



ACADEMIA ESPAÑOLA
DE DERMATOLOGÍA
Y VENEREOLOGÍA



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Patrocina:



2025

AEDV Highlights

34ª edición
17-20 sep
PARÍS

Brilla el futuro de *la dermatología*,
donde nace *la luz*



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■ Novedades en fotodermatosis y fotobiología

Oriol Yélamos Pena

Hospital de la Santa Creu i Sant Pau, Barcelona

@dryelamos



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INDIQUE SI TIENE ALGÚN CONFLICTO DE INTERÉS

**NO TENGO CONFLICTOS
DE INTERÉS**



2025

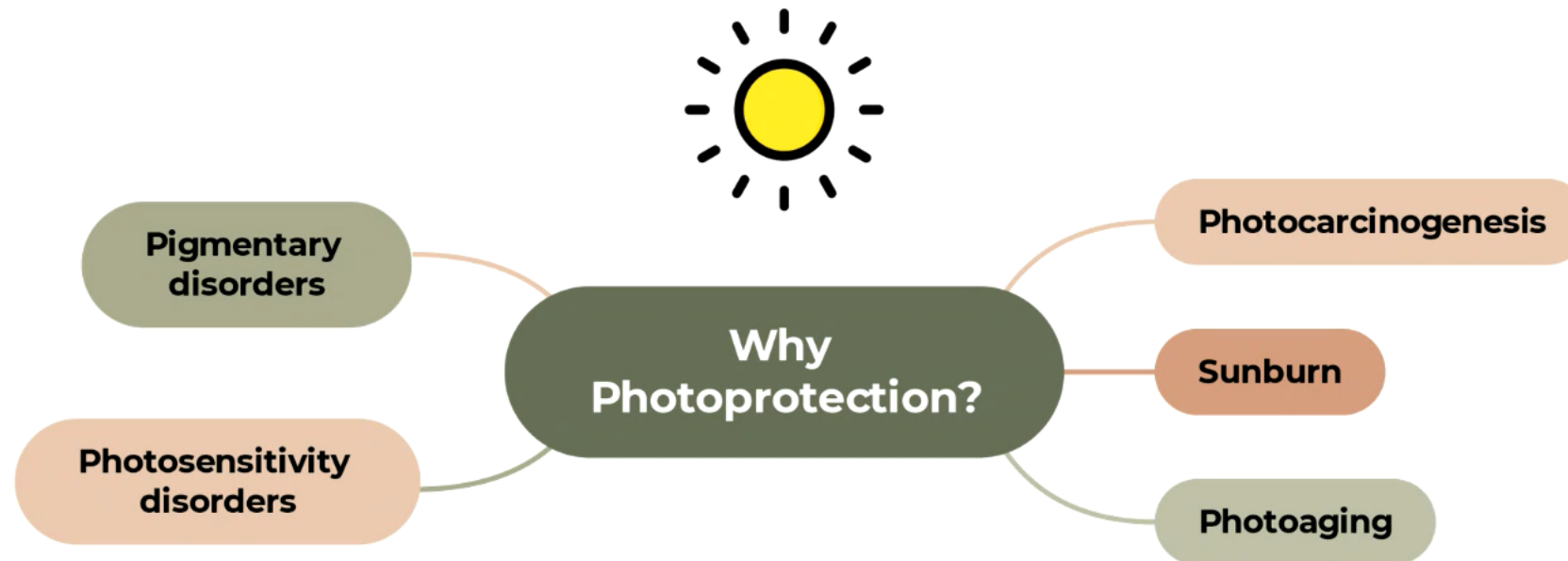
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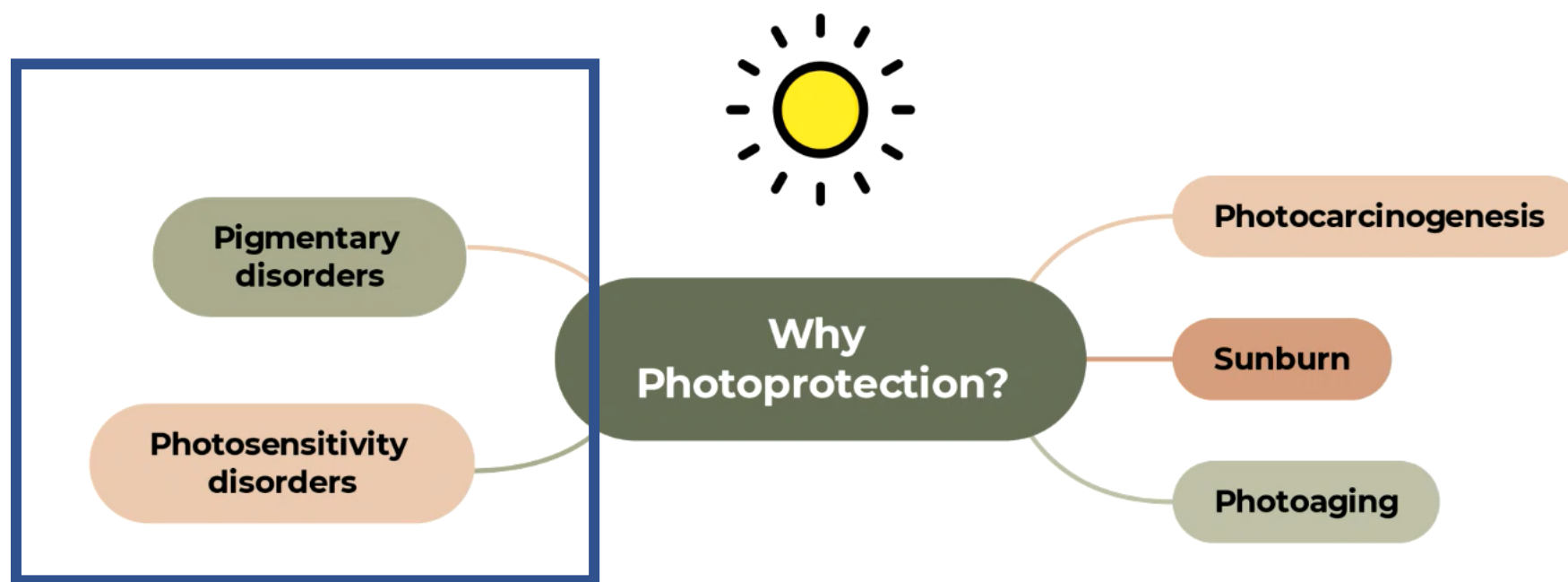
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FOTOPROTECCIÓN







Sunscreens for hyperpigmentation – Is there evidence?



- Dermatologists tend to **recommend sunscreens less often** in darker patients
- **Limited direct clinical evidence** for sunscreen efficacy via RCTs
- Some benefit for **melasma, lentigines, and PIH**
- Current broad spectrum sunscreens do not adequately protect against **UVAI and VIS**

- Dr Henry Lim, Dr Salvador González, Dr Harvey Lui, Dr

Photoprotection according to skin phototype and dermatoses

Fitzpatrick phototype	Description	Individual Typology Angle (ITA)	Skin color (ITA classification)	UVB protection (SPF)	UVA protection (UVA-PF)	High energy visible light protection (VL-PF)
I	Always burns, never tans	ITA° >55°	Very light	SPF50+	UVA-PF +++ (>1/3 labelled SPF)	
II	Burns easily, sometimes tans	41° <ITA° <55°	Light			
III	Sometimes burns, always tans	28° <ITA° <41°	Intermediate			
IV	Rarely burns, tans easily	10° <ITA° <28°	Tan			
V	Rarely burns tans easily; moderately pigmented	-30° <ITA° <10°	Brown			
VI	Rarely burns, tans promptly and intensely; highly pigmented	ITA° <-30°	Dark	SPF30+	UVA-PF +++ (> 2/3 labelled SPF)	VL-PF+++

Passerson et al. J EADV 2021

Lighter skin:
high risk of sunburn,
DNA damage, and skin
cancers
need protection against
UVB



Darker skin:
better natural protection
from UVB
more prone to
hyperpigmentation induced
by
UVA-I & VL



- Piel oscura no tolera el sol por un aumento del calor → ropa oscura

Why don't people with darker skin sunbathe?



- Socioeconomic barriers - \$\$\$
- Cultural factors – *no need to tan*
- Physiologic factors – *increased heat sensitivity with higher skin melanin*

Ono et al, Sci Rep 2017

Why do people fear sunscreens?



- Aversion to “**chemicals**”
- Contact **allergy**
- **Vitamin D** inhibition
- **Hormone disruption** by benzophenone
- **Nanoparticle** absorption
- **Benzene** contamination
- Safety in **children**
- **Environmental** effects

PRECIO

- Novedad mundial: la OMS declara los fotoprotectores una “medicina esencial”

WHO declares Sunscreens as an “Essential Medicine” in 2025



Photo courtesy of Standing Voice and ILDS



**World Health
Organization**

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FOTODERMATOSIS

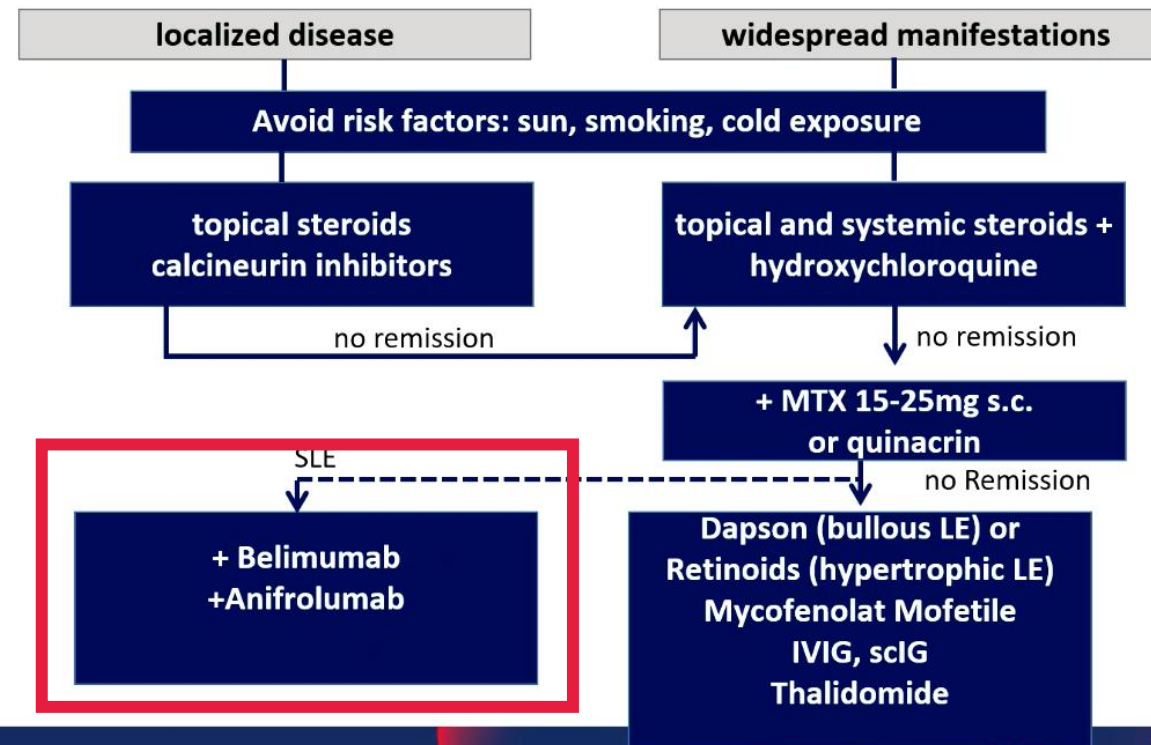


- Hypomelanosis session 19.9.2025 - Dr Picardo
- Hipomelanosis guttata:
 - La hipomelanosis se produce por senescencia del fibroblasto
 - Si el fibroblasto entra en senescencia, hace que el melanocito también lo haga
 - Explica por qué funciona el láser CO2 para la hipomelanosis guttata
- Melanogénesis y consumo de energía
 - Muchas dermatosis cursan con hipomelanosis secundaria
 - Melanogénesis consume mucha energía → si inflamación, se “apaga” melanocito para gastar menos
 - Cuando resuelta la inflamación, se vuelve a encender el melanocito → repigmentación

- Lupus eritematoso
 - Varios nuevos fármacos aprobados para lupus sistémico pero no para lupus cutáneo

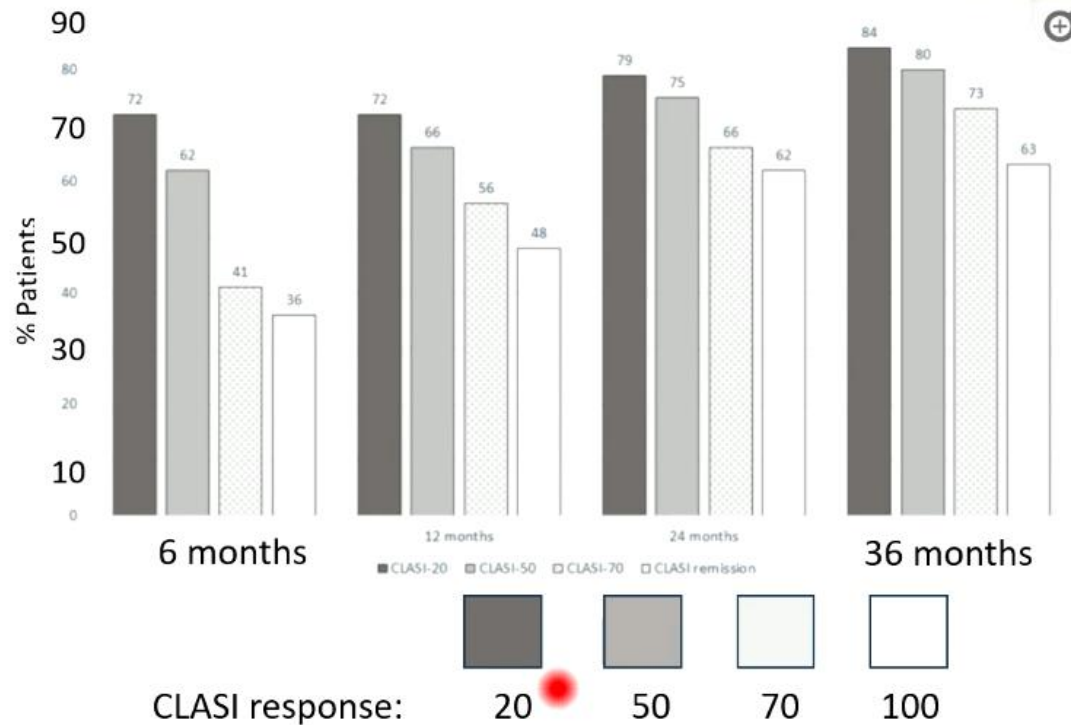
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Therapy of cutaneous lupus



Belimumab and anifrolumab in CLE

BeRLiSS-JS: 466 SLE pat, Italy, 6-36 months



- Nuevos fármacos para LES (no ensayos en formas cutáneas):

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**Novel BAFFR mAb:
lanalumab for SLE**

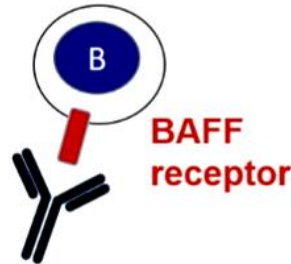
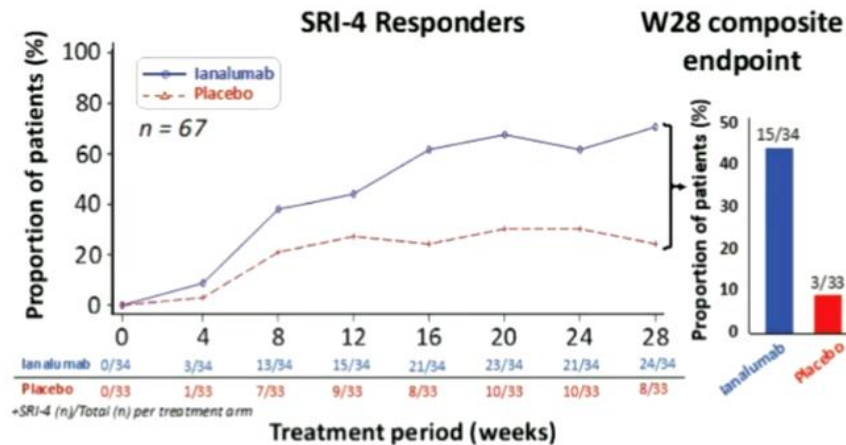


Figure: Proportion of patients with SRI-4 and achieving Week 28 composite endpoint



Recombinant TACI-Ig fusion protein: telitacicept for SLE

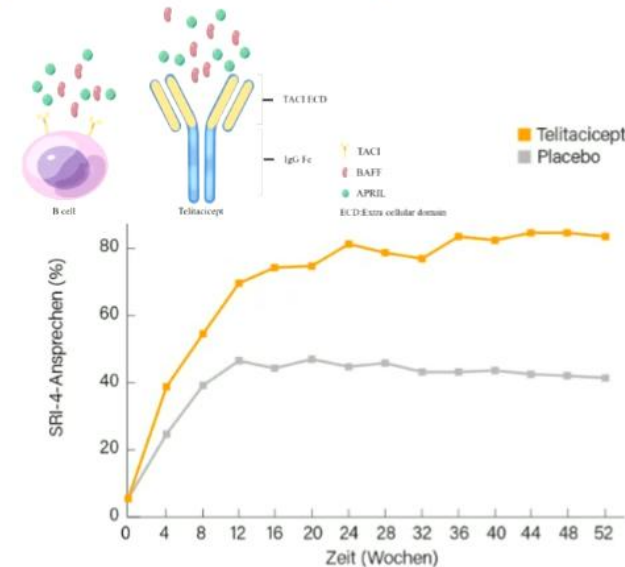


Abb. Phase-III-Studie: SRI-4-Ansprechen auf Telitacicept und Placebo bis Woche 52

Povetacicept: dual BAFF/APRIL inhibitor

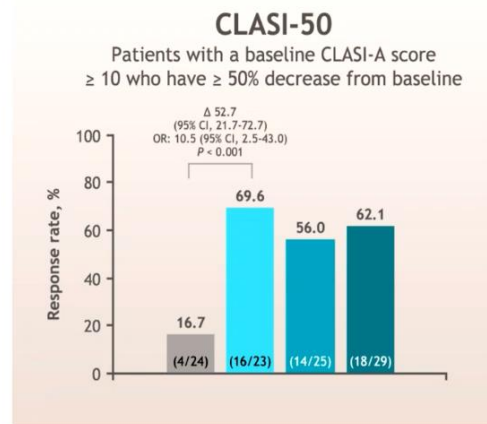
- Nuevos fármacos en investigación para lupus cutáneo:
 - Deucracitinib, a Tyk2 inhibitor seems promising with skin lesions, with improvement in 50% in some series, and there's actually a phase III trial undergoing for CLE.
 - cGAD inhibitors: good news for patients with rare forms of lupus such as familial chilblain lupus in which there is an alteration on the cGAS-STING pathway.
 - TLR7/8 inhibitors: Enpatoran, MHV370
 - IRAK4 inhibitors
 - BDCA2 inhibitors: Litifilimab which inhibits the release of type I IFN in plasmacytic dendritic cells, showing good results in CLE
 - CD19-CAR-T
 - BCMA-CD3 bispecific antibody: teclistimab attacks T cells and plasmacytoid cells
 - Bruton kinase (BTK) inhibitors: orelabrutinib
 - Combination of BTK and JAK inhibitors
 - JAK inhibitors: upadacitinib seems promising too
 - CD40-L: dapirolizumab inhibits T cells but no effects on the skin in a phase 3 trial
 - Gluconolactone cream: it restores the Treg function, very promising

Lupus erythematosus

Chairs: Branka Marinovic, Miloš Nikolić

Deucravacitinib in SLE

Tyk2 Inhibitor: inhibition of IFNAR, IL-12, IL-23



currently: phase III clinical trial in CLE

Morand E et al., Arthritis Rheumatism 2022
PAISLEY_Video_Abstract_v05 (brightcove.net)



Aw K et al., SAGE Open Med Case Rep 2025



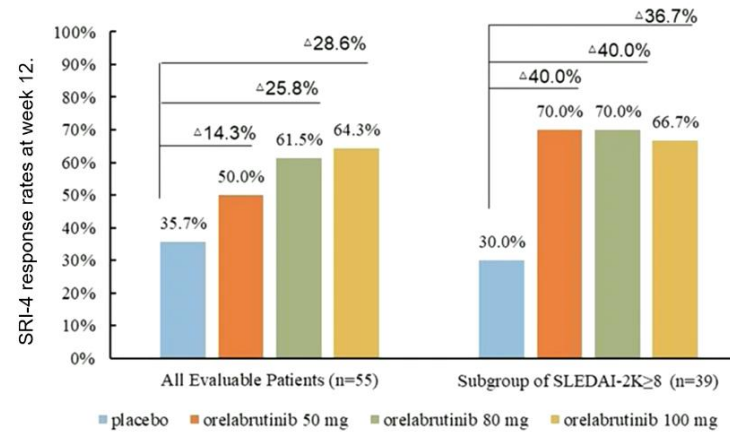
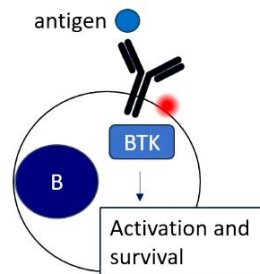
Claudia Günther
Novel and emerging cellular and
systemic treatments

Lupus erythematosus

Chairs: Branka Marinovic, Miloš Nikolić

Inhibition of Bruton Tyrosin Kinase (BTK) in SLE

ORELABRUTINIB, AN IRREVERSIBLE INHIBITOR OF BRUTON'S TYROSINE KINASE (BTK)
phase Ib/Ila, randomized, double-blind, placebo-controlled, dose-finding study



Li et al. Ann Rheum Dis 2022;81:210
Ahn et al., Front. Immunol., 2021

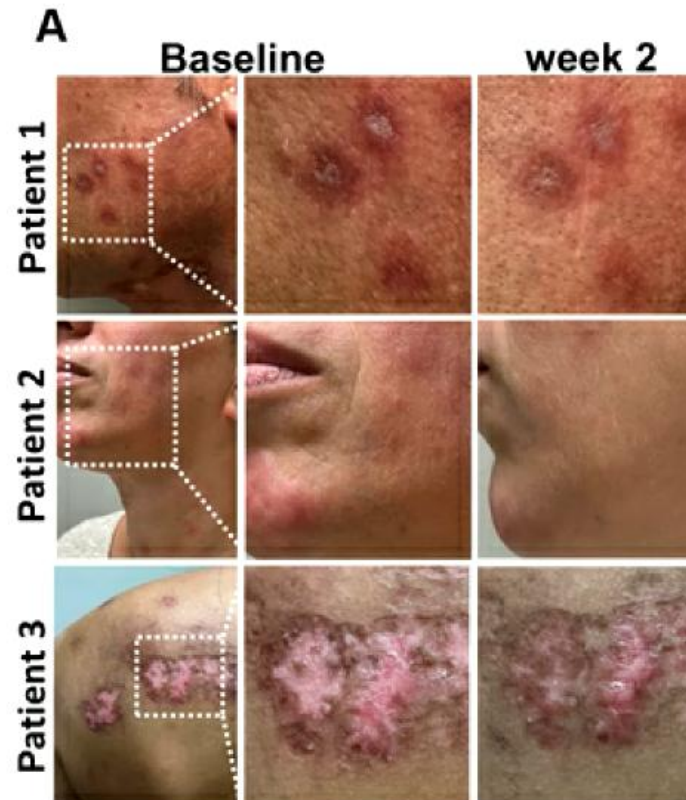
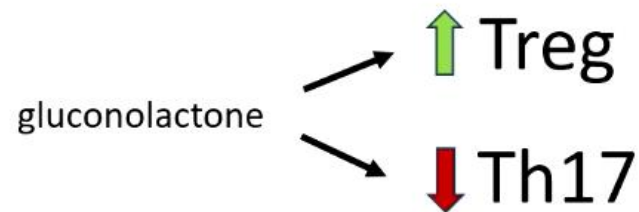
SRI= Systemic Lupus Erythematosus Responder Index 4



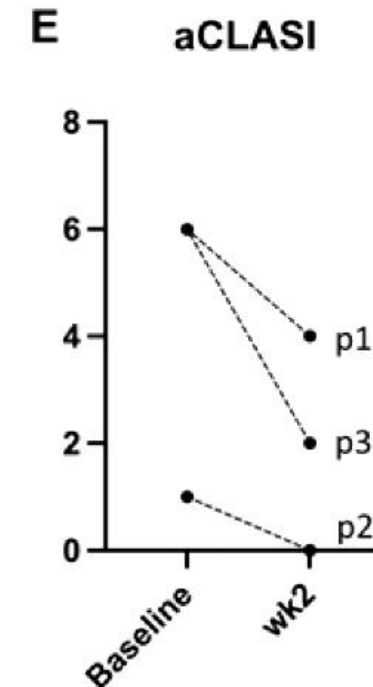
Claudia Günther

Novel and emerging cellular and systemic treatments

Restoration of Treg function improves CLE lesions



2 weeks 10% gluconolactone creme once daily



Adapted CLASI for erythema, non significant

Lupus erythematosus

Chairs: Branka Marinovic, Miloš Nikolić



Summary



- Possibilities to treat LE are limited! For CLE no licenced drugs except steroids
- Novel concept are approaching and many clinical trials are ongoing

Cellular targets:



litifilimab



Belimumab CLE
CD19CAR, BCMA CAR,
Teclistimab,
Orelabrutinib (BTK)



CD40L, dapirolizumab
Gluconolactone (Treg)



Selection, no complete list



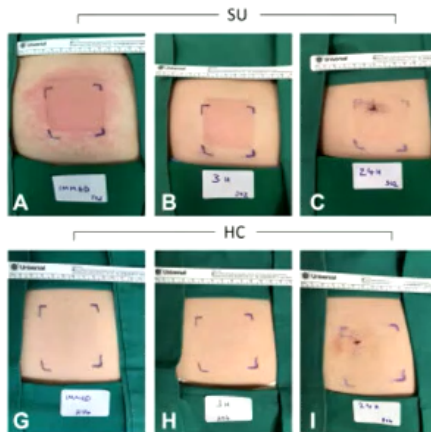
5/6



Claudia Günther

Novel and emerging cellular and
systemic treatments

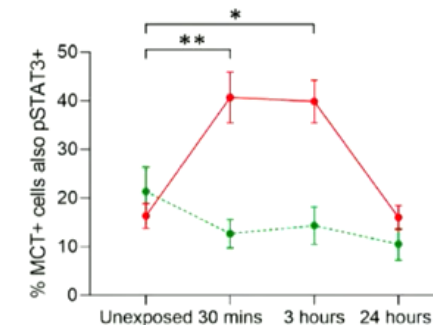
Cutaneous cellular and molecular events in the evolution of solar urticaria (SU) compared with healthy controls (HC).



Before – 30min – 3 hours – 24 hours after solar-simulated UV

Mast cell density (SU + HC)
STAT3 expression in mast cells
after 30min and 3 hours (SU)

- > Eosinophil counts (SU)
- > Dermal neutrophil counts (SU)



Solar urticaria is characterized by rapid STAT 3 activation in mast cells and involvement of multiple chemotactic and innate inflammatory pathways, with FcεRI engagement indicated as an early event.

Step 1: Sun protection

Avoid intense sunlight

Protective clothing & hats (UPF >30)

Broad-spectrum sunscreens (UVB, UVA, visible light)

Step 2: Antihistamines +/- Leukotriene receptor antagonist

non-sedating 2nd generation H1-antihistamines

bilastine, cetirizine, desloratadine, fexofenadine, levocetirizine,

Dose: up to four times the standard dose

Combination with LRAs: > CR



Step 3: Phototherapy

Action-Spectrum Hardening

Uses causative wavelength to induce tolerance

Inhibitory-Spectrum Therapy

Uses a non-urticarial wavelength

NB-UVB → SU induced by UVA1 or visible light

UVA-1 → SU induced by UVB or UVA2

Case reports and larger cohorts show efficacy

long-term maintenance with sun exposure 2-3x/week

Mechanism of tolerance: impairment or internalization of the mast cell IgE receptor



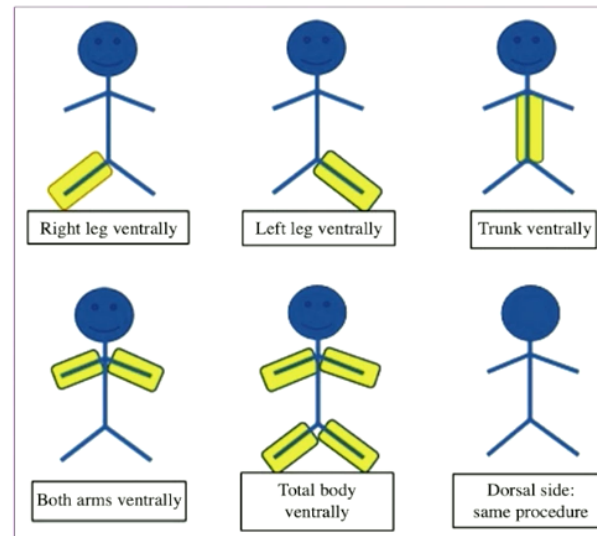
Beissert S, Ständer H, Schwarz T. UVA rush hardening for the treatment of solar urticaria. *J Am Acad Dermatol.* 2000;42:1030–1032

UVA rush Hardening with UVA, start 50% MUD
quadrant exposure at hourly intervals over 3 days
Duration of effect: 2–3 days

Percy Lehmann, Thomas Schwarz. *Dtsch Arztebl Int* 2011; 108(9): 135–41

PUVA beforehand, possibly UVA rust Hardening
Duration of effect 2–3 weeks

UVA-1 Hardening therapy



Adapted Beissert protocol

in 20 patients with UVA action spectrum

UVA/UVA-1 hardening:

intervals shorten to 15 min
+ daily UVB-311nm in 16 patients

Duration of inpatient rush hardening: 2–18 days

Maintenance: UVB 311 daily 1st week → 2–3×/week

Results: 17/20 patients adequately protected

Step 4: Omalizumab (off-label)

Recombinant humanised anti-IgE antibody

- lowers free IgE
- downregulates FcεRI on mast cells and basophils
- reduces mediator release after sun

Treatment: 300 - 600mg/every 4 weeks; s.c. injection

Snast I. et al. (n = 48): **79% improvement, 50% symptom-free¹**

Pesque D. et al. (n = 13): **all improvement, 1 patient was symptom-free²**
in 8 patients the extension of intervals (6-8 weeks) was possible

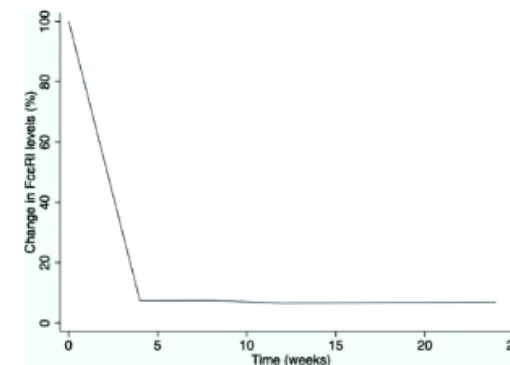


Fig. 2. Percentage reduction in high-affinity immunoglobulin E (IgE) receptor (FcεRI) baseline levels in patients after omalizumab initiation at weeks 0, 4, 8, 12 and 24.

Photodermatology

Chairs: Harvey Lui, Mariateresa Rossi

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Cases reports for SU treatment

- Antimalaria drugs
- Betacaroten
- Polypodium leukotomos
- Systemic glucocorticosteroids
- Azathioprine
- MTX
- Interferon
- **Cyclosporin (n = 11, PR 18%)**
- Ligelizumab (n=1, CR)
- **intravenous immunoglobulins (n=16, R 66,7- 71%)**
- **Plasmapheresis (n=8, R 62,5%),** Extracorporeal photopheresis (n=1, R)
- Afamelanotide (α -MSH, n=5)



Angelika Hofer

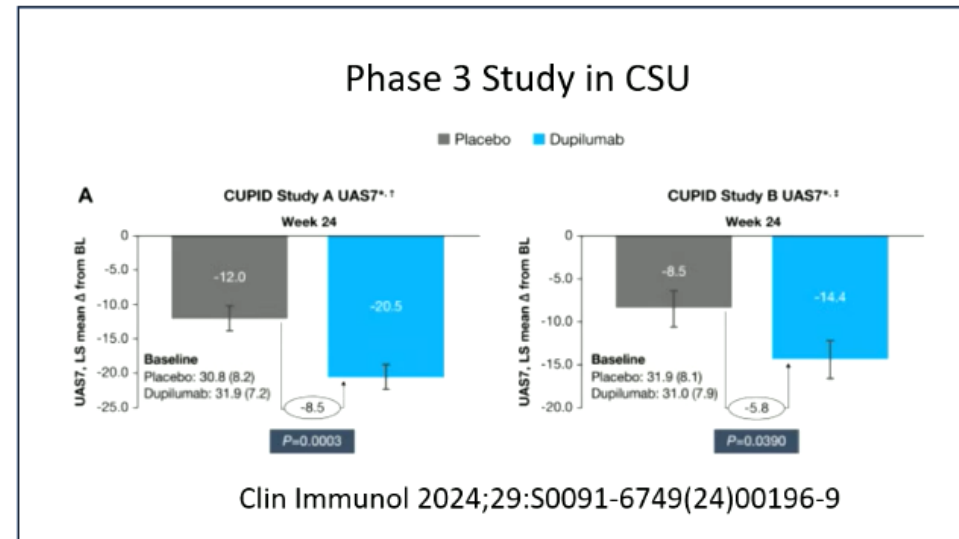
Solar urticaria

Dupilumab

Monoclonal antibody, approved for severe atopic dermatitis
inhibits the signaling of the IL4 and IL13 pathways
Directed against IL-4R α on mast cells

< Typ 2 Immunreaktion
< IgE-production

Systemic dermographism (phase 2 completed)
Cold urticaria (phase 2 completed)
Cholinergic urticaria (phase 2 completed)



Remibrutinib

Bruton Tyrosin Kinase (BTK) inhibitor (Small molecule), 25 mg twice daily p.o.
blocks BTK-mediated degranulation of mast cells and basophils downstream of FcεR1

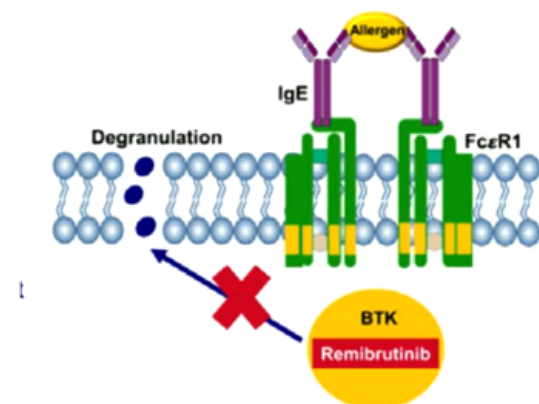
Phase 3 study (REMIX-1, REMIX-2) Remibrutinib vs placebo:
well-controlled chronic spontaneous urticaria (week 2 and 12)

Week 2: REMIX-1 (33.7% vs. 3.3%); REMIX-2 (30.0% vs. 5.9%)

Week 12: REMIX-1 (49.8% vs. 24.8%); REMIX-2 (46.8% vs. 19.6%)

Complete absence of itch and wheals at week 12

REMIX-1 (31.1% vs. 10.5%); REMIX-2 (27.9% vs. 6.5%)



BTK is essential for signalling through the FcεR1 receptor in mast cells and basophils

Fotodermatosis



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Briquilimab (anti-KIT mAbs)

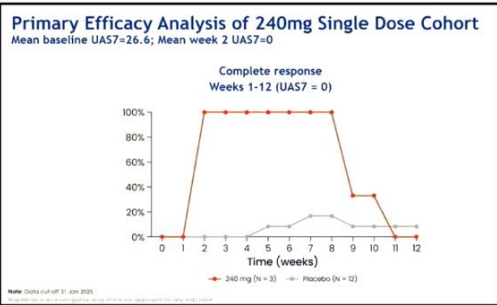
- Block stem cell factor binding
- Inhibits KIT signaling
- induces mast cell apoptosis

- Durable reduction of skin mast cells after a single dose

Briquilimab demonstrates rapid, clinically meaningful reduction in disease activity in adults with chronic spontaneous urticaria (CSU): Results from a Phase 1b/2a study

Thomas Casale¹, Edwin Tucker², Jinwei Yuan³, Daniel Adelman², David Ku², Annette Marcantonio², Patricia Carlor², Sophia R. Majumdar², Wendy W. Pang², Scott Fretzin¹, Martin Metz²

¹ Internal Medicine, Mount Sinai College of Medicine, University of South Florida, Tampa, FL, USA
² Janssen Therapeutics, Redwood City, CA, USA
³ Center for Dermatology, University of Michigan, Ann Arbor, MI, USA
⁴ Institute of Allergy, Charité-Universitätsmedizin Berlin, Germany



S040 Late breaking research: AAD 2025

JAKi therapy in CSU

frontiers | Frontiers in Immunology

Case report: Exploration of abrocitinib in the treatment of refractory chronic spontaneous urticaria: a case series

Na Du ^{# 1}, Dan Wang ^{# 2}, Jingyi Yang ¹, Yiwen Zhang ¹, Xinyan Lyu ¹, Wei Min ¹, Sicheng Zhao ³

Front Immunol. 2024;14:15:1466058

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Barzolvolimab (anti-KIT mAbs)

Open-label trial
Cold urticaria n=10
symptomatic dermographism n=10

one i.v. dose of barzolvolimab (3 mg/kg)
with a 12-week follow-up

CR: 10/10 cold urticaria, 9/10 symptomatic dermographism

Side effects: mild hematologic abnormalities, haircolor changes, taste alterations, headaches, and infections.

Allergy. 2023;78:1269–1279.

SHORT REPORT

DERMATOLOGIC THERAPY | WILEY

Efficacy of oral tofacitinib in refractory chronic spontaneous urticaria and urticarial vasculitis

Parvin Mansouri¹ | Nikoo Mozafari^{2,3} | Reza Chalangari⁴ | Katalin Martits-Chalangari⁴

Dermatologic Therapy. 2022;35:e15932

CHRONIC ACTINIC DERMATITIS DEFINITION

Immunologically-
mediated
photodermatosis

Eczematous eruption
predominantly affecting
photoexposed sites

Objective evidence
of broadband UVR
photosensitivity

Absence of a phototoxic
medication



Hiva Fassihi
Chronic actinic dermatitis



CHRONIC ACTINIC DERMATITIS IS CHANGING

BUT a lot has changed:

Mean age has fallen since 1990s from 65 to ~42 yrs

M=F

Skin phototypes I-IV : V-VI 2:1

Skin types I-IV more commonly elderly men

Patients below 30 years are often atopic, female, have type V-VI skin, and have acute flares



Hiva Fassihi

Chronic actinic dermatitis

CHRONIC ACTINIC DERMATITIS: PUVA TREATMENT

16 severe CAD patients

PUVA 2x per week for 10 weeks

Improved: 10

No change: 3

Flared: 3

PUVA effective in >60% of cases

PUVA was more effective in younger, predominantly male patients, with skin type IV–VI, mostly sensitive to only UVB on phototesting

Research letter

Chronic actinic dermatitis: successful treatment with psoralen–ultraviolet A photochemotherapy

DOI: 10.1111/bjd.15969

DEAR EDITOR, Chronic actinic dermatitis (CAD) is a debilitating photodermatosis. First-line therapy consists of strict photoprotection and topical corticosteroids. Second-line therapy uses systemic immunosuppression. However, an alternative is needed for patients with severe CAD who cannot use systemic immunosuppressants.^{1,2} Case reports and small case series suggest that psoralen–ultraviolet A (PUVA) photochemotherapy may be effective.^{3–6}

S N Chee, et al. Chronic actinic dermatitis: successful treatment with psoralen–ultraviolet A photochemotherapy. Br J Dermatol 2018 Mar;178(3):e189–e190.



Hiva Fassihi

Chronic actinic dermatitis



CHRONIC ACTINIC DERMATITIS: MANAGEMENT

Allergen/photoallergen avoidance

Topicals

Emollients, steroids, tacrolimus, pimecrolimus

Desensitisation

PUVA photochemotherapy (+/- oral steroids), low dose & increment regimen

Systemic agents

Oral prednisolone for acute exacerbations

Steroid sparing agents: methotrexate, azathioprine, ciclosporin, and mycophenolate mofetil



Hiva Fassihi

Chronic actinic dermatitis



DUPILUMAB IN CHRONIC ACTINIC DERMATITIS

There is weak, conflicting evidence on the use of Dupilumab for CAD in the literature

28/30 cases in the literature reported a partial to complete reduction in disease severity

However, only 14 cases were treated with Dupilumab as monotherapy

Case reports are not supported by pre and post Dupilumab phototesting

Complicated by the well-recognized exacerbated head and neck dermatitis from Dupilumab therapy

Holmes et al. Dupilumab for chronic actinic dermatitis: A case series and review of the literature. Australas J Dermatol. 2024 May;65(3):287-291



Hiva Fassihi

Chronic actinic dermatitis

JAK INHIBITORS IN CHRONIC ACTINIC DERMATITIS

10 cases in the literature with CAD
All 10 patients had a beneficial response to JAK inhibition

Five patients achieved a complete response

Further four patients experienced near complete resolution

Holmes et al. Upadacitinib for Chronic Actinic Dermatitis: A Case Report and Literature Review of JAK Inhibitor Use for Recalcitrant Disease. *Australas J Dermatol.* 2025 May;66(3):169-171.

Author	Age	Sex	Prev systemic agents	Concurrent systemic agents	JAK inhibitor (dose)	Treatment duration (months)	Beneficial response	Complete response
Pappa et al. [3]	75	M	OCS, CSA	Nil	Upadacitinib (15 mg OD)	8	Yes	Yes
<i>Majid and Akhtar [4]</i>								
1	65	M	MTX, AZA, HCQ, CSA, APR	Nil	Tofacitinib (5 mg BD)	6	Yes	Yes
2	55	M	MTX, AZA	Nil	Tofacitinib (5 mg BD)	12	Yes	Yes
3	58	M	OCS	Nil	Tofacitinib (5 mg BD)	12	Yes	Yes
Dev et al. [5]	50	M	OCS, MTX, AZA, ACI, HCQ	Nil	Tofacitinib (5 mg BD)	6	Yes	No*
Wang et al. [6]	46	M	OCS, HCQ	Nil	Tofacitinib (NR)	NR	Yes	NR
Zhong et al. [7]	58	M	Nil	Nil	Tofacitinib (5 mg BD)	6	Yes	No*
Vesely et al. [8]	60	M	OCS, HCQ, MTX, AZA, MMF, CSA, OMA, ACI, BEX, ECP	Nil	Tofacitinib (5 mg BD)	12	Yes	No*
Agud-Dios et al. [9]	53	F	OCS, CSA, HCQ, AZA	CSA, HCQ, AZA	Baricitinib (4 mg BD)	6	Yes	No*
Maguire et al. [10]	69	M	OCS, MMF, MTX, ACI, ALI	Nil	Baricitinib (2 mg BD)	16	Yes	Yes
Jin and Qiao [11]	70	M	HCQ	Nil	Abrocitinib (100 mg OD)	NR	Yes	No

Abbreviations: ACI, acitretin; ALI, aliretinoin; APR, apremilast; AZA, azathioprine; BEX, bexarotene; BD, twice daily; CSA, cyclosporine; ECP, extracorporeal photophoresis; F, female; HCQ, hydroxychloroquine; M, male; MTX, methotrexate; MMF, mycophenolate mofetil; OMA, omalizumab; mg, milligram; OD, once daily; NR, not reported; OCS, oral corticosteroids.

*Near complete response.



Hiva Fassihi
Chronic actinic dermatitis

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JAK INHIBITORS IN CHRONIC ACTINIC DERMATITIS



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Chronic actinic dermatitis

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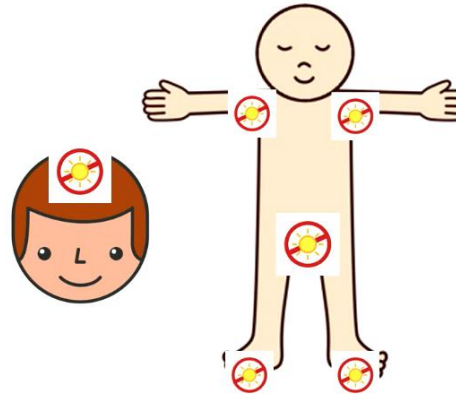
Photodermatology

Chairs: Harvey Lui, Mariateresa Rossi

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Limits of “conventional” Whole Body Phototherapy

- Ineffective in challenging «sanctuary» body areas:
 - Axillae, groin, genitalia
 - Soles
 - Hair-covered regions



Mariateresa Rossi
Phototherapy in challenging areas

Targeted UV phototherapy

- Indicated for skin diseases with limited skin involvement and/or «sanctuary» body areas
- Advantages upon topical drug treatments: non-messy, highly effective, convenient
- Evidence
- Practical considerations: supererythemogenic doses of energy can be delivered selectively to the lesions enhancing efficacy and achieving faster response, less carcinogenic activity

Photodermatology

Chairs: Harvey Lui, Mariateresa Rossi

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Targeted phototherapies

Principle: Light from an high output source is delivered to a small skin area via an optical fiber

• BROAD-BAND UVB LIGHT SOURCES

- Microphototherapy (Bioskin®)
- B-Clear®
- Theralight ®
- MultiClear® (Curelight)
- CUP-STRAHLER®



• NARROW-BAND UVB LIGHT SOURCES

- 308 nm Excimer Laser
- 308 nm Monochromatic Excimer Light (M.E.L.)



Mariateresa Rossi

Phototherapy in challenging areas



SKIN PATHOLOGY	THERAPEUTIC EFFECT
PSORIASIS	Effective in difficult to treat psoriasis subtypes Treatment option in refractory to systemic treatment psoriasis. Can achieve full remission in >50% of patients.
VITILIGO	Useful in depigmentation of <10% total body surface area.
ALOPECIA AREATA	Achieves cosmetically acceptable hair regrowth (>50% of regrowth). Resistant AA.
MYCOSIS FUNGOIDES	Useful in early stages (IA, IIA). Complete clinical response in 76.3% of cases.
ATOPIC DERMATITIS	In atopic dermatitis involving <20% total body surface area.
GRANULOMA ANNULARE	Complete remission has been seen in combination with topical/systemic CS
NODULAR PRURIGO	Complete remission in resistant cases
MORFEA	Useful to decrease lesion size.

INDICATIONS

Received: 25 July 2023 | Accepted: 25 February 2024
DOI: 10.1111/1346-8138.17191

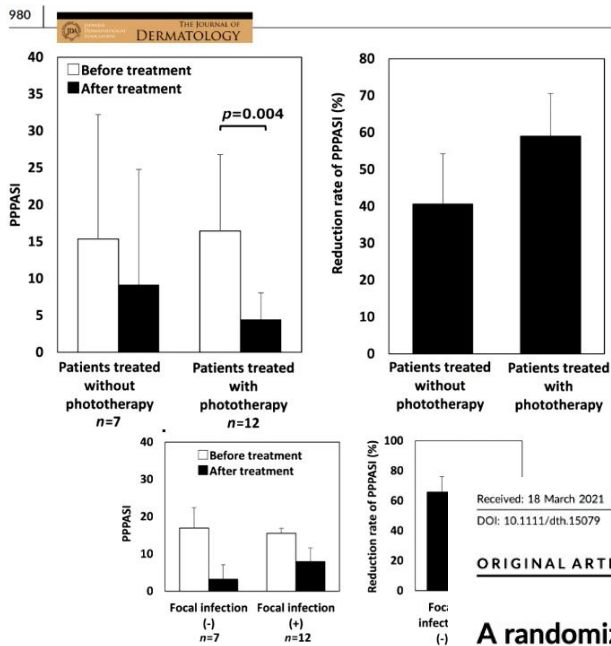
ORIGINAL ARTICLE

308-nm excimer light is effective for palmoplantar pustulosis regardless of the presence or absence of focal infection: Single-center real-world experience of treatment for palmoplantar pustulosis

Yoshiko Niimura | Masahiro Kamata | Takeko Ishikawa | Mayumi Nagata | Makoto Ito | Ayu Watanabe | Shota Egawa | Hideaki Uchida | Azusa Hiura | Saki Fukaya | Kotaro Hayashi | Atsuko Fukuyasu | Takamitsu Tanaka | Yayoi Tada

	Mean ± SD or number (%)
Female:male ratio	16:3
Age at onset (years)	48.8 ± 15.4
Duration of disease (years)	5.5 ± 5.9
Smoking	14/19 (73%) (never smoked, 5; quit smoking, 9; smoking, 5)
Pack-year	23.7 ± 17.6
Focal infection	13/19 (68%)
Dental infection	10/19 (52%)
Tonsillitis	6/19 (31%)
Sinusitis	2/19 (11%)
Allergy to metal	4/19 (21%)
Osteoarthritis	9/19 (47%) (8 women and 1 man)
PPPASI at the first visit	16.1 ± 13.1

Abbreviation: PPP, palmoplantar pustulosis; PPPASI, palmoplantar pustulosis area severity index; SD, standard deviation.



Received: 18 March 2021 | Accepted: 27 July 2021
DOI: 10.1111/dth.15079

ORIGINAL ARTICLE

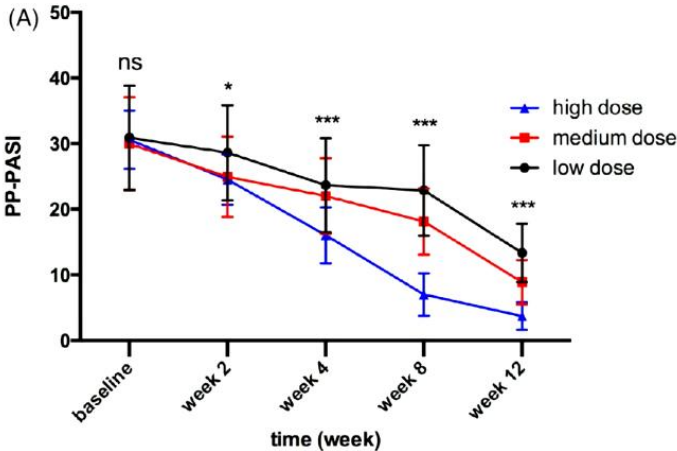
DERMATOLOGIC
THERAPY WILEY

A randomized prospective study of different dose regimens using the 308-nm excimer laser in the treatment of palmoplantar pustulosis

Chen Peng^{1,2} | Yifan Hu^{1,2} | Wenjuan Chen^{1,2} | Yangfeng Ding^{1,2} | Xingzi Li^{1,2} | Ning Yu^{1,2} | Jiajing Lu^{1,2} | Yuling Shi^{1,2}

Excimer dose-escalation regimens	Low dose regimen	Medium dose regimen	High dose regimen
Starting dose	200% of MED	400% of MED	600% of MED
Increment	20% increments with each treatment		
If adverse effects	Reduce to 10% increments		
Frequency of treatment	Three times weekly		

Adverse effects of erythema, blistering and erosions were more com-mon with the higher dose regimen



- Luz excimer no funciona en psoriasis ungueal (mejor PDL)

Al-Mutairi et al. Dermatol Ther (Heidelberg) (2014) 4:197-203

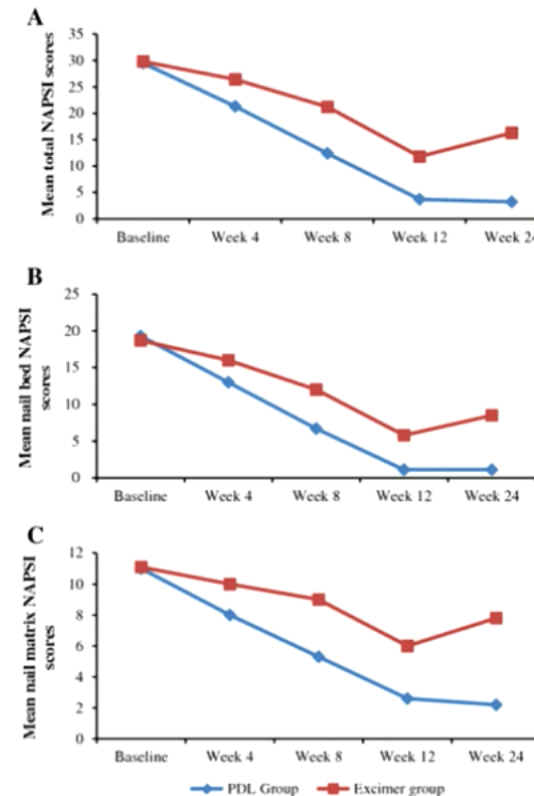


Single Blinded Left-to-Right Comparison Study of Excimer Laser Versus Pulsed Dye Laser for the Treatment of Nail Psoriasis

Week	PDL-treated nails		Excimer-treated nails	
	Mean NAPSI value	SD	Mean NAPSI value	SD
0 (baseline)	29.5	18.5	29.8	17.9
4	21.3	13.7	26.4	23.7
8	12.4	21.4	21.2	38.5
12	3.7	17.3	11.8	22.6
24	3.2	18.6	16.3	10.3

PDL pulsed dye laser, SD standard deviation

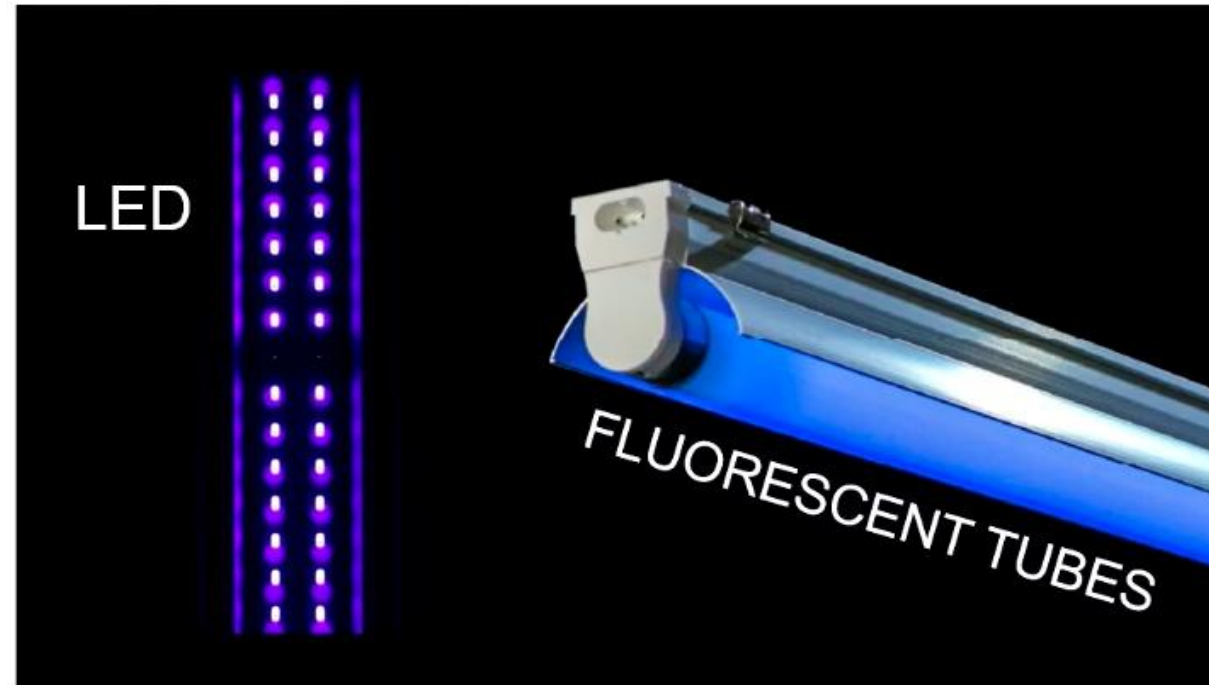
Although the excimer laser appears to be, on average, more efficacious than PDL for plaque psoriasis treatment, the contrary seems to be correct for nail psoriasis treatment.





New UV LED technology

- ✓ extremely long lifespan
- ✓ extremely energy efficient
- ✓ Very low maintenance costs and hassle
- ✓ can be much smaller than other lights
- ✓ faster switching (no warm-up or cool-down period)
- ✓ Adaptable to individual body shape

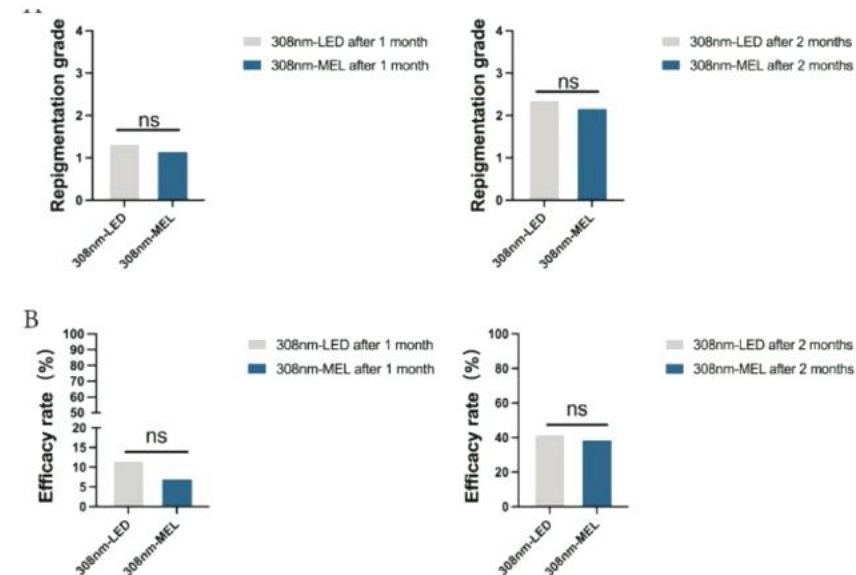
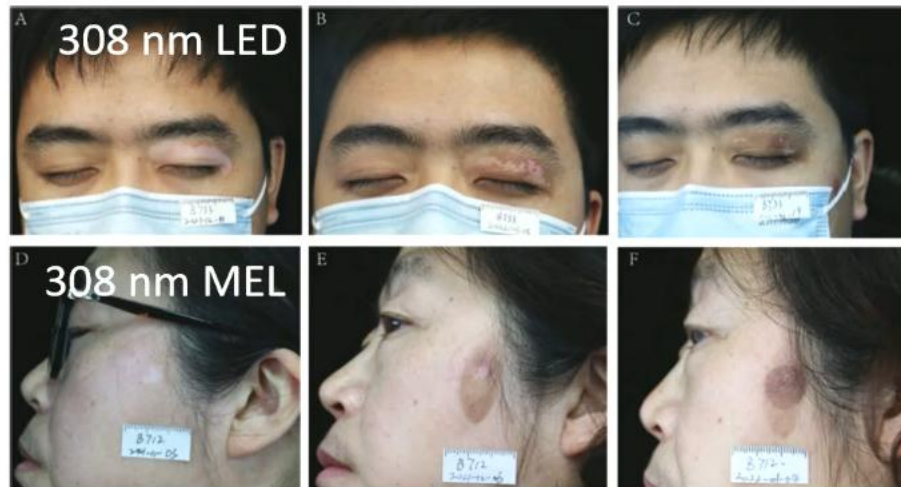


- ✓ contain toxic mercury
- ✓ short time to decay of fluorescent lamp
- ✓ Uneven field of irradiation



A randomized prospective study to compare the efficacy of 308-nm light-emitting diode and 308-nm excimer lamp in the treatment of facial vitiligo

Yu Hu² · Zhuohong Xu¹ · Lihao Liu¹ · Mei Ju² · Chao Luan² · Hongying Chen² · Lihao Chen² · Xiaoxi Dai¹ · Liangliang Zhang¹ · Dan Huang² · Jiaan Zhang² · Kun Chen²



76 VITILIGO PATCHES WITH LED, 69 WITH MEL

- Mejor la combinación de excimer con upadacitinib que upa solo



Archives of Dermatological Research (2025) 317:252
<https://doi.org/10.1007/s00403-024-03717-3>

ORIGINAL PAPER



Short-term efficacy and safety of upadacitinib combined with 308 excimer light versus upadacitinib alone and 308 excimer light alone in patients with progressing facial vitiligo

Progressing facial vitiligo
20 WEEKS

THERAPY	PATIENTS	VASI 100	>VASI 50	<VASI 50	VASI 0
UPA+MEL 308 NM	10	2 (20%)	7 (70%)	1 (10%)	0
MEL 308 NM	30	6 (20%)	9 (30%)	11 (36.7%)	4 (13.3%)
UPA	10	0	4 (40%)	6 (60%)	0



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Patrocina:



2025 AEDV Highlights

34ª edición
17-20 sep
PARÍS

Brilla el futuro de *la dermatología*,
donde nace *la luz*

La Academia Española de Dermatología y Venereología expresa su agradecimiento al patrocinador UCB, por su especial apoyo y contribución con la actividad formativa Highlights 2025.



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GRACIAS