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INHOUD

THEMANUMMER NVED-CONGRES
(gastredacteur: dr. Patrick Zeeuwen)

2 Programma
7 Abstracts oral presentations
23 Posters

ALGEMEEN

39 In het Kort

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Thursday 29 January 2026

09.30 - 10.15 **Registration and welcome with coffee/tea**

10.15 - 10.25 **Opening by the chair of the NVED**

10.25 - 11.30 **Session I - Biologics and JAK inhibitors in immunologic disease**
Session chair: Robert Rissmann (*CHDR*) and Marjolein de Bruin-Weller (*UMCU*)

1. Nikita Koster *CHDR*	Multi-omics analysis identifies common molecular signatures in psoriasis target lesions of varying severities during guselkumab treatment.
2. Emma Holtappels *AmsterdamUMC*	hiJAcK vitiligo: JAK3 and TEC family kinase inhibition in the pathogenesis and treatment of vitiligo.
3. Hidde Smits *UMCU*	Inhibition of the IL-4/ IL-13 signaling pathway induces type I immune activation in conjunctiva epithelial cells.
4. Anne-Lise Strandmoe *UMCG*	Altered pro-inflammatory B cell cytokine responses in pemphigus vulgaris: impact of prior rituximab treatment.
5. Margot Starrenburg *UMCU*	Impact of tralokinumab on circulating and skin immune cell landscape in patients with atopic dermatitis.

11.30 - 12.10 Guest lecture by dr. April Foster (*Wellcome Sanger Institute, United Kingdom*):
"Understanding human skin with spatial-temporal resolution across development and disease"

12.10 - 13:10 **Lunch**

13.10 - 14.15 **Session II – The skin barrier**
Session chairs: Shidi Wu (*LUMC*) and Barbara Horváth (*UMCG*)

6. Florentine de Boer *AmsterdamUMC*	Skin barrier biomarkers in patch-induced and clinical allergic and irritant contact dermatitis.
7. Lian van de Gang *UMCU*	Skin barrier changes in atopic dermatitis treated with biologics and JAK inhibitors.
8. Catherine Mergen *LACDR*	Stratum corneum ceramide alterations and barrier dysfunction in cutaneous T-cell lymphoma and their response to chlormethine treatment.
9. Noor van Hout *Radboudumc*	Construction of a minimal skin microbiome as a tool to study microbe-microbe interactions.
10. Florence Vroman *UMCU*	The differential effect of dupilumab and JAK inhibitors on the skin microbiome in patients with moderate-to-severe atopic dermatitis in daily practice: data from the BioDay registry.

14.15 - 14.35 **Come see my poster pitches (odd numbers)** Chair: Jeroen Bremer (*UMCG*)

14:35 - 15:35 **Poster presentation session I (odd numbers) with coffee and tea**

P1. Mariona Oliver (*CHDR*) Machine learning-based analysis of smartphone images for remote monitoring of psoriasis.
P3. Elise Beljaards (*LUMC*) Optical coherence tomography for non-invasive prediction of response to topical chlormethine in early-stage mycosis fungoides.
P5. Josephine Amkreutz (*LUMC*) The NORMA 1 study: NO Re-excision in pT1a MelanomA.
P7. Marie-Eline Debeuf (*MUMC+*) Biological changes of the skin after ablative laser therapy – a scoping review.
P9. Nienke Veldhuis (*UMCU*) Dupilumab-associated ocular surface disease in atopic dermatitis: results from a large prospective real-world cohort.
P11. Wouter Ouwerkerk (*AmsterdamUMC*) Biomarker-based diagnosis of contact dermatitis: a step towards more accurate and patient-friendly testing.
P13. Beatriz Oliveira Fagundes (*UMCG*) T cell exhaustion in chronic inflammatory diseases – a scoping review.

P15. Myrthe Moermans (*MUMC+*) Unmet care needs and experiences of patients with basal cell nevus syndrome and their parents: a qualitative interview study.
P17. Lindi Korpelshoek (*LUMC*) Incidence and outcome of other primary malignancies in 273 patients with primary cutaneous marginal zone lymphoma.
P19. Anastasiia Myronenko (*LUMC*) To what extent does behavioral immune activation influence itch contagion and public stigmatization of people with psoriasis? An experimental study.
P21. Otte Borghouts (*MUMC+*) Lack of consensus in reported outcomes for epidermal differentiation disorders: a scoping review.
P23. Juliette Farai Bollemeijer (*ErasmusMC*) Chronic pruritus in older adults: prevalence, associations, and pruritus-specific quality of life.
P25. Chen Liang (*UMCG*) A serum proteomics-based comparison between bullous pemphigoid and nonbullous pemphigoid.
P27. Angeliki Birmpili (*MUMC+*) Restoration of molecular profiles in psoriatic skin following guselkumab treatment
P29. Elise Leeman (*UMCG*) Evaluation of dupilumab in pemphigoid gestationis with placental pathology and literature review.

15.35 – 16.53 **Session III - Genetics in dermatology**
Session chairs: Antoni Gostynski (M*UMC+*) and Jos Smits (*Radboudumc*)

11. Rindert Venema *UMCG*	Development of a human anti-human-desmoglein-3 (hu-a-dsg3) monoclonal antibody for targeted systemic delivery to the skin.
12. Tara Urselmann *Radboudumc*	Multi-modal integrative single cell RNA-sequencing based atlas of chronic inflammatory skin disease.
13. Weixin Zhou *UMCG*	Transcriptome analysis reveals a distinct molecular profile of hyperkeratotic hand eczema.
14. Ashleigh Jimenez Lemus *MUMC+*	High prevalence of cutaneous postzygotic mosaicism of Patched 1 variants in patients developing multiple basal cell carcinomas.
15. Rosalie Baardman *UMCG*	Towards an optimal diagnostic strategy for epidermolysis bullosa (EB): the diagnostic and prognostic value of EB diagnostic modalities.
16. Fauve van Veen *MUMC+*	Reproductive dilemmas in genodermatoses: international perspectives of couples/patients, caregivers, dermatologists and clinical geneticists.

16:55 - 17:25 Marcel Jonkman lecture by **dr. Loes Hollestein** (*ErasmusMC*):
"From population-based data towards personalized prognostic models for skin cancer"

17.30 - 19.45 **Drinks and dinner**

19.45 - 20.30 **26th general assembly of the NVED**

20.30 - 01.00 **Party**

Friday 30 January 2026

09.00 - 10.05 **Session IV – Skin models to study human disease**
Session chairs: Sue Gibbs (*AmsterdamUMC*) and Martijn van Doorn (*ErasmusMC*)

17. Daphne Panocha *AmsterdamUMC*	A human immunocompetent LN-scaffold model to study human immune responses.
18. Alesha Louis *LUMC*	Dissimilar roles for papillary and reticular fibroblasts in skin pigmentation: insights from 3D *in vitro* human fibroblast derived matrix models.
19. Jaimy Klijnhout *Radboudumc*	A comparative analysis of N/TERT-derived epidermal equivalents.
20. Joliene Wichers-Schreur *LUMC*	Dissecting the impact of CTCL T-cell lines on epidermal structure and function.
21. Rens Peters *Radboudumc*	Combining experimental and AI-driven approaches for developing immunocompetent 3D skin models.

10.05 - 10.25 **Come see my poster pitches (even numbers)** Chair: Jeroen Bremer (*UMCG*)

10:25 - 11:20 **Poster presentation session II (even numbers) with coffee and tea**

P2. Joey Karregat (*AmsterdamUMC*) Incidence of tattoo-associated melanoma in the Netherlands (1991-2023): a nationwide registry study.
P4. Agnes Grutters (*UMCG*) The price of fragile skin: a scoping review on the economic burden of epidermolysis bullosa.
P6. Wandong Wang (*UMCG*) Spatial transcriptomic profiling of the tumour microenvironment of organ transplant–related versus sporadic cutaneous squamous cell carcinoma.
P8. Keneshka Atash (*UMCU*) Lebrikizumab in multi-therapy-refractory atopic dermatitis patients: a case series from the BioDay registry.
P10. Kim Daniëlle van der Gouw (*UMCG*) Improving the efficiency of antisense oligonucleotide-mediated exon skipping of COL7A1 to treat recessive dystrophic epidermolysis bullosa.
P12. Linda Godding (*Radboudumc*) Experiences of patients with generalised pustular psoriasis: a qualitative study.
P14. Fenna de Bie (*LUMC*) The effect of anthracycline treatment on primary cutaneous T-cell lymphoma cells.
P16. Julia Stankiewicz (*LUMC*) Investigating immunohistochemical markers in 101 early-stage mycosis fungoides patients: a retrospective study on disease progression and prognostic factors.
P18. Anna Patsea (*MUMC+*) Health-related quality of life in patients with basal cell nevus syndrome and high-frequency basal cell carcinoma: a questionnaire study
P20. Zixian Liang (*UMCG*) Complement fixation test in pemphigoid diseases: association with IgG1/IgG4 subclass profiles in a 1-year prospective study.
P22. Marleen de Winter (*LUMC*) Visualizing the effect of mogamulizumab on T regulatory cells in cutaneous T cell lymphoma using a multiplex immunofluorescence imaging approach.
P24. Marielle van der Peet (*CHDR*) Investigating the relationship between guselkumab treatment and signaling lipid levels in blister and plasma samples from psoriasis patients.
P26. Carin Smit (*UMCU*) Evaluating laboratory abnormalities in atopic dermatitis patients treated with JAK-inhibitors.
P28. Olivia Steijlen (*ErasmusMC*) The Dutch Squamous Cell Carcinoma and Metastasis (D-SQUAME) study: two nationwide cohorts with a nested case-control design for prognostic model development and validation.
P30. Clara Harrs (*UMCG*) Spatially resolved whole transcriptomic prolifing of aggressive cuteneous squamous cell carcinoma in epidermolysis bullosa.

11.20 - 12.25 **Session V – Cause and development in dermato-oncology**
Session chairs: Loes Hollestein (*ErasmusMC*) and Emoke Rácz (*UMCG*)

22. Marie Chevalier Florquin *LUMC*	Mapping the spatial immune landscape in Sezary syndrome: insights into moderate and progressive prognoses.
23. Andrya Reder Hollatz *ErasmusMC*	A prediction model for a first metachronous cutaneous squamous cell carcinoma: a 10-year nationwide cohort study.

	24. Inger Kreuger *LUMC*	Spatial gene expression and microenvironmental changes in the transition of nevus to melanoma.
	25. Alex Rooker *AmsterdamUMC*	Ptchflox/floxERT2+/– mouse model drives hair follicle neoplasms rather than basal cell carcinoma.
	26. Veerle Merkus *LUMC*	Stage-specific changes in the spatial immune landscape of mycosis fungoides.
12.25 - 13.00	**Lunch**	
13.30 - 14.10	Keynote Lecture by **Prof. dr. DirkJan Hijnen** (*Radboudumc*): **"A scientific journey through skin immunology: from Skin T cells to biologics in atopic dermatitis"**	
14.10 - 15.25	**Session VI – Clinical studies - innovations in treatment and monitoring** Session chairs: Juul van de Reek (*Radboudumc*) and Heike Röckmann (*UMCU*)	
	28. Juliette Simons *UMCU*	Performance of ciclosporin in omalizumab-naïve and omalizumab-refractory chronic urticaria in daily practice.
	29. Charlotte van Riel *Radboudumc*	Dose reduction of IL-17 and IL-23 inhibitors in patients with plaque psoriasis is non-inferior to usual care: an international pragmatic randomized controlled trial – the BeNeBio study.
	30. Sara van der Kamp *UMCU*	Performance of omalizumab in patients with mast cell-mediated angioedema.
	31. Ymke van Ginkel *UMCG*	Development of a fluorescence molecular imaging method for ustekinumab in psoriasis.
	32. Yara Valkenburg *MUMC+*	Dynamic vs. conventional optical coherence tomography for diagnosing basal cell carcinoma: a diagnostic cohort study.
15.25 - 15.40	**Awards for best presentation and poster**	
15.40	**Closure**	

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NVED abstracs 29-30 January 2026

Oral presentations

1 – NIKITA KOSTER

MULTI-OMICS ANALYSIS IDENTIFIES COMMON MOLECULAR SIGNATURES IN PSORIASIS TARGET LESIONS OF VARYING SEVERITIES DURING GUSELKUMAB TREATMENT

Nikita G. Koster[1,3,4], Robert Rissmann[1,3,4], Jannik Rousel[1], Menthe E. Bergmans[1], Catherine Mergen[3], Ingrid Tomljanovic[1], Marielle van der Peet[3], Thomas Hankemeier[3], Joke Bouwstra[3], Huma Shehwana[7], Johan Westerhuis[7], Tom Ederveen[5], Rob Vreeken[6], Eva Cuypers[6], Angiliki Birmpili[6], Martina Kutmon[6], Victor van der Valk[4], Boudewijn Lelieveldt[4], Marieke L. de Kam[1], Naomi B. Klarenbeek[1], Tessa Niemeyer-van der Kolk[1], Martijn B.A. van Doorn[2], the Next Generation ImmunoDermatology (NGID) consortium

[1]Centre for Human Drug Research, Leiden, The Netherlands; [2]Department of Dermatology, Erasmus Medical Centre, Rotterdam, The Netherlands; [3]Leiden Academic Center for Drug Research, Leiden University, Leiden, The Netherlands; [4]Leiden University Medical Center, Leiden, The Netherlands; [5]Radboud University Medical Center, Nijmegen, The Netherlands; [6]Maastricht Centre for Systems Biology and Bioinformatics (MaCSBio), Maastricht University, Maastricht, The Netherlands; [7]University of Amsterdam, The Netherlands.

Background Multi-omics profiling, including bulk transcriptomics, interstitial fluid (ISF) lipidomics, skin surface lipidomics, and spatial lipidomics, enables in-depth characterization of psoriasis pathobiology. Inter-individual differences in these profiles may influence treatment response.
Objective To determine whether phenotypically similar plaques in mild and moderate-to-severe psoriasis differ molecularly, and to assess the effects of IL-23 blockade with guselkumab on transcriptomic and lipidomic profiles across disease severities.
Methods Twenty patients with mild psoriasis (PASI ≤ 5) and six with moderate-to-severe disease (PASI ≥ 10) were enrolled, each with at least one target plaque on the extremities. Skin punch biopsies, tape strips, and peri-lesional suction blisters were collected before, during, and after 16 weeks of guselkumab therapy, alongside samples from ten healthy controls. Multi-omics analyses included RNA sequencing, LC-MS metabolomics, and mass spectrometry imaging.
Results At baseline, plaques were comparable across groups in erythema, scaling, and induration. Transcriptomic profiling revealed similar molecular signatures in both severity groups versus healthy controls, with elevated Th17- and modestly increased Th2-related gene expression. Only twelve genes differed significantly, all with small fold changes ($\log_2$FC < 1). Perilesional ISF metabolomics showed normalization of inflammatory lipid mediators (15-HETrE, S1P 18:2, S1P 16:1, 11-HETE) during treatment. Clinical, imaging, and molecular data indicated comparable therapeutic responses.
Conclusion Phenotypically similar plaques in mild and moderate-to-severe psoriasis share comparable molecular profiles, supporting IL-23 blockade with guselkumab as a relevant treatment for mild disease.

2 – EMMA HOLTAPPELS

HIJACK VITILIGO: JAK3 AND TEC FAMILY KINASE INHIBITION IN THE PATHOGENESIS AND TREATMENT OF VITILIGO

Emma Holtappels[1], Saskia Chielie[1], Nathalie van Uden[1], Tessa Licher[1], Marcel W. Bekkenk[1,2], Rosalie M. Luiten[1], Walbert J. Bakker[1]

[1]Amsterdam University Medical Center, Department of Dermatology, Netherlands Institute for Pigment Disorders, University of Amsterdam, Amsterdam Institute for Immunology and Infectious diseases, The Netherlands; [2]VU University of Amsterdam, The Netherlands.

Background Vitiligo is an autoimmune disease, characterized by depigmented skin-lesions due to melanocyte destruction. Current therapies have limited efficacy and often result in relapse, likely due to persisting tissue-resident memory T-cells (TRM), depending on IL-15 signaling via JAK3. Ritlecitinib is a novel, irreversible JAK3/TEC-family kinase-inhibitor through which TRM may be targeted in vitiligo.
Objective To elucidate the expression profile of JAK3/TEC-family kinases in vitiligo and assess whether ritlecitinib offers a durable treatment option.
Methods The JAK3/TEC family kinase (ITK/BTK/TEC) expression profile was analyzed on the RNA-level using publicly-available scRNAseq-datasets and on the protein-level using multiplex-immunohistochemistry, focusing on skin cells and T-cell subsets in healthy and vitiligo skin (n=13). Cellular effects of ritlecitinib were investigated in melanocyte/T-cell co-cultures and human vitiligo skin-explants.
Results In vitiligo skin, levels of JAK3/TEC-family kinase-expressing cells were increased compared to healthy skin. JAK3/TEC-family kinases were predominantly expressed by T-cells,

but, unexpectedly, keratinocytes also expressed JAK3/TEC-family kinases more frequently in vitiligo skin. Specific JAK3/TEC-family kinase inhibition using ritlecitinib effectively reduced *in vitro* cytotoxic activity of T-cells against melanocytes, showing a durable effect upon re-exposure to melanocytes without additional ritlecitinib treatment. Decreased melanocyte apoptosis was also seen in skin-explants. Ritlecitinib also reduced chemokine release from stimulated keratinocytes.
Conclusion We characterized expression of JAK3/TEC-family kinases and found significant, cell-type-dependent differential expression in vitiligo versus healthy skin. Ritlecitinib not only effectively inhibited T-cell activation, thereby reducing melanocyte apoptosis, but also reduced chemokine release from keratinocytes. Therefore, ritlecitinib has the potential to meet the requirements for effective repigmentation therapy in vitiligo.

3 – HIDDE SMITS

INHIBITION OF THE IL-4/L-13 SIGNALING PATHWAY INDUCES TYPE I IMMUNE ACTIVATION IN CONJUNCTIVA EPITHELIAL CELLS

H.M. Smits[1], A. MI Elfiky[1,2,3], C. Dekkers[2], M. van der Wal[1], J. Drylewicz[1], M. de Bruin-Weller[2], F. van Wijk[1]
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Background Both dupilumab and tralokinumab are monoclonal are highly effective in treating moderate-to-severe atopic dermatitis. Dupilumab blocks the shared IL-4Rα subunit, thereby inhibiting both IL-4 and IL-13 signaling, in contrast tralokinumab selectively binds the IL-13 molecule. The most common side effect of these treatments is ocular surface disease (OSD). The mechanisms underlying these adverse effects remain poorly understood.
Objective This study aimed to investigate the transcriptional mechanisms underlying OSD in non-immune conjunctival cells following dupilumab and tralokinumab treatment.
Methods Eyeprim samples were collected from 6 dupilumab-treated patients, 6 tralokinumab-treated patients (at baseline and after four weeks), and six non-atopic controls. Conjunctival epithelial cells were analyzed by single-cell RNA sequencing.
Results Computational analysis revealed three main epithelial populations: basal cells, superficial epithelial cells, and immune-activated superficial epithelial cells. Compared with non-atopic controls, showed no significantly enriched pathways in either population, although weak immune activation was evident through upregulation of HLA-DQA1 and IL6R in epithelial cells. Following dupilumab treatment, immune-activated superficial epithelial cells expanded and displayed an IFNγ-driven type 1 immune signature, characterized by increased CXCL1, CXCL6, CXCL9, CXCL10, CCL20 and HLA-DR expression, suggesting increased activation and immune cell recruitment. Tralokinumab treatment induced a similar but less pronounced response.
Conclusion IL-4/IL-13 pathway inhibition induces a conjunctival T1 immune response, which is more pronounced with dual IL-4/IL-13 blockade than with selective IL-13 inhibition by tralokinumab. These findings align with the clinical manifestations of ocular surface disease observed in treated patients and provide translational insights that may inform therapeutic decision-making.

4 – ANNE-LISE STRANDMOE

ALTERED PRO-INFLAMMATORY B CELL CYTOKINE RESPONSES IN PEMPHIGUS VULGARIS: IMPACT OF PRIOR RITUXIMAB TREATMENT

Anne-Lise Strandmoe[1,3], Nanthicha Inrueangsri[1], Inge M. Strating[1], Wayel H. Abdulahad[2], Kevin P. Mennega[1], Carlo G. Bonasia[2], Elisabeth Raveling-Eelsing[2], Jeroen Bremer[3], Barbara Horvath[3], Peter Heeringa[1]
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Background Pemphigus vulgaris (PV) is a B cell–mediated autoimmune blistering disease characterized by loss of epidermal and/or mucosal adhesion due to autoantibodies predominantly targeting desmoglein 3. Although B cell–depleting therapy with rituximab has revolutionized PV treatment, disease relapse remains common, and the immunological mechanisms underlying relapse are not fully understood.
Objective To assess whether differences in pro- and anti-inflammatory cytokine production by B cells distinguish patients with active PV from healthy controls, and to explore whether prior rituximab treatment or relapse-prone patients are associated with altered B cell functional profiles.
Methods PBMC samples were collected from 29 active PV patients and age/sex-matched healthy controls (HC). Among PV patients, 11 were rituximab-naive (nPV) and 18 were rituximab-non-naive (nnPV), having previously received rituximab and subsequently relapsed. At sampling, 72% of nPV and 22% of nnPV were receiving prednisone. PBMCs were cultured using CpG for 3 days, and BFA, PMA, and CaI for 5 hours for maximal stimulation. Intracellular cytokine (IL-10, TNFα, IL-6) production was analyzed by flow cytometry.
Results Patients with PV showed a reduced frequency of TNFα$^+$ B cells compared with HCs (8,8% vs. 18,6% of B cells). Within the PV cohort, nnPV patients displayed a significant decrease in TNFα$^+$ (6,3% vs. 20,7% of B cells) and IL-6$^+$TNFα$^+$ B cells (0,71% vs. 2,35% of B cells) compared with nPV patients.
Conclusion These findings suggest a lasting alteration in the pro-inflammatory capacity of B cells, which may reflect either long-term effect of prior rituximab treatment or features associated with relapsing patients.

5 – MARGOT STARRENBURG

IMPACT OF TRALOKINUMAB ON CIRCULATING AND SKIN IMMUNE CELL LANDSCAPE IN PATIENTS WITH ATOPIC DERMATITIS

Margot Starrenburg[1,2], Coco Dekkers[1,2], José ter Linde[2], Maria van der Wal[2], Kristof van Avondt[3], Mojtaba Amini[3], Daphne Bakker[1,2], Yvonne Vercoulen[3], Marjolein de Bruin-Weller[1#], Femke van Wijk[2#]

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Background Tralokinumab, an IL-13 targeting mAb, is an effective treatment for atopic dermatitis (AD). Th2 signaling disruption by IL-4Ra blockade had a strong functional immunological effect in AD patients, particularly on skin-homing T-cells. Skin resident T-cells have been associated with recurrent inflammatory skin diseases, such as AD.
Objective To study changes in (skin-homing) T-cell dynamics systemically and local (skin resident) T-cell function and distribution induced by selective IL-13 signaling blockade in AD.
Methods Blood samples of 22 AD patients and skin samples of 10 AD patients were collected longitudinally during tralokinumab treatment. PBMCs were characterized in flowcytometric assays, skin biopsies were studied using Imaging Mass Cytometry (IMC).
Results Mean EASI scores and serum TARC levels decreased significantly during treatment. Flow cytometry revealed a decrease in type 2 cytokine production and proliferation of skin-homing T-cells during treatment. IMC analysis enabled characterization of immune cell clusters in both the dermis and epidermis, including various myeloid cell types and several subsets of T-lymphocytes, amongst which OX40+, cytokine producing CD103+CD69+ skin resident CD4+ T-cells (CD4+ Tsr). The proportion of CD4+ Tsr decreased in the epidermis, but remained present in the dermis. Additionally, TARC producing myeloid cells and interferon gamma producing T-cells were significantly reduced in AD-lesional skin after tralokinumab treatment, while regulatory T-cell presence was increased.
Conclusion Systemically, tralokinumab reduces type 2 inflammation and serum TARC levels. Locally, attenuated AD activity by a reduction in TARC production and decreased cytokine producing CD4+ Tsr in the epidermis was observed, while dermal CD4+ Tsr persist.

6 – FLORENTINE DE BOER

SKIN BARRIER BIOMARKERS IN PATCH-INDUCED AND CLINICAL ALLERGIC AND IRRITANT CONTACT DERMATITIS

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Background Skin barrier impairment is central to irritant (ICD) and allergic contact dermatitis (ACD). *Stratum corneum* (SC) components cholesterol sulphate (CholSulph), glucosylcholesterol (CholGlc), and natural moisturizing factor (NMF) are critical for barrier function, but their changes in ICD and ACD remain underexplored.
Objective To measure CholSulph, CholGlc, NMF, and IL-1α, in patch-induced ICD and ACD, and in hand dermatitis (HD) diagnosed as ICD or ACD.
Methods SC samples were collected from HD patients undergoing patch testing. Biomarkers were analyzed in positive reactions to sodium lauryl sulfate (ICD, n = 44), allergens (ACD, n=113; nickel, chromium, methylisothiazolinone (MI)), lesional HD skin (n = 45) and control (empty chamber, n=121).
Results CholGlc was significantly elevated in patch-induced ICD and ACD. CholSulph increased in ICD and chromium- and MI induced ACD. NMF decreased in ICD, while IL-1α decreased in ICD and chromium ACD. Chromium induced the strongest response, nickel the weakest. In HD, ICD and ACD showed elevated CholGlc, reduced NMF and IL-1α, with CholSulph increased only in ACD. No biomarker differences were detected between clinical ICD and ACD.
Conclusion Both induced and clinical ICD and ACD show consistent SC biomarker changes reflecting barrier dysfunction, with no differences between clinical ICD and ACD.

7 – LIAN VAN DER GANG

SKIN BARRIER CHANGES IN ATOPIC DERMATITIS TREATED WITH BIOLOGICS AND JAK INHIBITORS

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Background Atopic dermatitis (AD) is strongly associated with impaired skin barrier function and altered *stratum corneum* lipid composition, particularly changes in ceramide levels. Skin barrier assessment can be clinically useful for lesion follow-up and therapy evaluation.
Objective To characterize skin barrier changes during biologic or Janus kinase inhibitor (JAKi) treatment using electrical impedance spectroscopy (EIS) and ceramide profiling.
Methods *Stratum corneum* impedance and deeper layer impedance were measured on lesional and non-lesional volar forearm skin of adult AD patients starting biologics (n=39) or JAKi (n=10) at baseline, week 4, and week 16. Controls included psoriasis patients (n=10) and healthy controls (n=10). Ceramides are currently being analyzed using liquid chromatography-mass spectrometry.
Results At baseline, both lesional and non-lesional EIS scores were significantly lower in AD patients compared to healthy controls and psoriasis patients. By week 16, both *stratum corneum* impedance and deeper layer impedance improved significantly, and *stratum corneum* impedance no longer significantly differed from healthy controls. When comparing treatment groups, no significant differences in EIS change after 16 weeks of treatment were found. However, JAKi induced greater EIS change at week 4 than biologics. Preliminary ceramide analysis shows partial normalization of the ceramide subclass composition and chain length with treatment.
Conclusion Systemic treatment with biologics and JAKi recovers skin barrier function in AD, as objectively measured by EIS. Barrier recovery was faster with JAKi, although differences levelled out by week 16. During treatment, EIS approached those of healthy controls, supporting its utility to monitor treatment response.

8 – CATHERINE MERGEN

STRATUM CORNEUM CERAMIDE ALTERATIONS AND BARRIER DYSFUNCTION IN CUTANEOUS T-CELL LYMPHOMA AND THEIR RESPONSE TO CHLORMETHINE TREATMENT

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Background Mycosis fungoides (MF) is the most common subtype of primary cutaneous T-cell lymphoma, in which malignant T lymphocytes infiltrate the skin, resulting in patches, plaques, and eventually tumors, which are associated with a reduced skin barrier function. The *stratum corneum* lipid matrix is essential for maintaining an intact skin barrier and alterations in lipid composition, especially ceramides, have been associated with a reduced barrier function in inflammatory skin conditions.
Objective To characterize the *stratum corneum* ceramide profile and skin barrier function in lesional and non-lesional skin of MF patients and in healthy volunteers, and to assess the effect of topical treatment with chlormethine gel on the ceramide profile.
Methods 21 early-stage MF patients and 10 healthy volunteers participated in the study. Ceramides were collected by tape stripping at baseline and after 16 weeks of treatment with chlormethine gel and analyzed using liquid chromatography-mass spectrometry. Barrier function was assessed by measuring transepidermal water loss.
Results The ceramide profile of lesional skin in MF was significantly different from non-lesional and healthy skin and correlated with the reduced barrier function. Specifically, lesional skin showed an altered ceramide subclass composition and a reduction in average ceramide chain length. These changes were partially normalized with chlormethine treatment, particularly in patients with a good treatment response.
Conclusion MF lesions exhibit a distinct and altered ceramide profile compared to non-lesional and healthy skin, which can partially be restored with topical chlormethine treatment.

9 – NOOR VAN HOUT

CONSTRUCTION OF A MINIMAL SKIN MICROBIOME AS A TOOL TO STUDY MICROBE-MICROBE INTERACTIONS

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Background The skin microbiome plays a key role in maintaining skin health, and its imbalance contributes to inflammatory skin diseases such as atopic dermatitis (AD), often marked by *Staphylococcus aureus* dominance.
Objective To investigate microbe-microbe interactions within a minimal skin microbiome by developing selective culture methods that allow quantification of individual bacterial strains from mixed communities after co-culture with skin models.
Methods Four skin-associated bacteria were selected, three commensal species (*Cutibacterium acnes*, *Staphylococcus epidermidis*, *Corynebacterium striatum*), and one pathogenic strain (*Staphylococcus aureus*). Mixed samples in 1:1:1:1 ratios were plated on selective agar plates under varying conditions to enable differentiation of colony-forming units per species. Tested parameters included pH modification, antibiotic overlays, incubation temperature, and colorimetric selection.
Results Acidification of LB agar to pH 4.6 selectively inhi-

bited *C. striatum* growth, facilitating separation from both *Staphylococcus* species. Overnight incubation at room temperature inhibits the growth of *S. epidermidis*, allowing to count the amount of *S. aureus*. Furthermore, an afabicin overlay effectively inhibited Staphylococci, allowing quantification of *C. acnes* and *C. striatum*. Because *C. acnes* only grows anaerobically, the amount of *C. striatum* can be determined under aerobic culturing conditions.
Conclusion By combining abiotic, antibiotic, and colorimetric selection strategies, we established a reproducible method to quantify individual alive bacterial populations within a defined minimal skin microbiome. These optimized separation techniques can support future studies on microbe–microbe interactions and the growth dynamics of potential therapeutic probiotic strains on human epidermal equivalents.

10 – FLORENCE VROMAN

THE DIFFERENTIAL EFFECT OF DUPILUMAB AND JAK INHIBITORS ON THE SKIN MICROBIOME IN PATIENTS WITH MODERATE-TO-SEVERE ATOPIC DERMATITIS IN DAILY PRACTICE: DATA FROM THE BIODAY REGISTRY

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Background Atopic dermatitis (AD) is associated with reduced skin microbial diversity and increased *Staphylococcus aureus* (*S. aureus*) colonization. Dupilumab has shown to improve skin microbial diversity and lower *S. aureus* abundance in AD patients; however, less is known on the effect during Janus kinase 1-selective inhibitor (JAK1-i) treatment.
Objective To evaluate the effect of JAK1-i compared to dupilumab on the skin microbiome of AD patients aged 12 years and older.
Methods Skin swabs were collected from AD patients: for dupilumab (n=20) lesional/non-lesional at baseline (T0) and week 16 (T16); for JAK1-i (n=15) lesional/non-lesional at T0, week 4 (T4) and 28 (T28); and for healthy controls (n=27). Relative abundance and microbial diversity were analyzed using shotgun sequencing.
Results In both groups, Eczema Area Severity Index (EASI) scores significantly decreased over time, indicating good clinical response. For dupilumab, in lesional skin, a significant decrease of *S. aureus* was observed at T16 compared to T0 (Log Fold Change (LFC) of 7.6). However, in JAK1-i treated patients, a less apparent, non-significant decrease of *S. aureus* was observed at T28 compared to T0 in lesional skin. In addition, during dupilumab treatment, a shift in microbial profiles and increase in diversity was observed, which revealed a shift towards that of HCs. This was less evident in JAK1-i treated patients.
Conclusion Although both dupilumab and JAK1-i treatment resulted in a comparable clinical effect, the skin microbiome of AD patients treated with dupilumab shifted towards that of healthy skin, while this effect was not observed during JAK1-i treatment.

11 – RINDERT VENEMA

DEVELOPMENT OF A HUMAN ANTI-HUMAN-DESMOGLEIN-3 (HU-A-DSG3) MONOCLONAL ANTIBODY FOR TARGETED SYSTEMIC DELIVERY TO THE SKIN

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Background Despite advances in skin gene therapies for genodermatoses such as recessive dystrophic epidermolysis bullosa (RDEB), effective delivery continues to be a major hurdle. Previously, we developed antisense oligonucleotide mediated exon skipping for RDEB, however, optimization of delivery is essential for exon skipping to be a viable approach for systemic treatment of RDEB. Therefore, we use exon skipping for RDEB as use case for the development of a universal targeted systemic delivery platform for the skin, which could be beneficial for RDEB and translated to other skin related diseases.
Objective Develop a universal targeted systemic delivery platform for the skin, by capturing the genetic sequence of hu-a-dsg3 targeting B-cells of Pemphigus Vulgaris (PV) patients.
Methods To capture the genetic sequence of hu-a-dsg3 B-cells, we isolated peripheral blood mononuclear cells from PV patients and stained for B-cell markers and AF647-labelled recombinant DSG3. Next, cells were single cell sorted by FACS and stimulated for antibody production for two weeks. A DSG3 ELISA was performed for IgG producing colonies, where positives were selected for RNA isolation, templated switching oligo cDNA conversion and nested PCR to obtain the B cell receptor sequence. Next, we re-expressed this sequence in plasmids designed for antibody production and produced the monoclonal hu-a-dsg3 delivery platform in HEK293T cells. Lastly, the hu-a-dsg3 antibody was characterized by extensive *in vitro* diagnostic assays.
Results/Conclusion We have successfully isolated and expressed a monoclonal hu-a-dsg3 antibody which lays the foundation for the development of a universal delivery platform for gene therapies as antisense oligonucleotides.

12 – TARA URSELMANN

MULTI-MODAL INTEGRATIVE SINGLE CELL RNA-SEQUENCING BASED ATLAS OF CHRONIC INFLAMMATORY SKIN DISEASE

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Background Current treatment of chronic inflammatory skin disease patients is trial-and-error based. This one-size-fits-all strategy fails to consider stratification biomarkers and overlooks potential shared mechanisms across diseases and repurposing of established drugs. Growing research efforts directed to atopic dermatitis (AD), Psoriasis (PSO), and more recently Hidradenitis Suppurativa (HS) have yielded multiple publicly available single-cell RNA-sequencing (scRNAseq) datasets of patient skin biopsies. Inclusion of both lesional and non-lesional samples, and treated patients enables high resolution investigations into cell-specific transcriptome-based mechanisms.
Objective To identify and integrate scRNAseq datasets of AD, PSO, and HS to build a computational tool able to visualize gene expression, including pathway activity scores and user-provided module scores to deepen atlas capabilities.
Methods Datasets were extracted from databases and processed through the Seurat pipeline. The data was integrated with Harmony and visualized by UMAP. Pathway activity was calculated with the PROGENy package.
Results We successfully constructed a single cell atlas through data curation and subsequent integration, resulting cells to cluster by cell type, enabling comparison between diseases. Pathway activity analysis was validated by JAK-STAT upregulation in AD, and revealing it as a shared mechanism across all three diseases. In contrast, TNFα pathway activity differentiated the diseases, showing elevated activity in keratinocytes from HS patients, while it was primarily upregulated in fibroblasts from PSO patients. Future research will focus on experimental validation and enrollment of the tool for public access.
Conclusion Our successful data integration enables identification of cell-specific mechanisms between chronic inflammatory skin diseases to improve personalized treatment.

13 – WEIXIN ZHOU

TRANSCRIPTOME ANALYSIS REVEALS A DISTINCT MOLECULAR PROFILE OF HYPERKERATOTIC HAND ECZEMA

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Background Hyperkeratotic hand eczema (HHE) is a clinical subtype of hand eczema, but emerging evidence suggests distinct pathophysiology.
Objective To profile the transcriptomes of HHE and compare these profiles with psoriasis and atopic dermatitis.
Methods Biopsies were obtained from lesional and non-lesional palmar skin of 11 adult patients with moderate-to-severe HHE and from the central palmar regions of 11 HC. Differentially expressed genes (DEGs) were identified using RNA sequencing (|fold change| > 2.0, false discovery rate < 0.01), with pathway enrichment analysis via KEGG/REACTOME databases. Gene set variation analysis (GSVA) facilitated comparison between lesional skin, non-lesional skin, and HC, as well as comparative analysis with GSE121212 dataset of psoriasis and atopic dermatitis transcriptomic data, obtained from body sites other than the hands.
Results RNA-seq revealed 2329 DEGs between lesional skin and HC and 318 between non-lesional skin and HC. Upregulated genes in lesional skin included T-cell activation markers (TNFRSF4, IL2RA), pro-inflammatory mediators (IL36G/20/23A), and chemokines (CXCL9/10, CCL18). Downregulated genes included lipid metabolism regulators (PLIN1, CIDEC) and barrier components (CLDN7/8/10). GSVA revealed increased Th1/Th2/Th17/Th22 activation, enhanced IL-12/23 and IL-36 signalling, increased tissue resident memory T cell (TRM) signatures and impaired lipid barrier and tight junction pathways in lesional skin. HHE exhibited broader T-helper activation than the Th17/IL-36-dominant profile of psoriasis and the Th2/JAK-STAT-driven signature of atopic dermatitis, with more pronounced TRM enrichment and barrier dysfunction.
Conclusion HHE is a distinctive entity with transcriptomic positioning between psoriasis and atopic dermatitis, implying a potentially unique pathophysiology.

14 – ASHLEIGH JIMENEZ LEMUS

HIGH PREVALENCE OF CUTANEOUS POSTZYGOTIC MOSAICISM OF PATCHED 1 VARIANTS IN PATIENTS DEVELOPING MULTIPLE BASAL CELL CARCINOMAS

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Background Basal cell nevus syndrome (BCNS) is a rare genetic disorder, characterized by multiple basal cell carcinomas (BCCs) and associated syndromic features. BCNS results from heterozygous pathogenic variants in the Patched 1 (PTCH1) tumor suppressor gene. Causal germline PTCH1 variants are well established in BCNS, however the prevalence of postzygotic mosaicism for PTCH1 variants in cutaneous BCC cases remains unclear.
Objective We aimed to investigate PTCH1 mosaicism prevalence in a cohort of patients with multiple BCCs and other BCNS features, lacking germline PTCH1 mutation in blood.
Methods Multiple different BCCs from 42 patients suspected having BCNS, lacking a germline causal PTCH1 variant in blood, were genotyped for PTCH1 using targeted next-generation sequencing. This cohort study was complemented by a literature review on PubMed, LOVD and EMBASE, to conceptualize the prevalence of de novo PTCH1 variants in BCNS.
Results Literature review demonstrated that the prevalence of de novo mutations in BCNS patients account for 35.8%. This suggests that mosaicism may be more prevalent in the general population than earlier acknowledged. Accordingly, in our cohort of patients with suspected BCNS, we found 33% of patients with postzygotic mosaicism in PTCH1, sharing a variant in the patient's BCCs. Remarkably, these patients frequently exhibit only multiple BCCs, with no other manifestations of BCNS.

Conclusion We demonstrate by using this analytic strategy, that many of the so called high frequency BCC patients are ultimately diagnosed as postzygotic PTCH1 mosaic cases. PTCH1 mosaicism may represent a significant proportion of patients with unexplained occurrence of multiple BCCs.

15 – ROSALIE BAARDMAN

TOWARDS AN OPTIMAL DIAGNOSTIC STRATEGY FOR EPIDERMOLYSIS BULLOSA (EB): THE DIAGNOSTIC AND PROGNOSTIC VALUE OF EB DIAGNOSTIC MODALITIES

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Background Epidermolysis bullosa (EB) comprises a heterogeneous group of rare disorders featuring mucocutaneous fragility. Currently, the diagnostic modalities for EB encompass: clinical assessment, immunofluorescence microscopy (IFM), transmission electron microscopy (TEM) and genome diagnostics (GD).
Objective To evaluate the diagnostic performance of clinical assessment, IFM, TEM, and GD in determining (1) main EB-type and (2) EB-subtype, and (3) to assess the added value of microscopy to GD.
Methods Patients with genetically confirmed EB-diagnoses who had IFM and TEM performed between 1988-2023 were retrospectively included. The proportion of cases in which the outcomes of the EB-diagnostic modalities matched for (1) main EB-type and (2) EB-subtype was calculated across the entire cohort, subdivided by EB-(sub)type and age. The reference standard for main EB-type was the identified pathogenic gene aligning with initial clinical phenotype and for EB-subtype the final EB-diagnosis. To evaluate the added value of IFM and TEM over GD, we assessed cases where IFM or TEM matched the final EB-diagnosis in cases where GD did not.
Results We included 202 patients. Initial clinical assessment, IFM and TEM matched main EB-type in 80.7%, 81.7% and 89.1%. Regarding EB-subtype: initial clinical assessment, IFM, TEM and GD matched in 41.1%, 29.7%, 12.9% and 55.4%. The added value of microscopy to GD in EB-subtyping was 6%. Additionally, IFM performed best in neonates and junctional EB.
Conclusion The EB-diagnostic modalities showed higher diagnostic than prognostic value, with GD excelling. Notably, IFM exhibited the highest prognostic value in neonates, highlighting its continued critical role in daily clinical practice.

16 – FAUVE VAN VEEN

REPRODUCTIVE DILEMMAS IN GENODERMATOSES: INTERNATIONAL PERSPECTIVES OF COUPLES/PATIENTS, CAREGIVERS, DERMATOLOGISTS AND CLINICAL GENETICISTS

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Background Inherited skin disorders (genodermatoses) impact patients' quality of life. Given the chronicity of genodermatoses and risk of transmission, couples/patients considering parenthood may face reproductive dilemmas. However, very little is known about this topic.
Objective To understand the reproductive decision-making (RDM) process: 1) Explore the impact of genodermatoses on couples/patients' RDM, their knowledge of and experience with reproductive options and counseling. 2) Examine clinical practice of professional support in RDM, from dermatologists and clinical geneticists' perspectives. 3) Assess the caregiver burden for those caring for patients with genodermatoses.
Methods Two qualitative studies were conducted: 1) with affected couples/patients, and 2) with dermatologists and clinical geneticists working in the Netherlands, Belgium, Australia and Japan. A scoping review was performed to investigate the perceived caregiver burden.
Results Interviews with couples/patients (n=30) revealed that most participants preferred to prevent transmission and considered reproductive options like pre-implantation genetic testing (PGT). RDM was influenced by negative experiences and fear about severe manifestations in offspring. Routine reproductive counseling was inadequate. Preliminary

findings from interviews with dermatologists and clinical geneticists (n=20) showed limited awareness among dermatologists regarding when to discuss reproductive options (e.g., PGT), alongside uncertainty about their counseling role for different genodermatoses. Clinical geneticists, while skilled in counseling, often lacked detailed knowledge of genodermatoses. The scoping review (54 included studies) showed variable attention from researchers per group of genodermatoses and a multifaceted impact on caregivers, influencing their quality of life and RDM.
Conclusion Genodermatoses substantially affect RDM, underscoring the need for routine reproductive counseling, improved education and interdisciplinary guidelines.

17 – DAPHNE PANOCHA

A HUMAN IMMUNOCOMPETENT LN-SCAFFOLD MODEL TO STUDY HUMAN IMMUNE RESPONSES

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Background Fibroblastic reticular cells (FRCs) arrange the dense lymph node (LN) architecture and are required for LN functioning. However, to date, current human *in vitro* LN models do not accurately mimic the aspects of human LN physiology while simultaneously allowing the long-term culture of FRCs in an *in vitro* model, which is needed to study the human immune system.
Objective In this study we aim to develop a human LN model using pre-printed scaffolds, comprising of FRCs and autologous immune cells from human LNs, for the long-term culture of FRCs and immune cells.
Methods Human FRCs and immune cells were isolated from LN biopsies. Pre-printed scaffolds were coated with collagen type 1 and first seeded with FRCs, followed by autologous immune cells. After 21 days of culture, flow cytometry, confocal imaging and cytokine/chemokine analysis were performed.
Results The LN-scaffold model resulted in viable cultures for up to 21 days and enabled close contact between FRCs and immune cells. FRC maintained their expression of important cell surface markers and the LN-scaffold supported the culture of several immune cell subsets. Furthermore, the microenvironment formed in the LN-scaffold model showed physiological similarities to the *in vivo* LN niche, consisting of extracellular matrix and the relevant cytokines and chemokines needed for immune homeostasis.
Conclusion This study demonstrates the relevance of LN-scaffolds for properly mimicking LN physiology. These findings support the suitability of the LN-scaffold model for multi-organ-on-chip set-ups, such as a skin-draining LN model to study human immune responses downstream from dermal pathology.

18 – ALESHA LOUIS

DISSIMILAR ROLES FOR PAPILLARY AND RETICULAR FIBROBLASTS IN SKIN PIGMENTATION: INSIGHTS FROM 3D IN VITRO HUMAN FIBROBLAST DERIVED MATRIX MODELS

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Background Human dermis is separated into the papillary (Pfs) and the reticular (Rfs) layer and with age a relative increase in the reticular layer has been observed. Skin aging is associated with the development of pigmented lesions, and since pigmentation is regulated by multiple pathways, distinct roles for Pfs and Rfs are hypothesized. Interestingly, transcriptomic studies suggest that the skin microbiome may influence pigmentation. Melanocytes can affect the composition of the skin microbiome, while the microbiome itself plays a role in melanocyte survival. Both the composition of the skin microbiome and the structure of the dermal layers changes with age, which may impact pigmentation. Therefore, in this study, we investigated the role of Pfs, Rfs and the skin microbiome on skin pigmentation.
Objective To explore the combined impact of Pfs, Rfs and microbiome shifts on skin pigmentation.
Methods Human skin equivalents (HSE) generated with Pfs or Rfs, and inoculated with *Staphylococcus (S.) epidermidis* were analyzed for epidermal morphogenesis and melanogenesis-related pathways.
Results Pf-HSE demonstrated an enhanced epidermal structure compared to Rf-HSEs. Rf-HSE have increased numbers of melanocytes in the basal layer, increased melanin and distinct expression of melanogenesis-related genes compared to Pf-HSE. *S. epidermidis* seem to increase melanocyte number although its exact role in melanocyte survival and skin pigmentation needs to be elucidated.
Conclusion We reveal dissimilar roles for Pfs and Rfs and the skin microbiome in skin pigmentation and melanogenesis-related pathways. Further investigation is warranted to validate the role of Pfs, Rfs and the skin microbiome in skin pigmentation.

19 – JAIMY KLIJNHOUT

A COMPARATIVE ANALYSIS OF N/TERT-DERIVED EPIDERMAL EQUIVALENTS

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Background N/TERT immortalized keratinocyte cell lines share key epidermal characteristics with primary keratinocytes in human epidermal equivalents (HEEs). Over the past 25 years, their widespread use in experimental dermatology has led to increasing variability in cell culture protocols, complicating cross-study comparisons.
Objective To compare N/TERT HEEs generated using EpiLife, KSFM, CELLnTEC or in-house developed media to *in vivo* epidermis based on morphology and epidermal gene- and protein expression.
Methods Formalin-fixed paraffin-embedded HEEs from two collaborating laboratories were assessed on morphology, and proliferation and differentiation protein markers, including Ki67, keratins 2, 10, 15 and 16, filaggrin, involucrin, transglutaminase-1 (TGM-1) and cathepsin V (CTSV). RNA sequencing was performed on EpiLife and CELLnTEC cultures to identify differentially expressed genes.
Results All culture protocols generate a multilayer stratified epidermis. EpiLife-generated HEEs have 5-6 epidermal layers and a cobblestone-like basal layer. Other models contain 6-8 cell layers and a less pronounced basal morphology. Ki67 staining confirms differences in proliferation rates between media and differentiation marker expression varies between models. A lower proliferation rate correlated with more *in vivo*-like differentiation patterns and higher expression of terminal differentiation proteins TGM-1 and CTSV. Transcriptomic analyses will aid in discovery of biological processes linked to observed phenotypes and correlations to culture medium composition.
Conclusion EpiLife-generated HEEs show the closest morphological resemblance to *in vivo* epidermis. Additional analyses on gene- and protein expression and barrier function are necessary and ongoing to draw final conclusions on best practices. Hereby we will steer our experimental dermatology field towards improved and reproducible epidermal models.

20 – JOLIENE WICHERS SCHREUR

DISSECTING THE IMPACT OF CTCL T-CELL LINES ON EPIDERMAL STRUCTURE AND FUNCTION

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Background Cutaneous T-cell lymphomas (CTCL) are a group of rare malignancies characterized by the presence of malignant T-cells in chronically inflamed skin lesions. In early stages, CTCL lesions often mimic benign inflammatory dermatoses. As the disease progresses, skin lesions evolve in tumors and/or generalized erythroderma. The pathogenesis and pathophysiology of CTCL is not fully understood. In particular, little is known about how the altered cytokine milieu in CTCL affects the epidermal compartment. This knowledge gap hampers the development of effective, targeted therapies and limits our ability to stratify patients based on disease behavior.
Objective This study aims to unravel the effect of cytokines secreted by malignant CTCL T-cell lines on epidermal morphogenesis, architecture, and barrier function, with the goal of clarifying their role in disease progression and skin pathology.
Methods 2D cell cultures and 3D human skin equivalents (HSEs) were generated and exposed to varying concentrations of conditioned media from malignant CTCL T-cell lines (HH, MyLa, and SeAx) during the final 72 hours before harvesting. Epidermal morphogenesis, structure and function were assessed based on morphology and epidermal markers.
Results Morphological analysis demonstrated that secreted cytokines from CTCL T-cell lines impaired keratinocyte differentiation. The expression of barrier markers was reduced indicating that cytokine exposure altered epidermal stratification and barrier formation.
Conclusion Cytokines from malignant CTCL T-cell lines impair epidermal morphogenesis, structure and function in both 2D keratinocytes cultures and 3D HSEs. Ongoing in-depth analysis aims to further validate the functional consequences of these cytokines and their role in CTCL-associated epidermal pathology.

21 – RENS PETERS

COMBINING EXPERIMENTAL AND AI-DRIVEN APPROACHES FOR DEVELOPING IMMUNOCOMPETENT 3D SKIN MODELS

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Background Common inflammatory skin disease, like atopic dermatitis (AD) are characterized by epidermal barrier disruption and T-helper cell-driven inflammation. Integrating relevant immune subsets into physiologically relevant 3D skin models enables disease-specific immune–epithelial cellular crosstalk, improving disease modeling and therapeutic testing.
Objective We aimed to establish an immunocompetent *in vitro* AD skin model incorporating Th2-polarized T-cells within a collagen-based dermal compartment.
Methods Systematic development of AI prompts, trained and validated on relevant datasets, enabled efficient data extraction by ChatGPT to map existing 2D and 3D immunocompetent skin models. Highlighted common practices and limitations in immune cell integration strategies were utilized to improve cell culture protocols for 2D co-cultures of keratinocytes with fibroblasts or T-cells, and 3D skin models with activated T-cells.
Results Literature revealed inconsistent approaches to Th2-

cell polarization and immune-cell integration, which guided the optimization of our model design. Protocols were optimized to successfully isolate keratinocytes, fibroblasts, and skin-resident T-cells from one single human skin biopsy, enabling donor-matched model construction. T-cells stimulated keratinocytes to secrete CXCL10, CCL2, and CCL20 chemokines in a dose-dependent manner. Activated T-cells induced epidermal differentiation disruption and upregulation of inflammatory markers, reflecting early AD-like pathology. Optimized polarization protocols yielded Th2 cells (IL-4 and GATA3 expression) in >50% of naïve $CD4^+$ T cells, to incorporate in co-culture models.
Conclusion By combining AI-guided literature searches and data extraction, with experimental validation, we established the foundation for immunocompetent 3D skin models incorporating Th2-polarized T cells—an essential step toward physiologically relevant *in vitro* models of atopic dermatitis.

22 – MARIE CHEVALIER FLORQUIN

MAPPING THE SPATIAL IMMUNE LANDSCAPE IN SEZARY SYNDROME: INSIGHTS INTO MODERATE AND PROGRESSIVE PROGNOSES

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Background Sezary syndrome is a rare (<5% of cutaneous T-cell lymphomas) and aggressive malignancy, characterised by erythroderma, lymphadenopathy, and clonally related neoplastic T cells ('Sezary cells') in skin, lymph nodes, and blood. Median survival is 32 months, with 5-year survival of 10–30% [1]. While the extent of peripheral blood involvement may affect prognosis [2], the prognostic significance of the skin tumour microenvironment (TME) remains unclear.
Objective To characterise the spatial TME in Sezary syndrome across patient samples with differing survival to identify immune cell dynamics and potential prognostic markers.
Methods Spatial transcriptomics was performed on 18 FFPE baseline biopsies from patients with long-term (≥5 years, n=4), intermediate-term (2–5 years, n=8), and short-term (≤2 years, n=6) survival, and 4 follow-up samples from patients with short-term survival showing progression under treatment (mogamulizumab, n=3; interferon, n=1). Analyses used the Xenium platform (10x Genomics) with an immune-oncology panel (n=380) and a custom panel (n=95). Cell segmentation was performed with the Xenium Cell Segmentation Kit, and cell type annotation combined reference-based typing [3] with clustering. Neighbourhood analyses will be performed using an in-house computational pipeline.
Results Preliminary spatial profiling of 18 samples suggests variation in immune and stromal cell organisation across prognostic groups, including differences in B-cell and cytotoxic cell levels. These findings are being explored in ongoing analyses to clarify biological and prognostic relevance.
Conclusion Spatial mapping of Sezary syndrome reveals prognostic group–specific variation in the skin TME. Further analyses will determine how these spatial immune patterns relate to disease progression and outcomes.

23 – ANDRYA REDER HOLLATZ

A PREDICTION MODEL FOR A FIRST METACHRONOUS CUTANEOUS SQUAMOUS CELL CARCINOMA: A 10-YEAR NATIONWIDE COHORT STUDY

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Background Following a first cutaneous squamous cell carcinoma (CSCC), one-third of patients develop new primaries, yet individualized absolute-risk estimates to guide follow-up are limited.
Objective To develop and internally validate a competing-risk model predicting the first metachronous CSCC after an index CSCC.
Methods A retrospective nationwide cohort study including patients with a first histologically confirmed CSCC in 2007–2008 (Netherlands Cancer Registry) with up to 10-years of follow-up. We developed a Fine–Gray competing-risk model for the first metachronous CSCC using prespecified predictors (age, sex, hematologic malignancy, basal cell carcinoma (BCC) and actinic keratosis (AK) history, synchronous CSCC, tumor location and differentiation). We evaluated model performance via time-dependent C-index and calibration measurements after 10-fold cross-validation.
Results Among 11,737 patients (median age 76 years; 57% male), 3,288 (28%) developed a metachronous CSCC. Strong predictors included AK history, ≥5 prior BCCs, and history of chronic lymphocytic leukaemia/small lymphocytic leukaemia; male sex, synchronous CSCC, sun-exposed sites, and poorer differentiation were also associated with higher risk. Cross-validated 5-year C-index was 0.64 with good calibration.
Conclusion A competing-risk model using routinely available clinical features provides well-calibrated absolute-risk estimates for metachronous CSCC, supporting future risk-based surveillance research despite modest discrimination.

24 – INGER KREUGER

SPATIAL GENE EXPRESSION AND MICROENVIRONMENTAL CHANGES IN THE TRANSITION OF NEVUS TO MELANOMA

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Background Melanoma is an aggressive skin cancer, which can arise from benign nevi. Enhancing our understanding of melanoma development from nevi could improve early diagnosis and treatment. However, studies of early melanoma stages are limited, and the existing studies were mainly performed using bulk RNA sequencing, involved only limited gene subsets, or lacked spatial context.
Objective Therefore, we have mapped the transition of nevi to melanoma using spatial transcriptomics.
Methods We analyzed nevus-associated melanoma FFPE samples from 18 patients using the 10X Genomics Visium Spatial Gene expression technology. Data analysis was conducted using Spaceranger, Semla, STdeconvolve and various R packages. Additionally, imaging mass cytometry was performed.
Results We identified the main transcriptomic signatures in the skin, as well as nevus an melanoma signatures and their spatial location. Differential gene expression analysis identified potential biomarkers and key pathways in melanoma. The pathways could be broadly classified into three major categories: promoting proliferation under metabolic stress, alterations in differentiation state, and modifications linked to microenvironmental remodelling. Additionally, two melanoma signatures within the same patient with their own spatial location were detected. Microenvironmental analysis further showed shifts in immune cells, with only M2-like macrophages in the nevus, but abundant immune cells around melanoma regions. Simultaneously, melanomas exhibited features associated with an immunosuppressive microenvironment.
Conclusion Spatial analysis revealed gene expression alterations and microenvironmental changes during the transition of nevus to melanoma. This study advances our understanding of melanoma development, thereby providing a framework for the identification of novel biomarkers and treatment targets in melanoma.

25 – ALEX ROOKER

PTCHFLOX/FLOXERT2+/– MOUSE MODEL DRIVES HAIR FOLLICLE NEOPLASMS RATHER THAN BASAL CELL CARCINOMA

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Background Basal-cell carcinoma (BCC) is the most common skin cancer. Primary BCC-risk factors are UV-exposure and aging. Although BCC rarely metastasizes, its high and increasing prevalence creates an enormous healthcare burden. Immunotherapies such as checkpoint inhibitors have recently been introduced for locally advanced BCC but the immune system's natural role in BCC control remains poorly understood.
Objective Here we aim to investigate *in vivo* if BCC can be treated using a vitiligo bystander immune reaction against melanocytes present in BCC.
Methods Using an previously reported BCC model, Ptchflox/floxERT2+/– mice were injected with tamoxifen inducing BCC formation on the ear. During BCC development mice were either treated with monobenzone/imiquimod/CpG therapy (MIC) or vaccinated with TRP-2 peptide to induce vitiligo-like immunity.
Results Sixty days after induction, mice developed swelling and scab-like lesions on the ears, indicative of BCC lesions. Additionally, blood analysis showed that the MIC treated mice had T-cell activation and the vaccinated mice had a TRP2 specific T-cell response. However, histological examination by trained (mouse) pathologists, showed that ear-lesions resembled hair follicle neoplasms rather than fully developed BCC. Additional immunohistochemical staining using discriminative markers CD10, SOX9, Melan-A and EpCam, indeed confirmed these findings.
Conclusion Although we showed that vitiligo-inducing therapies generated a melanocyte specific T-cells response in mice, resulting in fur-depigmentation, our histopathological analysis revealed that the Ptchflox/floxERT2+/– BCC model produces hair follicle neoplasms rather than BCC. These findings emphasize the need for a well-characterized mouse model representative of human BCC to advance research into pathogenesis and immunotherapy of BCC.

26 – VEERLE MERKUS

STAGE-SPECIFIC CHANGES IN THE SPATIAL IMMUNE LANDSCAPE OF MYCOSIS FUNGOIDES

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Background Mycosis Fungoides (MF) is characterized by the proliferation of malignant CD4+ T cells. Disease progress occurs from early stage plaques (IA-IB) to late stage tumors

(IIb) in approximately one-third of cases. While recent research has explored the role of the tumor microenvironment (TME) in MF progression, spatial interactions between cancer cells and surrounding immune cells remain poorly understood.
Objective We aimed to profile the spatial landscape of the MF TME across disease stages, providing insight into immune cell dynamics and potential therapeutic targets.
Methods We performed a high-dimensional analysis of cell composition and interactions across MF stages using a custom Imaging Mass Cytometry (IMC) panel to examine the spatial complexity of the TME. 27 skin biopsies from 20 patients with confirmed classical CD4+ MF (stages IA- IIb) were included.
Results The IMC panel enabled visualization and identification of immune cell subsets, and revealed the cellular composition of MF tumors and plaques. Our findings revealed stage-specific changes, with early-stage plaques enriched in percentage of cytotoxic CD8+ T cells, whereas late-stage tumors exhibited increased B cell infiltration. We identified a shift from CD8+ T cell-cancer cell and monocyte-cancer cell interactions in plaques to B cell-cancer cell interactions in tumors.
Conclusion A stage-dependent shift in cellular interactions from an effective anti-tumor immune responses to features consistent with immune evasion mechanisms is observed during MF progression. These insights underscore the importance of spatial context in understanding MF progression and highlight therapeutic targets that could lead to stage-specific (immuno)therapies, ultimately improving patient outcomes.

27 – JULIETTE SIMONS

PERFORMANCE OF CICLOSPORIN IN OMALIZUMAB-NAÏVE AND OMALIZUMAB-REFRACTORY CHRONIC URTICARIA IN DAILY PRACTICE

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Background Ciclosporin is currently a second-line treatment for chronic urticaria (CU) patients. Effectiveness is mainly investigated in omalizumab-naïve populations.
Objective We aim to investigate the effectiveness and safety of ciclosporin in omalizumab-refractory CU-patients and for comparison in omalizumab-naïve patients, including factors associated with effectiveness.
Methods All CU-patients prescribed ciclosporin in two Dutch tertiary centers were retrospectively included. Response to ciclosporin (based on UCT or physician estimation), treatment duration, reasons for discontinuation, drug survival and potential predictors (Log/Cox regression) were assessed.
Results 166 CU-patients treated with ciclosporin were identified (68.7% female, median age 34 years). Complete/good response to ciclosporin was observed in 48% (n=75)(57.4% omalizumab-refractory patients, 40.9% omalizumab-naïve (p=0.04)). Ciclosporin-omalizumab combination treatment was used in 41% (n=68; median 3.8 months), mostly omalizumab-refractory patients (n=42). In 35% complete/good response (n=27/75) was attributed to combination treatment. Ciclosporin treatment was discontinued in 133 patients (80%). Reasons for discontinuation differed between omalizumab-naïve and omalizumab-refractory patients (p=0.15) respectively: remission 24% vs. 43%, side-effects 27% vs. 14%, ineffectiveness 19% vs. 11%, combination side-effects/ineffectiveness 24% vs. 27%. Ciclosporin drug survival rates due to remission at 0.5, 1 and 2 years differed significantly between omalizumab-naïve and omalizumab-refractory patients: 94%, 83%, 72% versus 91%, 67%, 35%)(p=0.004). Angioedema was associated with complete/good response to ciclosporin (OR 3.1 (1.6-6.2), p=0.001) and a lower risk of discontinuation due to ineffectiveness (HR 0.5 (0.3-0.9), p=0.02).
Conclusion Omalizumab-refractory patients show more often complete/good response and a more favorable drug survival, compared to omalizumab-naïve patients. Side-effects and ineffectiveness are common reasons for discontinuation, especially in omalizumab-naïve patients.

28 – CHARLOTTE VAN RIEL

DOSE REDUCTION OF IL-17 AND IL-23 INHIBITORS IN PATIENTS WITH PLAQUE PSORIASIS IS NON-INFERIOR TO USUAL CARE: AN INTERNATIONAL PRAGMATIC RANDOMIZED CONTROLLED TRIAL – THE BENEBIO STUDY

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Background Dose reduction of biologics for psoriasis may lower costs and prevent overtreatment. Knowledge on dose reduction of the newest biologics, interleukin (IL)17 and IL23 inhibitors (i), is lacking.
Objective This pragmatic, non-inferiority randomized clinical trial evaluates whether dose reduction (DR) by stepwise interval prolongation of IL17i and IL23i in patients with psoriasis with stable low disease activity is non-inferior to usual care (UC).
Methods A total of 244 patients using IL17i/IL23i with stable low disease activity and good quality of life at inclusion, were randomized 2:1 to DR (stepwise interval prolongation to 67% and 50% of standard dose) or UC. Primary outcome: difference in cumulative incidence of persistent flares (PASI>5 for ≥3 months) after 18 months with a 15% non-inferiority margin. Secondary outcomes: proportion of patients with successful DR, course of PASI/DLQI, and safety.
Results At baseline: mean(±SD) age 51(±15) years, 67% male, 46% used IL17i, 54% used IL23i, 47% was biologic naive, and median[IQR] PASI and DLQI were 0.0 ([1.1] and [1.0], respectively). After 18 months, the difference in cumulative incidence of persistent flares for DR was non-inferior to UC (0.62% (95%CI [-5.84%; 4.64%]). Also, 74.5% of patients had a successful DR.

Mean PASI and DLQI scores were very low and did not significantly differ between DR and UC. No safety signals related to DR were detected.
Conclusion Dose reduction of IL17i and IL23i in psoriasis patients with low disease activity was non-inferior to UC and safe. At 18 months, successful DR was reached in ¾ of patients.

29 – SARA VAN DER KAMP

PERFORMANCE OF OMALIZUMAB IN PATIENTS WITH MAST CELL-MEDIATED ANGIOEDEMA

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Background Mast cell–mediated angioedema (AE-MC) is managed according to urticaria guidelines. Evidence on the efficacy and safety of omalizumab has mainly been derived from RCTs including patients with mainly wheals (with/without AE-MC) or small case series.
Objective To investigate long-term real-world performance of omalizumab in patients with isolated or predominantly AE-MC.
Methods In this retrospective multicenter cohort study, 14 international urticaria expertise centers included all AE-MC patients treated with omalizumab (data lock 2022). Treatment response was assessed using UCT and physician assessment. Drug survival by reason for discontinuation (Kaplan–Meier analysis) and predictors of treatment discontinuation (Cox regression) were analysed.
Results Of 148 patients with AE-MC (mean age 45; 74% female) who started omalizumab, 67 (45%) had isolated AE and 81 (55%) AE with subordinary wheals. The majority of all patients (77%, n=90/117) had good/complete response to omalizumab, without significant differences between subgroups. Overall, 60 (41%) patients discontinued, primarily due to well-controlled disease (63%, n=38); less frequent due to ineffectiveness (18%, n=11) or adverse effects (5%, n=3). Drug survival due to well-controlled disease was 82%, 67%, and 58% at 1, 2, and 5 years, respectively, independent of the presence of subordinary wheals. Longer disease duration prior to omalizumab (>2 years) predicted delayed discontinuation (HR 0.32), while fast response predicted earlier discontinuation (HR 2.71) due to well-controlled disease. No predictors for discontinuation due to ineffectiveness were found.
Conclusion Omalizumab is highly effective and safe in mast cell–mediated angioedema, independent from presence of wheals, supporting the management of AE-MC as part of CSU.

30 – IMKE VAN GINKEL

DEVELOPMENT OF A FLUORESCENCE MOLECULAR IMAGING METHOD FOR USTEKINUMAB IN PSORIASIS

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Background Fluorescence molecular imaging of the skin is a novel technique in dermatology, while its value has already been demonstrated in oncology and in gastroenterology. Promising results in molecular imaging of biologics for inflammatory bowel disease suggest similar potential in dermatological conditions.
Objective To develop a robust and reproducible imaging protocol enabling targeted imaging of biologics in psoriasis.

Methods Ustekinumab was conjugated with IRDye800CW under GMP conditions to ensure stability and suitability for clinical use. A custom-built in-house fluorescence camera was developed, and to enable quantitative analysis of the fluorescence signal, a complementary spectroscopy system was constructed. Additionally, a standardized protocol was established for ex vivo analysis of skin biopsies using fluorescence microscopy, allowing detailed visualization of tracer distribution in lesional and non-lesional skin. Subsequently, a clinical trial was initiated to evaluate the feasibility and performance of the complete imaging workflow in patients with psoriasis.
Results Eight imaging procedures have been successfully completed, with no adverse events reported. Preliminary analysis of fluorescence imaging data revealed strong light reflectance from the device on the skin, complicating image interpretation. However, spectroscopy measurements showed a clear increase in fluorescence intensity in lesional skin compared to non-lesional skin. Furthermore, tracer signal detection was successful using fluorescence microscopy.
Conclusion These preliminary data demonstrate the feasibility of detecting ustekinumab-800CW in patients with psoriasis with the developed method. The results highlight the potential of fluorescence molecular imaging as tool for visualizing drug distribution in dermatological conditions with potential implications for guiding personalized therapeutic strategies.

31 – YARA VALKENBURG

DYNAMIC VS. CONVENTIONAL OPTICAL COHERENCE TOMOGRAPHY FOR DIAGNOSING BASAL CELL CARCINOMA: A DIAGNOSTIC COHORT STUDY

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Background Optical coherence tomography (OCT) provides a non-invasive diagnostic alternative to biopsy for diagnosing basal cell carcinoma (BCC). Dynamic OCT (D-OCT), integrated into OCT-devices, visualizes vascular shapes and patterns using speckle-variance.
Objective This diagnostic cohort study evaluated whether D-OCT improves BCC detection and subtyping accuracy compared with OCT alone and which vascular shapes and patterns predict BCC presence and its subtype.
Methods Lesions clinically suspicious for BCC requiring biopsy were scanned using (D-)OCT. Scans were assessed in a paired order alongside clinical photographs; first the conventional scan, subsequently with visualization of vasculature at three standardized depths (150µm, 300µm, 500µm). Diagnostic confidence was assigned on a five-point confidence-scale, the predicted subtype was noted as well as the predominant vascular shape and pattern. Histopathology served as reference test.
Results A total of 321 patients with 424 lesions were included (BCC prevalence of 60.8%). D-OCT assessment resulted in a higher sensitivity for BCC detection compared to OCT (84.9% vs. 72.9%, respectively, p=<0.001), at comparable specificity (94.0% vs. 95.8%, respectively p=0.453). Diagnostic parameters for BCC subtyping were comparable between OCT and D-OCT assessment. Vascular shapes and patterns with either positive or negative associations for BCC detection and subtyping were identified.
Conclusion Using the dynamic functionality for blood vessel examination on OCT improves the sensitivity for BCC detection without compromising specificity. Although both positive and negative associations have been found between vascular shapes and patterns and the presence of BCC and its subtypes, D-OCT does not improve subtype classification accuracy compared to conventional OCT.

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Posters

P1 – MARIONA OLIVER

MACHINE LEARNING-BASED ANALYSIS OF SMARTPHONE IMAGES FOR REMOTE MONITORING OF PSORIASIS

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Background Psoriasis requires long-term monitoring to evaluate treatment effectiveness. Traditional tools such as the Psoriasis Area and Severity Index (PASI) are clinic based, time-consuming, and prone to inter-rater variability. Digital health and artificial intelligence (AI) may overcome these limitations by providing remote, objective, and consistent disease evaluation.
Objective This study evaluated the feasibility and accuracy of AI-assisted smartphone imaging for monitoring psoriatic lesions and treatment response.
Methods In a randomized, double-blinded, placebo-controlled trial, 26 patients with mild-to-moderate plaque psoriasis received guselkumab or placebo. Over a 16-week period, patients captured standardized, colour-calibrated images of target lesions at home via smartphone-based medical imaging platform. Physicians also recorded clinical images and evaluated severity with PASI and the Target Lesion Score (TLS). AI-assisted tissue analysis was applied to all images, and a predictive model combining erythema, scaling, and induration was trained on physician-assessed TLS sub-scores to estimate overall TLS.
Results Baseline comparisons showed no significant differences between clinic and home images for erythema and scaling. Guselkumab significantly reduced PASI and TLS compared to placebo, with AI-derived TLS (aiTLS) scores closely aligned with physician-assessed TLS. From week 8 onward, AI analysis detected significant reductions in erythema and scaling in the guselkumab group, consistent across home and clinic images.
Conclusion AI-assisted smartphone imaging offers a reliable, standardized method for remote assessment of psoriasis. By quantifying erythema, scaling, and lesional size, this approach generates comprehensive lesion scores, potentially reducing the frequency of in-clinic assessments and supporting remote monitoring in both research and clinical care.

P2 – JOEY KARREGAT

INCIDENCE OF TATTOO-ASSOCIATED MELANOMA IN THE NETHERLANDS (1991-2023): A NATIONWIDE REGISTRY STUDY

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Background Tattooing is an increasingly prevalent practice that is associated with various clinical complications. The carcinogenic potential of tattoo pigments remains unclear. While 45 case reports have described melanomas colocalizing with tattoos thus far, a pathogenetic link between tattoos and melanomas remains unproven. No nationwide epidemiological study has investigated the incidence of tattoo-associated melanoma (TAM).
Objective To determine the incidence of TAM in the Netherlands from 1991 to 2023, analyse TAM characteristics and patient demographics, and compare these findings with melanoma data from the general Dutch population during the same period.
Methods Data were obtained from the nationwide network and registry of histo- and cytopathology in the Netherlands (PALGA). Malignant and benign melanocytic lesions on the tattooed skin were included. Patient demographics and mela-

noma characteristics were extracted and analysed. Data from the Netherlands Cancer Registry were used for comparison.
Results From 1991 to 2023, 94 TAMs and 467 benign melanocytic lesions on tattoos were identified. The annual incidence of TAMs has increased over time. TAMs were diagnosed at an overall median age of 48.0 years, predominantly in males (64.9%). The median Breslow thickness was 0.9 mm, and most TAMs were TNM stage I (76.6%). The number-needed-to-excise was 6.0.
Conclusion This nationwide cohort study found no evidence supporting a causal relationship between tattoos and melanoma.

P3 – ELISE BELJAARDS

OPTICAL COHERENCE TOMOGRAPHY FOR NON-INVASIVE PREDICTION OF RESPONSE TO TOPICAL CHLORMETHINE IN EARLY-STAGE MYCOSIS FUNGOIDES

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Background Mycosis fungoides (MF) is the most common cutaneous T-cell lymphoma. Long-term treatment with topical chlormethine is recommended in early-stages, however, some patients fail to respond. Therapeutic response is monitored using invasive biopsies or subjective clinical scores including Composite Assessment of Index Lesion Severity (CAILS).
Objective To evaluate the potential of optical coherence tomography (OCT) as a non-invasive imaging technique to provide an objective, accurate and patient-friendly alternative.
Methods In this open-label interventional study, 21 early-stage MF patients (IA-IIA) and 10 healthy controls were included. Patients applied chlormethine gel for 16 weeks. Clinical scores and OCT imaging of lesional, non-lesional and matched skin sites in controls were performed at weeks -6 and 0 (observational phase) and weeks 4, 8, 12, and 16 (interventional phase). Skin biopsies were obtained at baseline (week 0) and study end (week 16).
Results Lesional skin showed significantly increased epidermal thickness at baseline compared to non-lesional and healthy skin ($p<0.001$), as measured by OCT and histology. Eight patients showed significant reduction in modified CAILS following treatment (Δ-10.5, $p<0.0001$). These responders had significantly thicker epidermis at baseline than non-responders (Δ-83.7 µm, $p<0.05$). OCT measurements correlated significantly with histology (Rrm=0.80, $p<0.001$) and CAILS (Rrm=0.40, $p<0.001$). A subepidermal low echogenic band was observed in seven responders, corresponding to extensive inflammatory infiltrate histologically.
Conclusion OCT reliably measures epidermal thickness and correlates with clinical and histological findings. Our results suggest that epidermal thickness correlates with clinical response to chlormethine, thereby supporting its role as a non-invasive tool for monitoring therapeutic response.

P4 – AGNES GRUTTERS

THE PRICE OF FRAGILE SKIN: A SCOPING REVIEW ON THE ECONOMIC BURDEN OF EPIDERMOLYSIS BULLOSA

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Background Epidermolysis bullosa (EB) comprises a phenotypically and genetically heterogeneous group of rare skin disorders characterised by mucocutaneous fragility. Currently, EB is incurable, and management focuses on wound care. Emerging genetic therapies offer promising avenues for EB management. However, the high cost of these therapies necessitates robust cost evaluations to support their integration into clinical practice.
Objective This scoping review aimed to provide a comprehensive overview of reported costs, financial magnitude and time investment of EB.
Methods A systematic literature search was performed in the databases MEDLINE, Embase, CINAHL, PsycINFO, Scopus, EBSCO interface and Web of Science Core Collection covering the period until February 2025. English full text published articles from any country or study setting on genetic EB and reporting on cost domains were included.
Results Twenty-two studies from 15 countries, published between 2013 and 2024, were included, encompassing 3128 patients. The majority (77%) employed patient or caregiver-completed questionnaires/interviews for data collection. Direct non-healthcare costs were reported in 73% of studies, direct healthcare costs in 68%, and indirect costs in 27%. Financial magnitude was addressed in 82% of studies, while 45% reported time investment. Annual mean total economic burden per patient ranged from €42,323 to €77,008. Average dressing changes requiring two hours daily.
Conclusion Both financial magnitude and time investment of EB care are substantial. We observed heterogeneity in reported cost domains, which complicated drawing definitive conclusions across EB subtypes. Clarifying the current economic burden of EB care is essential to enable future introduction of high-cost genetic therapies.

P5 – JOSEPHINE AMKREUTZ

THE NORMA 1 STUDY: NO RE-EXCISION IN PT1A MELANOMA

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Background Stage I melanoma accounts for the majority of new melanoma cases and shows the steepest increase in incidence. In the Netherlands, approximately 3,000 patients are diagnosed annually with pT1a melanoma. Standard treatment for pT1a melanoma includes primary excision followed by a re-excision with 1cm margins, aiming to eradicate microsatellites and prevent locoregional recurrence (LR). However, microsatellites are rare and there is insufficient evidence that a re-excision improves survival. The NORMA1-study (NO Re-excision in pT1a MelanomA) will evaluate the omission of re-excision in pT1a melanoma.
Objective To demonstrate that the 5-year LR rate (LRR) remains <5% after omitting re-excision in pT1a melanoma.
Methods The NORMA1-study is a large multicenter, single-arm, prospective interventional study to be conducted in the Netherlands. Adult patients with a completely resected pT1a melanoma choose between omitting or undergoing re-excision. They are monitored annually for 5 years. The experimental arm (no re-excision) requires 650 patients.
Results This abstract describes the study protocol and results are not available yet. The primary endpoint is the 5-year LRR in the experimental arm. Secondary endpoints include survival, quality of life (QoL), cost-effectiveness, as well as complication rates and the 5-year LRR after re-excision.
Conclusion The rationale for re-excision in melanoma treatment is increasingly questioned due to limited supporting evidence alongside its morbidity and healthcare costs. This particularly applies to low-stage melanoma patients with an excellent prognosis. Demonstrating that re-excision can be safely omitted could improve QoL and enhance healthcare efficiency, supporting a paradigm shift in the management of low-risk melanoma worldwide.

P6 – WANDONG WANG

SPATIAL TRANSCRIPTOMIC PROFILING OF THE TUMOUR MICROENVIRONMENT OF ORGAN TRANSPLANT–RELATED VERSUS SPORADIC CUTANEOUS SQUAMOUS CELL CARCINOMA

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Background Cutaneous squamous cell carcinoma (cSCC) is the second most common skin cancer worldwide. Organ transplant recipients (OTRs) have a 65–200-fold increased risk of developing cSCC compared with the general population. OTRs-cSCC is often more aggressive, with higher recurrence, metastasis, and disease-specific death rates. The mechanisms underlying this aggressiveness remain poorly understood, particularly regarding the tumour microenvironment (TME).
Objective Previous studies have shown that the TME in OTRs differs from sporadic cSCC in exhibiting immune dysfunction characterized by reduced immune cell infiltration, altered T-cell subset composition, and increased immune exhaustion. This study aims to further delineate tumour–stroma–immune interactions and identify spatially defined molecular features contributing to OTRs-cSCC aggressiveness and potential therapeutic targets.
Methods A tissue microarray (TMA) was constructed from formalin-fixed paraffin-embedded (FFPE) tumour samples of metastatic OTRs-cSCC (n=8) and sporadic cSCC (n=8). Tumour sites were matched as closely as possible (OTRs-cSCC: extremities 3/8, head and neck 5/8; sporadic cSCC: extremities 8/8). Sections were analysed using the GeoMx Digital Spatial Profiler, with morphology markers (PanCK, CD45, SYTO13) guiding region-of-interest (ROI) selection in the TME. Spatial transcriptomic profiling was performed using the GeoMx Human Whole Transcriptome Atlas, and sequencing data are currently being processed.
Results Preliminary analyses are expected to reveal differential immune and stromal compositions and distinct gene expression signatures between OTRs-cSCC and sporadic cSCC.
Conclusion We will present our (preliminary) data on the TME of OTRs-cSCC versus sporadic cSCC, and hope hereby to reveal mechanisms of tumour aggressiveness and inform future biomarkers and therapeutic development.

P7 – MARIE-ELINE DEBEUF

BIOLOGICAL CHANGES OF THE SKIN AFTER ABLATIVE LASER THERAPY – A SCOPING REVIEW

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Background Ablative laser therapy is a widely used interventional tool in dermatology, primarily for skin rejuvenation and scar treatment. The choice between fractional or fully abla-

tive modes is determined by specific indications and patient characteristics. Although research into the clinical effects is growing, the biomolecular mechanisms behind these different laser modes are not fully understood.
Objective To provide a comprehensive overview of the biomolecular changes induced by (fractional) ablative laser therapy on skin.
Methods A literature search was conducted on Pubmed, Embase and Web of Science covering studies from inception until September 2025. Studies focusing on changes in gene and protein expression after ablative laser therapy (10 600 nm CO2 laser, 2940 nm Er:YAG laser, 2790 nm Er:YSGG laser) were included.
Results Twenty studies were included, most focusing on skin rejuvenation. Both fractional and ablative lasers resulted in an inflammatory phase followed by dermal remodelling and neocollagenesis, as characterised by increases in metalloproteinases and collagen. High collagen levels persisted for up to 6 months. Decreases in elastin and increases in tropoelastin indicated a breakdown of old elastic fibres, followed by new elastogenesis, which was more pronounced after full ablation. Superficial Er:YAG micro-ablation, preserving the basal membrane, showed similar biomolecular changes compared to full ablation of the epidermis and superficial dermal layer.
Conclusion Both fractional and ablative laser induced dermal remodelling and neocollagenesis, while elastogenesis was more evident after full ablation. Microablation provided similar results compared to full ablation, highlighting the need for further research in this area.

P8 – KENESHKA ATASH

LEBRIKIZUMAB IN MULTI-THERAPY-REFRACTORY ATOPIC DERMATITIS PATIENTS: A CASE SERIES FROM THE BIODAY REGISTRY

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Background Lebrikizumab, a new interleukin-13 inhibitor, has demonstrated efficacy and overall favourable safety in phase-III clinical trials for atopic dermatitis (AD). However, real-world evidence (RWE) regarding lebrikizumab, particularly in multi-therapy-refractory patients is scarce.
Objective To evaluate real-world effectiveness and safety of 28-weeks lebrikizumab in moderate-to-severe AD refractory to multiple conventional/advanced systemic therapies.
Methods Within the BioDay-registry, a prospective case-series analyzed 28-week lebrikizumab outcomes in AD patients from an early access program, who had failed multiple conventional/advanced therapies. Eczema Area and Severity Index (EASI), Numeric Rating Scale (NRS) itch, pain and sleep deprivation, were assessed at baseline, week 4, 16 and 28 using a linear mixed model. Additional clinical outcomes, laboratory parameters, and adverse events (AEs) were analyzed descriptively.
Results Thirteen patients were included. Twelve patients (92.3%) had received prior conventional systemic therapy. All had previously been treated with biologics and/or oral JAK-inhibitors; 8 patients (61.5%) had used ≥ 3 such agents. At week 28, lebrikizumab resulted in a statistically significant mean reduction in EASI from 13.2 (95% CI: 10.3 – 16.2) to 8.4 (95% CI: 5.3 – 11.5); and in NRS itch from 6.7 (95% CI: 5.6 – 7.7) to 5.1 (95% CI: 4.0 – 6.2). In 12 patients (92.3%) 28 AEs occurred, mostly mild and moderate; most commonly ocular surface disease (n=7), myalgia/arthralgia (n=4) and transient eosinophilia (n=4).
Conclusion Lebrikizumab achieved modest yet clinically meaningful improvement in multi-therapy-refractory AD patients with overall tolerable safety. Larger RWE studies are needed to confirm long-term effectiveness and safety of lebrikizumab in broader AD patient populations.

P9 – NIENKE VELDHUIS

DUPILUMAB-ASSOCIATED OCULAR SURFACE DISEASE IN ATOPIC DERMATITIS: RESULTS FROM A LARGE PROSPECTIVE REAL-WORLD COHORT

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Background Dupilumab-associated ocular surface disease (DAOSD) is a frequently reported side effect in atopic dermatitis (AD) patients treated with dupilumab.
Objective To investigate the frequency and severity of DAOSD, and the effect of dupilumab on conjunctival goblet cells (GCs) in a large prospective real-world cohort.
Methods This prospective study included moderate-to-severe AD patients treated with dupilumab between February 2020 and January 2025 at the UMC Utrecht. Ophthalmological and dermatological examinations were performed at baseline (start of dupilumab), week 4, and week 28. Ocular surface disease (OSD) severity was assessed using the Utrecht Ophthalmic Inflammatory and Allergic disease (UTOPIA) score. DAOSD was defined as a ≥3-point increase from baseline. Conjunctival impression cytology was performed to study the quantity and function of conjunctival GCs.
Results OSD was present in 94.0% (n=141/150) of patients at baseline, while only 60.0% (n=90/150) reported ocular symptoms. During 28 weeks of dupilumab treatment, 30.7% (n=46/150) of patients developed DAOSD. At week 4 and week

28, 56.7% (n=85/150) and 64.7% (n=97/150) of patients regularly used ophthalmic medication, respectively. GC numbers remained stable between baseline and week 28, while Mucin 5AC (MUC5AC) production in Cytokeratin 19-CD45-MUC5AC+ cells significantly decreased.

Conclusion This study highlights the high prevalence of OSD in moderate-to-severe AD patients before dupilumab treatment. DAOSD was observed in 30.7% of patients, despite the potential protective effect of ophthalmic treatment. While conjunctival GC numbers remain stable but low, dupilumab seems to impair GC function.

P10 – KIM DANIËLLE VAN DER GOUW

IMPROVING THE EFFICIENCY OF ANTISENSE OLIGONUCLEOTIDE-MEDIATED EXON SKIPPING OF COL7A1 TO TREAT RECESSIVE DYSTROPHIC EPIDERMOLYSIS BULLOSA

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Background Recessive dystrophic epidermolysis bullosa (RDEB) is a severe blistering disease caused by mutations in the COL7A1 gene. This gene is mainly expressed by keratinocytes and encodes type VII collagen (C7), a protein that is essential for attaching the epidermis to the dermis. Previously, we demonstrated that antisense oligonucleotides (ASOs) can induce skipping of exon 105 of COL7A1 and restore C7 production in cultured keratinocytes from patients and in patient skin grafts on the backs of mice. However, exon skipping efficiency was only 2.5-5% and this might be too low for clinical benefit.

Objective To increase the efficiency of ASO-mediated exon skipping of COL7A1, by (1) further optimizing the sequence and chemistry of the ASO and (2) improving the delivery of the ASO to keratinocytes.

Methods We will transfect cultured keratinocytes with more than 100 tiled ASOs spanning exon 105 of COL7A1 to assess which ASO induces most exon skipping. We will also compare the efficiency of ASOs with different chemistries. Then, we will use the optimized ASO to explore targeted and non-targeted conjugation approaches. We will evaluate if, and to what extent, these approaches improve ASO delivery to keratinocytes and lead to more efficient exon skipping. Experiments will be performed *in vitro* (in HaCaT cells, primary keratinocytes and 3D human skin equivalents), *ex vivo* (in an intact human skin model) and ultimately *in vivo*.

Conclusion This study will support the development of ASO-mediated exon skipping of COL7A1 as a feasible treatment option for patients with RDEB.

P11 – WOUTER OUWERKERK

BIOMARKER-BASED DIAGNOSIS OF CONTACT DERMATITIS: A STEP TOWARDS MORE ACCURATE AND PATIENT-FRIENDLY TESTING

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Background Contact dermatitis (CD) is a highly prevalent inflammatory skin disorder, with two main types; irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD). Overlapping clinical features make subtypes difficult to distinguish. Currently, the gold-standard for distinguishing ICD from ACD is an epicutaneous patch test. Patch tests are a burden on patients, a positive test does not necessarily implicate the allergen and there is a high risk of false-negative results. There is an unmet need for more accurate, objective and patient-friendly diagnostics tools to rapidly distinguish between ICD and ACD.

Objective To develop a prediction tool to discriminate between ACD and ICD in both patch test induced reactions as well as in clinical chronic hand dermatitis.

Methods We collected *stratum corneum* tape strips from a positive patch test reaction to allergens (nickel, chromium, methylisothiazolinone), an irritant lesional skin and a control site of 153 patients. A broad panel of 32, including skin barrier and immunological, biomarkers was measured. We developed multiple classifiers using Bayesian and penalized regression and machine learning methods.

Results We were able to discriminate ACD and ICD in patch test data, with mean AUCs between 0.79 and 0.85. AUC was lower in the classifier validating on lesional data (AUC 0.69-0.72). We could not discriminate between different allergens

in patients with ACD (AUC ~0.5). Most important parameters were Cholesterol (Sulf/Glc), NMF, CEACAM-5, TRAIL, and Amphiregulin.
Conclusion Patch-induced ACD and ICD identified strong discriminators, particularly barrier-related biomarkers. When these patch-derived classifiers were applied to chronic hand-dermatitis, performance decreased.

P12 – LINDA GODDING

EXPERIENCES OF PATIENTS WITH GENERALISED PUSTULAR PSORIASIS: A QUALITATIVE STUDY

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Background The evolving insights and changing treatment landscape of the severe, chronic inflammatory skin disease generalized pustular psoriasis (GPP) increases the need for a deeper understanding of its disease burden and patients' preferences.
Objective To explore the disease burden and impact of GPP, in both the acute and chronic phase of the disease.
Methods A cross-sectional qualitative study was performed consisting of semi-structured interviews. Patients were recruited through the Dutch Psoriasis Patient Federation and at the outpatient clinic of the Radboud University Medical Centre. Interviews were audio-recorded and transcribed verbatim. Interviews were analysed by inductive thematic analysis using ATLAS.ti software.
Results A total of 10 patients were interviewed. Mean duration of interviews was 51 minutes. Patients had a mean age of 61 years (range 32-75) and a median disease duration of 6 years at the time of interview. Most patients were treated with a combination of topical and systemic treatments (90%), of which 56% with acitretin. Four patients had received spesolimab, a novel interleukin-36 targeting biologic. Based on preliminary results, GPP has a large impact on patients' lives and their social environment. GPP disease flares were reported as extremely distressing events, emphasizing the importance of controlling upcoming flares. The positive experiences of patients who were treated with the newest biologic spesolimab may result in a brighter future perspective for patients with GPP.
Conclusion At the 2026 NVED meeting, final emerging themes resulting from the interviews will be presented.

P13 – BEATRIZ OLIVEIRA FAGUNDES

T CELL EXHAUSTION IN CHRONIC INFLAMMATORY DISEASES – A SCOPING REVIEW

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Background T cell exhaustion, a dysfunction driven by chronic antigen exposure, is well established in persistent infections and cancer but underexplored in chronic inflammatory diseases. Clarifying how persistent immune activation drives T cell exhaustion may reveal therapeutic strategies. We hypothesize that persistent inflammation promotes exhaustion, contributing to chronic disease sustainment.
Objective To systematically map and summarize the available literature on phenotypic characteristics and functional aspects of exhausted T cells in chronic inflammatory diseases.
Methods A scoping review is being conducted in accordance to JBI methodology and PRISMA-ScR guidelines. PubMed, Embase, Web of Science and Scopus were searched in October 2025 for studies. Title, abstracts, and full texts will be screened independently by two reviewers using JBI SUMARI tool, capturing study characteristics, disease context, biomarkers, mechanisms, and therapeutic interventions. Extracted data will be analyzed descriptively and summarized in tabular and narrative form.
Results Preliminary screening suggests variability in how T cell exhaustion is defined across studies. Common markers include PD-1, TIM-3, CTLA-4, and LAG-3, yet interpretation and defining criteria vary widely. Reports in chronic inflammatory diseases show reduced proliferation and altered cytokine profiles. However, associations with disease severity, prognosis, or treatment response remain inconsistent.
Conclusion This scoping review will provide a comprehensive mapping of T cell exhaustion in autoimmune and chronic inflammatory diseases. By synthesizing evidence on mechanisms, biomarkers, and therapeutic implications, it aims to clarify conceptual boundaries, highlight underexplored conditions, and identify key knowledge gaps. These findings are expected to guide future research and inform the development of targeted immunotherapies.

P14 – FENNA DE BIE

THE EFFECT OF ANTHRACYCLINE TREATMENT ON PRIMARY CUTANEOUS T-CELL LYMPHOMA CELLS

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Background Cutaneous T-cell Lymphomas (CTCL) are a rare group of extra-nodal mature T-cell-derived lymphomas originating in the skin of which mycosis fungoides (MF) and Sézary syndrome (SS) are the most studied types. Several treatment modalities are available for early stage disease,

however more advanced disease is difficult to treat and requires new therapeutic options. Anthracyclines are widely used in the treatment of various hematologic malignancies and solid tumors. Anthracyclines can cause severe side effects in patients, such as dose-dependent irreversible cardiotoxicity. Aclarubicin (Acla) is an anthracycline that has no cardiotoxic side effects.

Objective Evaluate the cellular toxicity and anti-tumor effects of various anthracyclines on CTCL cell lines and ex vivo CD4+ T-cells derived from CTCL patients.

Methods From 5 CTCL patients of the LUMC Dermatology out-patient clinic peripheral blood was collected, CD4+ T cells were isolated and treated with 4 anthracyclines. Relative cell survival was evaluated by CellTiter-Blue assay and the components of healthy and tumor cells were assessed using spectral flow cytometry.

Results The range IC50 of Acla is 1.0-4.9 µM, of other anthracyclines 3.1-33.6 µM. Flow cytometry analysis revealed that both normal and malignant cells exhibited comparable levels of cell death at equivalent concentrations of anthracycline treatment.

Conclusion Aclarubicin has therapeutic activity against CTCL tumor cells with therapeutic efficacy at lower concentrations than other anthracyclines. These findings suggest that Acla could be an effective treatment in CTCL patients. Given the absence of cardiotoxic side effects of Acla, this treatment warrants further investigation for CTCL patients.

P15 – MYRTHE MOERMANS

UNMET CARE NEEDS AND EXPERIENCES OF PATIENTS WITH BASAL CELL NEVUS SYNDROME AND THEIR PARENTS: A QUALITATIVE INTERVIEW STUDY

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Background Basal cell nevus syndrome (BCNS) is a rare genetic disease, with a wide variety of clinical presentations, such as multiple basal cell carcinomas, keratocysts and other extracutaneous manifestations. The nature of the condition requires a rigorous and frequent trajectory of hospital visits and procedures, resulting in a substantial burden on quality of life (QoL).

Objective The objective of this study is to in-depth explore the impact of BCNS on the biological, psychological, and social domains of QoL in patients with BCNS and their parents.

Methods After purposive sampling, semi-structured, individual qualitative interviews were conducted. The interviews were recorded and transcribed verbatim. Interview data were coded and analysed using thematic content analysis in ATLAS.ti version 9.0.

Results Ten patients with BCNS and six parents of children with BCNS were interviewed. Thematic content analysis revealed five major themes related to the impact of BCNS on their QoL. These included: (1) daily impact and physical complaints, (2) experiences with hospital care, (3) being the parent of a child with BCNS, (4) impact on psychological well-being, and (5) impact on social network and relationships.

Conclusion BCNS affects all domains of QoL. However, the degree of impact is highly patient-dependent. Therefore, patient-tailored care should be pursued. Areas for future improvement include creating more disease awareness in the medical and general community, early confirmation of the diagnosis, and attention to psychological and genetic counselling. Patients may benefit from a patient organisation advocating their needs and from a combined patient and physician score to gain more insight into the disease burden.

P16 – JULIA STANKIEWICZ

INVESTIGATING IMMUNOHISTOCHEMICAL MARKERS IN 101 EARLY-STAGE MYCOSIS FUNGOIDES PATIENTS: A RETROSPECTIVE STUDY ON DISEASE PROGRESSION AND PROGNOSTIC FACTORS

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Background Mycosis fungoides (MF) is the most common subtype of cutaneous T-cell lymphomas (CTCLs). Most patients present with patches and plaques (stage Ia/b) and follow an indolent disease course. However, 20-30% of these patients progress to tumor stage (stage IIb) with a 5-year survival rate around 50%. The prognostic value of immunohistochemical markers in early-stage MF remains unclear.

Objective To identify immunohistochemical markers associated with disease progression and poor survival in early-stage MF patients.

Methods Clinical and immunohistochemical data from diagnostic skin biopsies in 101 early-stage MF patients were retrospectively analyzed. Patients were classified as having stable MF (sMF, n=50) or progressive MF (pMF, n=51) based on progression to tumor stage disease during a minimal follow-up of 12 months. Expression of CD4, CD8, CD3, CD5, CD56, TIA-1 and Granzyme B (Gr B) on the tumor cells was retrieved from files in both groups and correlated with disease specific survival (DSS), overall survival and progression free survival (PFS) using univariate and multivariate analysis.

Results Median follow-up time was 121 months. TIA-1 expression was significantly higher in the sMF group and independently associated with improved DSS (HR 0.3, p=.015). Gr B expression correlated with prolonged PFS but did not retain significance in multivariate analysis.

Conclusion Our results suggest that loss of TIA-1 in tumor cells is an independent predictor of worse DSS in early-stage

MF, suggesting its potential as a prognostic marker. Further research is warranted to confirm these findings and to develop a prognostic model integrating immunophenotypic markers and clinical data.

P17 – LINDI KORPELSHOEK

INCIDENCE AND OUTCOME OF OTHER PRIMARY MALIGNANCIES IN 273 PATIENTS WITH PRIMARY CUTANEOUS MARGINAL ZONE LYMPHOMA

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Background Primary cutaneous marginal zone lymphoma (PCMZL) is a low-grade B-cell lymphoma with an excellent prognosis. The incidence of other primary malignancies has been investigated in other types of cutaneous lymphoma, but not yet in PCMZL.
Objective This study investigates the incidence of other primary malignancies in PCMZL patients relative to the general population.
Methods We performed a retrospective cohort study including 273 patients diagnosed with PCMZL between 2000 and 2024. Lifetime pathology reports were retrieved from the nationwide Dutch pathology registry and screened for other primary malignancies, either before or after PCMZL diagnosis. Observed rates were compared with population-based expected rates from the Dutch National Cancer Registry. Systemic dissemination or transformation of PCMZL were not classified as OPM.
Results The median observation period was 57.3 years before and 10.3 years after PCMZL diagnosis. Incidence rates were significantly increased for basal cell carcinoma (SIR 1.9, 95%-CI 1.3-2.7) and squamous cell carcinoma (SIR 3.36, 95%-CI 1.4-6.9) before PCMZL diagnosis. After PCMZL diagnosis, significantly increased rates were found for basal cell carcinoma (SIR 3.7, 95%-CI 5.0), squamous cell carcinoma (SIR 3.4, 95%-CI 1.5-6.4), melanoma (SIR 6.1, 95%-CI 2.5-12.6), haematological malignancies (SIR 8.1, 95%-CI 4.9-12.5) and endocrine malignancies (SIR 13.7, 95%-CI 1.7-49.5). Notably, haematological malignancies, both low- and high-grade, predominantly occurred post-diagnosis.
Conclusion This study demonstrates that app. 40% of PCMZL patients is diagnosed with other primary malignancies, with an increased risk of cutaneous, haematological and endocrine malignancies compared with the general Dutch population. Clinicians should be aware of this risk.

P18 – ANNA PATSEA

HEALTH-RELATED QUALITY OF LIFE IN PATIENTS WITH BASAL CELL NEVUS SYNDROME AND HIGH-FREQUENCY BASAL CELL CARCINOMA: A QUESTIONNAIRE STUDY

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Background Patients with multiple Basal cell carcinomas (BCCs) require frequent interventions, which impact quality of life (QoL). Multiple BCCs develop from a genetic disease like basal cell nevus syndrome (BCNS) or without known genetic predisposition (high-frequency BCC, HF-BCC). Data on QoL in these patients and the influence of number of BCCs on QoL remain limited.
Objective To evaluate QoL in BCNS and HF-BCC and the impact of BCC burden on QoL.
Methods BCNS patients were recruited through the Maastricht University Medical Centre (MUMC+) and patient/professional associations. HF-BCC patients (≥9 BCCs in 3 years or ≥6 in 10 years) were recruited regionally. QoL was assessed with Skindex-29.
Results 75 BCNS and 59 HF-BCC patients were included. BCNS patients were younger (median 48 vs.74 years), female (61% vs. 34%) and had more BCCs (48% vs. 1% with >100 BCCs), compared to HF-BCC patients. BCNS patients reported more QoL impairment across emotions, functioning and total scores. The mean emotions score (32.2) exceeded the clinically relevant threshold (24). After adjusting for age and sex, the total score remained higher in BCNS, with a between-group difference of 10.0 (95%CI 2.5-17.5). Within BCNS, BCC burden was negatively associated with QoL, a trend absent in HF-BCC.
Conclusions BCNS patients experience greater QoL impairment than other patients with multiple BCCs, particularly in emotional and functional domains. A higher BCC burden further impacts QoL in BCNS, but additional challenges in this group may further impact their psychosocial well-being. These disease-related challenges should be addressed during consultations to improve patient support.

P19 – ANASTASIIA MYRONENKO

TO WHAT EXTENT DOES BEHAVIORAL IMMUNE ACTIVATION INFLUENCE ITCH CONTAGION AND PUBLIC STIGMATIZATION OF PEOPLE WITH PSORIASIS? AN EXPERIMENTAL STUDY

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Background The behavioral immune system (BIS) plays a key role in detecting and avoiding potential sources of pathogens, often triggering behavioral (e.g., avoidance) and emotional (e.g., disgust) responses to disease-relevant cues, facilitating survival. Previous research suggests involvement of BIS in the perception of contagious itch and stigmatization. However, experimental research on the causal role of BIS activation in contagious itch and stigmatization remains limited.
Objective This study examines whether BIS activation, through visual exposure to pathogen-themed information, increases itch contagion and stigmatizing attitudes toward a person with a chronic skin condition.
Methods In a video vignette experiment (target N = 136), all participants first viewed a person with psoriasis discussing a neutral topic, with lesions concealed. Then, participants were randomly assigned to watch a BIS-activating (pathogen-themed) or neutral control video. After this, participants watched a third video showing the same person from the first video, but now with visible lesions, scratching, and describing intense itch experiences. Outcomes included subjective itch ratings and stigma-related attitudes and behavioral avoidance (social distance).
Results Preliminary results (N = 60) show that participants experienced a significant increase in itch sensations and desire for social distance after viewing the third video when compared to the first video. These effects occurred regardless of prior exposure to the pathogen-themed video, indicating no additive effect of pathogen-themed priming.
Conclusion Preliminary results suggest that the combination of visual skin lesions, scratching, and talking about itch elicits both contagious itch and stigmatizing attitudes, without being amplified by pathogen-related threat cues.

P20 – ZIXIAN LIANG

COMPLEMENT FIXATION TEST IN PEMPHIGOID DISEASES: ASSOCIATION WITH IGG1/IGG4 SUBCLASS PROFILES IN A 1-YEAR PROSPECTIVE STUDY

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Background The Complement fixation test (CFT) is an indirect immunofluorescence technique performed on salt-split human skin to detect circulating IgG directed against the basement membrane (BMZ), capable of complement binding. Complement activation contributes to blistering in pemphigoid diseases and depends on IgG subclass, where IgG1 strongly activates complement, whereas IgG4 hardly does.
Objective To assess the association between CFT and IgG subclasses in pemphigoid diseases.
Method In this prospective study, serum samples from suspected pemphigoid patients at UMCG (2023) underwent indirect immunofluorescence on salt-split skin (IIF SSS) for IgG, IgG1, IgG4, and CFT. For CFT, patient serum was incubated on salt-split skin, followed by fresh human serum and fluorescent anti-C3 to detect C3 at the BMZ. Samples that showed IIF SSS-IgG staining intensity of 2+ or 3+ were selected for IgG1 and IgG4 staining.
Results Of 806 tested sera, 113 were strongly positive for IIF SSS-IgG; 61 of these were CFT-positive. In CFT(+) group (n=61), the most common expression pattern was both positive for IgG1 and IgG4 (59.02%, 36/61). However, CFT(-) group (n=52) predominantly showed positivity for only IgG4 (65.38%, 34/52). In CFT(+) group, 57.38% cases exhibited strong IgG1 staining intensity(2+, 3+), while only 1.92% cases (1/52) in the CFT(-) group did($P < 0.0001$). No significant difference in IgG4 staining intensity between the two groups.
Conclusion Complement fixation is strongly associated with IgG1 expression, but not IgG4 subclass. CFT may serve as a functional marker of complement-activating antibody profiles, stratifying patients for complement-targeted therapies, but lacks sensitivity for routine diagnostics.

P21 – OTTE BORGHOUTS

LACK OF CONSENSUS IN REPORTED OUTCOMES FOR EPIDERMAL DIFFERENTIATION DISORDERS: A SCOPING REVIEW

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Background Epidermal differentiation disorders (EDDs, formerly known as inherited ichthyosis) comprise a group of rare heterogeneous genodermatoses affecting quality of life.

Although no cure exists, novel treatments are increasingly being investigated. However, heterogeneity in reported outcomes hampers comparison across studies and slows therapeutic progress. Inconsistent or poorly defined outcomes may also contribute to failure to meet trial endpoints. Well-defined outcomes are therefore essential. The Core Outcome Set (COS) for Epidermal Differentiation Disorders aims to establish a minimum list of outcomes and baseline characteristics that should be measured and reported in EDD research.
Objective This scoping review aimed to identify previously reported baseline characteristics and treatment outcomes used in clinical and observational studies on EDDs, serving as a first step in COS development.
Methods The review followed Joanna Briggs Institute (JBI) and PRISMA-ScR guidelines. Medline, EMBASE, CINAHL, Cochrane Library, and Web of Science were searched. Two reviewers independently performed title/abstract and full-text screening, excluding reports on acquired EDDs or studies with fewer than three patients. Data were extracted using a predefined, pilot-tested form.
Results Eighty publications (49 published articles, 31 protocols) were included, reporting 396 baseline characteristics and 259 outcomes, with some degree of overlap. Outcomes were combined and grouped into 10 domains: demographics, anthropometrics, medical history, pregnancy/perinatal, congenital/genetic, skin, extracutaneous manifestations, life impact, resource use, and adverse events.
Conclusion Substantial heterogeneity exists in reported outcomes for EDDs. These findings form the basis for the subsequent e-Delphi and consensus process to define a COS, enabling improved comparability and advancing therapeutic development.

P22 – MARLEEN DE WINTER

VISUALIZING THE EFFECT OF MOGAMULIZUMAB ON T REGULATORY CELLS IN CUTANEOUS T CELL LYMPHOMA USING A MULTIPLEX IMMUNOFLUORESCENCE IMAGING APPROACH

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Background Cutaneous T-cell lymphomas (CTCL) are characterized by the proliferation of malignant T cells within the skin. In advanced stages, patients are commonly treated with mogamulizumab, a humanized monoclonal antibody that targets CCR4$^+$ cells. CCR4 is expressed on both malignant T cells and regulatory T cells (Tregs). In peripheral blood, mogamulizumab has been shown to effectively deplete CCR4$^+$ malignant T cells as well as Tregs. However, it remains unclear whether mogamulizumab exerts a similar depleting effect on Tregs within the skin.
Objective This study aims to elucidate the effects of mogamulizumab therapy on Tregs and malignant T cells in the skin of CTCL patients.
Methods A multiplex immunofluorescence antibody panel was used combining T-cell markers (CD3, CD5, CD8) with markers relevant to mogamulizumab's mechanism (FoxP3, identifying Tregs, and CCR4, the therapeutic target). Formalin-fixed paraffin-embedded (FFPE) skin biopsies were collected from CTCL patients before and four months after initiation of mogamulizumab treatment. Image analysis was performed to assess changes in CCR4$^+$ and FoxP3$^+$ cell populations within lesional skin.
Results Preliminary analyses indicate a depletion of CCR4$^+$ cells in the skin following mogamulizumab therapy. Further evaluation of specific cell subsets within the tissue is ongoing.
Conclusion These findings suggest that mogamulizumab effectively depletes CCR4$^+$ cells in the skin, supporting its ability to target CCR4+ cells within the tumor microenvironment.

P23 – JULIETTE FARAI BOLLEMEIJER

CHRONIC PRURITUS IN OLDER ADULTS: PREVALENCE, ASSOCIATIONS, AND PRURITUS-SPECIFIC QUALITY OF LIFE

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Background Chronic pruritus (itch lasting ≥6 weeks) is a burdensome condition that frequently affects older adults, yet its epidemiology and impact on health-related quality of life (QoL) in the ageing population remain underexplored.
Objective To examine the prevalence of chronic pruritus, its associated factors, and pruritus-specific QoL in the general ageing population.
Methods We included 4,474 participants (median age 72 years; range 48–99; 58.8% female) from the population-based Rotterdam Study. Questionnaires assessed current, 12-month, and lifetime chronic pruritus, along with ItchyQoL scores. Multivariable logistic regression identified factors associated with chronic pruritus, and linear regression assessed factors linked to pruritus-specific QoL. Principal component analysis (PCA) explored the dimensional structure of the ItchyQoL in this older population.
Results Prevalence of chronic pruritus was 8.6% (current), 10.5% (12-month), and 18.6% (lifetime). Female sex, older age, smoking, atopic dermatitis, psoriasis, self-reported dry skin, asthma, steatotic liver disease, polyneuropathy, depressive

symptoms, anxiety, and poor sleep were associated with higher odds of chronic pruritus. Among those with current chronic pruritus, pruritus-specific QoL was moderately impaired, with greater impairment among participants with atopic dermatitis and psychological symptoms. PCA identified four ItchyQoL dimensions, extending beyond the original three domains.
Conclusion Chronic pruritus is a prevalent, multifactorial condition in older adults, with significant psychological impact and implications for multidisciplinary management.

P24 – MARIELLE VAN DER PEET

INVESTIGATING THE RELATIONSHIP BETWEEN GUSELKUMAB TREATMENT AND SIGNALING LIPID LEVELS IN BLISTER AND PLASMA SAMPLES FROM PSORIASIS PATIENTS

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Background Psoriasis is a chronic inflammatory skin disease characterized by erythematous, scaly plaques caused by excessive keratinocyte proliferation and immune-mediated inflammation. The IL-23/Th17 axis drives disease pathogenesis through cytokines that promote keratinocyte activation. Guselkumab, a selective IL-23 inhibitor, effectively improves Psoriasis Area and Severity Index scores, but its broader molecular effects remain unclear. Metabolomics can be used to reveal integrated systemic and local disease mechanisms.
Objectives To compare local (suction blister fluid) and systemic (plasma) metabolite profiles to identify treatment-responsive pathways and elucidate psoriasis-related metabolic alterations.
Methods In a randomized, double-blind study, 26 psoriasis patients (20 guselkumab, 6 placebo) and healthy volunteers provided plasma and suction blister samples from non-lesional and peri-lesional skin. Samples were collected at baseline, and after 28 and 112 treatment days. A targeted HPLC-MS approach quantified 250 metabolites, including fatty acids, bile acids, oxylipins, endocannabinoids, and lysophospholipids.
Results We identified 114 metabolites in blister fluid and 127 in plasma. Baseline differences between patients and controls were most pronounced in peri-lesional blisters, emphasizing local metabolic dysregulation. After guselkumab treatment, these differences diminished, particularly in peri-lesional blister fluid, suggesting stronger local than systemic effects. Elevated sphingosine species decreased post-treatment, consistent with reduced keratinocyte activation. Oxylipin profiling indicated predominant lipoxygenase (LOX) over cyclooxygenase (COX) metabolism of arachidonic acid, with LOX activity declining after treatment while COX remained impaired.
Conclusion Guselkumab induces distinct local and systemic metabolic shifts, with pronounced normalization in skin. Paired lipidomic profiling offers mechanistic insight and potential biomarkers for monitoring therapeutic response in psoriasis.

P25 – CHEN LIANG

A SERUM PROTEOMICS-BASED COMPARISON BETWEEN BULLOUS PEMPHIGOID AND NONBULLOUS PEMPHIGOID

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Background Bullous pemphigoid (BP) is a common autoimmune subepidermal blistering disease that typically presents with tense bullae and intense pruritus. However, some patients do not develop bullae, a presentation referred to as nonbullous pemphigoid (NBP). The pathogenesis of BP is complex, and it remains unclear why some patients do not develop blisters. It is hypothesized NBP and BP differ in disease mechanism, or reflect a disease phase with differences in intensity of the inflammatory response.
Objective To explore and compare the proteomic profiles in serum of BP and NBP patients with the aim of elucidating their differences and uncovering the mechanisms that drive blister formation.
Methods Serum samples from 27 patients with BP, 29 patients with NBP, and 28 healthy controls (HCs) were analyzed using the multiplex Olink Reveal NGS based proteomics.
Results multiplex proteomics analysis revealed significant differences and trends related to proinflammatory mediators and immune function. In detail analysis will indicate the exact role in disease pathomechanism of BP and NBP.
Conclusion multiplex proteomics may contribute to elucidate potential differences in disease mechanism between the clinical phenotypes of BP and NBP.

P26 – CARIN SMIT

EVALUATING LABORATORY ABNORMALITIES IN ATOPIC DERMATITIS PATIENTS TREATED WITH JAK-INHIBITORS

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Background Real-world evidence on laboratory abnormalities in patients with atopic dermatitis (AD) treated with Janus kinase inhibitors (JAKi) is scarce.
Objective To evaluate the frequency, severity, and clinical impact of laboratory abnormalities in adults with moderate-to-severe AD treated with JAKis in routine clinical practice, providing insight for optimized patient monitoring.
Methods This multi-center observational study included adults with moderate-to-severe AD treated with a JAKi between January 1, 2021, and August 31, 2024, in five Dutch hospitals participating in the BioDay and TREAT NL registries. Laboratory parameters were assessed at baseline and at weeks 4, 16, 28, 40, and 52. The severity of abnormalities was graded according to the Common Terminology Criteria for Adverse Events (CTCAE).
Results 282 adult patients with a total of 404 treatment episodes were included. The most common CTCAE grade ≥2 abnormalities were elevated CPK (9.0%), increased triglycerides (4.3%), and decreased lymphocytes (2.6%). Severe abnormalities (grade ≥3) were rare, observed in 0.35% of all tests (49/13,927), mainly in patients receiving abrocitinib 200 mg, upadacitinib 15 mg, or baricitinib 4 mg. The most frequent severe findings were elevated CPK, increased triglycerides, and lymphopenia. Treatment discontinuation due to abnormalities occurred in two episodes (0.5%), both with upadacitinib 15 mg.
Conclusion This study showed that severe lab abnormalities are rare in AD patients treated with JAKis in daily practice, aligning with phase III trial data. These findings support reconsidering the need for routine laboratory monitoring of parameters with limited clinical relevance.

P27 – ANGELIKI BIRMPILI

RESTORATION OF MOLECULAR PROFILES IN PSORIATIC SKIN FOLLOWING GUSELKUMAB TREATMENT

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Background Psoriasis is a chronic inflammatory skin disease characterized by keratinocyte hyperproliferation and immune dysregulation. Alterations in skin lipid metabolism play a major role in barrier dysfunction and disease pathogenesis. Although Guselkumab, an anti–IL-23 monoclonal antibody, effectively restores clinical and histological features of psoriatic skin, its impact on the skin lipidome remains poorly understood.
Objective This study aims to investigate the molecular effects of Guselkumab treatment on the spatial distribution and composition of lipids in psoriatic skin using mass spectrometry imaging.
Methods Biopsies from psoriatic lesions were collected at baseline and after 16 weeks of Guselkumab therapy. Matrix-assisted laser desorption ionization time-of-flight (MALDI-TOF) MSI was employed to spatially map lipid species across skin layers. Data were analyzed by multivariate statistical analysis to identify treatment-related lipid changes relative to healthy control skin.
Results Guselkumab treatment markedly normalized the lipidome of psoriatic skin, with the vast majority of ceramide species restored toward healthy profiles. Principal component analysis (PCA) revealed a clear shift of post-treatment samples toward the healthy cluster, indicating molecular recovery. Nevertheless, several phospholipid and triglyceride species, particularly within the dermis and hypodermis, remained altered, suggesting incomplete metabolic normalization in deeper skin layers.
Conclusion In conclusion, guselkumab substantially restores psoriatic skin lipid composition, supporting the therapeutic normalization of epidermal lipid metabolism. Persistent lipid alterations in deeper layers may represent biomarkers of residual disease activity or potential targets for adjunctive therapy. MALDI-TOF MSI emerges as a powerful approach for monitoring molecular resolution in psoriasis treatment.

P28 – OLIVIA STEIJLEN

THE DUTCH SQUAMOUS CELL CARCINOMA AND METASTASIS (D-SQUAME) STUDY: TWO NATIONWIDE COHORTS WITH A NESTED CASE-CONTROL DESIGN FOR PROGNOSTIC MODEL DEVELOPMENT AND VALIDATION

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Background Current clinical risk stratification systems are limited in their predictive value for metastasis in cutaneous squamous cell carcinoma. Progress requires large representative datasets integrating clinical, pathological, and molecular data.
Objective To describe a nationwide study design that enables collection of two large and well-defined sets of CSCC samples with long follow-up and sufficient metastatic events to support prognostic research.
Methods Linked data of the Netherlands Cancer Registry and the Dutch Nationwide Pathology Databank were used to collect two nationwide CSCC cohorts and perform nested case–control studies. In the discovery cohort (first CSCC diagnosis 2007-2009), each metastatic case was matched to a non-metastatic control with similar metastatic risk. In the validation cohort (first CSCC diagnosis 2017-2018), each case was matched to both a random and a risk-matched control. Tissue sections were performed for Haematoxylin & Eosin staining, RNA/DNA sequencing, and spatial proteomics.
Results The discovery cohort included 19,120 CSCC patients with ten years of follow-up and 472 samples (236 case-control sets) with a median time to metastasis of 1.1 (IQR 0.5-2.1) years. The validation cohort included 25,921 CSCC patients with at least five years of follow-up and 349 samples (~175 sets with 2 types of controls). These datasets provide the basis for the development of absolute risk prognostic models that combine clinical, pathological, and molecular data, paving the way towards more personalised treatment approaches for CSCC patients.
Conclusion This design enabled large-scale CSCC sample collection with a sufficient number of events, supporting robust prognostic research.

P29 – ELISE LEEMAN

EVALUATION OF DUPILUMAB IN PEMPHIGOID GESTATIONIS WITH PLACENTAL PATHOLOGY AND LITERATURE REVIEW

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Background Pemphigoid gestationis (PG) is a rare autoimmune subepidermal blistering disease of pregnancy. PG likely results from loss of maternal immune tolerance, with antibodies to placental BP180 cross-reacting with maternal cutaneous BP180, driving eosinophil-mediated inflammation and blister formation. Given its Th2-skewed pathogenesis, dupilumab has emerged as a potential therapy.
Objective To report four cases of PG refractory to corticosteroids treated with dupilumab, including placental pathology, and a literature review.

Methods Prospective case series of pregnant women with PG by IgG and/or C3 deposits along the basement membrane zone by direct immunofluorescence and identification of circulating autoantibodies on salt-split skin and by ELISA against NC16A. Dupilumab was administered as a 600 mg loading dose followed by 300 mg biweekly (one patient weekly). Outcomes included clinical response, BP180 titers, placental pathology, and a review of reported PG cases treated with dupilumab.
Results Four women with severe PG were included. Dupilumab led to rapid improvement of itch, cessation of blistering, and corticosteroid tapering (prednisolone discontinued in three; one reduced topical use). Two women delivered healthy infants; two pregnancies are ongoing. BP180 titers increased in three of four patients during treatment and declined after delivery in one of two patients. Placental pathology in one case showed chronic villitis, multifocal histiocytic intervillositis, low-grade fetal vascular malperfusion, and chorangiosis. In total, eleven PG cases treated with dupilumab during pregnancy have been reported to date.
Conclusion Dupilumab appears to be an effective corticosteroid-sparing therapy for refractory PG. Placental abnormalities, possibly reflecting maternal immune activation, warrant further study.

P30 – CLARA HARRS

SPATIALLY RESOLVED WHOLE-TRANSCRIPTOMIC PROFILING OF AGGRESSIVE CUTANEOUS SQUAMOUS CELL CARCINOMA IN EPIDERMOLYSIS BULLOSA

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Background A serious complication in epidermolysis bullosa (EB) is the development of aggressive cutaneous squamous cell carcinoma (EB-cSCC), characterized by a high metastatic risk and poor survival. The biological behaviour of these tumours is more aggressive than conventional UV-induced cSCCs, but the underlying pathogenesis remains unclear. A permissive tumour microenvironment (TME) may play a critical role in driving the development of aggressive EB-cSCCs. Spatial transcriptomics is a novel technique enabling gene expression profiling of specific TME compartments, including immune or stromal cells. This approach may shed light on intrinsic and extrinsic TME processes contributing to EB-cSCC aggressiveness and can lead to the optimization of therapeutic interventions.
Objective Analysing spatial features of the TME in EB-cSCC to unravel the underlying pathogenesis and to identify novel biomarkers and anticancer targets.
Methods Spatial transcriptomics is being performed using the NanoString GeoMx Digital Spatial Profiler on formalin-fixed paraffin-embedded tissues of EB-cSCC (n=8) and UV-induced cSCC (n=8). The TME was segmented into tumour, immune, and stromal compartments based on staining with fluorescently labelled antibodies. These selected regions of interest were UV-illuminated to release oligonucleotide tags from RNA probes hybridized to target transcripts. These tags were collected and next-generation sequencing is currently conducted. Thereafter, data analysis will be performed, enabling spatial gene expression profiling within the defined regions to characterize and compare the TME between EB-cSCC and UV-induced cSCC.
Results/conclusions After finishing data analysis, it is estimated that preliminary results and conclusions of the spatial gene expression profiling can be presented at the NVED meeting.

Nicotinamide voor huidkankerpreventie: resultaten uit een groot cohortonderzoek

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Nicotinamide, een derivaat van vitamine B3, wordt steeds vaker ingezet als chemopreventie bij patiënten met eerdere huidmaligniteiten, maar grootschalige klinische gegevens zijn beperkt. In deze retrospectieve cohortstudie werden gegevens uit de Veterans Affairs Corporate Data Warehouse, een grote elektronische patiëntendatabase van Amerikaanse veteranen, geanalyseerd om de effectiviteit van nicotinamide in het voorkomen van nieuwe huidmaligniteiten te beoordelen.

In totaal werden 33.822 patiënten geïncludeerd met minstens één voorafgaande huidmaligniteit. Van hen gebruikten 12.287 patiënten gedurende minimaal 30 dagen nicotinamide 500 mg tweemaal daags. De gemiddelde leeftijd bedroeg 77 jaar, en vrijwel alle patiënten waren man en van Kaukasische afkomst. De primaire uitkomstmaat was de tijd tot ontwikkeling van een nieuwe huidmaligniteit.

Nicotinamidegebruik was geassocieerd met een 14% lagere incidentie van nieuwe huidmaligniteiten ten opzichte van niet-gebruik, met de grootste risicoreductie voor cutaan plaveiselcelcarcinoom (PCC, 22%). Het beschermende effect was het sterkst wanneer nicotinamide werd gestart na de eerste huidmaligniteit (circa 50% reductie) en nam af naarmate behandeling later werd ingezet. Voor basaalcelcarcinomen werd geen significant risicoverschil gezien in de totale populatie, maar wanneer nicotinamide direct na de eerste of tweede huidmaligniteit werd gestart, was er wel sprake van een duidelijk lagere incidentie. Bij solide-orgaantransplantatiepatiënten werd geen algemeen beschermend effect gevonden, maar bij zeer vroege start trad wel een significante daling van PCC -incidentie op.

De auteurs concluderen dat nicotinamide in de klinische praktijk geassocieerd is met een lagere incidentie van nieuwe huidmaligniteiten, met name PCC, en dat vroege behandeling het meeste voordeel biedt. Zij benadrukken tegelijkertijd de beperkingen van het observationele design en de beperkte generaliseerbaarheid van deze overwegend mannelijke, witte veteranenpopulatie. Prospectieve studies zijn nodig om de effectiviteit van nicotinamide in verschillende patiëntgroepen definitief te beoordelen.

Breglio KF, Knox KM, Hwang J, Weiss R, et al. Nicotinamide for skin cancer chemoprevention. JAMA Dermatol. 2025;17:e253238.

Een klinisch/dermatoscopisch risico scorend model voor vroeg melanoom op de voetzool: de iDscore_plantar

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Plantair vroeg melanoom (pVM) is zeldzaam, maar essentieel om niet te missen. Onder pVM werd melanoom in situ en dun invasief melanoom (stadium Ia/Ib/IIa) op de voetzool geschaard. Deze studie ontwikkelde een risico scorend model voor discriminatie tussen pVM en plantair atypische naevus (pAN).

De studie betreft een samenwerking met de EADV teledermatoscopie taskforce. De klinische gegevens, histologische diagnose, dermatoscopische en klinische beelden van patiënten met pVM (n=98) of pAN (n=392) uit 17 Europese centra werden geïncludeerd. Via voorwaartse stapsgewijze logistische regressie werd een scorend model ontwikkeld. Vervolgens werd het model gevalideerd, getest en vergeleken met patroon diagnose en BRAAFF checklist. Ook ondergingen 104 dermatologen en 56 aios een geblindeerde teledermatoscopische test.

Vier parameters werden gescoord: diameter, leeftijd, lokalisatie en dermatoscopie. Vervolgens werden er risicogroepen ingedeeld. S=0-3: geen risico, advies is follow-up. Er vielen geen pVM in deze groep. S=4-8: laag/medium risico, advies is biopt/korte follow-up. S=9-13: hoog risico, advies is excisie. S=14-17: zeer hoog risico, advies is wijde excisie op korte termijn. Er was slechts 1 pAN met een score van 14.

De laagste score (0) betreft een melanocytaire plantaire laesie ≤8 mm gelokaliseerd op de plantaire boog, zonder asymmetrie van kleur- of structuurpatroon bij een patiënt ≤40 jaar oud. De hoogste score (17) wordt toebedeeld aan melanocytaire plantaire laesies >12 mm gelokaliseerd op de hiel, met een asymmetrisch structuur- en kleurpatroon bij een patiënt ouder dan 50 jaar.

Door het model te volgen is er minder misdiagnose (-26%) en minder onnodige excisie (-28%) van pAN. Tevens een hoger aantal correct gediagnosticeerde pVM (+19%) en pVM waarbij excisie wordt geadviseerd (+9%). Tevens wordt pVM 16% vaker direct geëxcideerd in plaats van eerst gebiopteerd.

Conclusie

Het iDscore_plantar model kan behulpzaam zijn in de risico inschatting en besluitvorming rondom melanocytaire laesies op de voetzool.

Tognetti L, Lo Conte S, Leonardelli S, et al. A clinical-dermoscopic risk scoring model for early melanoma of the soles: The iDScore_plantar. J Eur Acad Dermatol Venereol. 2025;39:1934–1944.