

BIODERMA CONGRESS REPORTS

RADLA 2026

Reports drafted by Dr Silvina Maldonado, Argentina

The basics of trichology

Report drafted by Dr Silvina Maldonado, Argentina

Presenter: Dr Marlene Haudenschild.

Pruritus of the scalp.

This is the reason for 13-45% of our consultations. It is present in 75% of patients over 65 years old and 87% of atopic patients. Unfortunately, for 44.5% of them, we are unable to determine its origin. The scalp has different characteristics from the skin on the rest of the body, as it has numerous blood vessels and sensory nerves branching from the trigeminal nerve, more hair follicles and sebaceous glands, as well as a specific normal flora. Pruritus of the scalp can be acute or chronic and generalised or localised. There are four key mechanisms in its pathophysiology: pruritoceptive (triggered by activation of cutaneous afferent nerve fibres), neuropathic (resulting from direct neuronal damage), neurogenic (originating from nervous system mediators), and psychogenic (derived from psychological or psychiatric sources). A solid line of questioning can lead us to determine whether it is due to a dermatological pathology or is secondary to other underlying pathologies.

In addition to traditional treatments (glucocorticoids, calcineurin inhibitors, zinc, ketoconazole, selenium, etc.), the presenter highlighted the role of antihistamines, anticonvulsants, opioids, antidepressants, cyclosporin, and thalidomide. To conclude, she talked about emerging therapies, focussing on:

- IL-31 BLOCKING: the most promising being nemolizumab, which works by blocking the main mediator of pruritus, with rapid response. It is particularly promising for atopic dermatitis of the scalp, chronic pruritus, inflammatory trichodynia, and refractory pruritus.
- Th2 MODULATION
- JAK INHIBITORS: Upadacitinib and abrocitinib have rapid action inhibiting IL-4, IL-13, IL-31, and TSLP; useful in severe pruritus causing significant sleep disturbance and resistant inflammation.
- NEUROMODELLING: Gabapentin and pregabalin. Also dual inhibitors for serotonin and noradrenaline
- DISRUPTION OF THE NEUROINFLAMMATORY AXIS / INNOVATIVE TOPICAL THERAPIES: PDE-4 phosphodiesterase inhibitors, roflumilast, AhR (aryl hydrocarbon receptor) antagonists, and tapinarof (FDA approved). The AAD 2025 guidelines incorporate these therapies as new therapeutic options.

The speaker also spoke about DUAL THERAPIES currently being researched: SIMULTANEOUS IL-4/IL-13 + IL-31 BLOCKING, THERAPIES TARGETING THE NEUROIMMUNE AXIS, with the aim of simultaneously controlling inflammation and pruritus.

In conclusion, she noted that further research is needed to identify more effective antipruritic agents with fewer adverse effects. Emerging therapies are evolving, from non-specific therapies to targeted neuroimmunological strategies, especially on the IL-31 axis and JAK-STAT pathways.

Trichology today: details that redefine diagnoses.

Report drafted by Dr Silvina Maldonado, Argentina

Presenters: Dr Nestor Carreño, Dr Aldo Galvez Canseco, Dr Jeyce Reynoso, Dr Nelly Espinoza Gonzalez, and Dr Sebastian Metcau.

Alopecia of the eyebrows: key diagnostic criteria.

Presenter: Dr Nestor Carreño.

Alopecia of the eyebrows is a condition of great functional and aesthetic importance, with a significant psychological impact. The clinical presentation varies, sometimes with few indications; as such, doctors should look for other signs and consider the high prevalence of frontal fibrosing alopecia (FFA). The condition may have an autoimmune, endocrinological, infectious, genetic, neoplastic, nutritional, or traumatic origin. We must differentiate between scarring and non-scarring, looking for trichoscopic signs: hyperkeratosis, erythema, loss of follicular ostia, yellow dots, black dots, broken hairs, flame hairs, ribbon-like hairs, arborising or looped blood vessels, zigzag hairs, and corkscrew hairs.

- Among autoimmune diseases:
 - Alopecia areata is usually bilateral and in patches, with its typical exclamation-point hairs, cadaver hairs, hairs in a state of regrowth, and yellow and black dots.
 - FFA, which generally begins developing long before the frontal component appears. Trichoscopy shows hairs pointing in different directions, black and yellow dots, distracted hairs, pili torti, loss of follicular ostia, hyperkeratosis, and perifollicular scaling.

- Discoid lupus erythematosus (DLE): trichoscopy is poor, with loss of follicular ostia, pigmentation changes and arboriform or looped vessels.
- Endocrine: + Hypothyroidism: the classic indicator is Hertoghe's sign, or loss of the lateral third of the eyebrows, although the sign is non-specific.
- Infectious: mycotic (comma, spiral, bent, Morse-code, and zigzag hairs), viral (inflammation or scarring), leprosy, syphilis
- Trichotillomania (broken hairs of different lengths, black dots, flame hairs, tulip hairs, transverse fractures).
- Genetic: Lichen spinulosus and keratosis follicularis spinulosa decalvans, and Graham-Little-Piccardi-Lassueur syndrome.

This condition presents a great challenge; you must examine the rest of the face and body for signs. Do not avoid trichoscopy-guided scalp biopsy.

When everything is diffuse: how to determine the correct diagnosis.

Presenter: Dr Aldo Galvez Canseco.

The presenter noted three steps:

- 1.) Distribution: assess patches and patterns by comparing interparietal and occipital areas.
- 2.) Scarring or non-scarring, using fluorescence from new dermatoscopes: in non-scarring, pink dots signal *C. acnes* and green dots *Malassezia sp.* In scarring types, the perifollicular fluorescence appears bluish white.

3.) Trichoscopic keys and patterns: Telogen effluvium (hair in regrowth 95%), anagen effluvium, alopecia areata (the incognita type is very difficult), alopecia syphilitica (distracted, white, and circular hairs, and erythematous background), systemic lupus erythematosus (SLE), prominent arboriform blood vessels, pigmentation changes, scattered brown pigmentation, blue-grey hyperpigmentation and black dots, and dystrophic hairs.

In conclusion, the three-step approach shortens the diagnostic investigation, and in spite of technological advances, patient history and clinical suspicion still have important roles to play.

Red scalp: subtle signs of erythema.

Presenter: Dr Jeyce Reynoso.

The presenter stressed the importance of assessing whether the erythema is inter- or perifollicular, whether the scales are oily, dry, intermediate, or perifollicular, the shape of the blood vessels (arboriform, punctiform, telangiectatic, interfollicular) and the symptoms (pruritus, burning, trichodynia). When the erythema is interfollicular: think inflammatory, not scarring. If the erythema is perifollicular, it may be a sign of early scarring alopecia. If the scale is tubular-adherent, think scarring. In case of pain or burning and according to clinical findings, think of rosacea/trichodynia. The area to be biopsied should be an active area with perifollicular erythema.

Difficult cases in trichology: sources of confusion and enlightenment.

Presenter: Dr Nelly Espinoza Gonzalez.

The presenter proposes three levels of diagnostic confidence, using a pyramid whose base is NON-SPECIFIC FINDINGS (black dots, yellow dots, scaling, and erythema). The middle of the pyramid is distribution, vascular patterns, and perifollicular changes.

Finally, at the top of the pyramid are high-confidence findings: loss of ostia, orphan hairs, flame hairs, and blue-grey dots.

She noted that the diagnosis depends more on combined patterns than on individual signs. She emphasised that the most common errors are related to over-interpreting isolated signs, ignoring previous treatments, confusing late disease with active disease, not assessing loss of the ostia, and disconnecting trichoscopy from clinical topography.

Alterations of the hair strand: when trichoscopy saves the day.

Presenter: Dr Sebastian Metcau.

The doctor should assess whether fragility of the hair shaft is congenital or acquired. Among congenital causes, he mentioned monilethrix, pili torti, proximal trichorrhexis nodosa, trichorrhexis invaginata, and trichothiodystrophy. Among acquired causes, he mentioned distal trichorrhexis nodosa and bubble hair (physical and chemical factors).

Symposium: new prospects for the management of acne

Report drafted by Dr Silvina Maldonado, Argentina

Presenters: Dr Geraldo Magalhaes, Dr Cristina Echeverría, Dr Vitoria Azulay, and Dr Andrea Cortes.

The new biology of acne: from subclinical inflammation to dysbiosis.

Presenter: Dr Geraldo Magalhaes.

The presenter spoke of re-evaluating the pathogenesis of the pathology. The historical model focussed on the role of follicular hyperkeratosis, microcomedones, sebum accumulation, and colonisation by *C. Acnes*, along with inflammation.

The modern model focusses on inflammation being present throughout the cycle of the lesions. Talk of “inflammatory” and “non-inflammatory” lesions has become obsolete. The new prospects come from a shift in therapeutic focus, targeting immune modulation and tissue homeostasis. He showed that inflammation occurs before hyperproliferative changes are visible in the microcomedones. There is an early perifollicular infiltration with CD3+ and CD4+ T cells/micros in clinically normal follicles, with levels comparable to those of papules, refuting the theory that the inflammation is secondary to follicular rupture, so the prospect involves a “field treatment”. The role of *C. acnes* changes from a simple commensal to a key immunomodulator in acne. It stimulates the innate immune system by binding to toll-like receptors (TLR2 and TLR4), attracting neutrophils. In addition, it induces the production of IL-12, IL-8, and TNF-alpha in monocytes and keratinocytes. It also releases enzymes that activate the PAR-2 receptor, which stimulates the synthesis of MMP-9, which degrades collagen and influences scar formation. It should be noted that the severity of acne is not directly correlated with the total bacterial load, but rather with the immunogenic capacity of the strain involved. Dense biofilm formation explains the rapid recurrence after antibiotic use. As for inflammation of the sebaceous glands, sebocytes produce cytokines in response to chemical and bacterial stimuli. Squalene peroxidation generates by-products that stimulate nuclear factor kappa B, which in turn stimulates follicular keratinocyte proliferation, even before massive bacterial colonisation. Linoleic acid reduction impairs the skin's barrier function and triggers IL-1 release.

Regarding the neurocutaneous interface, corticotropin-releasing hormone (CRH) produces increased lipid synthesis and an inflammatory response, substance P stimulates follicular proliferation and cytokine stimulation; the stress-acne axis modulates the virulence of *C. acnes* via catecholamines, leading to therapeutic failure in patients with high emotional stress.

The presenter described the gland as an endocrine organ, producing local conversion of weak androgens to DHT. IGF-1 (insulin-like growth factor 1) causes cell proliferation and lipid synthesis by activating the mTORC1 pathway, resulting in the suppression of the transcription factor FOXO1. Patients with acne have elevated IGF-1 levels and signs of peripheral insulin resistance, even in the absence of obesity.

He also talked about the shift in perspective from “overpopulation” to “loss of diversity”, regarding eubiosis versus dysbiosis, with follicular dysbiosis worsening with antibiotic therapies. The *C. acnes* IA1 and IA2 phylotypes are lineages with greater association with inflammatory lesions and active virulence determinants. Phylotype IB is an intermediate genomic profile with variable expression of adhesion factors. Types II and III, less associated with inflammatory or severe acne and RT4/RT5 associated with acne, versus RT6 associated with health, with protective CRISPR systems. Regarding the involvement of *Malassezia* and *Staphylococcus*, we know that the skin microbiome is multi-species and the balance between the two is crucial for the stability of the follicular barrier, as the latter antagonises and competes with the former, regulating it, which also stimulates hyperkeratosis. The gut-skin axis shows that acne is influenced by increased intestinal permeability, which sensitises sebocytes via mTOR, so probiotics reduce the inflammatory burden by improving the barrier. He described inflammasomes as multi-protein sensor complexes of the innate immune system. The NLRP3 inflammasome is triggered by cholesterol crystals and oxidised lipids, opening up new targets for severe acne.

The exposome (total lifetime exposure) modulates acne, so strategies such as sunscreens and antioxidants are effective. He concluded by saying that acne is a SYSTEMIC and COMPLEX inflammatory disease, whose therapeutic future will be the restoration of eubiosis, inflammatory modulation, and epigenetic control.

Sebaceous regulation and the hormonal axis: beyond isotretinoin. Current prospects for the control of sebaceous secretion, hormonal impact, and new drugs modulating androgens and IGF-1.

Presenter: Dr Cristina Echeverría.

The presenter discussed sebum as an early and central link, as its excessive production driven by androgens and IGF-1 contributes to comedogenesis, proliferation of *C. acnes*, and inflammation. She showed that spironolactone was superior to doxycycline according to the FASCE 2024 study, representing a relevant systemic alternative for adult women, although it remains an off-label treatment for acne. Clascoterone (not yet available in Latin America) represents a topical innovation for both sexes. It is the first topical antiandrogen approved by the FDA (in 2020), acting locally without relevant systemic adverse effects proven in phase III trials. It opens up antiandrogen treatment to men and adolescents. Finally, metformin is an emerging option for the IGF-1/IR axis, with meta-analysis showing significant improvement in GAGS scores. It is potentially useful in patients with insulin resistance or metabolic disorders, although higher-quality evidence is still pending. Understanding sebaceous regulation and the hormonal axis allows us to stop treating lesions and start modulating mechanisms.

Cultural, dietary, and environmental factors: why acne is not the same all over the world. Influence of diet, pollution, regional microbiota, and cultural habits on the prevalence and clinical expression of acne in different populations.

Presenter: Dr Vitoria Azulay.

Different diets show that a high glycaemic index stimulates the proliferation of keratinocytes and sebocytes, increases sebum production, promotes androgen synthesis, and increases sensitivity to androgens. Composition of dairy products (protein versus fat) influences the acneigenic potential; caffeine produces an insulinotropic effect. Dysbiosis is improved with oral probiotics.

The real impact of new technologies on the treatment of active acne and scarring.

Presenter: Dr Andrea Cortes.

The presenter discussed the importance of using technology in the early stages of acne treatment to avoid major sequelae, suggesting a combination of technologies to control active acne and its sequelae.

SYMPOSIUM: Artificial intelligence and new technologies.

Report drafted by Dr Silvina Maldonado, Argentina

Presenters: Dr Cristobal Lecaros, Dr Enrique Majul, Dr Rodrigo Schwartz, Dr Leonel Hidalgo Acuña, Dr Gabriel Salerni.

Where we have come from and where we are headed with AI in dermatology.

Presenter: Dr Cristobal Lecaros.

Where have we come from? The history of AI in dermatology is the history of applied computer science. We are currently in a scenario of opinions. The facts show that AI produces similar diagnoses to dermatologists in dermoscopy of melanoma; it produces similar histopathological diagnoses of non-Spitzoid melanoma; and it can pass the American Board of Dermatology examination. The presenter discussed an augmented intelligence model, in which AI provides diagnostic support that improves the diagnostic accuracy of humans by 64% to 77%. He also talked about explainable artificial intelligence, showing studies in which the artificial intelligence, by explaining its reasoning, increased the confidence of dermatologists in their decision-making. Where are we headed? No one knows; there will likely be future benefits such as a reduced administrative burden, diagnostic access for populations that currently have limited access, and individualisation of care. The challenges will be tied to implementation, like other health technologies, ensuring that AI use improves care.

The augmented doctor: how AI is redefining the standard of care in all fields of medicine.

Presenter: Dr Enrique Majul.

From cognitive overload to multimodal accuracy in clinical practice. The speed of generation of medical information has surpassed our biological capacity for synthesis, forcing us into a role of overloaded data processors and pushing us into a zone of overload and burnout. The traditional medical doctor was a repository of memorised knowledge, using technology for passive record keeping, with manual data entry and assessment; diagnosis could be limited by observer bias. The “augmented doctor” becomes a synthesiser of complex biological systems. Their augmented relationship with technology transforms it into a cognitive partner and interactive copilot. The augmented focus enhances clinical judgement, empathy, and interpersonal connection, and the augmented capacity leads to quantitative analysis and hyper-pattern recognition. Dermatology is at the epicentre of disruption due to its hyper-visual nature: adapting is not an option, but rather an imperative for professional survival. Neural networks decode subclinical features imperceptible to human vision on its own. The automation of routine processes frees the professional from repetitive tasks so they can focus exclusively on highly complex, atypical cases. Ambient scribes allow technology to “disappear” so that human care “reappears”, saving 2 to 3 hours of monitor time and putting the patient back in the foreground, as ambient scribe technology listens to the consultation and structures the clinical notes without the need for robotic voice commands.

The multimodal ecosystem crosses data boundaries to predict the behaviour of pathologies such as tumours by submitting all the data to AI predictive algorithms and generating precision medicine with risk stratification, mathematical prediction of metastasis, and overall survival prognoses, as well as a proactive selection of therapies (BRAF/MEK inhibitors versus immunotherapy). But it's not all good news: there is a danger of algorithmic bias. Most AI tools are trained disproportionately with white populations, leaving out darker phototypes, leading to critical false negatives. Another important concept is "deskilling" or cognitive atrophy: this is the loss of skills due to delegated decision-making, with alarming evidence already demonstrated in young doctors and residents after three months of exclusive dependence on AI. Along with the loss of existing skills, it can also impede the learning of new skills. The legal liability remains with the doctor, who feels obligated to take decisions based on the tool's results, but there is no legal protection for that. We must take advantage of the technology, which is not here to replace us, but rather to give us back time so we can reconnect with our patients. To conclude, AI is a cognitive scalpel that does not replace dermatological expertise; it does, however, allow the doctor to return to their most irreplaceable function: empathy, care, and bioethics.

AI: between utopia and dystopia.

Presenter: Dr Rodrigo Schwartz.

The utopian vision would be the democratisation of health, as AI can overcome barriers related to poverty and distance, and improve access to health care. AI also improves early detection of diseases without the presence of a specialist in remote locations. It can help by predicting outbreaks of infectious diseases and identifying at-risk populations. However, at the same time, it can be an amplifier of health disparities, as has been previously described: WEIRD (Western-educated, industrialised, rich, democratic) bias.

In the particular case of melanoma, it has been found that the technology is six times more likely to make a diagnostic mistake with dark skin compared to light skin, due to training of the algorithm in predominantly white-skinned countries. This represents a major problem because white-skinned people are only 12% of the world's population, leading to misdiagnosis and potentially increased comorbidities. The presenter also spoke of the term "computernalism", which is health decision-making that relies exclusively on computers and AI, displacing human clinical judgement, creating a trilateral relationship. Another problem is that the use of AI changes the doctor's perception of legal liability. With regard to health insurers, one would expect them to use AI for more efficient management, but on the contrary, the rejection of claims has increased compared to when claim assessment was in the hands of people. As a result, California has already passed new legislation on the issue and prohibits AI-only claim refusals. The threat from AI consists of three pillars: the concentration of data by totalitarian governments, with systems for the mass surveillance of their populations, massive disinformation campaigns, and the consequent loss of freedom and democracy; the potential for unemployment and its consequences such as alcoholism, depression, increased sedentary lifestyles, suicide, etc.; and finally that artificial general intelligence uses autonomous reasoning and decision-making made possible by continuous learning from experience, eventually learning to evade any restrictions in its code and starting to developing its own goals. The presenter concluded by saying that the medical community must be actively involved in AI policy debates; it is not an option and is instead part of our responsibility as 21st-century doctors.

AI in the dermatologist's day-to-day work.

Presenter: Dr Leonel Hidalgo Acuña.

While apps for the assessment and management of skin conditions are on the rise, there is limited evidence to support their efficacy, with most lacking FDA approval (only two with disclaimers). There is a lack of transparency, oversight, and evidence for the algorithms, coupled with a risk for patients' privacy. The presenter cited various apps that are recommended. For use in online meetings: Fireflies and tl;dv. For reviewing literature: Consensus, SciSpace, Semantic Scholar, and Elicit. Others: Perplexity Health, Connected Papers, ResearchRabbit, Suites, Kleia, Vera Health, OpenEvidence (withdrawn from EU due to legislation). For content management: Mapify, Canva, FigureLabs, Gamma, Kimi, Genspark. Some more: Osmosis, Sketchy, YouLearn, Astra AI, Penseum. For transcription of medical care: Tandem, Heidi, Nabla, Twofold. He suggested avoiding Claude, Gemini, and ChatGPT. He also recommended NotebookLM. Among the final considerations, he suggested that users take care with sensitive information.

Is there a place for AI beyond diagnostics?

Presenter: Dr Gabriel Salerni.

Yes, of course. AI plays a key role in the recognition of pigmented lesions and skin cancer. It not only helps us in diagnosis, but also becomes important operationally (automatic report generation, clinical history summaries, automated follow-up, adaptive comparison, smart image search, assistant for scientific research and population screening). Augmented AI does not replace the dermatologist, but rather helps amplify their analysis and management capacities, optimising clinical time. Digital twins are virtual representations of individuals developed by integrating real-time data such as images, genetic information, lifestyle factors, and environmental influences.

The information is continuously updated and prediction or simulation scenarios are generated. The digital twin model has evolved from static to progressive, operational, and then autonomous, moving into a new era of personalised skin care. In dermatology, the main applications are:

- 1.) Personalised diagnosis and treatment, simulating treatment responses and helping predict which drugs or therapies will be most effective.
- 2.) For cosmetic dermatology and anti-ageing treatments, it simulates results, optimising personalised parameters and reducing adverse effects.
- 3) Predictive dermatology, anticipating the onset of skin diseases, detecting early changes in pigmented lesions through continuous digital monitoring, and favouring proactive prevention.

The challenges come from data privacy concerns, regulatory compliance, high implementation costs, and hardware/software knowledge gaps.

Plenary-session lectures.

Report drafted by Dr Silvina Maldonado, Argentina

Presenters: Dr Esther Freeman, Dr Melinda Gooderham, Dr Brigitte Dréno

Identifying the gaps: global access to skin health.

Presenter: Dr Esther Freeman.

The presenter spoke about the poor distribution of dermatologists worldwide, with there being places where there are one per 1 million inhabitants. This occurs in different parts of the world, even in the industrialised world. She described these places as “dermatological deserts”. The Global Access to Skin Health Observatory Study (under the ILDS) brings dermatological care to underserved areas.

The study’s actions led to the recognition of skin diseases as a global public health priority at the 78th World Health Assembly, which may enable a big step forward towards equity and recognition of the importance of skin health. It is worth highlighting the work of Dr Isabel Casas in Argentina’s Patagonia region, among many other doctors around the world.

New therapies for atopic dermatitis.

Presenter: Dr Melinda Gooderham.

The presenter underscored the importance of updated guidelines such as EuroGuiDerm AD, which include the latest biologics and JAK options. Dermatologists should review these guidelines to form a new baseline for the systemic care of atopic dermatitis. Among the most effective options, she highlighted biologics (IL-4/13, IL-13, IL-31) and JAK inhibitors, which provide doctors with more effective and tolerable systemic options than ever before, backed by direct comparison and network meta-analysis (NMA) data.

She discussed managing risk reflexively, as oral JAK inhibitors for AD are not associated with the elevated CV/MACE (major adverse cardiovascular events) risk seen in RA patients; however, doctors must remain alert to the risk of HZV, lab changes, and acne. Patient selection is important. In conclusion, the treatment landscape for atopic dermatitis has changed a lot in the last 30 years. Biologics are considered first-line systemic treatments because they are targeted, long-lasting, and well-tolerated. JAK inhibitors stand out for their rapid onset of action, oral administration, and manageable safety for select patients. In development: new avenues are being explored to broaden the therapeutic menu, including: OX40/OX40L, IL-22, BTK, STAT degraders, and YTE-engineered biologics.

How to manage adverse skin effects induced by cancer drugs.

Presenter: Dr Brigitte Dréno.

The presenter spoke about the important role of the dermatologist within the multidisciplinary care team, helping prevent adverse skin effects. As skin specialists, dermatologists can contribute to the success of treatment by helping avoid the premature discontinuation of therapies. Alteration of the skin barrier and the microbiome predisposes the patient to inflammation. The management of these effects depends on the synergy between the pharmacological treatment and dermo-cosmetic support. Management can reduce inflammation, irritation, and xerosis, thereby increasing treatment adherence and quality of life. Alteration of skin pH directly modulates kallikrein-5 activity, leading to skin barrier dysfunction and increased filaggrin; therefore, it is recommended that patients use cleansers that maintain skin pH. Moisturisers help prevent and correct neurogenic inflammation. Dr Dréno invited all to familiarise themselves with the ISKIMO (International Skin Management in Oncology) guidance.

In conclusion, she noted that the prevention of adverse effects will be crucial in the context of upcoming adjuvant and neoadjuvant treatments, for which skin care will have a key role to play. Treatment of adverse skin effects improves patients' quality of life and has a great psychological impact. It is important to educate patients so they can take an active role in fighting their illnesses, together with their doctors.

Therapeutics: the classic, the new, and the future.

Report drafted by Dr Silvina Maldonado, Argentina

Presenters: Dr Alejandra Larre Borges, Dr Patricia Della Giovanna, and Dr Manuel Dario Franco.

Isotretinoin: real risks versus historical fears.

Presenter: Dr Alejandra Larre Borges.

The first historical fear is depression: temporal association is often confused with causation, and adolescence is associated with a natural window for psychiatric disorders. Severe acne itself is associated with depression and suicidal ideation, and often the improvement of the acne improves the psychological state. No consistent increase in suicide has been demonstrated and many studies even show a reduction in depressive symptoms following acne improvement. In patients with a history of depression and/or psychosis, continuous psychiatric monitoring is required. Another fear is inflammatory bowel disease (IBD): after litigation and alarm in the media, no evidence of a consistent increase was found in modern meta-analyses. The real risks are:

1.) mucocutaneous effects such as cheilitis, xerosis, facial dermatitis, blepharoconjunctivitis/dry eye, skin fragility, pruritus, epistaxis, nasal and oral dryness, as well as initial exacerbation of acne.

2.) Neurological effects such as headache, dizziness, insomnia, lethargy, paraesthesia, weakness and syncope, intracranial hypertension, especially with concomitant use of tetracyclines, seizures, stroke.

3.) Endocrine-metabolic disorders such as decreased appetite, weight fluctuations, and blood glucose alterations. Most effects are dose-dependent, reversible, and manageable with supportive measures and adequate monitoring.

4.) Regarding teratogenesis: the risk of miscarriage is 20% and the risk of embryopathy is up to 28.6%. The greatest therapeutic risk is up to four weeks post-LMP. Neurobehavioural sequelae have been documented, even in the absence of structural malformations. In men, it is not teratogenic, although it can produce hormonal changes such as a slight decrease in testosterone and LH, which are reversible. The laboratory values to be monitored would be lipids and liver panel at the start of treatment and at 56 and 62 days, respectively. The blood panel is very rarely altered, so it may not be necessary; however, Dr Larre Borges suggests doing it anyway. Additional monitoring in patients with metabolic risk factors, high or borderline baseline values, concomitant use of hepatotoxic medication, significant dose increase, or suspected adverse effects. To minimise risks, low doses (0.5 mg/kg/day) are suggested. Regarding the decrease in height due to premature epiphyseal closure, there is no evidence that it is permanent, only reducing the growth rate transiently and without affecting the final adult height. Premature epiphyseal closure was observed at high doses given for prolonged periods of time.

In adolescents involved in repetitive high-impact sports, growth plate injuries and spondylolisthesis may occur. Moderate activity and plenty of hydration should be recommended, and a baseline CPK lab test should be done for monitoring, especially in elite athletes.

In relation to the risk of defective healing from dermatological procedures and surgeries, there is only evidence of a risk of necrosis in the case of muscle flaps, and mechanical dermabrasion and fully ablative laser treatments should be avoided.

Treatment of genodermatosis.

Presenter: Dr Patricia Della Giovanna.

The presenter discussed keratinisation disorders, neurocutaneous syndromes, overgrowth syndromes, and pigmentation disorders. In epidermal naevi with acanthosis nigricans, besides surgery, which can have major sequelae, and laser treatments, after which the condition can return, she recommended topical sirolimus 0.1% daily for its indirect mechanism acting on the FGFR3 pathway, which plays a fundamental role in the regulation of cell proliferation. Another study proposes the same drug at 2% for sebaceous naevi and epidermal naevi. Everolimus 1% cream is more water-soluble and therefore has better skin penetration. For CHILD Syndrome, she pointed to treatment with topical simvastatin and lovastatin 2% as a monotherapy, with great results based on its pathogenesis (defects in cholesterol synthesis). For ILVEN, she recommended secukinumab with 300 mg as an induction therapy, and then administration every four weeks. Complete resolution was obtained in five weeks. With 32 weeks of treatment at 300 mg every eight weeks, there was complete, sustained resolution of plaques and keratoderma leaving hyperpigmentation. Ustekinumab is also used. Response with both drugs has been observed in patients with the CARD14 mutation, but also in those without it. For CAPE syndrome, researchers observed an excellent response to ustekinumab at 90 mg every 12 weeks. For neurofibromatosis, she proposed selumetinib, approved by the FDA in May 2020, showing an overall response of 68% in children with symptomatic inoperable NF1-PN, with improvement in pain and a manageable adverse event profile. It is used as a neoadjuvant to surgery. The dose is 25 mg/m² twice daily, 13 hours apart.

For tuberous sclerosis complex, mTORC1 is sensitive to rapamycin. For naevic dermal melanocytosis (Ota/Ito/Hori/Hodano), she pointed to Q-switched Nd:YAG 1064 nm laser, suggesting starting before five years old for better results and no recurrences in those achieving 95% elimination. For PROS syndrome, she proposed PI3K inhibitors, and for CLOVES syndrome, alpelisib.

Metformin, beyond diabetes.

Presenter: Dr Manuel Dario Franco.

Classically used to lower blood glucose levels, this simple biguanide from the 1950s had been pigeon-holed for type-2 diabetes. The drug's pleiotropy has now been demonstrated: a single molecule with multiple hidden mechanisms capable of regulating the entire state of the human body, beyond glycaemia. This is made possible by activating AMPK when under metabolic stress, which inhibits mTORC1, ordering cellular austerity and halting inflammation by blocking NF. This reduces pro-inflammatory cytokines (IL-1, IL-6, TNF-alpha), suppressing the NF-kB pathway. It also decreases IGF-1, reducing free testosterone levels, regulating hormonal chaos. Another function is anti-angiogenic, slowing down the excessive proliferation of blood vessels by blocking VEGF and MMP-9. Finally, it combats cellular oxidative stress (ROS) by increasing enzymes such as SOD. As the skin is a highly metabolic organ, improving insulin sensitivity and curbing lipogenesis "turns off" inflammation at its systemic root, not just at the surface. Because of all this, it is used in polyendocrine metabolic ovarian syndrome (formerly PCOS), producing insulin sensitisation, which lowers IGF-1 and androgen levels, so that the sebaceous glands stop their hyperproduction; ovulation cycles are regulated and inflammatory lesions disappear. It is equally effective in reducing acne lesions compared to oral contraceptives, with no increased risk of venous thromboembolism. This makes it useful in refractory and late-onset acne.

For HS, it inhibits mTORC1 and directly blocks NLRP3, halting the destructive cascade that causes deep inflammatory lesions, restoring quality of life. For rosacea, it reduces VEGF and promotes MMP-9 degradation, eliminating erythema. For psoriasis, it significantly improves PASI by targeting the underlying metabolic syndrome and reducing oxidative stress. For melasma, it became an unexpected breakthrough with its topical use at 30%, reducing the accumulation of cAMP in cells, which suppresses MITF, the master survival gene of the melanocyte, by shutting off the transcription of colour-producing proteins (tyrosinase, TRP-1, TRP-2), leading to cell whitening. For acanthosis nigricans, aggressive doses of 1500 to 2000 mg per day for more than six months are used to directly attack the metabolic root. Its side effects are manageable, predominantly mild gastrointestinal problems at the start of treatment, and its safety is very high.

Plenary-session lecture. severe cutaneous adverse reactions to medication: towards precision diagnostics and targeted therapies.

Report drafted by Dr Silvina Maldonado, Argentina

Presenter: Dr Lars French.

The skin is one of the organs most frequently affected by the side effects of drugs. Skin side effects affect 0.1-1% of patients, rising to 1-5% for some high-risk drugs (NSAIDs, antibiotics, anticonvulsants), and in hospitalised patients it reaches 5-10%. Predisposing factors include extreme age, polymedication, immunosuppression, viral infections, history of HLA in some cases genetically linked to severe skin reactions and enzyme defects.

Of these side effects, 98% are benign and 2% are severe. Dr French suggests doctors should be concerned about certain features that may increase the suspicion of severity: facial oedema, marked eosinophilia, mucosal lesions or conjunctivitis, burning sensation in the eyes or pain in the skin, greyish lesions and epidermal detachment or erosions. He spoke about acute generalised exanthematous pustulosis (AGEP), drug reaction with eosinophilia and systemic symptoms (DRESS), Stevens-Johnson syndrome (SJS), and toxic epidermal necrolysis (TEN). For AGEP, beta-lactam antibiotics, macrolides, cephalosporins, and sulphonamides are involved in >80% of cases. Other drugs: Hydroxychloroquine, terbinafine, diltiazem. Onset is rapid (<48 hours), accompanied by fever and neutrophilia, while recovery can be rapid and spontaneous. Presenting a new paradigm (2025-2026), he spoke of AGEP as a variant of drug-induced generalised pustular psoriasis (GPP), forming part of a unified spectrum of IL-36-related pustulosis; the immune response patterns and immune cell composition of the two are indistinguishable. Non-drug-induced GPP differed from AGEP only by increased Th17-related gene expression and higher neutrophil counts. On the other hand, DRESS appears within three weeks after the patient begins taking the drug responsible, and is accompanied by eosinophilia and systemic symptoms. The most commonly implicated drugs are: antiepileptics, allopurinol, sulphonamides, and other antibiotics, including vancomycin. Mortality can be as high as 10%, with predictive risk factors being age >57 years, renal failure, ICU admission, and again, facial oedema is a marker of severity. Multicentre cohort studies found that a low haemoglobin to red blood cell size variation ratio, elevated platelet to lymphocyte ratio, and lower monocyte counts predict greater severity.

As a new development, a study by von Wachter et al. (2025) used serum proteomics and identified three immune endotypes: Group 1, characterised by increased IL-17 and milder immune activation, being older patients with shorter drug latency, but longer hospitalisation. Group 2, marked by a pronounced Th17/type 3 response, with increased toll-like receptor signalling and mainly HIV-positive patients.

Group 3, defined by elevated eosinophil counts and elevated type 2 cytokines/eosinophil-associated mediators (MCP-4/CCL13). All patients showed increase in the type 2/eosinophil axis (IL-4, IL-5, IL-13) and proinflammatory mediators (IL-6, IFN-gamma, CXCL9) compared to the healthy control group. If validated, this could make patient stratification and a targeted treatment approach possible. Another study proposed that tofacitinib intervention can be used for disease control, as can antivirals, which suppressed drug-induced T-cell proliferation in vitro, supporting the role of JAK-STAT and HSV pathways in mediating adverse drug effects, as they found that certain immune cells (CD4+ lymphocytes) were enriched with HSV-6b DNA. Also in 2025, a study on prolonged (>8 weeks) versus short-duration DRESS was published, showing that JAK inhibitors (tofacitinib and upadacitinib) decrease in vitro CCL1 and CXCL10 release, successfully treating patients with prolonged DRESS, with JAK inhibitors thus representing a targeted therapy for prolonged or corticosteroid-refractory DRESS. In a publication of five cases treated with IL-5 inhibitors (mepolizumab or benralizumab) there was a rapid decrease of eosinophils in a mean of 1.4 days, with no recurrence during prolonged follow-up. Notably, one patient who had a recurrence with mepolizumab achieved long-lasting suppression by rotating to benralizumab with only one dose.

Regarding SJS and TEN, progression is very rapid and can be fatal within days; rapid biopsy is imperative for differential diagnosis. In recent years, the drugs suspected of causing these conditions have changed considerably, with mogamulizumab, cobimetinib, pralatrexate, vemurafenib, nivolumab, and pembrolizumab joining the traditional list. Adequate supportive care management in the acute stage immediately after discontinuation of the implicated drug is very important. He spoke about finding consensus based on the Delphi method. There is no specific therapy, with systemic corticosteroids being the statistically significant therapy; cyclosporin and ETN with cyclosporin are statistically unstable. In another study, JAK inhibitors showed efficacy in TEN patients, albeit off-label (clinical study in preparation).

They used methylprednisolone at 80 mg per day between days zero and four and then 60 mg per day between days four and eight, associating abrocitinib at 200 mg between days four and eight, and then 100 mg between days eight and 14, stopping the death of keratinocytes and showing that this therapy could change the course of the deadly disease by offering rapid control of the lesions. He also pointed to a study in which tofacitinib-baricitinib or abrocitinib-upadacitinib were indicated, showing that JAK inhibitors are safe and effective, representing a potential targeted curative therapy. In conclusion, progress in deciphering the pathogenesis of SCARs opens the prospect for therapeutic precision: - AGEP: steroids as a first line and IL-17 or IL-36 antagonists as a second line. - DRESS: steroids as a first line; IL-5 antagonists/JAK inhibitors as a second line. Finally, SJS/TEN: JAK inhibitors a potential first-line treatment.

NAOS SYMPOSIUM: Advanced photoprotection: new challenges and clinical relevance throughout life

Report drafted by Dr Silvina Maldonado, Argentina

Presenters: Dr Sergio Shalka and Dr Carla Castro.

The symposium focussed on the importance of visible light (VL), as recent research has shown that this part of the solar spectrum impacts the skin in ways that traditional sunscreens (focussed only on UV rays) do not cover. Dr Shalka began by discussing the key points of the 2025 Consensus Conference in Amsterdam, which brought together experts to define harmonised standards and methods for assessing the protection offered by photoprotectors against visible light (400-500 nm).

It was shown that only visible light from the sun has a photobiological effect on the skin (refuting speculation about light from electronic devices), inducing cutaneous oxidative stress, inflammation, and direct DNA damage. Its effects are: hyperpigmentation in phototypes III and above, erythema in all skin types, and at least currently there is no evidence that visible light contributes to photoageing and photocarcinogenesis. Photoprotection against visible light should be recommended for people with a high phototype, for those suffering from or at risk of melanocytic hyperpigmentation disorders, and for certain types of dermatoses. Then, Dr Castro discussed the issue of visible light exposure in childhood and adolescence, which is of crucial importance as most sun damage is acquired at these stages. Animal studies have shown that for squamous cell carcinomas, the cumulative UV dose is significant, whereas for melanoma, the crucial factor is sunburn, particularly in childhood. Dangerous trends are emerging in the form of new behaviours and challenges promoted on social media: the myth of the “sun callus”, “sunburn art”, the inverse use of the UV index, and movements claiming that sunscreens contain harmful chemicals. Doctors must fight a daily battle to educate patients and their parents.

Regarding the safety of new sunscreens, Dr Shalka said that the 500 daltons rule is imposed for lower absorption; as for the risk of inorganic particulate sunscreens, penetration is minimal; however, they should be banned in aerosols and for the lips because of systemic pulmonary and genotoxicity risk. In conclusion, the studies that point to hormonal and ecological risks were carried out with doses much higher than those used in practice. None of the international regulatory agencies have banned the use of chemical sunscreens. He then spoke about vitamin D and photoprotectors. Different studies have shown that they can reduce vitamin D production, but do not lead to deficiency: rather, it is necessary to consider their misapplication in terms of amount, areas, and the time of exposure, as users believe they are protected and remain exposed for longer periods of time.

He then mentioned that to prevent photoageing, there should be high UVA protection; sunscreens can contain antioxidants and age-fighting active ingredients, preferably with colour to prevent melasma and lentigines. For skin cancer, SPF >60, as high as possible, is suggested, in addition to physical measures and oral photoprotection all year round. For melasma, SPF >60, UVA protection as high as possible, VL protection is a must, physical measures and oral photoprotection all year round. For post-inflammatory hyperpigmentation, SPF >60, UVA protection as high as possible, VL protection is a must, use at least 15 days before procedures or after signs of skin inflammation have disappeared; opaque skin protectors may be recommended for high-risk patients and oral photoprotection is highly recommended. To conclude, Dr Castro spoke about the risks related to structural differences between infant and adult skin, about melanogenesis as a marker of actinic damage and a marker of melanoma risk (lower at 6-12 months compared to 16-24 months).

As for the number of melanocytic naevi, their development is a sign of actinic damage and a marker of melanoma risk: an epidemiological study in Australia showed that the presence of more than 10 melanocytic naevi of at least 5 mm multiplied by 9.9 the risk of melanoma in childhood, and a comparative study of children of the same age in Australia versus Great Britain showed a higher number of naevi (seven times higher) in areas of greater exposure and in more irradiated areas of the body. It was also observed that the presence of BRAF mutations in both acquired melanocytic naevi and melanoma is associated with increased exposure in childhood. With regard to risks associated with immunosuppression, animal studies have shown that UV radiation has immunomodulatory effects, both negative (favouring carcinogenesis) and positive (preventing autoimmune processes), although the impact on children is unknown. Regarding the risk of topical or systemic adverse effects, she again said that there is no evidence of this as they are relatively large molecules, which limits their absorption beyond the epidermis and their interaction with the immune system.

She recommended inorganic sunscreens such as zinc dioxide and titanium dioxide, even if their cosmetic qualities are not as desirable. Regarding the risk of vitamin D deficiency, supplements are recommended for infants under one year of age on the day of birth or by use of fortified formulas, and for older children or adolescents who do not ingest 400 IU per day in their diet. Finally, she suggested adding an organic sunscreen to the inorganic sunscreen to ensure SPF 50.

SYMPOSIUM: game changers: leaders transforming dermatology. ilds wcd 2027

Report drafted by Dr Silvina Maldonado, Argentina

Presenters: Dr Lars French and Dr Luca Borradori

Cutaneous adverse reactions.

Presenter: Dr Lars French. (See above).

Autoimmune blistering diseases: What's new with autoimmune blistering diseases, Focus on pemphigus and pemphigoid.

Presenter: Dr Luca Borradori.

Fast and correct diagnosis, as early as possible, is essential and the aim is to minimise the damage caused by prolonged use of corticosteroids and immunosuppressants, and to prevent relapses. The lack of effective and safe therapies results in a lack of options for medium- and long-term management, with an unsatisfactory profile of current therapies and only partial responses.

For pemphigus, based on the guidelines published in the JEADV in 2020, the first-line treatment is rituximab, with two IV infusions of 1 g each, with an interval of two weeks between them, and maintenance with reinforcements of 0.5 g to 1 g in months 12 and 18; and prednisolone 0.5 mg/kg to 1.5 mg/kg per day, with gradual reduction in mild cases over a period of four months, and in moderate to severe cases over a period of six months. Second-line treatment: azathioprine, MMF, MTX, dapsone. As a third-line treatment (for refractory or complex cases): IVIG 2 g/kg/month, cyclophosphamide, immunoadsorption.

The combination of treatments has been shown to be superior to corticosteroids alone. Rituximab has been highlighted as a game-changer for treatment, with a higher survival rate and lower relapse rate. With regard to pemphigoid, the 2022 S2K guidelines propose, for moderate to severe forms, topical superpotent corticosteroids on the whole body once daily and prednisone 0.5 to 0.75 mg/kg/day for nine to 12 weeks. As a second-line treatment, consider doxycycline, dapsone, MTX, AZA, MMF, rituximab, IVIG, and immunoadsorption, although these may have inconsistent responses, difficult treatments, side effects, and frequent relapses. He pointed to the NanoString transcriptomics study of patients with bullous pemphigoid: it analysed gene expression and identified a strong Th2 immune response, justifying the use of targeted therapies to inhibit Th2 response and inflammatory cell activity. A 2024 review studied dupilumab 600 mg and then 300 mg every 2 weeks as a monotherapy or in combination, controlling 70-100% of cases in only four weeks and with complete response in 30-100%. Approximately 50% were able to discontinue corticosteroids by the sixth month. The risk of relapse after one year is 75%, usually before the fourth month; if the four-weekly dosing interval is maintained, the relapse rate drops dramatically to 5%. It is therefore considered a “game changer”, although off-label, and may be very useful for difficult cases, cicatricial pemphigoid, and immunotherapy-triggered pemphigoids. Finally, there is talk of promising results with omalizumab, with 55-85% response, but high recurrence rates (up to 80%). It is an off-label option for difficult cases.