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免疫軸再平衡｜止癢三軸：AD · PN · CSU 的精準照護

Rebalancing the Immune Axis | Three Axes of Itch: Precision Care across AD, PN, and CSU

時　　間：115 年 6 月 28 日(星期日) 08:00~12:25
地　　點：臺北榮民總醫院 致德樓第三會議室

08:00-08:25	***Opening Remarks***	張雲亭教授 Yun-Ting Chang
	座長：陳志強 副教授 (Chih-Chiang Chen)	
08:30-08:55	Lebrikizumab 治療異位性皮膚炎之三年長期療效與安全性：聚焦 IL-13 抑制機轉 Lebrikizumab in Moderate to Severe AD Management: What Matters to Taiwanese Clinicians?	陳奕先醫師 Yi-Hsien Chen
08:55-09:05	***Panel discussion-Q & A***	
	座長：張雲亭 教授 (Yun-Ting Chang)	
09:05-09:30	頭頸部異位性皮膚炎的未被滿足需求：部位特異性免疫異質性與治療意涵 Unmet Needs in Head and Neck Atopic Dermatitis: Site-Specific Immune Heterogeneity and Therapeutic Implications	烏惟新醫師 Wei-Hsin Wu
09:30-09:40	***Panel discussion-Q & A***	
	座長：洪誌聰 副教授 (Chih-Tsung Hung)	
09:40-10:05	Dupilumab 於第二型發炎皮膚疾病的整合治療角色：從異位性皮膚炎到相關共病 One Solution Across Type 2 Inflammation: The Expanding Role of Dupilumab in AD and Beyond	陳俊賓副教授 Chun-Bing Chen
10:05-10:15	***Panel discussion-Q & A***	
10:15-10:30	***Coffee Break***	
	座長：李婉若 教授 (Woan-Ruoh Lee)	
10:30-10:55	異位性皮膚炎治療新進展：從分子機制到臨床應用 Advancing Atopic Dermatitis Management: From Molecular Insights to Clinical Practice	張華景醫師 Hua-Ching Chang
10:55-11:05	***Panel discussion-Q & A***	

座長：蔡雅竹 醫師 (Ya-Chu Tsai)

11:05-11:30	照護不退場，治療再突破　Cosentyx PsA 全面守護 Care Never Quits: Above and Beyond PsA Care with Cosentyx	俞佑醫師 Peter Yu Yu
11:30-11:40	***Panel discussion-Q & A***	

座長：蔡呈芳 教授 (Tsen-Fang Tsai)

11:40-12:05	糖尿病患者的皮膚照護：神經醯胺的角色 Simple Approaches to Managing Skin Problems in Diabetes Mellitus：The Role of Ceramides	卓雍哲醫師 Yung-Tsu Cho
12:05-12:15	***Panel discussion-Q & A***	
12:15-12:25	***Closing Remarks***	陳志強副教授 Chih-Chiang Chen
12:25-	***會後便當***	

Lebrikizumab in moderate to severe AD management: What matters to Taiwanese clinicians?

Lebrikizumab 治療異位性皮膚炎之三年長期療效與安全性：聚焦 IL-13 抑制機轉

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Patients with moderate-to-severe atopic dermatitis (AD) commonly suffer from itch (90.1%), sleep disturbances (49.3%), and anxiety/depression. They experience an average of 9 flares per year, each lasting about 15 days, which significantly impacts their quality of life (DLQI). The HOME organization has established Long-Term Control (LTC) as a core outcome domain for clinical trials, emphasizing it as a multidimensional, patient-centered, holistic assessment. AD is an IL-13-dominant disease; IL-13 levels are elevated in the skin of AD patients across all age groups and serve as the key cytokine driving the pathogenesis of the disease. Lebrikizumab selectively binds to IL-13 with high affinity and is characterized by a slow dissociation rate.

Lebrikizumab: Clinical Efficacy and Long-Term Control

Lebrikizumab is a biologic that selectively inhibits IL-13. In Taiwan, it is approved for the systemic treatment of patients aged 12 and older (weighing ≥ 40 kg) with moderate-to-severe AD who are inadequately controlled by, or intolerant to, topical therapies.

ADvocate 1/2 Monotherapy (Week 16) Results:

- IGA 0/1: 38.1% (vs. 11.7% placebo)
- EASI-75: 55.4% (vs. 17.2% placebo)
- EASI-90: 34.5% (vs. 9.2% placebo)
- Pruritus NRS improvement ≥ 4 points: 42.9% (vs. 12.2% placebo)

Long-Term Maintenance (ADvocate 1/2 → ADjoin):

An Observed Case (OC) analysis of Q4W (every 4 weeks) maintenance dosing showed that clinical responses—including IGA (0/1) and EASI-75/90/100—were sustained through Week 152 (approx. 3 years). Notably, in the Q4W group (N = 99), only 24.2% required any rescue medication, and only 9.1% required systemic rescue therapy. These data support the concept of “Long-Term Stable Control” with a lower treatment burden. The safety profile remained consistent, with most adverse events being mild to moderate in severity.

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Unmet needs in head and neck atopic dermatitis: Site-specific immune heterogeneity and therapeutic implications

頭頸部異位性皮膚炎的未被滿足需求：部位特異性免疫異質性與治療意涵

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Head and neck (H&N) involvement represents a clinically distinct and often underrecognized phenotype of atopic dermatitis (AD), associated with disproportionately high disease burden, visible skin involvement, and significant impact on quality of life. Despite advances in systemic therapies, H&N dermatitis remains a persistent challenge, highlighting a critical unmet need in AD management.

The pathophysiology of H&N-predominant AD is complex, with site-specific immune heterogeneity playing a central role. Increased expression of key cytokines, including IL-4, IL-13, IL-17, and IL-22, is thought to amplify local inflammation. Notably, the development or persistence of H&N dermatitis in patients receiving biologic therapies—particularly IL-4/IL-13 inhibitors—has drawn increasing attention. This observation suggests a potential shift toward alternative inflammatory pathways, underscoring the limitations of targeting a single axis in certain patient populations.

From a therapeutic perspective, management of H&N AD requires a personalized and mechanism-based approach. In patients who experience suboptimal response or de novo head and neck dermatitis during biologic treatment, treatment optimization or switching to alternative systemic therapies may be warranted.

Targeted therapies with broader immunomodulatory effects, such as Janus kinase (JAK) inhibitors, modulating multiple cytokine pathways simultaneously. Targeting multiple cytokines may translate into better disease control in difficult-to-treat areas such as the head and neck, as well as more rapid and consistent symptom relief.

In conclusion, H&N involvement highlights the need for a more refined and patient-centric treatment strategy. Addressing the site-specific immune heterogeneity behind H&N will be essential to improve clinical outcomes.

One solution across type 2 inflammation: The expanding role of Dupilumab in AD and beyond

Dupilumab 於第二型發炎皮膚疾病的整合治療角色：從異位性皮膚炎到相關共病

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Type 2 inflammation represents a common pathophysiological pathway underlying multiple dermatological conditions. Dupilumab (Dupixent), a monoclonal antibody targeting IL-4 receptor alpha, effectively blocks IL-4 and IL-13 signaling—key drivers of type 2 inflammation—offering a unified treatment approach across diverse conditions.

Failure to recognize and address type 2 comorbidities in patients with AD may compromise treatment effectiveness, resulting in persistent pruritus, disease relapse, and diminished long term disease control. For dermatologists, understanding the systemic nature of type 2 inflammation is therefore essential to optimize treatment strategy and improve long term patient outcomes.

In atopic dermatitis (AD), dupilumab demonstrates remarkable efficacy in reducing disease severity, improving quality of life, and providing sustained itch relief. Clinical trials show significant improvements in EASI scores and patient-reported outcomes.

Beyond AD, dupilumab extends to prurigo nodolaris (PN), reducing nodule count and itch intensity for patients with this challenging condition. The therapeutic benefits also apply to chronic spontaneous urticaria (CSU), addressing the shared inflammatory pathways underlying these conditions.

This three-axis approach to itch management—addressing AD, PN, and CSU—represents precision medicine in dermatology. By understanding the shared pathophysiology of type 2 inflammation, we provide targeted care that addresses root causes rather than symptoms.

Dupilumab exemplifies how one solution can address multiple type 2 inflammatory conditions, offering patients comprehensive relief and enhanced quality of life across the type 2 inflammation spectrum.

Advancing atopic dermatitis management: From molecular insights to clinical practice

異位性皮膚炎治療新進展：從分子機制到臨床應用

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Atopic dermatitis (AD) is a heterogeneous and multifactorial inflammatory disease that extends beyond a single immune pathway. While type 2 (Th2) inflammation plays a central role, increasing evidence demonstrates the involvement of additional immune axes, including Th17, Th22, and Th1 signaling, particularly in Asian patients. This complex immune landscape contributes to diverse clinical phenotypes, disease severity, and variable treatment responses. These insights from molecular and translational studies provide a strong biological rationale for broader immunomodulatory approaches. Janus kinase (JAK) inhibitors, by targeting intracellular signaling common to multiple cytokine pathways, can simultaneously modulate Th2-, Th17-, and Th22-driven inflammation contributing to the immunopathology observed in Asian AD. In clinical practice, JAK inhibitors have demonstrated rapid and profound improvements in key disease domains of AD, particularly pruritus control and overall disease severity. Importantly, they address several unmet needs observed with biologic therapies, including residual disease activity, suboptimal responses in certain anatomical areas, and delayed onset of action. Switching from biologics to JAK inhibitors has emerged as a rational strategy for patients with inadequate response, enabling deeper and faster disease control. This presentation will bridge molecular insights with clinical evidence to demonstrate how JAK inhibitors alleviate symptom burden, enhance quality of life, and support long-term disease management of AD.

Care never quits: Above and beyond PsA care with Cosentyx

照護不退場，治療再突破 Cosentyx PsA 全面守護

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Psoriatic arthritis (PsA) is a heterogeneous, immune-mediated disease that extends beyond skin involvement to affect peripheral joints, axial structures, entheses, and multiple organ systems. Ongoing inflammation not only drives pain and disability, but also leads to progressive and often irreversible structural joint damage. Achieving early, sustained, and comprehensive disease control is therefore essential for improving long-term outcomes in patients with PsA.

This symposium reviews current insights into PsA pathophysiology, highlighting interleukin-17A (IL-17A) as a central and unifying driver of inflammation across skin and musculoskeletal domains. IL-17A is produced by diverse innate and adaptive immune cells via both IL-23–dependent and IL-23–independent pathways, positioning it as a key amplifier of chronic inflammation. Its activity links inflammatory burden with joint damage, bone erosion, and pathological new bone formation—hallmark features of both peripheral and axial PsA.

Secukinumab, a fully human monoclonal antibody that selectively targets IL-17A, directly inhibits this core inflammatory pathway. By neutralizing IL-17A regardless of its cellular source, secukinumab provides robust and consistent control of both skin and joint manifestations. Clinical and imaging evidence demonstrates that IL-17A inhibition is associated not only with rapid and durable symptom improvement, but also with favorable effects on structural outcomes, including reduced progression of erosions and enthesophytes.

Aligned with EULAR and GRAPPA recommendations, this session emphasizes a treat-to-target strategy in PsA, underscoring the importance of early recognition, holistic assessment across disease domains, and timely escalation to biologic therapy when appropriate.

To sum up perspectives above, targeting pure IL-17A mechanisum, for example, Secukinumab, goes beyond symptom control—addressing the inflammatory core of PsA to protect joints, preserve structure, and support long-term patient outcomes.

Simple approaches to managing skin problems in diabetes mellitus：The role of ceramides

糖尿病患者的皮膚照護：神經醯胺的角色

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Diabetes mellitus is a common systemic disease that frequently affects the skin, with up to 30–70% of patients experiencing dermatologic manifestations. Among these, xerosis and pruritus are particularly prevalent and can significantly impair quality of life. These skin changes are largely driven by impaired epidermal barrier function, altered lipid composition, and reduced skin hydration, which may also increase the risk of fissures, infections, and delayed wound healing. Early recognition and appropriate management of diabetes-associated skin conditions are essential components of patient care. Basic skincare interventions, including the use of gentle cleansers and effective moisturizers, play a key role in restoring barrier function and alleviating symptoms. Emerging evidence suggests that formulations containing physiological lipids, such as ceramides, may further support barrier repair and improve clinical outcomes. This lecture will provide an overview of common diabetic skin changes, underlying mechanisms, and practical, evidence-based skincare strategies that can be incorporated into routine patient education to improve both skin health and overall well-being.