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Studies on Vitamin A Signaling in Psoriasis Intercellular Calcium-mediated Cell Signaling in Keratinocytes Cultured from Patients with NF1 Or Psoriasis Role of MyD88 Signaling in the Imiquimod-induced Mouse Model of Psoriasis: Focus on Innate Myeloid Cells *Therapy and Prevention of Atopic Dermatitis and Psoriasis* **Psoriasis and Psoriatic Arthritis Psoriatic Arthritis and Psoriasis The Role of Connexin Mediated Signalling in the Pathogenesis of Psoriasis Establishment of a Novel in Vivo Mouse Model of the IL-17 Signaling Pathway Suitable for Explorations of PK/PD Relationships A Hyper Th17 Response Connects the Psoriasis-associated ACT1 Variant to Skin Inflammation Cytokines as Potential Therapeutic Targets for Inflammatory Skin Diseases** *Novel Genomic Signature Predicts Response to Ruxolitinib Cream in Psoriasis* **Investigation of Phospholipase C/protein Kinase C Signalling Pathways in Skin Relevant to the Pathogenesis of Psoriasis** Targeting Human Inflammatory Skin Diseases With Natural Products: Exploring Potential Mechanisms and Regulatory Pathways Clinical and Basic Immunodermatology Psoriasis and Psoriatic Arthritis **Analysis of the Calcineurin/NFAT Signal Transduction Pathway in Human Keratinocytes, Human Skin and Psoriasis** Therapeutic Advancements in Psoriasis and Psoriatic Arthritis **Model of pathogenesis of psoriasis. Part 1. Systemic psoriatic process.** *Downregulated Caveolin-1 Expression in Monocytes May Contribute to the Pathogenesis of Psoriasis What's New in the Skin?* Psoriatic Arthritis, An Issue of Rheumatic Disease Clinics 41-4, **Psoriatic Arthritis, An Issue of Rheumatic Disease Clinics** *Oxford Textbook of Psoriatic Arthritis Das Psoriasis-Syndrom* In Vitro Th-17 Inflammatory Skin Models as a Promising Tool for Psoriasis Research and Drug Development *Distinct Gene Expression Signatures Differentiate Clinical Response to Ustekinumab Compared to Adalimumab in Psoriasis* POL202 Ameliorates IMQ-induced Psoriasis-like Skin Lesions In Mice By Inhibiting Janus Kinase Pathway **Role of the IL-23/IL-17 Pathway in Chronic Immune-Mediated Inflammatory Diseases: Mechanisms and Targeted Therapies** *Cytokines and Their Signaling in Chronic Inflammatory Diseases and Beyond* **S100 Proteins as Calcium Signaling Mediators and Transglutaminase Substrates in Keratinocyte Biology Psoriasis: Advances in Knowledge and Care, An Issue of Dermatologic Clinics, IL-6 Signals Through PStat3 to Prevent Functional Immune Suppression by Human Regulatory T Cells** *Neutrophil Extracellular Traps (NETs) in der Interleukin-23-induzierten psoriasiformen Dermatitis Role of Apoptosis and Key Canonical Pathways in Psoriasis Plaque Clearance in Response to UVB Phototherapy Psoriasis: New Insights for the Healthcare Professional: 2013 Edition* **IL-36 Receptor Antagonistic Antibodies Inhibit Inflammatory Response in IL-23 Model of Psoriasiform Dermatitis Advances in Psoriasis Immunology of Psoriatic Disease** *Retinoids in Dermatology* **Textbook of Psoriasis**

Downregulated Caveolin-1 Expression in Monocytes May Contribute to the Pathogenesis of Psoriasis Oct 10 2021 Caveolin-1 (Cav-1) is a membrane protein that is essential for the formation of ufb02ask-shaped membrane invaginations known as caveolae. Cav-1 regulates a variety of signaling molecules and receptors, and aberrant Cav-1 expression is involved in a variety of diseases. Our previous study revealed aberrant reduction of Cav-1 in the epidermis of psoriasis patients, which leads to enhanced phosphorylation of STAT3 and cytokine production, suggesting that decreased Cav-1 may contribute to psoriatic inflammation. Not only keratinocytes, but also immune cells are major players in psoriasis. Migrated immune cells produce a variety of effector cytokines which affect keratinocytes proliferation and induce psoriatic inflammation. The purpose of this study was to determine whether Cav-1 is involved in the leucocyte function of psoriasis and investigate their role in the pathogenesis of psoriasis. We first determined Cav-1 expression levels in peripheral blood mononuclear cells (PBMCs) and polymorphonuclear cells (PMNs) isolated from 20 patients with psoriasis and 20 control subjects by quantitative PCR and immunoblotting. Immunocytochemistry and magnetic cell isolation were also conducted to identify which cells were aberrant on Cav-1 expression in PBMCs. mRNA and protein levels of Cav-1 in PBMCs and PMNs were significantly lower in psoriasis patients than control subjects. In PBMCs, significant reduction of Cav-1 was observed in CD14 positive monocytes. In addition we assessed function of Cav-1 decreased monocytes on cytokine production and migration. Cav-1 RNA interference in isolated monocytes resulted in elevated levels of IL-1u03b2 and IL-6 under LPS stimulation, compared to control monocytes. Furthermore, a migration assay revealed that Cav-1 silenced monocytes had enhanced chemotaxis activity induced by MCP-1. Our results suggest that aberrant Cav-1 expression in monocytes may be involved in the pathogenesis of psoriasis by mediating cell migration and cytokine production.

Role of the IL-23/IL-17 Pathway in Chronic Immune-Mediated Inflammatory Diseases: Mechanisms and Targeted Therapies Jan 01 2021

Psoriatic Arthritis, An Issue of Rheumatic Disease Clinics Jul 07 2021 Some people with psoriasis can also develop psoriatic arthritis, when the immune system attacks the joints as well, causing inflammation. Like psoriasis, psoriatic arthritis symptoms flare and subside, vary from person to person, and even change locations in the same person over time. Psoriatic arthritis can affect any joint in the body, and it may affect just one joint, several joints or multiple joints. For example, it may affect one or both knees. This issue will include articles on Genetic and Epigenetic aspects of psoriatic arthritis, Clinical features and diagnostic considerations in psoriatic arthritis, Natural history, prognosis and socioeconomic aspects of psoriatic arthritis, Etiology and pathogenesis of psoriasis and many more! *Oxford Textbook of Psoriatic Arthritis* Jun 06 2021 Psoriatic arthritis, or PsA, is now acknowledged the second most prevalent and important inflammatory arthropathy worldwide. The addition of this new textbook on PsA is a fitting and important inclusion to the Oxford Textbooks in Rheumatology series, written to reflect the significant advances in the field in recent years. With the recent advances in the understanding of pathogenesis, and the development of novel therapies, the Oxford Textbook of Psoriatic Arthritis provides a comprehensive overview of the disease. Each chapter is written by leading clinicians and scientists in the field of psoriatic arthritis, to provide a contemporary view of PsA, and a look into the future directions of research. Covering everything from epidemiology and diagnosis to genetics and pathology, detailed sections on treatment and outcomes provide an invaluable resource for the clinician. The book is also highly illustrated with both clinical images such as x-rays and histological photographs to aid clinical knowledge, and diagrams of the immunology and genetics that underlie the disease. Practical and all-inclusive, with summary boxes to distil the most important information, the Oxford Textbook of Psoriatic Arthritis will prove an invaluable resource for rheumatologists, dermatologists, trainees, and all members of the multidisciplinary team who are interested in recent advances in PsA. *Neutrophil Extracellular Traps (NETs) in der Interleukin-23-induzierten psoriasiformen Dermatitis* Jul 27 2020 Psoriasis is a chronic recurrent inflammatory disease of the skin and joints, often associated with significant morbidity. In spite of its clinical importance, the pathogenesis of psoriasis is not fully understood. Recently, it was discovered that Neutrophil Extracellular Traps (NETs) may be involved in the inflammatory process of this disease, as these networks of chromatin and antimicrobial peptides, expelled by neutrophilic granulocytes, can be found in psoriatic plaques and are colocalized with Interleukin 17, a keyplayer in the signal pathway of Psoriasis. Therefore, the TLR dependency...

IL-6 Signals Through PStat3 to Prevent Functional Immune Suppression by Human Regulatory T Cells Aug 28 2020 Human autoimmune diseases such as psoriasis,

multiple sclerosis, and rheumatoid arthritis are characterized by systemic T cell dysfunction, resulting in chronically activated Th1 and Th17 cells which are insufficiently suppressed by regulatory T cells. The data presented herein demonstrate that the pro-inflammatory cytokine IL-6 directly inhibits human regulatory T cell function, similar to its functions in the mouse. IL-6 is over-expressed in tissue and serum of patients with autoimmune diseases and is produced in high amounts by dermal dendritic cells and endothelial cells in the lesional skin of psoriasis patients. Upon T cell entry to the lesional skin, cells are exposed to high levels of IL-6, which likely contribute to their escape from regulation by Treg cells. We sought to determine the mechanism of action for the anti-tolerigenic properties of IL-6 by examining the signaling pathways downstream of IL-6R in primary human T cells. We show that in both effector and regulatory T cells, IL-6 preferentially signals through Stat3, with moderate activation of Stat1 and minimal MAPK/Erk activation. Inhibition of Stat3 signaling in mixed lymphocyte cultures containing IL-6 restores Treg-mediated suppression, demonstrating that IL-6-mediated loss of Treg suppression requires phosphorylation of Stat3. Criss-cross experiments, in which either effector or regulatory T cells were pre-treated with Stat3 inhibitors, show that pStat3 is required in both T cell populations for IL-6-mediated reversal of Treg function. Similarly, IL-21, which also signals preferentially through pStat3, induces a loss of Treg suppression, in contrast to signaling by IL-27 and IFN-gamma, which signal preferentially through Stat1 and do not inhibit suppression. Lastly, effector T cells stimulated strongly through the TCR are resistant to suppression by Treg and have concurrent Stat3 phosphorylation; inhibition of pStat3 restores functional suppression by Treg. Collectively, our data suggest that Stat3 signaling is crucial for mediating effector cell resistance to Treg suppression and that kinase-specific inhibitors may hold therapeutic promise for the treatment of autoimmune disease.

Cytokines and Their Signaling in Chronic Inflammatory Diseases and Beyond Nov 30 2020

Investigation of Phospholipase C/protein Kinase C Signalling Pathways in Skin Relevant to the Pathogenesis of Psoriasis May 17 2022

POL202 Ameliorates IMQ-induced Psoriasis-like Skin Lesions In Mice By Inhibiting Janus Kinase Pathway Feb 02 2021 Psoriasis pathophysiology is characterized by abnormal keratinocyte proliferation and immune cell infiltration involving the innate and adaptive immune systems in the dermis and epidermis. As the number of patients with psoriasis continues to increase, the therapeutic agent beyond the concept of symptom relief has not been released other than steroids and biologics. JAK signaling pathway is implicated in the pathogenesis of inflammatory diseases, recent and ongoing psoriasis related studies focus on JAK inhibition (ex:Tofacitinib). Our platform technology, POLIGOTM is ASO (antisense-oligonucleotide) based technology using PNA (peptide nucleic acid). POLIGOTM is chemically improved properties (stability, delivery ability) of PNA and regulated PNA particle size to nano-grade. We evaluated in vivo therapeutic efficacy of POL202 (cream type of JAK1/2 targeted POLIGOTM) after topical treatment in 5% IMQ-induced C57BL/6 mouse ear. The group of 2 nmole POL202 (daily for 2 weeks) improved psoriasis pathogenesis and demonstrated more higher efficacy than positive control, LIDOMEX. Rapid decreases in clinical score (PASI), trans-epidermal water loss (TEWL) and JAK1/2 expression level were observed. In H&E data, POL202 was decreased epidermis thickness and inhibited keratinocyte proliferation. Furthermore, histological analyses of POL202 cream group showed the reduction in Ki67+ epidermal hyperplasia, CD11c+ neutrophils and CD3+ filtration T cell compared with positive control. In addition, POL202 group significantly reduced serum level of IL-17A, IgE and the abundance of CCR6+ T cell (the major IL-17A producer) derived from spleen. Following our technology POL202, not only have ability to targeted treatment, but also enhances the topical delivery by using cream formulation. Finally, POL202 cream mainly alleviates IMQ-induced psoriasis-like pathogenesis through inhibition of JAK1/JAK2 involving IL-17A related Th17 immune response.

Textbook of Psoriasis Dec 20 2019 Textbook of Psoriasis is a comprehensive and highly illustrated guide to this dermatological disorder. Enhanced by over 300 full colour images and illustrations. The inclusion of several recent dermatology research studies pertaining to psoriasis make this book and authoritative and up to date resource for dermatologists.

IL-36 Receptor Antagonistic Antibodies Inhibit Inflammatory Response in IL-23 Model of Psoriasiform Dermatitis Apr 23 2020 Psoriasis vulgaris (PV) results from activation of IL-23/Th17 immune pathway and is further amplified by skin responses. Among skin derived pro-inflammatory cytokines, IL-36 family members are highly upregulated in PV patients and play a critical role in general pustular psoriasis. However, there is scant data showing crosstalk between the IL-23 and IL-36 pathways in PV. Herein we interrogate if functional inhibition of IL-36 receptor (IL-36R) in the IL-23-induced mouse models of psoriasiform dermatitis drives down the skin inflammation. Anti-IL-36R antibodies (mAbs) were validated in vitro by inhibiting IL-36uf061 induced secretion of mouse CXCL1 from NIH 3T3 cells. In vivo antibody target engagement was validated by inhibition of CXCL1 production in an acute model of IL-36uf061 systemic injection. In addition, anti-IL-36R mAbs were able to inhibit tissue inflammation and inflammatory gene expression in an IL-36uf061 ear injection model of psoriasiform dermatitis demonstrating adequate target coverage in the ear skin. To elucidate the possible role of IL-36 signaling in IL-23/Th17 pathway we tested the ability of anti-IL-36R mAbs to inhibit skin inflammation in both IL-23 ear injection and IL-23 mini-circle mouse models. We show that inhibiting the IL-36 pathway results in significant but modest attenuation of skin thickening, inflammatory cell infiltration and psoriasis-relevant gene expression in both models. Taken together, our data suggests a role for IL-36 signaling in the IL-23/Th17 signaling axis in PV.

Establishment of a Novel in Vivo Mouse Model of the IL-17 Signaling Pathway Suitable for Explorations of PK/PD Relationships Sep 21 2022 The IL-17 signaling pathway is a key driver of psoriasis and a validated target for treatment of the disease. The purpose of the present study was to establish a mechanistic in vivo mouse model of IL-17 signaling, suitable for modelling of PK/PD relationships for new psoriasis therapies inhibiting this pathway.To obtain an IL-17 induced response in skin, which is the relevant tissue for psoriasis translations, an intradermal cytokine injection model was selected, with proximal biomarkers of clinical relevance as the primary endpoints. The biomarkers were measured in ear tissue lysate 0.5-24 hours after cytokine injection, analyzing both mRNA and protein levels.When IL-17A was injected intradermally in one ear of BalbC mice, a clear upregulation of several proximal biomarkers was seen, including CXCL1 and CCL20. However, the response was only evident at very high concentrations of IL-17A. To obtain a response with lower, clinically relevant IL-17 levels, TNFuf061 - which is known to act in synergy with IL-17 - was co-injected with IL-17A. TNFuf061 significantly potentiated the response of IL-17A, resulting in robust upregulation of proximal biomarkers at IL-17A concentrations that were within the concentration range reported in skin samples from psoriasis patients. The upregulated biomarkers included CXCL1 and CCL20, with the peak response observed 1-4 hours after the intradermal cytokine injection.To confirm that the biomarker response was dependent on IL-17A, we determined the therapeutic effect of an anti-IL17A antibody. Indeed, antibody doses ranging from 1 to 10 mg/kg inhibited the biomarker response. In conclusion, we have established a mechanistic mouse model of the IL-17 signaling axis which enables in-depth investigations of the PK/PD relationships of therapeutic interventions targeting this signaling pathway.

Das Psoriasis-Syndrom May 05 2021 Psoriasis ist eine entzündliche, chronische Hauterkrankung, die auffällige Assoziationen mit verschiedenen internistischen Erkrankungen und einer Anzahl genetisch fixierter Risikofaktoren zeigt. Hier wird der Versuch unternommen, ein zusammenfassendes Modell des Psoriasis Symptomkomplexes, das Psoriasis-Syndrom, vorzustellen. Es beschreibt, wie die spezifisch psoriatischen Veränderungen der Haut, die durch das Zusammentreffen unterschiedlicher Risikofaktoren und Trigger ausgelöst werden, auf zellulärer Ebene durch eine gestörte Regulation des second messenger cyclo-Adenosinmonophosphat (cAMP) und eine defiziente cAMP-Versorgung entstehen. Es wird dargestellt, wie cAMP-Defizienz und die Dysregulation des nukleären Faktors KappaB (NF-κB) und seines Inhibitors IκBα miteinander verbunden sind, wie beides die Immunantwort beeinflusst und die Hauterkrankung mit ihren Komorbiditäten verbindet. Erhärtet wird diese ätiologische Sichtweise durch den Nachweis, dass sowohl Psoriasis auslösende Medikamente, als auch Psoriasis-Therapeutika sämtlich, wenn auch mit umgekehrten Vorzeichen, in ihrer den cAMP-Spiegel bzw. die cAMP-abhängigen Funktionen beeinflussenden Wirkung

konvergieren. Die Arbeit konkretisiert die zentrale Bedeutung von cAMP für die zellulären Calciumsignale, die calciumabhängigen Differenzierungsprozesse, den Aufbau des epidermalen Calciumgradienten und den hier vorgestellten epidermalen Calciumkreislauf. Es wird dargestellt, wie sich aus einer fatal blockierten cAMP-Versorgung unter Aktivierung der Hedgehog-Transkription die psoriatische Läsion entwickelt, und in wiefern bereits die präsymptomatische psoriatische Haut u.a. durch eine vermehrte Expression NF-κB-abhängiger Zellprodukte vom Gesunden abweicht. Konkrete Forschungsvorschläge sollen die Entwicklung neuer, ergänzender Therapien anregen.

Analysis of the Calcineurin/NFAT Signal Transduction Pathway in Human Keratinocytes, Human Skin and Psoriasis Jan 13 2022

Model of pathogenesis of psoriasis. Part 1. Systemic psoriatic process. Nov 11 2021

Distinct Gene Expression Signatures Differentiate Clinical Response to Ustekinumab Compared to Adalimumab in Psoriasis Mar 03 2021 There is a need to optimise biologic therapy for psoriasis. The aim of this study was to identify gene expression signatures that predict response to two commonly prescribed biologics. Bulk RNAseq analysis was performed on skin biopsies of lesional and non-lesional skin of 82 psoriasis patients initiating treatment with adalimumab (Humira; TNF inhibitor) or ustekinumab (Stelara; IL-12/23 inhibitor) at baseline, 1 week and 12 weeks. Clinical response was defined by percentage reduction in psoriasis severity area index (PASI) at week 12 compared to baseline (u0394PASI). A Limma-Voom pipeline was used to identify differentially expressed genes (DEGs) at all three time points that associate with absolute PASI (aPASI); and u0394PASI in both drug cohorts. Principal component analysis of sample features driving transcriptome differential expression showed that tissue, time, and PASI (in that order) were the main drivers of transcriptome variation. Ingenuity pathway analysis of DEGs in both analyses showed common regulatory signals in both drug cohorts. Nevertheless, downregulation of the NFkB and p38 pathways showed stronger associations with aPASI in the adalimumab compared to the ustekinumab cohort. Downregulation of interferon (IFN) signalling at baseline strongly associated with u0394PASI in the ustekinumab but not the adalimumab cohort; downregulation of IFN signalling at weeks 1 and 12 associated with u0394PASI in both drug cohorts. This suggests that dampening of IFN signalling is a common mechanism involved in clearing psoriasis, but the activation state of this pathway at baseline has greater power in predicting response to ustekinumab compared to adalimumab. However, downregulation of inflammasome signalling at baseline and week 1 associated with u0394PASI in the adalimumab cohort but not the ustekinumab cohort. This work highlights the potential for stratification of biologic therapy for psoriasis based on gene expression at baseline and during the early phases of treatment.

Psoriasis and Psoriatic Arthritis Feb 14 2022 First comprehensive book on this topic: Textbooks on psoriasis and psoriatic arthritis in one Newest information on Psoriasis. Written by well-known international experts. Well-structured with reader-friendly format.

S100 Proteins as Calcium Signaling Mediators and Transglutaminase Substrates in Keratinocyte Biology Oct 30 2020

Psoriatic Arthritis and Psoriasis Nov 23 2022 Educational advancement in the field of psoriatic arthritis which this book will provide is consistent with GRAPPA's aims and objectives leading to a productive synergy. GRAPPA (Group for Research and Assessment of Psoriasis and Psoriatic Arthritis), is recognized world wide as the leading international society for the study and promotion of awareness of psoriatic arthritis. GRAPPA is an association of leading rheumatologists, dermatologists, representatives of patient service leagues and other stakeholders focused on psoriasis and PsA.Psoriatic Arthritis (PsA) ranks with rheumatoid arthritis and axial spondyloarthritis as one of the most prevalent inflammatory arthropathies worldwide. There is now a significant global awareness among Rheumatologists, Dermatologists, Internal Medicine Specialists, Gastroenterologists, General Practitioners, Family Practitioners, Physiotherapists, Nurse Specialists, Immunogenetics and many other Health Care Professionals with regards to the importance of psoriatic arthritis.

Immunology of Psoriatic Disease Feb 20 2020 Psoriasis is a chronically relapsing inflammatory skin disorder affecting about 2% of the worldwide population. The disease is associated with important systemic manifestations, including cardiovascular comorbidities and metabolic syndrome. In addition, about 30% of patients develop joint inflammation known as psoriatic arthritis (PsA). Our knowledge on the pathogenesis of psoriasis has dramatically expanded in the last decade, suggesting the existence (or co-existence) of both auto-immune and auto-inflammatory components. Skin lesions develop from a complex interplay between keratinocytes, vascular endothelium, dendritic cells, and T cells, generating a self-sustaining inflammatory cycle. Within this cycle, epidermal CD8+ T lymphocytes specific for self-antigens may represent the major autoimmune mechanism. Despite the recent progress in the comprehension of the pathogenesis of psoriasis many questions remain open, ranging from the plaque-initiating events to the characterization of the autoimmune /autoinflammatory components of the disease. The mechanisms that link cutaneous psoriasis to its extra-cutaneous and systemic manifestations also remain vague. In this Research Topic we invited top scientists to summarize the front-line research in the field of immunology of cutaneous psoriasis and its systemic and joint manifestations. Our intention was to integrate the pillar concepts of psoriasis immunopathology with the most novel insights, aiming at providing an advanced view of this rapidly evolving and fascinating field.

Psoriatic Arthritis, An Issue of Rheumatic Disease Clinics 41-4, Aug 08 2021 Some people with psoriasis can also develop psoriatic arthritis, when the immune system attacks the joints as well, causing inflammation. Like psoriasis, psoriatic arthritis symptoms flare and subside, vary from person to person, and even change locations in the same person over time. Psoriatic arthritis can affect any joint in the body, and it may affect just one joint, several joints or multiple joints. For example, it may affect one or both knees. This issue will include articles on Genetic and Epigenetic aspects of psoriatic arthritis, Clinical features and diagnostic considerations in psoriatic arthritis, Natural history, prognosis and socioeconomic aspects of psoriatic arthritis, Etiology and pathogenesis of psoriasis and many more!

What's New in the Skin? Sep 09 2021

Psoriasis: Advances in Knowledge and Care, An Issue of Dermatologic Clinics, Sep 28 2020 Psoriasis is the most prevalent autoimmune disease in the U.S. • ~125 million people worldwide have psoriasis. Commonly misunderstood and interpreted as a "cosmetic problem, psoriasis is a complex and potentially debilitating disease; nearly 60% of people with psoriasis reported their disease to be a large problem in their everyday life. This issue of Dermatologic Clinics devoted exclusively to psoriasis is edited by two leaders of the International Psoriasis Council, Dr Alan Menter and Dr Chris Griffiths. Topics include: Genetics of psoriasis; Immunopathogenesis of psoriasis; Phenotypical expressions of psoriasis; Psoriasis as a systemic disease with multiple comorbidities; Outcomes measures in the assessment of psoriasis in clinical practice and trials; Update on topical therapies for mild to moderate psoriasis; Phototherapy and photochemotherapy (PUVA) for psoriasis; Current and future oral systemic therapies for psoriasis; Current biological therapies for psoriasis; Future biological therapies for psoriasis; Psoriatic arthritis for the dermatologist; Pharmacogenomics and the future of psoriasis therapies. This information that goes to the heart of clinical practice is equally appropriate, beyond dermatologists, for internal medicine clinicians, primary care physicians, bone & joint specialists; hematologists/oncologists, and researchers in genetic targeted therapy development.

Cytokines as Potential Therapeutic Targets for Inflammatory Skin Diseases Jul 19 2022 Cytokines and cytokine receptors remain an area of great interest for the development of targeted therapies for cutaneous inflammatory diseases. Anti-TNF therapeutics have proven to be effective in the treatment of psoriasis, and clinical investigations have now begun for other cytokine-directed therapies, such as those targeting IFN-g, IL-12p40, and IL-18. In addition to therapeutics that target cytokines directly, strategies that target cytokine signaling pathways are in development. This book summarizes the findings of the 56th International Workshop of the Ernst Schering Research Foundation that focused on "Cytokines as Potential Therapeutic Targets for Inflammatory Skin Diseases".

Studies on Vitamin A Signaling in Psoriasis Apr 28 2023

Targeting Human Inflammatory Skin Diseases With Natural Products: Exploring Potential Mechanisms and Regulatory Pathways Apr 16 2022

The Role of Connexin Mediated Signalling in the Pathogenesis of Psoriasis Oct 22 2022

Role of Apoptosis and Key Canonical Pathways

in Psoriasis Plaque Clearance in Response to UVB Phototherapy Jun 25 2020 Psoriasis is a chronic inflammatory, immune mediated skin and systemic disease with debilitating and life limiting effects. Currently patients are treated on a trial and error basis highlighting the need for predictive biomarkers. Phototherapy is an effective therapy and may induce remission. Previous work showed that 311nm UVB (effective in clearing psoriasis) but not by 290nm UVB (ineffective in clearing psoriasis) induced apoptosis in lesional psoriatic skin. The aim of this study was to investigate the molecular mechanisms regulating psoriasis plaque resolution in response to UVB and to identify potential biomarkers.Bioinformatic analysis was performed on microarray data derived from skin biopsies (n=48) obtained 4h and 18h after irradiation of psoriatic skin with 311nm or 290nm UVB. Differentially expressed genes (DEGs) associated with 311nm but not 290nm were analysed for their upstream regulators (IPA). Validation, localisation patterns and quantification of biomarkers was determined using immunofluorescence and Volocity.Eliminating genes induced by 290nm resulted in 755 and 795 DEGs regulated by 311nm at 4h and 18h respectively. Upstream analysis showed that the commonest pathways regulated by 311nm (but not 290nm) in psoriasis skin were apoptosis and necrosis which appeared to be occurring in a variety of cell types. In terms of canonical pathways, we found differential modulation of acute phase signalling (including IL-1 and p38), p53, IL17a and atherosclerosis signalling by 311nm compared to 290nm UVB. FOSL1, JUNB, GDF15 and p21, proteins implicated in apoptosis, were differentially induced by 311nm compared to 290nm.Our microarray analysis confirmed apoptosis and necrosis as key cellular processes differentially induced by 311nm UVB compared to 290 but also identified pathways relevant to psoriasis and its associated co-morbidities. Together with our earlier work, these data support a key role for apoptosis and necrosis in addition to psoriasis cytokine networks in plaque clearance.
Psoriasis: New Insights for the Healthcare Professional: 2013 Edition May 25 2020 Psoriasis: New Insights for the Healthcare Professional: 2013 Edition is a ScholarlyEditions™ book that delivers timely, authoritative, and comprehensive information about Genetics. The editors have built Psoriasis: New Insights for the Healthcare Professional: 2013 Edition on the vast information databases of ScholarlyNews.™ You can expect the information about Genetics in this book to be deeper than what you can access anywhere else, as well as consistently reliable, authoritative, informed, and relevant. The content of Psoriasis: New Insights for the Healthcare Professional: 2013 Edition has been produced by the world's leading scientists, engineers, analysts, research institutions, and companies. All of the content is from peer-reviewed sources, and all of it is written, assembled, and edited by the editors at ScholarlyEditions™ and available exclusively from us. You now have a source you can cite with authority, confidence, and credibility. More information is available at http://www.ScholarlyEditions.com/.
Novel Genomic Signature Predicts Response to Ruxolitinib Cream in Psoriasis Jun 18 2022 Psoriasis is a chronic immune-mediated skin disease affecting up to 3% of the population that is associated with a significantly impaired quality of life. Lesional psoriatic skin is primarily characterized by significant Th1- and Th17-mediated inflammation, resulting in increased Janus kinase (JAK) signaling. Ruxolitinib cream (Rux Cream) is a potent, topically applied, selective inhibitor of JAK1 and JAK2 of the JAK-STAT signaling pathway. Treatment with Rux Cream was associated with significant therapeutic benefit in patients with mild to moderate psoriasis with some patients experiencing greater benefit than others. This study aims to identify biomarkers to predict response to Rux Cream in participants with plaque psoriasis from RNA-Seq data.
Advances in Psoriasis Mar 23 2020 It has become increasingly clear that psoriatic disease, both of the skin and joints, can be a significant diagnostic and therapeutic challenge for the physician and a debilitating illness for the patient. Genetic and immunologic advances have increased our understanding of the pathophysiology of psoriasis and psoriatic arthritis and there is a need for practically oriented evidence based references to describe the management options open to clinicians. The speed at which developments are occurring in the field also necessitates a novel approach to keeping up with these changes in practice and the need is for a reference that that be updated regularly as the subject requires. Psoriasis is an incredibly fast-moving discipline within dermatology. Guidelines, treatment options and management all change at incredible speed. There is a requirement to provide a comprehensive reference resource to provide practical, user friendly information for the dermatology profession to aid in the decision-making process. Psoriasis is a graphical subdiscipline of medicine and therefore this will have copious illustrations. As a fast moving discipline the emphasis must be on annual updates to ensure that readers are kept up to date on the important areas of development.
Role of MyD88 Signaling in the Imiquimod-induced Mouse Model of Psoriasis: Focus on Innate Myeloid Cells Feb 26 2023
Retinoids in Dermatology Jan 21 2020 This up-to-date reference on the use of retinoids in dermatology presents how retinoids function in the skin, how they can best be used to treat and prevent various skin diseases, and how they can be monitored effectively. The text will provide an in-depth update on the pharmacology, clinical use, side effects, and follow-up of retinoid therapy in dermatology. This source also addresses topics related to retinoid use in special circumstances, such as vulnerable populations, concomitant surgery, and aesthetic procedures.
In Vitro Th-17 Inflammatory Skin Models as a Promising Tool for Psoriasis Research and Drug Development Apr 04 2021 This last decade, it has been established that antagonism of IL-17 is sufficient to reverse psoriasis features. IL-17 mainly secreted by Th-17 T-cells is now considered as the psoriasis main driver (Guttman-Yassky and Krueger, 2017).In the search for specific strategies to improve skin resilience towards inflammation, we designed a 3D in vitro human reconstructed epidermis (RHE) model that recapitulates activated CD4+ T cells-mediated conditions of psoriasis driven by Th-17 cytokines (IL-17A and TNF-uf061). Our data demonstrated an increase of psoriasis biomarkers at the mRNA levels, including IL-19, IL-23, DEFB4 and S100A7, as well as IL-19 and IL-23 levels in tissue culture supernatants. Full-transcriptome testing identified additional deregulated genes from 4 major psoriasis-related pathways, i.e. antimicrobial defenses, chemotaxis and inflammatory response (mainly linked to the NF-u03baB pathway).Signal transduction was further characterized based on primary keratinocytes, which were challenged following the same protocol as for the RHE. We demonstrated the activation of NF-u03baB by transactivation assay, and identified STAT3 and PPAR through the transcription factor activation profiling technology.Recent publications recognized Iu03baBu03b6, an atypical nuclear Iu03baB protein encoded by the NFKBIZ gene (nuclear factor of u03ba light polypeptide gene enhancer in B cells inhibitor u03b6), as a key driver in the development of psoriasis (Johansen et al., 2015). Iu03baBu03b6 is critically involved in the downstream effects of IL-17 and was identified as a key transcriptional regulator of psoriasis signature genes (Muller et al., 2018). Furthermore, NFKBIZ has been identified as a psoriasis susceptibility locus (Tsoi et al., 2015). We demonstrated from both RHE and monolayer keratinocytes that a synergistic stimulation with IL-17 and TNF-uf061 strongly up-regulates NFKBIZ and results in Iu03baBu03b6 accumulation.The relevance of the model was further demonstrated through the pre-clinical development of TEM1657, a first-in-class API candidate targeting Iu03baBu03b6 for the treatment of psoriasis.
Clinical and Basic Immunodermatology Mar 15 2022 This updated volume provides a "user-friendly" reference for dermatologists, dermatology residents and students, as well as for health care workers in related fields to better understand immune-mediated skin diseases and their therapies. The focus is on what is needed by the physician/resident or student for betting understanding the pathophysiology of the disease as well as the mechanisms of action of the therapies. The reader can easily read about groups of related diseases as well as groups of related therapies. The level of complexity of the book is such that it has practical applications on a daily basis but can also be used by the resident as a teaching tool and as a handy source of review for the boards. In addition, it can be used the practicing dermatologist to study for recertification. The scope of the book is immunology, immunogenetics, immunopathology and immunopharmacology as they relate to clinical dermatology.
A Hyper Th17 Response Connects the Psoriasis-associated ACT1 Variant to Skin Inflammation Aug 20 2022 IL-17 is a pro-inflammatory cytokine implicated in the pathogenesis of autoimmune diseases including psoriasis. ACT1 is an essential adaptor molecule in the IL-17 signaling pathway. Here we found that ACT1 is a client protein of the molecular chaperone Hsp90. A missense single nucleotide polymorphism (rs33980500; SNP-D10N) that resulted in the substitution of an asparagine for an aspartic acid at position 10 of

ACT1 (ACT1-D10N) is associated with psoriasis susceptibility. We demonstrated that ACT1-D10N was defective in its interaction with hsp90, which resulted in a global loss of ACT1 function. Act1-deficient mice modeled the mechanistic link between loss of Act1 function and susceptibility to psoriasis. Although Act1 was necessary for IL-17-mediated inflammation, Act1-deficient mice had a hyperactive response of the Th17 subset of helper T cells and developed spontaneous IL-22-dependent skin inflammation. In the absence of IL-17-signaling, IL-22 was the main contributor to skin inflammation. Due to alternative splicing in humans, however, SNP-D10N encodes two mutated ACT1 proteins, ACT1-D10N and ACT1-D19N. Though both ACT1 isoforms are Hsp90 `client' proteins, the nine additional amino acids in ACT1-D19N provide an additional Hsp90 binding site that is absent in ACT1-D10N. Therefore, while ACT1-D10N is a dead protein that is unable to transduce IL-17 signals for gene expression, ACT1-D19N is fully responsive to IL-17. Intriguingly, the two ACT1 isoforms are differentially expressed in ACT1D10N/D10N fibroblasts and T cells. Fibroblasts express both isoforms equally, enabling ACT1-D19N to compensate for the loss of ACT1-D10N function. ACT1D10N/D10N T cells, however, express predominantly ACT1-D10N. Lacking this compensatory mechanism, ACT1D10N/D10N T cells behave like ACT1-deficient T cells, exhibiting a dysregulated and hyperactive Th17 phenotype with overproduction of IL-22 and IL-17. The hyperactive Th17 response combined with fully responsive fibroblasts likely synergized to contribute to psoriasis susceptibility in SNP-D10N patients.

Psoriasis and Psoriatic Arthritis Dec 24 2022 Psoriasis is a life-long chronic autoimmune disease characterized by thick scaly skin lesions and often associated with severe arthritis. In psoriasis, lesions skin cells, keratinocytes, grow too quickly, resulting in thick, white, silvery or red patches on skin. Normal skin cells grow gradually and flake off about every four weeks, but psoriasis causes new skin cells to move rapidly to the surface of the skin in days rather than weeks. Psoriasis symptoms often appear on the elbows, scalp, feet, knees, hands, or lower back, or as flaking or patches on the skin. It is most common in adults, but teenagers and children can also suffer from psoriasis. Psoriasis is not only a skin condition; it is a chronic disease of the immune system. Chronic psoriasis is associated with other health conditions such as psoriatic arthritis, several inflammatory disorders, type 2 diabetes, and cardiovascular disease. This book provides extensive coverage of psoriasis and psoriatic arthritis. It features information on epidemiology and etiology of psoriasis, pathogenesis, genetics of psoriasis, clinical manifestations, and treatment options using cutting-edge drugs including adalimumab and tofacitinib. Natural phytochemicals and nutraceuticals have demonstrated efficacy in ameliorating psoriasis. The book dedicates comprehensive coverage of nutraceutical therapeutic options including antioxidants, bioactive peptides, carotenoids, alpha lipoic acid, curcumin, and whey protein. These inexpensive natural therapeutics are not associated with any known adverse side effects. *Therapy and Prevention of Atopic Dermatitis and Psoriasis* Jan 25 2023 Atopic dermatitis and psoriasis are common inflammatory skin diseases. The excellent therapeutic success of anti-IL-4/IL-13 biologics for atopic dermatitis and anti-TNF-α/IL-23/IL-17A biologics for psoriasis highlights the major pathogenic roles of these cytokines in the respective diseases. Although atopic dermatitis and psoriasis are distinct clinical entities, they share similar inflammatory processes, including increased Th17, Th22, and Th1 signatures. In addition to skin inflammation, both skin diseases exhibit a significant association with systemic diseases such as cardiovascular diseases, metabolic syndrome, and autoimmune diseases. In contrast to atopic dermatitis, males are more susceptible to psoriasis than females. Differential risk factors may be involved in the development and exacerbation in atopic dermatitis and psoriasis. In this issue, we will publish cutting-edge information regarding skin inflammation, clinical aspects, risk factors, microbiome, and therapeutics related to psoriatic and atopic inflammation.

Intercellular Calcium-mediated Cell Signaling in Keratinocytes Cultured from Patients with NF1 Or Psoriasis Mar 27 2023

Therapeutic Advancements in Psoriasis and Psoriatic Arthritis Dec 12 2021