Lanc 80/0.016; 75(95) 120 (88) 0.001 0.003 0.81 – 0.004 0.001 0.014 – 0.24 0.014 0.25 0.52 – – 0.19 0.03 0.051 0.003 0.003 0.63 0.63 0.051 0.003

Psoriasis Gene to Clinic
Psoriasis is a chronic inflammatory skin disease with both local and systemic components. Genome-wide approaches have identified more than 60 psoriasis-susceptibility loci, but genes are estimated to explain only one-third of the heritability in psoriasis, suggesting additional, yet unidentified, sources of heritability. Epigenetic modifications have been linked to psoriasis, suggesting additional, yet unidentified, sources of heritability. Our exploratory study represents a start-}

**Ps08** Genome-wide DNA methylation profiling identifies differential methylation in uninvolved psoriatic epidermis

D. Verma, A.-K. Ekbom, C.Ø. Eding and C. Enerbäck

Aim: We have performed an exhaustive genome-wide DNA methylation profiling using reduced representation bisulfite sequencing, which interrogates the methylation status of around 1,200,000 strongly differentially methylated sites (DMSs) were identified, and a striking overrepresentation of the Wnt and cadherin pathways among the DMSs was found. In particular, we observed a strong differential methylation in several psoriasis candidate genes. A substantial number of DMSs present in uninvolved vs healthy epidermis suggest the presence of a psoriatic epigenetic signature. Our exploratory study represents a starting point for identifying biomarkers for psoriasis-prone skin before disease onset.

**Ps09** Gene expression changes induced by individual interleukin (IL)-17 family cytokines signalling through IL-17RA

D. A. Ewald, P. Lovato, T. Skak-Nielsen and L. Skov

We performed full-thickness punch biopsies of healthy skin were cultured in keratinocyte cultures was induced by IL-17A, IL-17F and IL-17C in synergy with tumour necrosis factor-α, a high, interleukin and low level, respectively. The induction of DEFB4A by IL-17A, IL-17F and IL-17C was downregulated by IL-17RA antagonism. The above findings indicate that IL-17A, IL-17F and IL-17C, and to a lesser extent IL-17C, can all induce gene expression changes similar to those observed in psoriatic skin lesions. This suggests that combined inhibition of IL-17 family cytokines, for example by targeting of the IL-17RA receptor, could be a favourable mechanism for normalization of the psoriasis-associated gene expression signature.

**Ps010** Validation of self-reported psoriasis in a Danish birth cohort study

C. Blievad, T. E.T. Nielsen, C. Zachariae, L. Cordingley and on behalf of the PSORT study group

The interleukin (IL)-23/IL-17 immune axis is of key importance for driving skin inflammation in psoriasis. However, the importance of IL-17 family cytokines in psoriasis other than IL-17A has not been fully elucidated. In this study, we investigated the global transcriptional profile induced by IL-17 family cytokines signalling through IL-17RA. For this purpose, full-thickness punch biopsies of healthy skin were cultured in the presence of individual IL-17 family cytokines for 24 h. Gene expression profiling was carried out on whole-transcript arrays (Affymetrix Human Gene 2.1 ST). We contrasted those profiles with those of untreated controls, and compared the resulting differential expression profiles with those of psoriatic lesional vs. nonlesional skin. Ranked-list comparison and comparative gene set enrichment analysis indicated that the IL-17A-, IL-17A/F- and IL-17F-induced expression profiles overlapped to a lower degree with the psoriasis signature, whereas the IL-17E-induced expression profile showed a comparable overlap with the psoriasis signature. The global expression findings were further confirmed by quantitative reverse-transcriptase polymerase chain reaction on selected genes. The expression of SELP was strongly upregulated in children (94.8%) with a positive DNRC psoriasis status were examined, and of these only 28 (48%) could be confirmed as having psoriasis with onset before the 11-year questionnaire. In regards to the mothers, we could confirm the DNRC psoriasis status in 97 out of 120 (80.8%) from the ‘mother with psoriasis’ group. Our study shows that the validity of self-reported psoriasis in an adult female population is high in comparison with the validity of the psoriasis status reported in their 11-year-old children. The main reason for this is probably the use of physician-diagnosed vs. self-diagnosed psoriasis. Furthermore, psoriasis is difficult to diagnose in children due to mild symptoms.

**Ps011** Intentional and unintentional medication nonadherence in psoriasis: the role of patients’ medication beliefs and habit strength

R. Thrane, C.E.M. Griffiths, R. Emsley, D. Ashcroft, I. Kristensen and on behalf of the PSORT study group and BADBIR

Medication nonadherence is a missed opportunity for therapeutic benefit. Patients’ beliefs about their medication are key drivers of nonadherence, however, there is a lack of high-quality data on nonadherence to systemic therapies used for psoriasis outside of clinical trials. As part of the Psoriasis Stratification to Optimise Relevant Therapy (PSORT) consortium, we have prospectively evaluated levels of self-reported nonadherence to conventional and biological systemic therapies and evaluated psychological and biological factors associated with nonadherence using multivariable analysis. Cross-sectional data from 811 patients with moderate-to-severe psoriasis using a conventional systemic (35.3%) or biological therapy (64.7%) were collected from 35 dermatology centres across England. All patients were enrolled in the British Association of Dermatologists Biologic Interventions Register (BADBIR). A questionnaire assessed patients’ illness and medication beliefs (Revised Illness Perception Questionnaire and Beliefs about Medicines Questionnaire), psychological distress (Hospital Anxiety and Depression Scale), the strength of the patient’s routine or habit for using their medication (Self-Reported Habit Index) and medication adherence (Medication Adherence Report Scale, MARS), with a score ≤ 38 out of 40 on the MARS indicating nonadherence. Patients’ biomedical data were obtained from the registry. A significant proportion of patients using conventional systemic (methotrexate, ciclosporin, acitretin, fumaric acid esters) or biological therapies (stanozolid, adalimumab) were classified as nonadherent (MARS < 22, odds ratio 4.47). Patients who reported nonadherence, such as deliberately altering the dose, timing or frequency of their therapy, and those who reported unintentionally nonadherence, such as forgetting to take their therapy, were significantly more likely to be classified as nonadherence (odds ratio 2.32, 95% confidence interval 1.6–4.7). Patients who reported nonadherence, such as deliberately altering the dose, timing or frequency of their therapy, and those who reported unintentionally nonadherence, such as forgetting to take their therapy, were significantly more likely to be classified as nonadherence (odds ratio 9.02, 95% confidence interval 0.89–9.09). Medication nonadherence needs to be assessed when determining factors influencing treatment response. Medication beliefs and habit strength are important modifiable targets for strategies to improve adherence and clinical outcomes in the management of psoriasis. C.E.M.G. is a National Institute for Health Research Senior Investigator. PSORT is funded by the Medical Research Council, grant NR/1101180/1.

**Ps012** Prognostic effect of psoriasis and psoriatic arthritis in patients with suspected coronary artery disease assessed by cardiaccomputed tomography: a multicentre cohort study

K.J. Hjortekilde, S. Winther, M. Battcher and L. Iversen

Department of Dermatology, Aarhus University Hospital, Aarhus, Denmark and Department of Cardiology, Hospital Unit West, Høng, Denmark. Epidemiological population-based studies assessing cardiovascular disease (CVD) in patients with psoriasis have been based on registries containing diagnoses coded by clinicians. However, detailed clinical information is still needed about the correlation between disease activity in both psoriasis and psoriatic arthritis (PsA) and the risk of CVD. This study was based on a data from a subregistry to the Western Denmark Heart Registry (WDHR), the Western Denmark Cardiac Computed Tomography Registry. This clinical registry contains information on approximately 60 000 cardiac computed tomography (CT) scans from 2008 to 2015 and has shown both high completeness and high internal validity. Furthermore, WDHR contains data on clinical information seldom available in registries. All nine sites collaborating in WDHR register patients consecutively in a catchment area of 3.3 million inhabitants (55% of the Danish population). The purpose of this study was to acquire detailed clinical information on the association between both psoriasis and PsA and coronary artery disease, coronary interventions and major adverse cardiac events in a large population-based cohort. This study cohort included...
### Psoriasis: Safety of guselkumab in plaque psoriasis for 2 years: a pooled analysis from VOYAGE 1 and VOYAGE 2

K. Reins,1 K. Papp,2 A.W. Armstrong,3 Y. Wasfi,4 S. Jin,5 K. Yen,6 Y.K. Shen,7 B. Randazzo,8 M. Song9 and A.B. Kimball1

1Department of Dermatology and Skine Research Institute, Columbia University, New York, NY, USA; 2Clinical Research, Waterloo, ON, Canada; 3Probity Medical Research, Winnipeg, MB, Canada; 4Research and Development, Janssen Research & Development, LLC, Spring House, PA, USA; 5Department of Dermatology, University of Western Ontario, London, ON, Canada; 6University of Maryland, Baltimore, MD, USA; 7Biologics Research and Development, Janssen Research & Development, LLC, Spring House, PA, USA; 8Dermatological Sciences, University of British Columbia, Vancouver, BC, Canada; 9Janssen Research & Development, LLC, Spring House, PA, USA.

The study objective was to assess efficacy responses based on absolute Psoriasis Area and Severity Index (PASI) improvement through 2 years of guselkumab treatment in the VOYAGE 1 trial. VOYAGE1 is a phase III, randomized, double-blinded, placebo–active comparator-controlled trial. Eligible patients (age ≥ 18 years) had moderate-to-severe plaque psoriasis for ≥ 6 months, an Investigator’s Global Assessment score ≥ 3, PASI score ≥ 12, ≥ 10% body surface area involvement, and were candidates for systemic or phototherapy. In total 837

#### Table: Week 52 PASI response

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#### Table: Week 240 PASI response

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**Values are reported as event rates per 100 patient-years. All adverse event, MAEC, major adverse cardiac event, NMSC, non-melanoma skin cancer.**

*Abbreviations*. ABP: absolute Psoriasis Area and Severity Index (PASI) improvement through 2 years of continuous treatment; CBP: continuous Q2W group; IC: interquartile range; MAEC: major adverse cardiac event; NMSC: non-melanoma skin cancer. **With 95% confidence interval.**
Psoriasis is an interleukin (IL)-23-driven T-cell-mediated disease requiring prolonged treatment at high cost. The goal of our research was to find out the cost of topical treatment of the patients with mild-to-moderate psoriasis and its impact on their monthly income. For this purpose 101 patients with psoriasis were interviewed about 40.2% female and 55.7% male, with 65.6% (± 6.6%) from rural populations. Overall 74% of the patients were age 25-76 years. The diagnosis in 97.9% of patients was mild-to-moderate chronic plaque psoriasis with an affected body surface area < 10%. These patients used only self-treatment with topical medicines and had poor contact with their dermatologists. The questionnaire included the type and amount of topical medications used by the patients with psoriasis, the frequency of showering, their skin care products, and their monthly income. The results showed that 99% (102) of the patients used only the strongest strength of topical steroid – cloristerol 0.05% (available over the counter in Georgia) – not only for the period of exacerbation but also during the remission to prolong it, using four to five tubes (each 25g) per month. At the same time as the topical corticosteroid treatment, only 15 patients (14.6%) used other topical medicines (calcipotriol 50 mg + betamethasone 50 mg ointment, coal tar solution 5%) because of their high costs. The majority of the patients used only topical medicines (90.7%). The patients showered every day and 29.4% showed two to three times per week. The average income of the interviewed patients was the highest among pensioners (36.7%); and 3.8% among the other groups. The low income of the patients, the availability of over-the-counter topical corticosteroids in Georgia, their low costs compared with other topical medicines and their effectiveness determine the priority of their usage. According to the National Statistics Office of Georgia, 21.3% of the population was under the absolute poverty line in 2016, therefore further study of economic impact of the treatment of psoriasis in Georgia would provide data on the disease burden on society.

PsO8 Looking beyond 52 weeks: long-term drug survival and safety of secukinumab in world psoriasis patients J.R. Georgakopoulos1, A. Ighani2, M. Phung3 and J. Yeung4,5

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The discovery of type 17 helper T (Th17) cells and their role in the pathogenesis of psoriasis raise the possibility of Th17-directed therapies in psoriasis. The clinical success of guselkumab (GUS) and ixekizumab (IXE) in psoriasis has renewed interest in biological switching, particularly switching from anti-tumor necrosis factor (TNF) agents to anti-interleukin (IL)-17A agents.

From early findings identifying interleukin (IL)-17A as a plausible target in the immunopathogenic mechanisms of psoriasis to phase III randomised controlled trials (RCTs) demonstrating secukinumab to be highly efficacious and safe, much excitement has surrounded the approval of the first IL-17A antagonist. However, at this time, no study has looked at long-term outcomes for real-world patients with psoriasis, who are believed to be more challenging to treat than those enrolled in RCTs. We aimed to investigate the efficacy and safety of secukinumab at week 52 or the time of treatment discontinuation for individuals who were Psoriasis Area and Severity Index (PASI) 75 responders at week 12. These findings will provide much-needed insight into whether real-world patients with psoriasis achieve similar long-term outcomes to individuals enrolled in RCTs. A multicentre retrospective chart review was conducted using data from medical records of adult patients treated with secukinumab 300 mg for plaque psoriasis. Efficacy (PASI 75 or Physician’s Global Assessment (PGA) of 0 or 1) and safety (reported adverse events) were assessed following 12- and 52-week treatment periods or at the time of treatment discontinuation. Forty-one patients had moderate-to-severe plaque psoriasis, achieved PASI 75 or PGA 0/1 at week 12 and were followed up week 52 or the time of secukinumab discontinuation. Overall, 32 (78%) patients continued secukinumab treatment for all 52 weeks; 4 (10%) had moderate-to-severe plaque psoriasis, achieved PASI 75 or PGA 0/1 at week 12 and were followed up week 52 or the time of secukinumab discontinuation; and 6 (15%) were discontinued in the interim. Twenty-eight (68%) maintained PASI 75 or PGA 0/1 at week 52. Of the 13 (32%) patients who did not achieve efficacy outcomes treated with secukinumab 300 mg for plaque psoriasis (<PASI 75) and 11 (27%) stopped treatment prior to week 52 due to lack of efficacy (n = 10, 24%) or intolerance (n = 1, 2%). The mean treatment duration for individuals who discontinued treatment between weeks 12 and 52 was 40.0 weeks (range 26.1–51.0). Additionally, adverse events after week 12 were documented in 17% (n = 7) of patients. Commonly reported adverse events included dermatitis of the skin (n = 6, 5%) and respiratory tract infections (n = 2, 5%). Our results suggest that fewer patients with psoriasis in real-world clinical practice achieved efficacy outcomes at week 53 than those enrolled in RCTs. Furthermore, discontinuation of treatment appears to recur around week 40, similarly to time of treatment discontinuation seen with other biological therapies. Overall, these findings will greatly improve dermatologists’ ability to monitor secukinumab efficacy and safety beyond week 12.

PsO9 Efficacy and safety of ixekizumab in secukinumab nonresponders: therapeutic options for nonanti-interleukin-17A naïve patients J.R. Georgakopoulos,1 M. Phung, A. Ighani2 and J. Yeung3

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PsO6 Guselkumab treatment results in more effective and durable inhibition of T helper (Th)17 and Th2 cells and downstream effectors compared with adalimumab K. Tsagareishvili and N. Chijavadze

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Psoriasis is a chronic disease requiring prolonged treatment at high cost. The goal of our research was to find out the cost of topical treatment of the patients with mild-to-moderate psoriasis and its impact on their monthly income. For this purpose 101 patients with psoriasis were interviewed and 47 (45.2%) female and 55 (54.8%) male, with 65 (63.1%) from urban and 38 (38.8%) from rural populations. Overall 74% of the patients were age 25-76 years. The diagnosis in 97.9% of patients was mild-to-moderate chronic plaque psoriasis with an affected body surface area < 10%. These patients used only self-treatment with topical medicines and had poor contact with their dermatologists. The questionnaire included the type and amount of topical medications used by the patients with psoriasis, the frequency of showering, their skin care products and their monthly income. The results showed that 99% (102) of the patients used only the strongest strength of topical steroid – cloristerol 0.05% (available over the counter in Georgia) – not only for the period of exacerbation but also during the remission to prolong it, using four to five tubes (each 25g) per month. At the same time as the topical corticosteroid treatment, only 15 patients (14.6%) used other topical medicines (calcipotriol 50 mg + betamethasone 50 mg ointment, coal tar solution 5%) because of their high costs. The majority of the patients used only topical medicines (90.7%). The patients showered every day and 29.4% showed two to three times per week. The average income of the interviewed patients was the highest among pensioners (36.7%); and 3.8% among the other groups. The low income of the patients, the availability of over-the-counter topical corticosteroids in Georgia, their low costs compared with other topical medicines and their effectiveness determine the priority of their usage. According to the National Statistics Office of Georgia, 21.3% of the population was under the absolute poverty line in 2016, therefore further study of economic impact of the treatment of psoriasis in Georgia would provide data on the disease burden on society.
Psoriasis
Long-term, real-world efficacy of infliximab for psoriasis
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Infliximab has been licensed for the management of severe psoriasis. Infliximab data on its long-term, 6-year, efficacy. The aim of this study is to report long-term, real-world clinical data of patients with psoriasis on infliximab. A single-centre retrospective observational study was conducted. Patients with psoriasis maintained on infliximab for 6 years or longer were included. Clinical data, including Psoriasis Area and Severity Index (PASI) and Dermatolog Life Quality Index (DLQI), were collected prior to start- ing infliximab and at each subsequent outpatient visit. A total of 11 patients with psoriasis (seven male and four female) with an average age of 54.5 years (range 43–84) were maintain- ed on infliximab at 5 mg kg⁻¹ every 6–8 weeks (average 7.6) for ≥ 6 years. The average age of onset of psoriasis was 24.2 years (range 11–63) and none had psoriatic arthritis. The most common comorbidities, other than psoriatic arthritis, were hypertension, cardiovascular disease and ankylosing spondylitis. The median time between 2005 and 2011 with a mean treatment duration of 9.3 years (range 6–12). The mean baseline PASI and DLQI scores were 24.2 and 18, respectively. At years 1, 4, 8 and 12 the respective mean PASI scores were 13, 12, 8 and 6 and the mean DLQI scores were 3, 4, and 4, respectively. There were no serious side-effects reported, with recurrent respiratory and skin infec- tions being the most common. Most patients flared up after postponing treatment due to surgery, infections or time abroad, and three patients were on concomitant low-dose methotrexate (average dose 10 mg per week). To our knowl- edge this is the longest reported study on patients with psoria- sis maintaining efficacy and safety on infliximab for up to 12 years. Further studies need to elucidate why these patients continue to have control of their psoriasis on long-term infliximab.

Ps11
Fine mapping and subphenotyping implicates ADRB2 gene variants in psoriasis in a Chinese population
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Previous genome-wide association studies (GWAS) have iden- tified more than 40 independent genome-wide significant posi- tions susceptibility loci. We identified a genomic region on 1q21.3 harboring variants associated with psoriasis and the DLQI in our own GWAS and replication between the IL12B and PTG1 genes. However, there are many potential causal variants in this genomic region. To examine further the influence of vari- ants on psoriasis we used the recently published reference haplotypes to impute additional variants in the region in our study samples to increase the marker density in this region to 1121 genetic variants. We then used lasso-based regression analysis to assess the independent contributions of these variants to psoriasis and found evidence of association for 62 of the 2171 single-nucleotide polymorphisms (SNPs) in this region (seven SNPs around IL12B, 12 SNPs around PTG1 and 43 SNPs between the two genes). To evaluate these 62 SNPs further, we tested them for association with different clinical psoriasis subtypes, psoriasis severity and psoriasis age of onset. These analyses revealed slight differences between the SNPs exhibiting associations based on the whole case-con- trol analysis and the subphenotype, case-only analyses. The most significant locus with the largest number of SNPs was located in the ADRB2 gene, which is between the IL12B and PTG1 genes in the 5q33.3 region. Variants in ADRB2 were most strongly associated with the plaque subgroup, and showed a stronger association with the moderate-to-severe skin disease group and an earlier age at onset of psoriasis. Using genotyping data, we found the 62 calculated princi- dently associated loci in the ADRB2 gene in total explained 39.5% of the phenotype variance of psoriasis under the most common minor allele hypothesis. These results indicate the contribution of ADRB2 to psoriasis in the Chinese popula- tion. We found no evidence to support the notion that vari- ants in the IL12B and PTG1 genes are associated with age of onset of psoriasis in our study, suggesting that the IL12B and PTG1 genes might have only a weak relation to psoriasis. However, we did find evidence that variants in the ADRB2 gene residing between IL12B and PTG1 are associated with psoriasis. This could explain why variants in the region have been found to be associated with psoriasis previously, although more studies confirming this should be pursued.

Ps12
Prospective study of childhood psoriasis
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Psoriasis is a chronic immune-mediated inflammatory skin dis- ease that occurs in about 3.5% of the general population. It begins in childhood in approximately one-third of the cases. Most children manifest with plaque-type psoriasis vulgaris, which is the most common form in childhood. Psoriasis vulgaris is a chronic, relapsing inflammatory skin disease with unpredictable natural history. The disease may affect any part of the body, but is most often located on the scalp, elbows, knees, and buttocks. The disease is characterized by red, scaly patches with raised borders. The disease may affect anywhere from a few percent of the body to most of the body. The disease may be mild or severe, and it may last for years, decades, or even a lifetime. The disease may occur at any age, but it is most common in children and young adults. The disease may be triggered by a variety of factors, including stress, infection, and exposure to the sun. The disease is usually treated with topical or systemic medications, but there is no cure. The disease may be treated with phototherapy, systemic medications, or a combination of both. The disease may be treated with phototherapy, systemic medications, or a combination of both. The disease may be treated with phototherapy, systemic medications, or a combination of both. The disease may be treated with phototherapy, systemic medications, or a combination of both.
we proposed a similar mechanism for IL-8 regulation. We performed an in silico analysis and found a 100-nucleotide-long possible interaction site between the mRNA of IL-6 and PRINS. An in vitro assay confirmed this predicted interaction with a very high affinity of PRINS binding to IL-6 mRNA (K_d = 1.04 nM L^-1). To validate the functionality of this interaction on the cellular level we created a PRINS reporter sequence, containing a scrambled sequence on the possible IL-8 interaction site, and performed the overexpression experiment with PRINS. While PRINS decreased the expression of IL-6, PRINS did not show the same effect; however, the level of CCL5 (having a different interaction site) decreased to the same level after overexpression of PRINS or PRINS. Based on our results we propose that PRINS acts as a regulator for both IL-6 and CCL5. These results link PRINS to inflammatory processes, and indicate the significance of its higher expression in psoriatic uninvolved epidermis. Funding: K111885, GINOP-2.1.2-15-2016-00007 and GINOP-2.3.2-15-2016-00015.

PS16 Early changes in peripheral lymphocyte populations during oral dimethylfumarate treatment
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Dimethylfumarate (DMF) is a common first-line drug used for the treatment of moderate-to-severe psoriasis. The immunosuppressive effect of DMF is associated with a reduction in the peripheral blood lymphocyte count, which, when uncontrolled, can result in severe lymphopenia. However, early changes in the lymphocyte compartment are relatively unexplored. Thus, in the current study we have assessed the changes in blood lymphocytes in a cohort of patients with psoriasis during the first 3 months of DMF treatment. Using multicolour flow cytometry we assessed the frequencies of CD4 and CD8 T cells and neutrophils. Our results showed that the majority of DMF-treated patients had a reduction in absolute lymphocytes after only 1 month of treatment. When analysed in detail, we found that a loss of peripheral neutrophils was primarily responsible for this reduction, particularly in the first month of treatment. In most patients, the reduction in neutrophils persisted at all time points - cell counts were generally increased compared with baseline after 1 month of treatment. Hence, the number of peripheral CD4 T cells was increased in 69% of patients with a mean increase to 55.8 ± 36.7% of the pretreatment baseline. Increases in both CD4 and CD8 T cells accounted for this change. At the same time point, a drop in the number of T cells was seen in 23% of patients. For the PIC group overall (P = 0.001), however, there were no changes in B-cell subsets. As no differences were observed between groups regarding safety, we hypothesize that DMF induces and maintains psoriasis pathology. To understand the feedback loop between the IL-17 and IL-36 cytokines that drive psoriatic inflammation via a synergistic mechanism, we used an ear injection model in vivo to investigate psoriasis. Our results strengthen the thesis that neutrophils are affected by oral DMF from an early time point to such a degree that neutrophils are critical for psoriasis pathogenesis, it is likely that these changes are linked to the disease ameliorating effect of DMF.

PS17 Randomized controlled trial of patient-initiated care for patients with psoriasis
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Treatment and care of moderate-to-severe psoriasis often revolves around consultation with dermatologists with close monitoring of systematic treatments. Moreover, psoriasis is a fluctuating disease and the patient’s healthcare needs depend on the current severity of their psoriasis, indicating that healthcare services play a more significant role during some periods of patients’ lives. We hypothesize that a more flexible system may reduce inappropriate follow-up consultations, release resources to difficult consultations, and improve patient quality of life and satisfaction with healthcare. We therefore, aimed to determine the effect of patient-initiated care (PIC) for patients with psoriasis in a dermatology outpatient clinic. A prospective randomized controlled trial was set up. Patients on well-controlled systematic treatment were randomly assigned to either (i) the PIC group, where patients received one yearly scheduled consultation with a dermatologist and with no routine follow-up consultations, but patients were able to initiate consultations when needed or (ii) the control group, where patients received consultation routinely every 12-16 weeks. The main outcome was Dermatology Life Quality Index (DLQI), and secondary outcomes were clinical status, safety and patient satisfaction with healthcare assessed at baseline and after 1 year. In total 150 patients were included in the study (73 in the PIC group and 77 in the control group). Overall 58.0% were treated with biologics, 37.9% with methotrexate and 4.0% with acitretin. In the PIC group, a mean reduction of 6.7 ± 5.5 points was observed compared with the control group (P = 0.001). However, there were no changes for safety, DLQI: -1.6 ± 5.9 vs. -4.7 ± 5.4 (P = 0.75) and in patient satisfaction with healthcare: 0.23 ± 0.35 vs. 0.13 ± 0.36 (P = 0.23). There was also no change in the number of consultations: 2.3 ± 1.4 vs. 2.3 ± 1.5 (P = 0.75). PIC offers some clinical benefits compared with routine care, giving patients more control over their disease, time while taking the necessary consultation. The intervention adds no harm to monitoring systematic psoriasis treatment, and patients report high quality of life and satisfaction with healthcare. Providing unnecessary consultation releases resources to organize a more patient-centred approach to dermatology services, providing time to support patients during vulnerable periods and psoriasis relapse, and decreases comorbidity risk. This study has been registered with ClinicalTrials.gov, flno. NCT01321698.

PS18 Establishment of an intradermal ear injection model of interleukin (IL)-17A and IL-36γ as a tool to investigate psoriasis inflammation
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Psoriasis is a chronic skin disease caused by the excessive secretion of inflammatory cytokines, and affects 2–3% of the Western population. The proinflammatory interleukin (IL-) 17A is a key cytokine in psoriasis. However, accumulating evidence has revealed that IL-36γ also plays a pathogenic role in this disease. So far, using keratinocyte monolayers and a three-dimensional psoriasis model we have already detected a feedback loop between the IL-17 and IL-36 cytokines that induces and maintains psoriasis pathologies. To understand more precisely the role of the IL-17A-IL-36γ cytokine network in skin pathology, we used an ear injection model in our present study. Therefore, we injected IL-17A and IL-17A alone into the ear in combination into the ear pinna of mice. After 4 days of consecutive treatment mice were euthanized and histological and immunohistochemical stainings were performed. The intradermal delivery of IL-17A and IL-36γ resulted in a significant increase in the number of inflammatory cells over time. Histological evaluation of IL-17A- and IL-36γ-treated skin showed a strong accumulation accompanying hyperkeratosis and hyperplasia. We found the same histological features in mice that underwent injections with IL-36γ alone, but to a lesser extent. IL-17A on its own was not able to induce psoriasis-like changes in the mouse skin. Moreover, the expression of generic encoding antimicrobial peptides, like mi100A8, mi102B4 (orthologue of human β-defensin 2), mi01A7A and mi01B14 (orthologue of human β-defensin 3) were upregulated after treatments with IL-36γ. IL-17A and IL-36γ in the ear pinna of mice provides an in vivo model to investigate psoriasis. Our results strengthen the thesis that IL-17A and IL-36γ-driven cytokines regulate psoriasis via a synergistic interaction. Our established intradermal ear injection model of IL-17A and IL-36γ can be utilized in future to monitor effects of various inhibitors of this cytokine network.

PS19 Pharmacogenomic signature of response to genetic therapy for psoriasis: Genistein in vitro and in vivo and its mechanism of action
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Itch and pain perception and epidemiology in patients with psoriasis: results from a prospective, multicentre study
N. Maas, K. Tötz, U. Mrowietz, V. Oijj, S. Ständer, S. Gerthoux, L. Cordingley and on behalf of the PSORT study group and BADBIR
University of Manchester and Manchester Academic Health Science Centre, Manchester, U.K.
High levels of psychological distress can influence long-term outcomes for people with psoriasis via psychophysiological and behavioural pathways such as alcohol use or nonadherence to medication. Beliefs about illness affect how people

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High levels of psychological distress can influence long-term outcomes for people with psoriasis via psychophysiological and behavioural pathways such as alcohol use or nonadherence to medication. Beliefs about illness affect how people
cope and adjust to psoriasis, but few studies have explored these relationships in those using biological therapies. We assessed levels of general and psoriasis-specific psychological distress and levels of anger experience in patients using conventional systemic and biological therapies. We evaluated factors associated with distress using regression analyses. Cross-sectional data from 811 patients using conventional systemic and biological therapies for the treatment of moderate-to-severe psoriasis and enrolled in the British Association of Dermatologists Biologic Interventions Register (BADBIR) were collected from 35 dermatology centres across England. We measured anger expression (State-Trait Anger Expression Inventory), distress (Hospital Anxiety and Depression Scale, HADS), beliefs about psoriasis (Revised Illness Perception Questionnaire) and medication (Beliefs about Medicines Questionnaire). A score ≥ 8 on the HADS indicates a possible or probable caseness for anxiety (40%) and/or depression (24%), with two-thirds reporting strong negative psoriasis-specific distress and almost half (43%) reporting strong feelings of anger towards their psoriasis. In total, 11.5% reported a high level of anger suppression. Stronger suppression of anger was associated with anxiety (standardized β = 0.40, P ≤ 0.001), depression (β = 0.40, P ≤ 0.001) and negative psoriasis-specific distress (β = 0.11, P = 0.001). Holding strong beliefs that psoriasis has negative consequences was associated with negative psoriasis-specific distress (β = 0.49, P ≤ 0.001). There were different drivers of anxiety and depression with worries about appearance associated with anxiety (β = 0.13, P = 0.006), whereas concerns that psoriasis is noticeable to others was associated with depression (β = 0.17, P ≤ 0.001). Patients who expressed the strongest medication concerns were more likely to report higher depression scores (β = 0.10, P = 0.002). Psychological distress remains high for many patients using systemic therapies. Some patients using biologics may require additional interventions to target long-held beliefs and address emotion-focused coping strategies in order to improve clinical outcomes and quality of life. C.E.M.G. is a National Institute for Health Research Senior Investigator. PSORT is funded by the Medical Research Council, grant MR/1011808/1.
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