Advancements in Atopic Dermatitis:
Diagnosis, Drug Candidates, and Trial Enhancements

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Introduction

Atopic dermatitis (AD), also known as eczema, is a chronic inflammatory skin disease marked by itchy and inflamed skin. It is one of the most common skin diseases, especially among children, but it can persist into adulthood or start at an older age.

Historically, AD was first distinguished from other forms of dermatitis in the early 20th century, but it has been described in medical literature for centuries under various names such as "prurigo diathesique".

Over the decades, research has evolved from viewing AD merely as an allergic condition to understanding its complex pathophysiology involving epidermal barrier dysfunction, immune dysregulation, and environmental and psychological factors.

This shift has been pivotal in shaping current therapeutic strategies which aim to restore barrier function, reduce immune dysregulation, and manage environmental and lifestyle factors.

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Key Characteristics and an Evolving Understanding

Key Characteristics

- **Chronic and Relapsing Nature:** AD is known for its long-term course punctuated by periods of flare-ups and remissions.
- **Intense Itchiness (Pruritus):** The intensity of the itch often leads to scratching, which exacerbates the skin condition**.**
- **Skin Dryness:** This dryness contributes to the susceptibility of the skin to irritants and pathogens.
- **Eczematous Lesions:** The skin of individuals with AD may show red, inflamed, and swollen areas.
- **Distribution of Lesions:** In infants, lesions commonly appear on the face, scalp, and extensor surfaces of limbs. In children and adults, the lesions typically occur in the flexural areas
- **Skin Flaking and Cracking:** The skin may flake off or crack, which can lead to bleeding and increases risk of infection.
- **Associated Conditions:** AD often coexists with other atopic disorders such as asthma and allergic rhinitis (hay fever).
- **Vulnerability to Infections:** The compromised skin barrier and immune dysregulation associated with AD make patients more susceptible to skin infections.

Perspectives

- **Biological Factors:**
  Atopic dermatitis (AD) is heavily influenced by genetics, particularly mutations in the filaggrin gene affecting skin barrier function. Immunologically, AD involves an imbalance in T-helper cells, with Th2 cells prominent in acute phases and Th1 cells in chronic stages, contributing to its chronic, immune-mediated symptoms.
- **Psychological Factors:**
  Stress plays a significant role in the exacerbation of AD. Stress can worsen symptoms and provoke flare-ups. The release of stress hormones such as cortisol can lead to an altered immune response, exacerbating the Th2-skewed response.
- **Environmental Factors:**
  Environmental influences, such as exposure to allergens (dust mites, pet dander, pollen and certain foods), and microbial elements (specific bacteria and viruses), play a significant role in the manifestation and severity of AD symptoms. Climate factors such as low humidity can dry out the skin further, worsening symptoms.

Diagnostic Evolution and Criteria

ICD-11 Criteria for Atopic Dermatitis

The WHO's 11th Revision of the International Classification of Diseases (ICD-11) classifies atopic dermatitis under "Diseases of the skin" . The criteria focus on chronic or recurrent episodes of itchy skin (pruritus) and typical morphological findings, such as eczema.

Diagnostic Criteria for Atopic Dermatitis

The diagnostic criteria for AD, primarily guided by clinical observation rather than laboratory tests, include:

- **Essential Features:**
  Persistent or recurrent eczema characterized by itching.
- **Typical Morphology and Distribution:**
  Eczema involving flexural surfaces in adults and facial and extensor involvement in infants and children.
- **Chronic or Relapsing History:**
  A history of skin symptoms that follow a chronic or relapsing course.
- **Exclusion of Other Diagnoses:**
  AD should be diagnosed after excluding other more specific forms of dermatitis and skin disorders.

Evolution of Diagnostic Criteria

Historically, the diagnostic criteria for AD have evolved significantly. Earlier definitions were vague and often did not distinguish between various types of eczemas. Over the decades, criteria have been refined to improve specificity and sensitivity, incorporating more detailed descriptions of symptom patterns, associated features, and typical age of onset. The recognition of the familial tendency and atopic background has also been included in more recent criteria.

Impact on Clinical and Research Perspectives

- **Clinical Practice:** Enhanced diagnostic accuracy has improved the management of AD, allowing for more tailored and effective treatment strategies. It has also helped in distinguishing AD from conditions with similar presentations which require different management approaches.
- **Research:** More precise criteria have streamlined the selection of study participants, enhancing the reliability of clinical trials and epidemiological studies. This has facilitated a more accurate understanding of the disease's pathophysiology and the development of targeted therapies.

Epidemiology of Atopic Dermatitis

Prevalence and Incidence

Atopic dermatitis is one of the most common skin conditions worldwide, affecting approximately 20% of children and up to 3% of adults. The prevalence of AD has been increasing over recent decades, particularly in industrialized countries, suggesting the influence of environmental and lifestyle factors. This rise is most noticeable in urban areas and in populations with higher socioeconomic status.

Age-Related Distribution

AD often begins in early childhood, with approximately 60% of cases being diagnosed by the age of one and about 85% by the age of five. While many children outgrow the condition, a significant number continue to experience symptoms into adulthood or see the condition re-emerge later in life. The manifestation of the disease can change with age.

Racial and Ethnic Disparities

Studies have shown that AD is more prevalent among African American and Asian populations compared to Caucasian populations. These differences may be influenced by genetic factors, environmental exposures, and access to healthcare. African American children are often reported to have more severe and persistent forms of the disease.

Geographic Variation

Geographic factors also play a significant role in the epidemiology of AD. Higher rates of AD are observed in urban settings and in colder climates, where there are reduced levels of sunlight exposure and drier air conditions, which can exacerbate skin dryness and barrier dysfunction. Conversely, rural environments and exposure to a wider range of microbes during early childhood have been associated with lower rates of AD, supporting the "hygiene hypothesis" which suggests that increased microbial exposure may protect against allergic diseases.

Geographic Variation

Genetics play a substantial role in the risk of developing atopic dermatitis. Having one or more family members with AD or other atopic diseases (asthma or allergic rhinitis) significantly increases an individual's risk. Specific genes, such as those involved in the skin barrier function (e.g., filaggrin) and immune response, have been linked to AD. Twin studies suggest a strong heritable component, with concordance rates significantly higher in monozygotic compared to dizygotic twins.

Key Clinical Trial Endpoints in Atopic Dermatitis

Primary Endpoints

1. Investigator's Global Assessment (IGA):
The IGA is the FDA-preferred primary endpoint for atopic dermatitis trials. It assesses the overall severity of the disease at a single point in time, combining clinical signs like erythema, edema, and lichenification into a standardized scale.

2. Scoring Atopic Dermatitis (SCORAD) Index:
This index is specifically designed to measure the extent and severity of AD and includes an assessment of subjective symptoms such as pruritus and sleep loss, which are particularly problematic in AD.

3. Patient-Oriented Eczema Measure (POEM):
A patient-reported outcome measure developed for AD. It assesses the frequency of symptoms such as itching and sleep disturbance over a seven-day period, reflecting the patient's perspective on the severity and impact of their disease.

4. Eczema Area and Severity Index (EASI):
While also used in other skin conditions, EASI is particularly pivotal in AD trials due to its detailed assessment of eczema severity and coverage, incorporating both patient and clinician observations.

Secondary Endpoints

1. Atopic Dermatitis Severity Index (ADSI):
ADSI is a composite score that includes the assessment of five signs each rated on a scale of 0-3.

2. Pruritus and Sleep Scales:
Specific scales for assessing pruritus and sleep disturbances are frequently used due to the high prevalence and impact of these symptoms.

3. Transepidermal Water Loss (TEWL):
While not a direct clinical outcome, TEWL measurements are often used in AD research to assess the integrity of the skin barrier.

4. Quality of Life Instruments:
Such as the Dermatitis Family Impact (DFI) questionnaire or the Children's Dermatology Life Quality Index (CDLQI).

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Challenges in Determining Endpoints in Atopic Dermatitis Clinical Trials

1. Scoring Atopic Dermatitis (SCORAD) Index:

- **Subjectivity:** The SCORAD Index includes subjective components which can vary significantly between patients.
- **Complexity in Scoring:** Accurately scoring the extent and severity of lesions requires trained professionals, which can be a limitation in settings lacking specialized training.

2. Patient-Oriented Eczema Measure (POEM):

- **Patient Bias:** POEM is subject to bias based on individual tolerance, perception of disease, and even the patient's mood.
- **Limited Clinical Parameters:** POEM does not directly measure clinical signs of improvement that might be visible to a healthcare provider.

3. Eczema Area and Severity Index (EASI):

- **Inter-observer Variability:** Different clinicians may score severity and extent slightly differently, which can lead to variability in results, especially in multicenter trials.

4. Atopic Dermatitis Severity Index (ADSI):

- **Complexity:** ADSI involves multiple clinical signs, each requiring separate assessment, which can complicate the scoring process.

5. Pruritus and Sleep Scales:

- **Subjective Nature:** Both pruritus and sleep quality are self-reported and can be influenced by external factors unrelated to the treatment or disease state.
- **Lack of Standardization:** Various scales exist to measure itch and sleep, and differences between these scales can affect comparability of data across studies.

6. Transepidermal Water Loss (TEWL):

- **Environmental Influences:** TEWL measurements can be affected by external environmental conditions such as humidity and temperature, which need to be controlled during testing.
- **Equipment and Expertise:** Reliable measurement of TEWL requires specialized equipment and technical expertise, which may not be available in all research settings.

7. Quality of Life Instruments Tailored to AD:

- **Cultural Sensitivity:** Quality of life instruments may not be equally applicable in different cultural or demographic contexts, which can affect the generalizability of the findings.
- **Longitudinal Relevance:** Changes in quality of life may occur over longer periods than those typically assessed in short-term clinical trials, requiring extended follow-up to capture meaningful data.

Commonly prescribed FDA approved treatments for Atopic Dermatitis

Drug Name	Year of Approval	Sponsor	Mechanism of Action	Therapeutic Effects
Dupilumab	2017	Sanofi/Regeneron	Interleukin-4 and Interleukin-13 inhibitor	Reduces inflammation and improves symptoms of atopic dermatitis, including itch and rash coverage. Suitable for moderate to severe cases.
Crisaborole	2016	Pfizer	Phosphodiesterase 4 (PDE-4) inhibitor	Reduces skin inflammation and is used for mild to moderate atopic dermatitis. Appropriate for patients aged two years and older.
Pimecrolimus	2001	Novartis	Calcineurin inhibitor	Specifically targets immune responses that cause skin inflammation and flare-ups. Used for short-term and non-continuous chronic treatment of mild to moderate forms.
Tacrolimus	2000	Astellas Pharma	Calcineurin inhibitor	Reduces itching and inflammation. Used for moderate to severe atopic dermatitis and as second-line therapy for the prevention of flare-ups.
Abrocitinib	2022	Pfizer	JAK1 selective inhibitor	Approved for the treatment of moderate to severe atopic dermatitis in adults and pediatric patients 12 years and older. It works by modulating the immune response to reduce inflammation and alleviate symptoms.

Approved Product Analysis for Dupilumab

Efficacy Results of DUPIXENT With or Without Concomitant TCS at Week 16 (FAS)

Trial	Endpoint	Scales Used	Time Point	Sample Size	Magnitude of Improvement
Trial 1	IGA 0 or 1	IGA	16 weeks	DUPIXENT: 224, Placebo: 224	DUPIXENT: 38%, Placebo: 10%
	EASI-75	EASI	16 weeks	DUPIXENT: 224, Placebo: 224	DUPIXENT: 51%, Placebo: 15%
	EASI-90	EASI	16 weeks	DUPIXENT: 224, Placebo: 224	DUPIXENT: 36%, Placebo: 8%
	≥4-point improvement in NRS	Peak Pruritus NRS	16 weeks	DUPIXENT: 213, Placebo: 212	DUPIXENT: 41%, Placebo: 12%
Trial 2	IGA 0 or 1	IGA	16 weeks	DUPIXENT: 233, Placebo: 236	DUPIXENT: 36%, Placebo: 9%
	EASI-75	EASI	16 weeks	DUPIXENT: 233, Placebo: 236	DUPIXENT: 44%, Placebo: 12%
	EASI-90	EASI	16 weeks	DUPIXENT: 233, Placebo: 236	DUPIXENT: 30%, Placebo: 7%
	≥4-point improvement in NRS	Peak Pruritus NRS	16 weeks	DUPIXENT: 225, Placebo: 221	DUPIXENT: 36%, Placebo: 10%
Trial 3	IGA 0 or 1	IGA	16 weeks	DUPIXENT + TCS: 106, Placebo + TCS: 315	DUPIXENT + TCS: 39%, Placebo + TCS: 12%
	EASI-75	EASI	16 weeks	DUPIXENT + TCS: 106, Placebo + TCS: 315	DUPIXENT + TCS: 69%, Placebo + TCS: 23%
	EASI-90	EASI	16 weeks	DUPIXENT + TCS: 106, Placebo + TCS: 315	DUPIXENT + TCS: 40%, Placebo + TCS: 11%
	≥4-point improvement in NRS	Peak Pruritus NRS	16 weeks	DUPIXENT + TCS: 102, Placebo + TCS: 299	DUPIXENT + TCS: 59%, Placebo + TCS: 20%

Approved Product Analysis for Dupilumab (cont.)

Efficacy Results of DUPIXENT With or Without Concomitant TCS at Week 16 and 52

Endpoint	Scales Used	Time Point	Sample Size	Magnitude of Improvement
Responder at Week 16 and 52	IGA 0 or 1	Weeks 16 & 52	DUPIXENT+TCS: 89, Placebo+TCS: 264	DUPIXENT+TCS: 22%, Placebo+TCS: 7%
Responder at Week 16 but Non-responder at Week 52	IGA 0 or 1	Week 16 to 52	DUPIXENT+TCS: 89, Placebo+TCS: 264	DUPIXENT+TCS: 20%, Placebo+TCS: 7%
Non-responder at Weeks 16 and Responder at Week 52	IGA 0 or 1	Week 16 to 52	DUPIXENT+TCS: 89, Placebo+TCS: 264	DUPIXENT+TCS: 13%, Placebo+TCS: 6%
Non-responder at Weeks 16 and 52	IGA 0 or 1	Weeks 16 & 52	DUPIXENT+TCS: 89, Placebo+TCS: 264	DUPIXENT+TCS: 44%, Placebo+TCS: 80%
Overall Responder Rate at Week 52	IGA 0 or 1	Week 52	DUPIXENT+TCS: 89, Placebo+TCS: 264	DUPIXENT+TCS: 36%, Placebo+TCS: 13%

Approved Product Analysis for Crisaborole

Primary Efficacy Outcomes in Subjects with Mild to Moderate Atopic Dermatitis at Day 29

Trial	Endpoint	Scales Used	Time Point	Sample Size	Magnitude of Improvement
Trial 1	Success in ISGA	ISGA	Day 29	EUCRISA: 503, Vehicle: 256	32.8% vs 25.4% achieved ISGA Clear or Almost Clear with ≥2-grade improvement
Trial 2	Success in ISGA	ISGA	Day 29	EUCRISA: 513, Vehicle: 250	31.4% vs 18.0% achieved ISGA Clear or Almost Clear with ≥2-grade improvement

Approved Product Analysis for Pimecrolimus

Combined Efficacy Results at Endpoint for Two 6-week Trials of ELIDEL Cream

Trial	Endpoint	Scales Used	Time Point	Sample Size	Magnitude of Improvement
ELIDEL Trials	Global Assessment: Clear	Physician's Global Evaluation	6 weeks	ELIDEL: N=267, Vehicle: N=136	ELIDEL: 10%, Vehicle: 4%
ELIDEL Trials	Global Assessment: Clear or Almost Clear	Physician's Global Evaluation	6 weeks	ELIDEL: N=267, Vehicle: N=136	ELIDEL: 35%, Vehicle: 18%
ELIDEL Trials	Global Assessment: Clear to Mild Disease	Physician's Global Evaluation	6 weeks	ELIDEL: N=267, Vehicle: N=136	ELIDEL: 67%, Vehicle: 40%

Approved Product Analysis for Abrocitinib

Efficacy Results of CIBINQO at Week 12 in Subjects with Moderate-to-Severe Atopic Dermatitis

Trial	Endpoint	Scales Used	Time Point	Sample Size	Treatment Arms	Magnitude of Improvement
AD-1	IGA response, EASI-75 response	IGA, EASI	12 weeks	N=387	- CIBINQO 200 mg QD - CIBINQO 100 mg QD - Placebo	IGA 0 or 1: 44% (200 mg), 24% (100 mg) vs 8% (Placebo); EASI-75: 62% (200 mg), 40% (100 mg) vs 12% (Placebo)
AD-2	IGA response, EASI-75 response	IGA, EASI	12 weeks	N=391	- CIBINQO 200 mg QD - CIBINQO 100 mg QD - Placebo	IGA 0 or 1: 38% (200 mg), 28% (100 mg) vs 9% (Placebo); EASI-75: 61% (200 mg), 44% (100 mg) vs 10% (Placebo)
AD-3	IGA response, EASI-75 response	IGA, EASI	12 weeks	N=837	- CIBINQO 200 mg QD - CIBINQO 100 mg QD - Placebo - Dupilumab 300 mg Q2W SC	IGA 0 or 1: 47% (200 mg), 34% (100 mg) vs 14% (Placebo); EASI-75: 68% (200 mg), 58% (100 mg) vs 27% (Placebo)

Drugs That Did Not Achieve Their Intended Endpoints in AD Trials

Name of Drug	Clinical Trial Identifier	Findings
Pimecrolimus	GDCT0372631	• There is no difference in the number of flares experienced and in the trend of adverse events between elidel 1% and vehicle, with an increase in infection over a year compared to vehicle
Montelukast Sodium	NCT00559546	• Montelukast is ineffective and is not recommended as a general drug to treat all the symptoms of atopic syndrome, but, should be considered as a major drug for asthma and rhinitis
Betamethasone	GDCT0350218	• Whole-body treatment with topical corticosteroids leads to systemic exposure but appears not to compromise glucose metabolism during short-term use, which may be a result of reduced systemic inflammatory activity. The negative impact on bone formation can be regarded an adverse effect of topical corticosteroids
Tradipitant	NCT03568331	• The study did not meet its primary endpoint in reduction of pruritus across the overall study population and produces a large and rapid antipruritic effect in mild atopic dermatitis
Maxtrex	NCT00809172	• Methotrexate is less effective compared to cyclosporine in moderate to severe intensity atopic dermatitis but with a better safety profile

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Common Failure Points in Atopic Dermatitis Clinical Trials

1. Variability in Disease Severity and Presentation:

This heterogeneity makes it challenging to standardize treatment effects, measure consistent outcomes, and often leads to mixed results.

2. Inconsistent Use of Scoring Systems:

The use of different scoring systems to assess disease severity and treatment response can lead to inconsistency in results.

3. Subjectivity in Symptom Assessment:

Variability in patient perception and reporting can skew results and complicate the objective evaluation of a treatment's effectiveness.

4. Difficulty in Blinding:

Effective blinding can be difficult, especially in those involving topical treatments, and the control can reveal treatment allocation to the patient or investigator. This can introduce bias in the results.

5. High Placebo Effect:

The psychological impact of receiving care in a trial setting can significantly improve symptoms, making it hard to demonstrate a significant difference between treatment and placebo groups.

6. Compliance Issues:

Non-adherence to treatment regimens is a significant issue in AD trials, particularly with topical treatments that require frequent application.

7. Environmental and Lifestyle Factors:

Controlling for variables in external factors, such as climate and allergens, is challenging but crucial, as their influence can mask true treatment effects.

8. Recruitment and Retention Challenges:

Patients may experience periods of remission due to the fluctuating nature of AD, during which they may drop out of the trial, affecting the study's ability to detect treatment effects.

Safety Concerns and Standard of Care

Safety Concerns

Topical Treatments:

- Corticosteroids: While effective, prolonged use can cause skin thinning, discoloration, and potential systemic absorption.
- Calcineurin Inhibitors: Concerns include potential burning or stinging sensations at the application site.

Systemic Treatments:

- Immunosuppressants: These drugs can be effective in severe cases but carry risks such as kidney damage, increased susceptibility to infections, and potential liver toxicity.
- Biologic Therapies: Side effects can include eye issues such as conjunctivitis, as well as inflammation of the eyelid.

Phototherapy:

- Ultraviolet Light Therapy: Long-term use has been associated with an increased risk of skin cancer and premature skin aging.

Phototherapy:

- There is a risk of systemic allergic reactions, especially in highly sensitive individuals.

Standard of Care

Initial Management:

- The cornerstone of AD treatment involves skin hydration and the use of emollients to maintain skin barrier function. Mild topical corticosteroids are typically used for flare management.
- Education about trigger avoidance and proper skin care techniques is essential for all patients.

Moderate to Severe Cases:

- For more severe manifestations, stronger topical steroids, calcineurin inhibitors, or systemic treatments may be necessary. Choice of treatment is often based on the severity of the symptoms, patient age, and impact on quality of life.
- Biologics like dupilumab have been transformative for many patients, providing targeted immune modulation with fewer systemic side effects.

Maintenance Therapy:

- Long-term management usually includes intermittent use of topical therapies to prevent flares and maintain skin health, combined with ongoing skin care practices.
- Regular follow-up appointments are crucial to adapt the treatment plan as the disease progresses or improves.

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Enhancing Clinical Trial Protocols: Towards FDA Approval

The advancement of treatment options for Atopic Dermatitis relies heavily on the design and execution of effective clinical trials. These trials must not only meet rigorous scientific and ethical standards, but also align with regulatory expectations to ensure approval.

1. Streamlining Protocol Design

Robust Inclusion and Exclusion Criteria:

- Targeted Patient Selection
- Address Comorbidities

Optimized Treatment Regimens:

- Personalized Dosing
- Standardized Application for Topicals

2. Enhancing Endpoint Reliability

Selection of Clinically Relevant Primary Endpoints:

- Exclusion of vIGA (recent validations aim to improve reliability across clinical settings)
- Integration of Patient-Reported Outcomes (PROs)

Incorporation of Biomarkers:

- Biomarkers for Mechanistic Insights

3. Promoting Patient Compliance and Retention

Effective Patient Engagement Strategies:

- Comprehensive Patient Education
- Enhanced Support Systems

Flexible Study Designs:

- Adaptive Design Elements
- Considerations for Dropout Rates

4. Meeting Regulatory Requirements

Comprehensive Data Management:

- High-Quality Data Collection
- Advanced Data Systems

Proactive Regulatory Engagement:

- Early and Ongoing Dialogue
- Transparent Reporting

Notable Key Opinion Leaders in Atopic Dermatitis Research

Name	Specialty	Designation	Location	Brief Bio
Dr. Lynda J Spelman (MBBS, FACD)	Dermatology	Founder, Queensland Institute of Dermatology	Queensland, Australia	Dr. Spelman is the founder and chairperson at the Queensland Institute of Dermatology and the Queensland Skin and Cancer Trust Dr. Spelman has made significant contributions through her extensive research and clinical practice in dermatology. Her work significantly impacts patient care and advances in dermatological therapies.
Dr. Rowland Noakes (MBBS (QLD), FRACGP, FACD)	Dermatology	Dermatologist, Queensland Institute of Dermatology	Queensland, Australia	Dr. Rowland Noakes is a distinguished dermatologist associated with the Queensland Institute of Dermatology in Brisbane, Australia. After joining the Australasian College of Dermatologists Dr Noakes followed his interests in the treatment and prevention of all diseases of the skin, hair and nails and rural and remote health by working as a visiting dermatologist at Gympie Private Hospital, Mater Hospital in Mackay, and Rockhampton Mater Hospital whilst practicing in Brisbane.
Dr. John C Su (MB, BS, MA, MSt, MEpi, MBA, FRACP, FACD)	Pediatric dermatology; Epidemiology; Biostatistics	Director, Eastern Health Australia	Victoria, Australia	Dr. Su is the clinical director of dermatology at Eastern Health, one of Victoria's largest public health services. He is also involved in teaching, particularly at the Eastern Clinical School of Monash University, where he contributes to the education of medical students. Dr. Su is known for his work in pediatric dermatology and has a special interest in eczema. He has contributed to numerous research studies and clinical trials aimed at improving treatments for skin conditions in children. His research and clinical work significantly impact patient care by advancing treatment protocols and improving the quality of life for his patients.
Prof. Deirdre (Dedee) Murrell (MA (Cantab), BMBCh (Oxon), FAAD (USA), MD (UNSW), FACD)	Dermatology	Director, St George Private Hospital	New South Wales, Australia	Professor Murrell serves as the Chair of the Department of Dermatology at St George Hospital in Sydney. She is also a professor at the University of NSW, where she is involved in both teaching and research. Professor Murrell's contributions are significant in the field of dermatological research, particularly concerning blistering skin diseases. She has authored over 400 peer-reviewed articles and several books, significantly influencing the understanding and treatment of skin diseases. She also runs a number of clinical trials particularly in the field of acne and has developed guidelines that are used globally for the treatment of skin conditions.
Dr. Johanna M Kuchel (MB.BS, M.Med, FACD)	Dermatology	Consultant Dermatologist, Skin & Cancer Foundation Australia	New South Wales, Australia	Dr. Johanna M. Kuchel is a consultant dermatologist with over 10 years experience at the Skin and Cancer Foundation in Australia, where she provides specialized care in dermatology and engages in research and education related to skin health and cancer prementioned. Kuchel runs general dermatology clinics, in addition to sub-specialty clinics in hyperhidrosis, cosmetic dermatology, psoriasis and eczema. She is the Principal Investigator of several clinical trials studying novel biological agents for the treatment of psoriasis and eczema.

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If you would like further information on how iNGENū CRO can assist you to conduct your clinical trial, please contact Dr Sud Agarwal or Adam Moodie:

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