2025
16th KSID Research Camp

Research & Recharge:
From the Stories of the Past to Science for the Future

2025. 9. 26(금)-27(토)
힐튼호텔 경주 체리룸(지하 1층) 및 파인룸(1층)

대한피부연구학회
The Korean Society for Investigative Dermatology

인사말

KSID 회원 여러분께,

안녕하세요. 이번에 KSID 회장직을 맡게 된 강희영입니다.
2009년에 시작된 KSID 리서치캠프가 올해로 16회를 맞이하게 되었습니다. 올해 캠프는 천년의 고도, 힐튼호텔 경주에서 9월 26일부터 27일까지 이틀간 개최될 예정입니다.

KSID 리서치캠프는 완성되지 않은 데이터를 바탕으로 자유롭게 토론하고, 서로 조언을 나누는 자리입니다. 부디 많은 분들께서 부담 갖지 마시고, 진행 중인 데이터를 가지고 오셔서 활발한 논의에 함께해 주시면 좋겠습니다.

특히 올해 경주는 APEC 개최를 앞두고 도시 곳곳이 새롭게 단장되고 있습니다. 경주문화유산을 경험하시기 바라며 특별히 학회에서는 이성낙 가천대학교 전 총장님의 '조선시대 초상화, 그 아름다운 흠집' 특강도 준비하였습니다. 가족 동반 참석도 환영하며, 아이들과 함께 들으셔도 좋은 시간이 될 것입니다. 리서치캠프의 취지에 맞게 정장보다는 편안한 복장으로 오셔서, 자유롭고 열정적인 분위기 속에서 뜻깊은 교류의 시간이 되기를 기대합니다.

9월 경주에서 여러분을 뵙기를 고대합니다.
감사합니다.

2025년 9월

KSID 회장 **강 희 영**

일정표 (Sep. 26 ~ Sep. 27)

KSID Research Camp: Sep. 26 (Fri), 힐튼호텔 경주 체리룸

Time	Program
13:00–14:00	Registration
14:00–15:00	Free Communication (1)
15:00–15:30	Coffee Break / Poster View / Group Photo / Booth Visit
15:30–16:30	Free Communication (2)
16:30–18:00	Free Communication (3)
18:00–	General Assembly & Closing Ceremony

KSID Research Camp: Sep. 27 (Sat), 힐튼호텔 경주 체리룸 & 파인룸

Time	Program
09:00–09:10	Registration & Light Breakfast
09:10–09:20	Welcome Message from the KSID President
09:20–10:30	Special Lecture (1)
10:30–11:30	Coffee Break / Poster Walk – Pine Room (1F)
11:30–12:00	Special Lecture (2)
12:00–12:30	General Assembly & Closing Ceremony

KSID Research Camp 프로그램 [Day1]

Sep. 26 (Fri), 힐튼호텔 경주 체리룸

13:00–14:00	Registration
14:00–15:00	**Free Communication (1)** **Skin Barrier Science: From Pathophysiology to Therapeutic Strategies**
	좌장 | 성균관의대 **이동윤,** 연세원주의대 **홍승필**
14:00-14:15	Senescent melanocytes impair skin barrier function 아주의대 **김진철**
14:15-14:30	Chronic dry skin induces hippocampal neurogenic impairment and cognitive decline via neural stem cell depletion 서울의대 **윤경노**
14:30-14:45	Targeting IL-24 signaling to restore skin barrier function: Discovery of penta-O-galloyl-β-D-glucose via in silico screening 연세의대 **이은정**
14:45-15:00	Dissolving microneedles as a strategy to bypass the skin barrier in skin diseases 연세대 생명공학과 **정형일**
15:00–15:30	Coffee Break / Poster View / Group Photo / Booth Visit
15:30–16:30	**Free Communication (2)** **Integrative Immunity in Skin: From Cellular Crosstalk to Inflammation**
	좌장 | 성균관의대 **이종희,** 연세의대 **김도영**
15:30-15:45	Analysis of epigenetic dysregulation and apoptosis-related gene silencing in autoimmune skin diseases 경희의대 **안혜진**
15:45-16:00	Spatial transcriptomic profiling reveals antiviral NK and cytotoxic T Cell responses associated with increased severity in DRESS syndrome 서울의대 **이지수**
16:00-16:15	Induction of neurotensin in sensory neurons drive itch and inflammation in a murine model of atopic dermatitis 연세의대 **이상은**
16:15-16:30	Identification of T helper type 1–like, Foxp3+ regulatory T cells in alopecia areata 중앙의대 **석 준**

KSID Research Camp 프로그램 [Day1]

Sep. 26 (Fri), 힐튼호텔 경주 체리룸

16:30–18:00	**Free communication (3)** **Innovative Platforms and Systems Biology in Dermatology**
	좌장 | 전남의대 **윤숙정,** 가톨릭의대 **김혜성**
16:30-16:45	Single-cell and spatial transcriptomics reveals immune dynamics and dermal immune niche underlying palmoplantar pustulosis 서울의대 **이한재**
16:45-17:00	Disruption of tight junction integrity by staphylococcus aureus via TLR2–JAK2–STAT3 signaling and its reversal by the pseudo-ceramide, DDSS in the 3D skin model 차의대 **김해빈**
17:00-17:15	Probiotic administration attenuates HS-like skin inflammation via modulation of gut microbiota and immune responses in mice 차의대 **서다혜**
17:15-17:30	Keloid pathogenesis from the aspect of skin site: based on single-cell RNA sequencing 성균관의대 **여은혜**
17:30-17:45	Specific biomarkers for melanoma revealed by spatial transcriptomic analysis and single-cell RNA sequencing 성균관의대 **이태민**
17:45-18:00	AI-driven discovery of novel HSP47 activating peptides for skin anti-aging 인코스팜 **정세규**
18:00–	General Assembly & Closing Ceremony

KSID Research Camp 프로그램 [Day2]

Sep. 27 (Sat), 힐튼호텔 경주 체리룸 & 파인룸

09:00–09:10	Registration & Light Breakfast	
09:10–09:20	Welcome Message from the KSID President	
09:20–10:10	**Special Lecture (1)**	**좌장** | 아주의대 **강희영**
09:20–10:00	조선시대 초상화, 그 아름다운 흠집	가천대학교 전 총장 **이성낙**
10:00–10:10	Q&A	
10:10–11:10	**Coffee Break / Poster Walk – Pine Room (1F)**	
		좌장 | 충남의대 **이 영,** 서울의대 **조성진,** 한림의대 **김혜원**
	Chronic protein leakage as an unrecognized sequela of chronic spontaneous urticaria	부산의대 **이병혁**
	Site-specific skin surface lipid–microbiome dysregulation in pediatric mild atopic dermatitis	한림의대 **김혜원**
	Personalized modeling of Th2 immunity in atopic dermatitis using AVATAR mice	연세의대 **박창욱**
	Validation of C3H/HeN mice as an alopecia areata animal model	연세의대 **김은혜**
	Minoxidil sulfate suppresses JAK/STAT pathway and restores mitochondrial function in IFN-γ and poly(I:C)-stimulated ORS cells: Implications for alopecia areata	충남의대 **이 영**
	ATG7 dysfunction in senescent melanocytes and hypopigmented skin: Reversal by metformin	아주의대 **김영은**
	The role and molecular mechanisms of melanophilin in skin cell aging	연세의대 **박서현**
	Protective role of *Lactiplantibacillus plantarum* ferment lysates on PM-Induced skin aging	중앙의대 **권도연**
11:10–11:40	**Special Lecture (2)**	**좌장** | 경북의대 **이원주**
	특허, 연구자를 발명자로 만드는 첫걸음	특허청 과장 **양인수**
11:40–12:10	General Assembly & Closing Ceremony	

Free Communication (1):
Skin Barrier Science: From Pathophysiology to Therapeutic Strategies

Sep. 26th 14:00-15:00

좌장 | 이동윤(성균관의대), 홍승필(연세원주의대)

Senescent melanocytes impair skin barrier function
아주의대 **김진철**

Chronic dry skin induces hippocampal neurogenic impairment and cognitive decline via neural stem cell depletion
서울의대 **윤경노**

Targeting IL-24 signaling to restore skin barrier function: Discovery of penta-O-galloyl-β-D-glucose via in silico screening
연세의대 **이은정**

Dissolving microneedles as a strategy to bypass the skin barrier in skin diseases
연세대 생명공학과 **정형일**

FC1: Skin Barrier Science: From Pathophysiology to Therapeutic Strategies

FC1-1. Senescent melanocytes impair skin barrier function

Jin Cheol Kim[1], Yeongeun Kim[1,2], So Yeon Myeong[1,2], Agnes Tessier[4], Gaëlle Gendronneau[4], Nada André[4], Tae Jun Park[2,3], Hee Young Kang[1,2]

[1]Dermatology, Ajou University School of Medicine, Suwon, Korea
[2]Inflamm-Aging Translational Research Center, Ajou University School of Medicine, Suwon, Korea
[3]Biochemistry and Molecular Biology, Ajou University School of Medicine, Suwon, Korea
[4]Innovation Research and Development, Chanel Parfums Beaute, Pantin, Paris, France

Melanocyte senescence primarily occurs in sun-exposed skin of elderly, contributing to skin aging. In this study, we investigated the impact of senescent melanocytes on skin barrier function. UV-induced senescent melanocytes downregulated the expression of barrier-associated genes and differentiation markers in keratinocytes and ex vivo skin. Moreover, conditioned medium derived from senescent melanocytes significantly reduced transepithelial electrical resistance (TEER), indicating impaired barrier permeability. Senescent melanocytes exhibited a distinct secretory profile enriched with interleukins, interferons, and redox-related factors, which led to the activation of JAK signaling in surrounding keratinocytes. Notably, inhibition of these signaling pathways using JAK inhibitors alleviated the barrier dysfunction induced by senescent melanocytes. These findings suggest that the secretome of senescent melanocytes plays a key role in age-related skin barrier impairment and may serve as a novel therapeutic target.

FC1-2. Chronic dry skin induces hippocampal neurogenic impairment and cognitive decline via neural stem cell depletion

Kyeong-No Yoon[1,2,3], Seong-Jun Kang[4], Seon Min Lee[3], Ji Su Lee[3], Hyunsun Park[3], Hyun Je Kim[4], Yong-Seok Lee[4,5,6,7], Dong Hun Lee[1,2,3*]

[1]Laboratory of Cutaneous Aging Research, Biomedical Research Institute, Seoul National University Hospital, Seoul, Korea
[2]Institute of Human-Environmental Interface Biology, Medical Research Center, Seoul National University, Seoul, Korea
[3]Department of Dermatology, Seoul National University Hospital, Seoul National University College of Medicine, Seoul, Korea
[4]Department of Biomedical Sciences, Seoul National University Graduate School, Seoul, Korea
[5]Department of Physiology, Seoul National University College of Medicine, Seoul, Korea
[6]Neuroscience Research Institute, Seoul National University College of Medicine, Seoul, Korea
[7]Wide River Institute of Immunology, Seoul National University, Hongcheon, Korea

The bidirectional communication between skin and brain, known as the skin-brain axis, represents a critical pathway in maintaining physiological homeostasis and neurological health. Chronic skin disorders have been increasingly associated with neurological and cognitive manifestations, suggesting a profound interconnection between cutaneous health and central nervous system function. Dry skin conditions, characterized by impaired epidermal barrier function and chronic mild inflammation, trigger cascades of inflammatory responses including the release of pro-inflammatory cytokines that may traverse the blood-brain barrier. The hippocampus, a brain region crucial for learning and memory formation, exhibits remarkable plasticity through adult neurogenesis. However, this process is highly sensitive to chronic stress and inflammatory signals, making it vulnerable to peripheral pathological conditions. In this study, we investigated whether chronic dry skin conditions impair hippocampal neurogenesis and cognitive behavior using a well-established murine model. A chronic dry skin condition was established by repeated topical application of acetone/ether and water (AEW) to the dorsal skin of mice for three weeks, mimicking human dry skin pathology. Comprehensive behavioral assessments revealed significant deficits in novel object recognition (NOR) and object place recognition (OPR) tests, indicating impaired hippocampal-dependent memory function. Detailed immunohistochemical analysis demonstrated a marked reduction in $Nestin^+/SOX2^+/GFAP^+$ neural stem cells (NSCs), along with decreased expression of neurogenesis and proliferation markers (DCX^+ and $Ki\text{-}67^+$) and reduced neuronal activity ($c\text{-}Fos^+$) in the hippocampus. These findings demonstrate that chronic dry skin compromises adult neurogenesis and neural plasticity through depletion of hippocampal NSCs and disruption of the neurogenic microenvironment. This study reveals the detrimental effects of chronic cutaneous dryness on hippocampal function and provides compelling evidence that the skin–brain axis plays a pivotal role in mediating systemic neuroplastic decline through disruption of the neural stem cell niche, with potential implications for understanding cognitive decline in dermatological patients.

FC1-3. Targeting IL-24 signaling to restore skin barrier function: Discovery of penta-O-galloyl-β-D-glucose via in silico screening

Ji Young Kim[1,5], Eun Jung Lee[1,5], Geunhyuk Jang[2,5], Seohyun Park[1], Hye-won Na[2], Nari Cha[2], Yu Jeong Bae[1], Shwinwon Hwang[1], Il Joo Kwon[1], Jamal Mohammed Alqahtani[3], Jinu Lee[4], Hyoung-June Kim[2], Sang Ho Oh[1*]

[1]Department of Dermatology and Cutaneous Biology Research Institute, Severance Hospital, Yonsei University College of Medicine, Seoul, Korea
[2]Research and Innovation Center, AMOREPACIFIC, Yongin, Korea
[3]Department of Dermatology, Imam Abdulrahman Bin Faisal University, Saudi Arabia
[4]College of Pharmacy, Yonsei Institute of Pharmaceutical Sciences, Yonsei University, Incheon, Korea
[5]These authors contributed equally to this work.

Skin barrier dysfunction is a key feature of various inflammatory skin diseases, often accompanied by aberrant cytokine signaling. Interleukin-24 (IL-24) is a cytokine belonging to the IL-10 family. This cytokine binds to type I (IL-20RA/IL-20RB) and type II IL-20 receptors (IL-22RA1/IL-20RB) to activate Janus kinase 1 (JAK1), signal transducer and activator of transcription 3 (STAT3), and mitogen-activated protein kinase (MAPK) signaling, leading to pro-inflammatory cytokine production. IL-24 has been reported to play crucial roles in wound healing and allergic diseases, such as atopic dermatitis. Specifically, IL-24-treated keratinocytes displayed downregulation of filaggrin and loricrin causing skin barrier dysfunction. In this study, we aimed to identify novel small molecule inhibitors targeting IL-24 signaling to restore skin barrier function. Using *in silico* virtual screening and molecular docking approaches, we discovered Penta-O-Galloyl-β-D-Glucose (PGG) as a promising candidate predicted to bind IL-24 with high affinity. Subsequent *in vitro* and *ex vivo* results demonstrated that PGG treatment led to significant upregulation of key genes involved in maintaining skin barrier homeostasis, such as filaggrin, loricrin, and involucrin. These molecular changes correlated with functional improvements in epithelial barrier properties in relevant cell models. Collectively, PGG, screened out from *in silico* system, was identified as an IL-24 signaling inhibitor, which enhances skin barrier function by suppressing the STAT3 pathway. Therefore, PGG can play a vital role in the restoration of barrier function by inhibiting the action mechanism of IL-24.

FC1-4. Dissolving microneedles as a strategy to bypass the skin barrier in skin diseases

Hyungil Jung[1,2*], Jiwoo Shin[1], Youseong Kim[1], Chansol Jeon[2], Mingyu Jang[2], Huisuk Yang[2]
[1]Department of Biotechnology, Yonsei University, Seoul, Korea
[2]Juvic Inc., Seoul, Korea

Traditional drug delivery methods such as topical administration and injections often suffer from drawbacks including low patient compliance, high systemic exposure, and undesirable side effects. These limitations are particularly evident in the treatment of dermatological conditions such as atopic dermatitis and androgenetic alopecia, where localized drug delivery remains a challenge due to the barrier function of the stratum corneum Similar limitations exist in cosmetics, where topical products often fail to deliver active ingredients through the skin. To address these issues, microneedles (MNs) have emerged as a promising transdermal drug delivery platform offering a minimally invasive and targeted alternative. Among various types of MNs, dissolving microneedles (DMNs), composed of biocompatible and biodegradable polymers, have garnered attention due to their ability to encapsulate therapeutic agents and release them directly into the skin as the MNs dissolve upon insertion. This study aimed to explore the potential of DMNs to overcome the limitations of conventional therapies by enhancing transdermal delivery and improving treatment outcomes for skin diseases. We focused on corticosteroid-loaded DMNs for atopic dermatitis and hair growth–promoting DMNs for androgenetic alopecia. We also evaluated their ability to enhance transdermal absorption of cosmetic actives. Our findings suggest that incorporating drugs such as triamcinolone acetonide into DMNs can enhance skin penetration and local anti-inflammatory effects while reducing systemic exposure and application frequency. For alopecia, DMNs that penetrate the hair-bearing scalp delivered agents near hair follicles and promoted regrowth through localized delivery of vasodilators or follicle-activating agents. In cosmetic use, combining DMNs with serum application enhanced skin hydration and tone. These outcomes demonstrate that DMNs can bypass the skin barrier and enable site-specific delivery. In conclusion, DMNs offer a versatile and effective platform for transdermal delivery in inflammatory, follicular, and cosmetic skin applications. Ongoing research will optimize their formulation and design for broader dermatologic therapies.

Free Communication (2):
Integrative Immunity in Skin: From Cellular Crosstalk to Inflammation

Sep. 26th 15:00-16:30

좌장 | 이종희(성균관의대), 김도영(연세의대)

Analysis of epigenetic dysregulation and
apoptosis-related gene silencing in autoimmune skin diseases
경희의대 **안혜진**

Spatial transcriptomic profiling reveals antiviral NK and cytotoxic
T Cell responses associated with increased severity in DRESS syndrome
서울의대 **이지수**

Induction of neurotensin in sensory neurons drive itch and inflammation
in a murine model of atopic dermatitis
연세의대 **이상은**

Identification of T helper type 1-like, Foxp3+ regulatory T cells
in alopecia areata
중앙의대 **석 준**

FC2-1. Analysis of epigenetic dysregulation and apoptosis-related gene silencing in autoimmune skin diseases

Hye-Jin Ahn[1], Ki-Heon Jeong[1], Min Kyung Shin[1], Mi Kyung Park[2,3]
[1]Derpartment of Dermatology, Kyung Hee University College of Medicine, Kyung Hee University Hospital, Seoul, Korea
[2]Department of Cancer Biomedical Science, Graduate School of Cancer Science and Policy, National Cancer Center, Goyang, Korea
[3]Department of Biomedical Science, Hwasung Medi-Science University, Hwaseong, Korea.

Background: Epigenetic alterations, particularly aberrant DNA methylation, are emerging as key regulators in autoimmune skin disorders. To identify disease-specific DNA methylation signatures in vitiligo and alopecia areata(AA), and to functionally validate the role of differentially methylated genes in modulating apoptosis using disease-model cell lines.
Methods: Genome-wide DNA methylation analysis was performed in patients from Kyung Hee University Hospital with vitiligo (n = 10), AA (n = 10) with active disease within one year of onset, and healthy controls (n = 10), using the Twist Human Methylome Panel Kit with subsequent methyl-capture sequencing and differential analysis. Based on these results, ADGRA2 (GPR124) was identified as the candidate gene for AA, while DUSP22 and FAAP20 were selected as candidate genes for vitiligo. To investigate the functional significance of these genes, siRNA-mediated knockdown experiments were conducted in appropriate disease model cell lines—Jurkat T cells for AA, and Melan-A melanocytes and HaCaT keratinocytes for vitiligo. After gene knockdown, cells were subjected to oxidative stress using hydrogen peroxide (H_2O_2), and the following assays were conducted to assess the effects: cell viability was measured by MTT assay, apoptosis was evaluated via FACS analysis, and the expression of pro-apoptotic genes was examined using qPCR.
Results: Knockdown of ADGRA2 resulted in a significant increase in the expression of pro-apoptotic genes indicating an enhancement of apoptotic processes. Similarly, silencing of DUSP22 and FAAP20 led to upregulation of apoptotic gene expression, reduced cell viability, and increased apoptotic cell populations when cells were exposed to oxidative stress conditions. These findings collectively suggest that disease-specific epigenetic silencing of these target genes sensitizes skin cells to apoptosis. Disease-specific epigenetic silencing of ADGRA2 in AA and DUSP22/FAAP20 in vitiligo enhances apoptosis in a cell type–dependent manner.
Conclusion: These findings highlight novel epigenetic mechanisms underlying autoimmune skin diseases and suggest potential therapeutic targets for intervention.

FC2-2. Spatial transcriptomic profiling reveals antiviral NK and cytotoxic T cell responses associated with increased severity in dress syndrome

Ju Young Jin[†1], Ji Hwan Moon[†2], Hyun Woo Kim[1], Ji Su Lee[*3,4], Hyun Je Kim[*1,5,6,7,8], Dong Hun Lee[*3,5]

[1]Department of Biomedical Sciences, Seoul National University Graduate School, Seoul, Korea
[2]Samsung Genome Institute, Samsung Medical Center, Seoul, Korea
[3]Department of Dermatology, Seoul National University College of Medicine, Seoul, Korea
[4]Department of Dermatology, Seoul Metropolitan Government - Seoul National University (SMG-SNU) Boramae Medical Center, Seoul, Korea
[5]Department of Dermatology, Seoul National University Hospital, Seoul, Korea
[6]Department of Microbiology and Immunology, Seoul National University College of Medicine, Seoul, Korea
[7]Cancer Research Institute, Seoul National University College of Medicine, Seoul, Korea
[8]Interdisciplinary Program in Artificial Intelligence (IPAI), Seoul National University

Background: Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) syndrome is a life-threatening drug-induced hypersensitivity syndrome. Molecular mechanisms underlying its severity remain poorly understood.
Objective: This study aimed to investigate the pathogenic mechanisms associated with increased disease severity in DRESS syndrome.
Methods: Six lesional skin tissues were collected across three patients with DRESS syndrome. Disease severity was classified as mild, moderate, and severe based on the DDS severity scoring system. Spatial transcriptomic analysis was performed separately for epidermal and dermal regions of interest (ROIs) to compare transcriptomic profiles across different severity groups.
Results: A total of 15 epidermal ROIs and 20 dermal ROIs were included in the analysis. In CD45+ immune cell–rich dermal areas, we identified 45 upregulated and 11 downregulated differentially expressed genes (DEGs) in the severe group compared to the mild group. Protein–protein interaction (PPI) analysis revealed activation of interferon-responsive signatures associated with natural killer (NK) cells and cytotoxic T cells in the severe group. Conversely, pathways related to skin barrier function and epithelial differentiation were downregulated in the severe group. In pancytokeratin+ epidermal areas, comparison between severe and mild groups revealed 6 upregulated and 38 downregulated DEGs. PPI analysis showed enrichment of antiviral response pathways and decreased expression of genes involved in skin barrier function and epithelial differentiation in the severe group.
Conclusion: Our data suggest that increased cytotoxic immune responses and impaired skin homeostasis are key features of severe DRESS syndrome. Enhanced antiviral cytotoxic activity, involving NK cells and cytotoxic T cells, may play a pivotal role in exacerbating disease severity in DRESS syndrome.

FC2-3. Induction of neurotensin in sensory neurons drive itch and inflammation in a murine model of atopic dermatitis

Song-Ee Kim, Yu Mi Jang, Sang Eun Lee
Department of Dermatology and Cutaneous Biology Research Institute,
Gangnam Severance Hospital, Yonsei University College of Medicine, Seoul, Korea

In the pathogenesis of atopic dermatitis (AD), the neuronal component not only initiates itch and consequent scratch-induced inflammation but also contributes to neuroinflammation through crosstalk with immune cells. However, little is known about the molecular alterations in sensory neurons during AD. In addition, stress is an important exacerbating factor in AD, yet the precise neuronal changes induced by stress also remain poorly understood. To investigate this, we utilized a murine model of AD induced by topical application of MC903, which recapitulates hallmark features of AD including inflammation, itch, and barrier dysfunction. Trigeminal ganglia (TG) were isolated at multiple time points and subjected to bulk RNA-sequencing. MC903 treatment induced transcriptional changes in the peripheral nervous system. Notably, expression of the *neurotensin (Nts)* gene increased as early as day 3, followed by upregulation of the NP3 neuron marker *Nppb* at day 4 and *Il31ra* at day 5. Inflammatory genes such as *Ccl8, Cxcl9,* and *Ptgs2* also showed marked elevation from day 4. Both 3-day restraint and chronic unpredictable stress upregulated *Nts* expression in TGs, indicating stress-induced neurotensin regulation. Functionally, intradermal injection of neurotensin into cheek skin induced rapid-onset scratching behavior, sustained up to 60 minutes. This acute pruritic response was significantly suppressed by co-treatment with an neurotensin receptor 1 (NTSR1) antagonist. To assess chronic effects, neurotensin or vehicle was intradermally co-administered daily during the 6-day topical MC903 treatment. Neurotensin significantly enhanced scratching on days 3, 4, and 6 and exacerbated dermatitis, including increased mast cell infiltration. Finally, pharmacologic inhibition of NTSR1 via intraperitoneal injection of SR-48692 attenuated both spontaneous scratching and skin inflammation in MC903-treated mice. Collectively, our findings identify neurotensin as a neuromodulator of both acute and chronic itch and reveal its functional role in stress- and inflammation-driven pathogenesis of AD.

FC2-4. Identification of T helper type 1-like, Foxp3$^+$ regulatory T cells in alopecia areata

Joon Seok[1,2*], Su-Young Kim[1,2], Beom Joon Kim[1,2]
[1]Department of Dermatology, Chung-Ang University Hospital, Chung-Ang University College of Medicine, Seoul, Korea
[2]Department of Medicine, Graduate School, Chung-Ang University, Seoul, Korea

It is known that CD4$^+$CD25$^+$Foxp3$^+$ regulatory T cells (T_{reg}) play a crucial role in establishing and maintaining immune regulation in both humans and mice. However, it is known that even fully differentiated T_{reg} cells can undergo conversion into Th1, Th2, or Th17 cell subsets under specific inflammatory conditions, leading to the loss of their suppressive function. Alopecia areata (AA), an autoimmune disease, occurs when NKG2D$^+$CD8$^+$ T cells attack hair follicle cells. Therefore, there is a need for research on T_{reg} cells to understand their role in regulating cytotoxic activity, especially in the context of autoimmune disorders like AA. Immunohistochemistry and flow cytometry were used for analysis method. In the skin of AA mice, T_{reg} cells had significantly decreased compared to WT mice, while the infiltration of CD8$^+$ T cells had shown a significant increase. Additionally, the ratio of T_{reg} cells to CD8$^+$ T cells had decreased as the severity of AA symptoms worsened. However, in the skin-draining lymph nodes (SDLN), the ratio of T_{reg} cells had increased. Moreover, various surface markers in T_{reg} cells from AA mice had been upregulated compared to T_{reg} cells in WT mice. We hypothesized that AA T_{reg} cells undergo change into Th1-like T_{reg} cells and consequently fail to perform their appropriate suppressive functions. As supporting evidence, the increased expression of CXCR3 and heightened responsiveness to IL-12 in T_{reg} cells from AA suggest the possibility that these cells have converted into Th1-like T_{reg} cells, potentially resulting in the loss of their suppressive function. However, further studies are needed to explore the plasticity of T_{reg} cells in AA.

Free Communication (3):
Innovative Platforms and Systems Biology in Dermatology

Sep. 26th 16:30-18:00

좌장 | 윤숙정(전남의대), 김혜성(가톨릭의대)

Single-cell and spatial transcriptomics reveals immune dynamics and dermal immune niche underlying palmoplantar pustulosis
서울의대 **이한재**

Disruption of tight junction integrity by staphylococcus aureus via TLR2-JAK2-STAT3 signaling and its reversal by the pseudo-ceramide, DDSS in the 3D skin model
차의대 **김해빈**

Probiotic administration attenuates HS-like skin inflammation via modulation of gut microbiota and immune responses in mice
차의대 **서다혜**

Keloid pathogenesis from the aspect of skin site: Based on single-cell RNA sequencing
성균관의대 **여은혜**

Specific biomarkers for melanoma revealed by spatial transcriptomic analysis and single-cell RNA sequencing
성균관의대 **이태민**

AI-driven discovery of novel HSP47 activating peptides for skin anti-aging
인코스팜 **정세규**

FC3: Innovative Platforms and Systems Biology in Dermatology

FC3-1. Single-cell and spatial transcriptomics reveals immune dynamics and dermal immune niche underlying palmoplantar pustulosis

Hanjae Lee[1,2], Juyoung Lee[2], Hyeok Ahn[2], Kyunghyuk Park[2], Bukyung Cha[2], Cheol Lee[3], Ohsang Kwon[1,2], Jong-Il Kim[2,4*], and Seong Jin Jo[1,2*]

[1]Department of Dermatology, Seoul National University College of Medicine, Seoul, Korea
[2]Genomic Medicine Institute (GMI), Medical Research Center, Seoul National University, Seoul, Korea
[3]Department of Pathology, Seoul National University College of Medicine, Seoul, Korea
[4]Department of Biochemistry and Molecular Biology, Seoul National University College of Medicine, Seoul, Korea
*Both authors contributed equally to this paper as co-corresponding authors.

Palmoplantar pustulosis (PPP) is a chronic, relapsing inflammatory disorder of the palms and soles with incompletely defined pathogenesis. Using single-cell RNA sequencing and high-resolution spatial transcriptomics, we chart the immune landscape across healthy skin, non-pustular lesions, and pustular lesions. We observe heightened JAK–STAT signaling in keratinocytes and fibroblasts, alongside a stepwise expansion of myeloid dendritic cells and Th17 cells across disease stages. Ligand–receptor analyses highlight *CCL19–CCR7* communication between fibroblastic reticular cell (FRC)-like fibroblasts and LAMP3+ migratory dendritic cells, and *CCL22/CCL17–CCR4* signaling between dendritic cells and $CD4^+$ T cells, including Th17 subsets. Spatial mapping validates these interactions in situ and delineates a lymphoid-like, *CCL19+* immune niche in the upper dermis. Keratinocytes within and surrounding pustules, together with the pustular compartment itself, emerge as major sources of *CXCL1/6/8* engaging *ACKR1+* endothelial cells to recruit neutrophils. These coordinated circuits form an amplifying network that establishes a spatially organized, dense immune niche during PPP progression. Finally, transcriptome-guided drug effect prediction nominates JAK and PDE4 inhibitors as multi-target therapeutic candidates. Collectively, our findings provide a comprehensive view of the immune dynamics in PPP and offer insights into effective treatment strategies.

FC3-2. Disruption of tight junction integrity by *Staphylococcus aureus* via TLR2-JAK2-STAT3 signaling and its reversal by the Pseudo-ceramide, DDSS in the 3D skin model

Haivin Kim[1], Aram Kim[1], Hanyoung Kim[3], Dahye Seo[1], Suji Son[1], DongHyun Kim[2,4] and Jung U Shin[2,*]

[1]Department of Biomedical Science, CHA University, Seongnam, Korea
[2]Department of Dermatology, CHA Bundang Medical Center, CHA University School of Medicine, Seongnam, Korea
[3]Central Research Laboratories Aekyung Co., Ltd., Daejeon, Korea
[4]StratumBio Inc., Seongnam, Korea

Background: *Staphylococcus aureus* (*S. aureus*) colonization is frequently observed in atopic dermatitis (AD) and is closely associated with epidermal barrier dysfunction and inflammatory responses. However, the molecular mechanisms by which *S. aureus* compromises tight junction (TJ) integrity in the skin remain incompletely understood.
Objective: This study aimed to elucidate the signaling pathways involved in *S. aureus*-induced skin inflammation and TJ disruption and inflammation, and to evaluate the therapeutic potential of the pseudo-ceramide Dodecenyl Stearylsuccinamide (DDSS).
Methods: We utilized both 2D keratinocyte cultures and 3D skin models to assess TJ integrity and inflammatory responses after *S. aureus* treatment, with or without Th2 cytokines. Transcriptomic analysis, Western blots, RT-qPCR, and signaling inhibition experiments were conducted to investigate the underlying mechanisms and the effects of DDSS.
Results: In both keratinocytes and 3D skin models, *S. aureus* penetrated deeper into the epidermis under Th2-skewed conditions, aggravating the disruption of TJ proteins and loss of scaffolding proteins such as Membrane Associated Guanylate Kinase, WW and PDZ Domain Containing 1 (MAGI1). This promoted robust increases in pro-inflammatory cytokines, including *TNFA, IL1B, IL6,* and *IL8.* These effects were associated with activation of the TLR2–JAK2–STAT3 signaling axis. Inhibition of TLR2, JAK2, or STAT3 restored MAGI1 expression and the structure of ZO-1, thereby improving TJ integrity and reducing cytokine expression. Notably, DDSS treatment reinforced the skin barrier, effectively suppressed *S. aureus* penetration, inhibited TLR2–JAK2–STAT3 activation, and significantly attenuated inflammatory cytokine production. Collectively, these results highlight a synergistic barrier-disruptive and inflammatory effect of *S. aureus* and Th2 cytokines, and demonstrate that targeting this axis with DDSS can restore barrier function and reduce inflammation.
Conclusion: Our findings demonstrate that *S. aureus* impairs epidermal barrier integrity and enhances inflammation via TLR2–JAK2–STAT3-mediated downregulation of MAGI1 and TJ disruption. The DDSS attenuates these effects by blocking bacterial infiltration, suggesting its potential as a therapeutic agent in AD-associated skin inflammation.

FC3-3. Probiotic administration attenuates hs-like skin inflammation via modulation of gut microbiota and immune responses in mice

Da Hye Seo[1], Hai Vin Kim[1], Jae Sang Ryu[2], Young Su Jang[1], Hee Jung Lee[2], Dong Hyun Kim[2], Yun Kyung Jang[2], Jung U Shin[2,*]

[1]Department of Biomedical Science, College of Life Science, CHA University, Seongnam, Korea
[2]Department of Dermatology, CHA Bundang Medical Center, CHA University School of Medicine, Seongnam, Korea

Hidradenitis suppurativa (HS) is a chronic inflammatory skin disorder characterized by recurrent painful nodules and abscesses, driven by follicular occlusion and aberrant immune activation. Emerging evidence suggests that the gut–skin axis plays a critical role in the pathogenesis of inflammatory skin diseases, including HS. This study aimed to investigate the effects of Probiotics on HS-like skin inflammation induced in mice. HS-like inflammation was induced by oral administration of the γ-secretase inhibitor LY-411575 combined with repeated tape-stripping of the dorsal skin. Immune cell composition was assessed by flow cytometry, skin immune cell infiltration was evaluated by immunohistochemistry, inflammatory gene expression was quantified by qRT-PCR, and fecal microbiota composition was analyzed by 16S rRNA full-length sequencing. Probiotic treatment significantly reduced epidermal thickness, macrophage and neutrophil infiltration, and the expression of *Il1b, Tnfa, Il6, Il36a, Il36g, Nlrp3, S100a8, S100a9, Ifng*, and *Il17a* in the skin. In addition, probiotic treatment markedly decreased the proportions of IFN-γ^+ and IL-17A$^+$ CD4$^+$ T cells in the mesenteric and axillary lymph nodes. Notably, probiotic administration increased the relative abundance of microbial taxa associated with short-chain fatty acid production. These findings suggest that probiotic administration may modulate both systemic and skin inflammation by altering gut microbiota composition, highlighting its potential as a novel therapeutic approach for inflammatory skin diseases such as HS.

FC3-4. Keloid pathogenesis from the aspect of skin site: Based on single-cell RNA sequencing

Eunhye Yeo, Junesoo Jeon, Se Jin Oh, Ji Hye Park, and Jong Hee Lee*

Department of Dermatology, Samsung Medical Center, School of Medicine, Sungkyunkwan University, Seoul, Korea

Human skin maintains extensive spatial heterogeneity with distinct positional identity across anatomical sites. Keloids exhibit pronounced site-specific clinical manifestations, yet the molecular mechanisms underlying this anatomical heterogeneity remain poorly understood. This study aimed to characterize site-specific molecular signatures and identify distinct pathogenic mechanisms between two anatomically representative keloid sites. We performed single-cell RNA sequencing on keloid tissues from chest (n=6) and earlobe (n=6) sites to elucidate anatomically distinct pathways. Fibroblast subpopulations were identified through unsupervised clustering, followed by differential gene expression analysis and pathway enrichment. Cell-cell communication networks were reconstructed using ligand–receptor databases. Chest keloids demonstrated an inflammatory circuit modulated by adipocyte-derived signaling, with fibroblasts exhibiting upregulated prostaglandin synthesis genes and diverse prostaglandin receptors, suggesting competing pro- and anti-fibrotic influences. Elevated expression of pattern recognition and immune regulatory genes further established self-sustaining adipocyte–immune signaling loops, reinforcing chronic inflammatory activation. Conversely, earlobe keloids showed enhanced mesenchymal-to- myofibroblast transition with upregulated contractile genes and matrix remodeling factors, indicating myofibroblast differentiation as the primary pathogenic driver. Our findings reveal anatomically distinct keloid mechanisms: chest lesions are driven by adipocyte-linked inflammatory feedback, whereas earlobe keloids involve direct activation of fibrogenic myofibroblasts. These differences support site-specific therapeutic strategies and provide a molecular rationale for precision medicine approaches in keloid treatment.

FC3-5. Specific biomarkers for melanoma revealed by spatial transcriptomic analysis and single-cell RNA sequencing

Tae Min Lee, Joonho Shim, Jihye Park, Se Jin Oh, Jong Hee Lee, Dongyoun Lee
Department of Dermatology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

There are limitations in histopathological diagnosis for borderline melanocytic tumors, and ancillary tests, such as immunohistochemistry, can provide greater diagnostic confidence. However, the markers currently in use are melanocyte markers rather than melanoma-specific markers. Our aim was to identify melanoma-specific biomarkers using molecular techniques and to characterize the differences between benign nail melanocytic lesions and early nail unit melanomas (melanoma in situ). Patients diagnosed with acral melanoma or benign melanocytic tumors via skin biopsy were included in the study. We spatially profiled regions of interest, including acral melanoma, benign melanocytic tumors, and control normal skin tissue, using digital spatial profiling. Additionally, we performed single-cell RNA sequencing on fresh specimens. Biomarker candidates were selected based on the data. We found that several genes were specifically highly expressed in melanoma cells compared to benign melanocytic lesions. Among these, we identified PRAME as a potential diagnostic marker for melanoma cells through spatial transcriptomics and single-cell RNA sequencing, and validated its RNA expression in melanoma cells via RNA-ISH. Furthermore, we confirmed PRAME expression in the nuclei of most melanoma cells across all 15 nail melanoma in situ cases. In our study, comparative spatial transcriptomic and single-cell analyses of melanoma revealed several potential diagnostic biomarkers for distinguishing melanoma from benign melanocytic tumors. These molecular techniques are valuable tools for discovering melanoma biomarkers.

FC3-6. AI-driven discovery of novel HSP47 activating peptides for skin anti-aging

Seokjeong Yoon, Yeonjae Kim, Hwa-jee Chung and Sekyoo Jeong*
R&D Center, Incospharm Corporation, Daejeon, Korea

Artificial intelligence (AI)-driven technologies are exerting a profound influence across industries and are rapidly expanding into both academia and everyday life. Recently, we have established an AI-guided peptidomimetic discovery system, termed AMPed (AI-Maneuvered Peptidomimetic Design), designed to identify novel peptide candidates targeting specific proteins. The AMPed system consists of two main modules: a "similarity coefficients" calculator, which screens a library of 67 million peptide molecules to identify the candidates with similar chemical structure to the reference molecule based on the chemical fingerprints; and a "protein-peptide binding" simulator, which predicts the binding affinity of the selected peptide to the target protein. Lead peptides with the highest predicted binding property were synthesized and subsequently subjected to bioactivity evaluation. Heat shock proteins (Hsp) are a highly conserved family of proteins produced in response to various cellular stressors, including elevated temperature, ultraviolet radiation, inflammation, and hypoxia. Hsp47, also known as Serpin H1, is a collagen-specific molecular chaperone required for the correct folding of procollagen in vertebrate cells. Reduced expression of Hsp47 in aged mice skin and increased type I collagen expression in dermal fibroblast upon Hsp47 stimulation suggest its potential as a therapeutic target for anti-wrinkle strategy. In this study, we explored the applicability of the AMPed system in developing novel anti-wrinkle peptide candidates with Hsp47 stimulating activity using series of *in vitro* and *ex vivo* studies.

Special Lecture (1)

Sep. 27th 09:20-10:10

좌장 | 강희영(아주의대)

조선시대 초상화, 그 아름다운 흠집

가천대학교 전 총장 이성낙

Special Lecture (1)

조선시대 초상화, 그 아름다운 흠집

이성낙
가천대학교 전 총장

약력:

- 독일 Marburg 의대(예과), Muechen의대 졸업. 같은 대학에서 박사학위를 받다.
- Frankfurt 의대에서 피부과학 전문의자격 및 교수자격(Habilitation)을 취득하다.
- 연세대 의대피부과주임교수
- 아주대 의대초대학장 및 의무부총장
- 가천대학교 총장
- 국제베체트학회 회장
- 명지대학교 대학원 미술사학과에서 〈조선시대 초상화에 나타난 피부병변 연구〉로 미술사학 박사학위.
- 前 (사)현대미술관회 회장
- 前 한국의·약사평론가회장.
- 現 (재)간송미술문화재단이사.
- 독일연방공화국대통령 수여
- 십자공로훈장

Special Lecture (2)

Sep. 27th 11:10-11:40

좌장 | 이원주(경북의대)

특허, 연구자를 발명자로 만드는 첫걸음

특허청 과장 양인수

Special Lecture (2)

특허, 연구자를 발명자로 만드는 첫걸음

양인수
특허청 과장

학력:

- 한양대학교 화학공학 졸업
- 미국 워싱턴 주립대학교 법학석사
- 한국개발연구원(KDI) 공공정책대학원 공공정책학 석사
- 충남대학교 일반대학원 법학 박사
- KAIST 미래과학기술정책과정

경력:

- 특허청 심사관, 심판관
- 국제특허출원(PCT) 심사과장, 의료기술, 식품, 바이오 분야 심사과장
- 부정경쟁조사팀장
- 대전지방검찰청 특허자문관
- 대법원 특허조사관

논문 및 저서:

〈부정경쟁방지법 판례 백선〉, 〈영업비밀의 비공지성에 관한 연구〉, 〈신규성 상실의 예외 규정과 자유 실시 디자인의 항변에 관한 연구〉, 〈고유성(inherency)에 의한 신규성 부정 여부〉, 〈진보성 판단에서 발명의 효과〉, 〈부정경쟁방지법상 데이터 부정사용행위에 관한 행정조사 매뉴얼〉, 〈파리조약(Paris Convention) 우선권의 기초가 된 선출원의 최초 명세서 등에 기재된 사항의 의미〉, 〈디자인 침해판단에 있어서 공지 부분을 포함하는 디자인의 유사판단에 관한 연구〉, 〈부정경쟁방지법 상품형태 모방 규정에 있어서 통상적인 형태의 의미에 관한 연구〉, 〈수치한정(Numerical-limitation) 관련 발명의 보호범위 해석에 관한 연구〉, 〈제조방법 한정 물건 청구항(Product-by-Process Claim)의 청구범위 해석에 관한 연구〉, 〈수치한정 관련 발명의 균등론에 관한 연구〉, 〈특허법상 물과 방법 카테고리에 대한 고찰〉 등

수훈:

- 우수공무원 대통령 표창
- 특허심판원 판례연구논문 공모전 최우수상
- 한국지식재산연구원 등재학술지 '지식재산연구' 우수논문상 등

Posters

Poster Walk: P1~P5

좌장 | 이영(충남의대), 조성진(서울의대), 김혜원(한림의대)

Posters

Poster Walk P1. Chronic serum protein leakage as a prognostic biomarker in chronic spontaneous urticaria

Byunghyuk Lee[1], Jungsoo Lee[1,2,3], Hoon-Soo Kim[1], Byung-Soo Kim[1], Moon-Bum Kim[1], Hyun-Chang Ko[1,2,3*], Kihyuk Shin[1,2,3*]

[1]Department of Dermatology, College of Medicine, Pusan National University, Busan, Korea
[2]Department of Dermatology, Pusan National University Yangsan Hospital, Yangsan, Korea
[3]Research Institute for Convergence of Biomedical Science and Technology, Pusan National University Yangsan Hospital, Yangsan, Korea

Chronic spontaneous urticaria (CSU) is a debilitating inflammatory dermatosis affecting 0.5-1% of the global population. To investigate the relationship between vascular permeability in CSU patients and their correlation with disease severity, we analyzed 138 CSU patients and 138 healthy controls for serum protein markers and inflammatory mediators using OLINK proteomic profiling. Disease severity was assessed using UAS7 scores, and patients were classified as antihistamine responders or non-responders. CSU patients exhibited significant decreased serum total protein levels, while the BUN/creatinine ratio was increased compared to healthy controls. Protein reduction correlated negatively with UAS7 scores and was more pronounced in severe cases and antihistamine non-responders. Notably, most inflammatory mediators showed significantly decreased serum levels in CSU patients, demonstrating extravasation and it correlates with disease severity. In particular, serum IL-2 levels showed a significant inverse correlation with UAS7 scores. Taken together, CSU is characterized by enhanced vascular permeability leading to protein leakage. Our findings suggest that various serum proteins resulting from chronic protein extravasation may contribute to the development of comorbidities.

Poster Walk P2. Site-specific skin surface lipid–microbiome dysregulation in pediatric mild atopic dermatitis

Han Bi Kim, Ji Young Um, Bo Young Chung, Chun Wook Park, Hye One Kim*
Department of Dermatology, Kangnam Sacred Heart Hospital, Hallym University College of Medicine, Seoul, Korea

Skin surface lipids and microbiota are crucial for barrier and immune function. However, site-specific lipidomic data in pediatric atopic dermatitis (AD) remain limited, despite known regional differences in skin lipid composition. This study aimed to analyze site-specific skin lipid composition to identify key lipid alterations, AD-specific microbial profiles, and potential lipid-based therapeutic targets. A total of 36 pediatric subjects (AD and healthy controls) were enrolled, with AD diagnosed using the Hanifin and Rajka criteria. Skin samples from the nose (oily), abdomen (dry), and antecubital fossa (wet) using tapping methods. Lipidomic analysis was performed using LC-MS/MS, and microbial composition and functional profiles were examined through shotgun metagenomic sequencing. Lipidomic profiling consistently revealed reduced levels of long-chain FFAs (LCFAs)/VLCFAs in AD skin, especially FFA 22:0 (behenic acid), which showed the most pronounced depletion at the antecubital fossa. This was accompanied by altered microbiome composition, where higher levels of FFA 22:0 correlated with commensal, barrier-supportive taxa such as *Streptococcus salivarius* and *Prevotella copri*, while lower levels were associated with increased abundance of pathobiont species including *Prevotella timonensis* and *Streptococcus thermophilus*. Functional pathway predictions further demonstrated that key microbial lipid metabolic routes—especially fatty acid elongation via ELOVL1 and ELOVL7—were significantly downregulated in AD skin. Together, these findings suggest that microbial functional impairment may contribute to the depletion of critical lipid species, thereby exacerbating skin barrier disruption in AD. Site-specific alterations in skin surface lipid composition and microbial profiles were observed in pediatric AD skin, particularly at the antecubital fossa. Reduced levels of LCFAs, such as FFA 22:0, were associated with shifts in microbial communities and downregulation of lipid-related metabolic pathways. These findings suggest a potential mechanistic link between skin surface lipid depletion and microbial functional changes in AD, supporting further investigation into lipid–microbiome interactions in skin barrier dysfunction.

Poster Walk P3. Personalized modeling of Th2 immunity in atopic dermatitis using AVATAR mice

Hye Li Kim[1,2], Kelun Zhang[1,2], Su Min Kim[1,3], Ryeo Won Kim[1], Wan Jin Kim[1], Yeon Woo Jung[1], Chang Ook Park[1,2*]

[1]Department of Dermatology and Cutaneous Biology Research Institute, Yonsei University College of Medicine, Seoul, Korea
[2]Brain Korea 21 PLUS Project for Medical Sciences, Yonsei University College of Medicine, Seoul, Korea

Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by complex and heterogeneous immune mechanisms. Despite the availability of Th2-targeted therapies, current treatment strategies remain generalized, often following a 'one-size-fits-all' approach. To address this limitation, we established a humanized AVATAR mouse model that reflects the immunopathology of individual AD patients. $CD3^+$ T cells from house dust mites (HDM)-sensitized AD patients were intravenously administered as pathogenic cells, while CD3-depleted PBMCs from the same patients were intradermally administered as antigen-presenting cells (APCs) into immunodeficient NOD-scid IL2Rγ null (NSG) mice. This approach enabled the AVATAR mouse to develop AD-like inflammatory responses upon HDM exposure. Single-cell RNA sequencing analysis of the AVATAR mice revealed an expansion of *IRF4*$^+$*MRC1*$^+$ type 2 conventional dendritic cells (cDC2s) and increased expression of Th2-associated genes, mirroring transcriptional profiles observed in human AD skin. Furthermore, TCRβ deep sequencing identified shared T cell clones between HDM-sensitized AVATAR mice and HDM-sensitized patients with AD. Notably, a correlation in clinical and laboratory parameters was observed between individual AD patients and their corresponding matched AVATAR mice. Specifically, patients with severe AD exhibited elevated $CD3^+$IL-13^+T cells, reflecting heightened Th2 immune responses, which were faithfully recapitulated in their matched AVATAR mice. In summary, these findings demonstrate that the AVATAR mouse model accurately reflects the antigen-specific immune profiles of individual AD patients, providing a valuable platform for advancing personalized, patient-tailored therapeutic strategies in AD.

Poster Walk P4. Validation of C3H/HeN mice as an alopecia areata animal model

Eun Hye Kim[1], Soyeon Park[1], Dongju Ahn[1], Mei Zheng[2], Jong-Hyuk Sung[2*], Do-Young Kim[1*]

[1]Department of Dermatology and Cutaneous Biology Research Institute, Yonsei University College of Medicine, Seoul, Korea,
[2]Epi Biotech Co., Ltd., R&D Center, Incheon, Korea

The C3H/HeJ mice have been predominantly used as a model to investigate the pathogenesis of alopecia areata (AA) and evaluate therapeutic interventions. The C3H/HeN strain has remained relatively underexplored despite its recent application in AA-related studies. Unlike C3H/HeJ, which is null for toll-like receptor 4 (TLR4), the C3H/HeN strain is TLR4 wild-type, and thus its validity as an AA model remains to be fully established. This study aimed to validate C3H/HeN as a reliable AA model by examining disease development and its immune pathogenesis. C3H/HeN mice began to develop hair loss within 10 weeks after transfer of cultured skin-draining lymph node (SDLN) cells, and the majority developed AA by 20 weeks, at a rate similar to C3H/HeJ. In AA-affected C3H/HeN mice, $CD44^{s\text{-}hi}CD49d^{lo}$ $CD8^+$ T cells and $NKG2D^+$ $CD8^+$ T cells in the SDLN were significantly increased. Skin lesions showed marked infiltration of $CD45^+$ leukocytes and $CD8^+$ T cells. To assess the cytokine-induced differentiation efficiency of $CD44^{hi}$ T cells into $CD44^{s\text{-}hi}$ T cells, we cultured sorted $CD44^{hi}$ $CD8^+$ T cells with IL-12, IL-15, and IL-18. Upon cytokine stimulation, $CD44^{hi}$ T cells from both C3H/HeN and C3H/HeJ mice differentiated into $CD44^{s\text{-}hi}$ T cells. Additionally, we investigated the effect of a preceding illness on the subsequent development of AA by inducing mild systemic inflammation via LPS prior to AA induction. Following LPS injection, only C3H/HeN exhibited weight loss and increased $CD44^{hi}$ $CD49d^{lo}$ $CD8^+$ T cells in the SDLN cells, whereas C3H/HeJ mice did not. However, AA incidence and progression in LPS-stimulated HeN mice were comparable to the non-stimulated group. Our study validated C3H/HeN as a reliable AA model, demonstrating a comparable AA induction rate to C3H/HeJ and identifying AA-associated pathogenic immune cells in the SDLNs and skin. Future studies should consider modifying the timing of stimulation and applying more clinically relevant factors.

Poster Walk P5. Minoxidil sulfate suppresses JAK/STAT pathway and restores mitochondrial function in IFN-γ and poly(I:C)-stimulated ors cells: Implications for alopecia areata

Jung-Min Shin, Kyung Eun Jung, Chang Deok Kim, Young Lee
Department of Dermatology, Chungnam National University School of Medicine, Daejeon, Korea

Background: Minoxidil sulfate (MXS), the active metabolite of minoxidil, is widely used to treat androgenetic alopecia due to its hair growth-promoting properties. However, its immunomodulatory potential remains poorly characterized, particularly in alopecia areata (AA), an autoimmune disease marked by the collapse of hair follicle immune privilege.
Objectives: This study aimed to investigate the anti-inflammatory and mitochondrial-protective effects of MXS in human outer root sheath (ORS) cells under inflammatory stress that mimics the AA microenvironment. Additionally, we evaluated the combinatorial effect of MXS with baricitinib, a JAK inhibitor approved for AA treatment.
Methods: Human ORS cells were exposed to interferon-gamma (IFN-γ) and polyinosinic:polycytidylic acid (poly[I:C]) to simulate an inflammatory AA-like environment. Cells were pretreated with MXS, and subsequent changes in JAK/STAT signaling, MHC class I expression, IFN-γ-induced chemokine production (CXCL9, CXCL10, CXCL11), and mitochondrial function were analyzed. Mitochondrial parameters included reactive oxygen species (ROS) levels, mitochondrial DNA (mtDNA) damage, and membrane potential. A co-treatment condition using baricitinib was also assessed.
Results: MXS significantly inhibited the phosphorylation of STAT1 and STAT3, key effectors in the JAK/STAT pathway. It downregulated MHC class I expression and suppressed the IFN-γ-induced chemokines CXCL9, CXCL10, and CXCL11. MXS also reduced cytosolic and mitochondrial ROS levels, attenuated mtDNA damage, and restored mitochondrial membrane potential, indicating mitochondrial protection. Notably, co-treatment with baricitinib resulted in synergistic suppression of pro-inflammatory signaling and chemokine expression.
Conclusions: MXS exerts dual immunomodulatory and mitochondrial-stabilizing effects in ORS cells exposed to inflammatory stimuli. These findings highlight the potential of MXS as an adjunctive therapeutic strategy alongside JAK inhibitors in the treatment of alopecia areata.

Poster Walk P6. ATG7 dysfunction in senescent melanocytes and hypopigmented skin: Reversal by metformin

Jin Cheol Kim[1], Yeongeun Kim[1,2], Tae Jun Park[2,3], Hee Young Kang[1,2]
[1]Dermatology, Ajou University School of Medicine, Suwon, Korea
[2]Inflamm-Aging Translational Research Center, Ajou University School of Medicine, Suwon, Korea
[3]Biochemistry and Molecular Biology, Ajou University School of Medicine, Suwon, Korea

Melanocyte senescence predominantly occurs in sun-exposed skin of elderly individuals and contributes to hypopigmentary disorders. Through single-cell transcriptomic and time-course analyses, we identified that autophagy dysregulation is an early event preceding glycolytic reprogramming during UV-induced melanocyte senescence. Among these changes, downregulation of ATG7 emerged as the earliest molecular alteration and was consistently observed in both senescent melanocytes and hypopigmented aged skin. Metformin treatment restored autophagic activity, including ATG7 upregulation, and mitigated oxidative stress, thereby delaying melanocyte senescence. These findings highlight the critical role of impaired autophagy in melanocyte aging and suggest that targeting the autophagy–oxidative stress axis may serve as an effective early intervention strategy to prevent melanocyte senescence and related hypopigmentary disorders.

Poster Walk P7. The role and molecular mechanisms of melanophilin in skin cell aging

Seohyun Park[1], Eun Jung Lee[1], Ji Young Kim[1], Yu Jeong Bae[1], Shinwon Hwang[1], Il Joo Kwon[1], Jamal Mohammed Alqahtani[2], Jinu Lee[3], Sang Ho Oh[1*]
[1]Department of Dermatology and Cutaneous Biology Research Institute, Severance Hospital, Yonsei University College of Medicine, Seoul, Korea.
[2]Department of Dermatology, Imam Abdulrahman Bin Faisal University, Saudi Arabia
[3]College of Pharmacy, Institute of Pharmaceutical Sciences, Yonsei University, Incheon, Korea

Melanophilin (Mlph) is a key component of melanosome transport in melanocytes. Clinically, mutations in melanin transport proteins cause different subtypes of Griscelli syndrome (GS). In particular, GS type 3, caused by mutations in *MLPH,* is characterized solely by hypopigmentation without any systemic abnormalities, highlighting its melanocyte-specific function. Although previous studies have reported that reduced expression of melanin transport proteins in aged melanocytes, it remains unclear whether suppression of these proteins can induce melanocyte senescence. Therefore, we investigated the role of Mlph in melanocyte senescence. This study aims to elucidate the role and molecular mechanisms of Mlph in skin cell aging. We hypothesize that *MLPH* knockout (KO) causes cellular senescence in melanocytes through intracellular melanosome accumulation, which may influence the aging of neighboring skin cells. We established *MLPH* KO MNT1 cells using CRISPR/Cas9 lentiviral vectors. In *MLPH* KO cells, tyrosinase protein expression and melanin content were increased, with perinuclear melanin accumulation. HaCaT cells co-cultured with *MLPH* KO cell revealed reduced gp100 expression, indicating impaired melanin transfer to keratinocyte. Upon etoposide treatment, *MLPH* KO cells showed elevated levels of DNA damage marker p-H2AX compared to controls. In addition, senescence markers p16 and p21 were significantly upregulated in *MLPH* KO cells. Our findings suggest that suppression of Mlph leads to intracellular melanin accumulation and impaired melanin transfer, which may contribute to DNA damage and melanocyte senescence.

Poster Walk P8. Protective role of *Lactiplantibacillus plantarum* ferment lysates on PM-induced skin aging

Do Yeon Kwon[1], Yoon Jin Roh[1], Hye Won Song[2], Seung Hyeon Kim[2], Soeun Jeon[1], Ji Yeon Hong[1], Joon Seok[1], Mi-Kyung Lee[2], Kui Young Park[1*]
[1]Department of Dermatology, Chung-Ang University College of Medicine, Seoul, Korea
[2]Department of Laboratory Medicine, Chung-Ang University College of Medicine, Seoul, Korea

Airborne particulate matter (PM) is a major environmental pollutant that accelerates skin aging by inducing inflammation, oxidative stress, and degradation of extracellular matrix (ECM). *Lactiplantibacillus plantarum* (formerly Lactobacillus plantarum) is known for its skin-beneficial properties, including anti-inflammatory effects and enhancement of the skin barrier function. Therefore, the objective of this study was to evaluate the anti-aging and anti-inflammatory effects of *Lactiplantibacillus plantarum* ferment lysates (LFLs) using a PM induced- skin aging model based on a co-culture system of human keratinocytes (HaCaT) and dermal fibroblasts (HDFs). To assess the protective effects of LFLs, we first measured the cytotoxicity of PM in HaCaT cells using a WST assay and selected a concentration that did not significantly reduce cell viability. LFLs were also confirmed to be non-cytotoxic in HDFs. Based on these results, LFL (10 μg/mL) was applied for 48H in the PM treated co-culture model. The mRNA expression levels of inflammatory cytokines, COX-2, and matrix metalloproteinases (MMPs) were measured by real-time quantitative PCR (RT-qPCR). Cellular senescence was examined through senescence-associated β-galactosidase (SA-β-gal) staining. Western blotting was performed to analyze the protein levels of aging markers, collagen-related proteins, and signaling molecules, including NF-κB and MAPKs. LFL treatment significantly reduced the expression of pro-inflammatory cytokines, COX-2 and MMPs. The number of SA-β-gal-positive cells were also markedly decreased in the LFL-treated group compared to the PM-only group. At the protein level, LFL effectively inhibited PM-induced activation of NF-κB and MAPKs signaling, modulating inflammation and aging markers. These findings support the translational potential of L. plantarum ferment lysates (LFLs) for future dermatological and cosmetic applications through their ability to regulate inflammatory responses, prevent collagen degradation, and reduce cellular senescence.

P9. Objective morphological analysis of hair abnormalities in liph-deficient mice

Mi Eun Choi[1], Hye Young An[1], Ji Won Byun[1], Jeonghyun Shin[1], Gwang Seong Choi[1], Hyun-Tae Shin[1,2*]
[1]Department of Dermatology, Inha University Hospital, Incheon, Korea
[2]Research Center for Controlling Intercellular Communication (RCIC), Inha University School of Medicine, Incheon, Korea

Objective and reproducible evaluation methods are essential for analyzing hair phenotypes in mouse models of hair loss or follicular disorders. LIPH encodes PA-PLA1α, which produces lysophosphatidic acid (LPA), activating P2Y5 and EGFR signaling pathways vital for normal hair shaft and inner root sheath formation. Liph knockout (KO) mice display visible coat and wavy vibrissae abnormalities, but quantitative analysis of these phenotypes remains limited. To establish an ImageJ-based quantitative method for assessing morphological differences in vibrissae angle, dorsal hair regrowth area, and vibrissae growth rate between wild-type (WT) and Liph KO mice. Vibrissae and dorsal hairs were collected from WT and Liph KO mice. Each vibrissa angle was individually measured using ImageJ under standardized imaging conditions. Dorsal hair and right-side vibrissae were shaved and photographed weekly for 5 weeks to analyze regrowth area and length. ImageJ's semi-automated thresholding and distance tools were used for quantification. Structural analysis of hairs and cuticle integrity was performed using scanning electron microscopy (SEM). Liph KO mice showed significantly greater vibrissae angles and reduced regrowth rates of vibrissae and dorsal hair compared to WT mice. SEM analysis revealed distinct cuticle damage and structural abnormalities in KO hairs. Liph deficiency disrupts follicular integrity, resulting in slower hair regrowth and vibrissae cuticle damage. ImageJ-based morphometric analysis offers an objective and reproducible approach for evaluating complex hair phenotypes in genetically modified mouse models.

P10. The effect of adipose stem cell derived exosome on the alopecia areat and JAK/STAT pathway

Byung Cheol Park, Mi Soo Choi, Myung-Wha Kim
[1]Department of Dermatology, Dankook University Hospital, Cheonan, Korea

Background: Mesenchymal stem cells have been reported to regulate the JAK/STAT pathway in cellular and hair follicle organ cultures, suggesting their potential use in treating alopecia areata. However, specific clinical applications in alopecia areata have not yet been reported. In this study, we aimed to evaluate the clinical efficacy of adipose stem cell-derived exosomes (ASCE+HRLV), which possess stem cell-like properties, in patients with alopecia areata and to investigate the underlying mechanisms related to the JAK/STAT pathway.
Materials and Methods: The clinical study included six patients with alopecia areata. Among them, three were unresponsive to baricitinib treatment for more than 12 months, and the other three did not respond to conventional therapies such as cyclosporine, steroid. Patients received microneedling therapy (MTS) combined with exosome (ASCE+HRLV) treatment every two weeks for 48 weeks. Clinical photographs and SALT scores were evaluated at weeks 12, 24, 36, and 48. Safety assessments were conducted through blood tests and local scalp reaction. For in vitro experiments, human dermal papilla cells and outer root sheath (ORS) cells were primarily cultured. Cells were treated with interferon-r(IFN-r), followed by either JAK inhibitors or exosomes, and their proliferative recovery was assessed.
Results: Among the six patients, the three who were unresponsive to baricitinib showed no improvement with exosome treatment. Of the three patients who did not respond to conventional therapies, one showed an increase in SALT score from 0 to 70, and another improved to a SALT score of 30. Notably, two patients experienced complete regrowth of their eyelashes and eyebrows. In the cell experiments, treatment with IFN-γ reduced the proliferation of both dermal papilla cells and outer root sheath (ORS) cells. However, when treated with JAK inhibitors or exosomes, cellular proliferation was restored.
Conclusion: Exosomes were found to inhibit inflammation by blocking the JAK/STAT pathway associated with IFN-γ

in alopecia areata, thereby promoting clinical improvement.

P11. Anti-photoaging effects of a vascular-derived ECM filler containing polynucleotides and hyaluronic acid

Yoon Jin Roh[1], Do Yeon Kwon[1], Hye Won Song[2], Seung Hyeon Kim[2], Soeun Jeon[1], Ji Yeon Hong[1], Joon Seok[1], Mi-Kyung Lee[2], Kui Young Park[1*]
[1]Department of Dermatology, Chung-Ang University College of Medicine, Seoul, Korea
[2]Department of Laboratory Medicine, Chung-Ang University College of Medicine, Seoul, Korea

Polynucleotides (PN) and hyaluronic acid (HA) are widely utilized as dermal fillers and skin boosters, with well-established efficacy and mechanisms in promoting skin regeneration and hydration. Recently, a novel filler formulation incorporating vascular-derived extracellular matrix (VdECM) with PN and HA has been developed, aiming to enhance regenerative outcomes while maintaining biocompatibility and safety. The rheological characteristics of the new VdECM filler were assessed. Thirty-five female hairless mice (SKH1) underwent photoaging induction via ultraviolet B (UVB) irradiation for 12 weeks. The mice were randomly divided into seven groups and intradermally injected with 100 μl of either phosphate-buffered saline, VdECM/PN/HA filler, VdECM/PN filler, VdECM/HA filler, HA filler, or PN alone into the dorsal region. To evaluate the effects of the fillers on photoaged skin, dermoscopic examination was performed. Additionally, histological evaluations, including immunohistochemical staining, were conducted at 14 weeks to assess biocompatibility and collagen formation. Real-time quantitative polymerase chain reaction (qPCR) and western blot analyses were performed to measure the expression of type I/III collagen, matrix metalloproteinases (MMPs), and transforming growth factor. Histological analysis revealed significant differences in collagen synthesis among the VdECM/PN/HA and VdECM/HA groups. No signs of inflammation were observed during the experimental period. The VdECM/PN/HA and VdECM/HA fillers notably induced type I/III collagen production and downregulated the expression of MMP-1 and MMP-3. Our results suggest that the incorporation of VdECM enhances the anti-photoaging effects of conventional PN/HA fillers and may offer a promising therapeutic option for photoaged skin. Based on these preclinical results, further well-controlled clinical studies are required.

P12. The investigation of immunological mechanism in the treatment of alopecia areata with 308-nm excimer laser

Eojin Kim[1], Jihye Heo[1,2], Su-Young Kim[1,2], Sin Woo Sung[1], Hyun Joo Lee[1,2], Ayeon Choi[1,2], Joon Seok[1,2*]
[1]Department of Dermatology, Chung-Ang University Hospital, Chung-Ang University College of Medicine, Seoul, Korea
[2]Department of Medicine, Graduate School, Chung-Ang University, Seoul, Korea

Alopecia areata (AA) is a T cell-mediated autoimmune disease characterized by non-scarring hair loss and collapse of hair follicle immune privilege. Although Janus kinase (JAK) inhibitors are clinically effective, their systemic side effects and cost underscore the need for alternatives. The 308-nm excimer laser shows promise in AA through localized immunomodulation, but its underlying mechanism remains unclear. AA mouse model was established by adoptive transfer (AT) of activated skin-draining lymph node (SDLN) cells into C3H/HeJ recipients. Disease severity was categorized into "severe AA" and "after AT" groups. Mice were treated with 308-nm excimer laser twice weekly for 12 weeks, during which hair loss areas were quantified, followed by histological and immunohistochemical analyses. 308-nm excimer laser treatment significantly delayed AA progression and induced partial hair regrowth in both severe AA and after AT groups. The hair regrowth areas showed a marked reduction in $CD8^+$ T cells and a significant increase in $FOXP3^+/CD4^+$ Treg proportion, while lesional skin remained highly infiltrated. This shift in immune cell profile indicates restoration of local immune balance in hair regrowth area following excimer laser treatment. This study provides novel evidence that 308-nm excimer laser therapy mitigates AA progression and promotes hair regrowth by reducing cytotoxic $CD8^+$ T cells and enhancing Treg-mediated immune regulation.

P13. Association between periodontitis and rosacea: A nationwide population-based cohort study in Korea

Gwang-Il Kim[1,2], Min-Gu Kang[4], Kyung-Hwa Nam[1,2], Jong-Seung Kim[2,3,4] and Jin Park[1,2*]

[1]Department of Dermatology, Jeonbuk National University Medical School, Jeonju, Korea
[2]Research Institute of Clinical Medicine of Jeonbuk National University-Biomedical Research Institute of Jeonbuk National University Hospital, Jeonju, Korea
[3]Department of Otorhinolaryngology-Head and Neck Surgery, Jeonbuk National University Medical School, Jeonju, Korea
[4]Department of Medical Informatics, Jeonbuk National University, Jeonju, Korea

Background: Periodontitis and rosacea are chronic inflammatory diseases which can be affected by other systemic diseases, or vice versa, and respond to antibiotics such as tetracyclines. Despite their common features, the relationship between periodontitis and rosacea has not been studied yet.

Objective: We investigated to determine the relationship between periodontitis and rosacea from nationwide population-based database.

Methods: Patients diagnosed with periodontitis between 2002 and 2004 were recruited as an experimental group from National Health Insurance Service database. It was divided into treated- and untreated-periodontitis group. We performed 1:1 propensity score matching between control and experimental groups. Each of individuals were monitored until 2019 or the diagnosis of rosacea. The incidence and the adjusted hazard ratio (aHR) of rosacea were calculated with Cox proportional hazards model.

Results: Compared to the control group, in the periodontitis with treatment group and periodontitis without treatment group, the crude HRs for developing new onset of rosacea were 1.59 (95% CI: 1.24-2.04) and 1.55 (95% CI: 1.21-1.99), respectively. The characteristics associated with an increased incidence of rosacea were as follows: female sex, 20-39 years old, 40-59 years old, hypertension (HTN) group, and cardiovascular disease (CVD) group. A characteristic that showed association with decreased incidence of rosacea was those with BMI over 25, which compared to those with BMI below 23. In sensitivity analyses, by giving a variation to design factors one by one, we confirmed that the results did not change based on the matching variables, recruitment periods for the periodontitis group, or matching ratios (all p values < 0.05).

Conclusion: Periodontitis may increase the risk of development of rosacea.

P14. Rab7A regulates melanosome uptake in keratinocytes via PAR2 membrane trafficking

Yu Jeong Bae[1], Ji Young Kim[1], Eun Jung Lee[1], Seohyun Park[1], Shinwon Hwang[1], Il Joo Kwon[1], Jamal Mohammed Alqahtani[2] and Sang Ho Oh[1*]

[1]Department of Dermatology and Cutaneous Biology Research Institute, Yonsei University College of Medicine, Seoul, Korea
[2]Department of Dermatology, Imam Abdulrahman Bin Faisal University, Saudi Arabia

Melanin synthesized by melanocytes is transferred to neighboring keratinocytes to maintain skin pigmentation and protect against ultraviolet (UV) damage. Rab proteins, a family of small GTPases, are known regulators of intracellular membrane trafficking and have been implicated in melanosome biogenesis and transport within melanocytes. However, their role in melanosome transfer and trafficking in keratinocytes remains unclear. This study aimed to investigate the function of Rab7A in melanosome uptake and trafficking in keratinocytes. Preliminary single-cell RNA sequencing of black and gray hair tissues from two donors revealed reduced Rab7A expression in the hair matrix cells of gray hair compared to black hair. To explore the role of Rab7A, Rab7A-knockout HaCaT keratinocytes were generated. Upon treatment with isolated melanosomes, Rab7A-knockout cells showed decreased expression of gp100, a melanosome marker, compared to control HaCaT cells, suggesting reduced melanosome uptake. Furthermore, the expression of protease-activated receptor 2 (PAR2), known to be essential for melanosome internalization in keratinocytes, was also diminished in Rab7A-deficient cells. Time-course assays confirmed that Rab7A-knockout cells exhibited impaired uptake of melanosomes, with a greater amount remaining in the culture medium. Notably, membrane-localized PAR2 was reduced in Rab7A-knockout cells, indicating defective trafficking to the plasma membrane. These findings suggest that Rab7A regulates melanosome uptake in keratinocytes by modulating the membrane trafficking of PAR2, potentially contributing to pigmentation abnormalities.

P15. UVB-induced senescence in primary human melanocytes and the possible therapeutic role of circadian clock-enhancing small molecule

Jiyun Lim[1,2], Soo Hong Seo[1], Hyo Hyun Ahn[1], Im Joo Rhyu[2], Jong-Wha Jung[3], Gi Hoon Son[4*], and Dai Hyun Kim[1*]

[1]Department of Dermatology, Korea University College of Medicine, Seoul, Korea
[2]Department of Anatomy, Korea University College of Medicine, Seoul, Korea
[3]Research Institute of Pharmaceutical Sciences, College of Pharmacy, Kyungpook National University, Daegu, Korea
[4]Department of Biomedical Science, Korea University College of Medicine, Seoul, Korea

Disruption of the circadian rhythm due to harmful environmental factors could contribute to the aggravation of major metabolic, cardiovascular, and skin diseases. In particular, it has been proven that dysfunction of the circadian clock in the skin leads to damage to the skin barrier and accelerates skin aging. Circadian clock-enhancing small molecules (CEMs) have been known to be promising agents for restoring skin barrier homeostasis and senescence-associated negative changes including aging pigmentation. Previous studies demonstrated that treatment with CEMs improved cellular senescence and pigmentation in an Ultraviolet-B (UVB) irradiated photoaging *in vitro* model using B16F10 murine melanocytes. However, murine cell lines have limited clinical relevance to human skin due to differences in cellular physiological characteristics. Therefore, this study aimed to evaluate the effects of CEMs on cellular senescence and pigmentation improvement using primary human melanocytes (HEMn-MP). Ultraviolet-B (UVB) irradiated photoaging *in vitro* model with HEMn-MP cells was treated with CEMs, and the improvement in cellular senescence and pigmentation was evaluated by β-galactosidase (Gal) concentration, melanin content and tyrosinase activity assay. The mRNA and protein levels of senescence- and melanosome transport-related markers evaluated using molecular biological methods. As a result of UVB irradiation, it was observed that β-Gal positive cells and melanin contents were significantly increased in the UVB irradiated group compared to the control group. In addition, the expression of senescence- and melanosome transport biomarkers were meaningfully upregulated in the UVB irradiated group. Treatment of CEMs on senescent human melanocytes significantly reduced β-Gal positive cells and degree of melanin contents and downregulated the expression of senescence- and melanosome transport-related markers. The application of CEMs in the UVB-induced senescence model in our study provided positive results on human melanocyte senescence and aging pigmentation. Primary human melanocytes have a more limited proliferative capacity than cell lines and show greater donor-to-donor variability, but they more accurately reflect physiological characteristics and have greater clinical relevance. Therefore, the use of primary human melanocyte models is expected to be potentially useful in the development of anti-aging and anti-pigmentation therapeutics.

P16. Senomorphic properties of Korean red ginseng-derived components in human dermal fibroblast aging

Hee-Seok Seo, Hannah Hong, Sung Ha Lim, Hyun Kang, Seung-Phil Hong*
Department of Dermatology, Yonsei University Wonju College of Medicine, Wonju, Korea

Senescent cells gradually accumulate in tissues and promote further senescence in neighboring cells through the senescence-associated secretory phenotype (SASP), contributing to tissue dysfunction and aging. While senolytic agents selectively eliminate senescent cells, senomorphic agents suppress senescence-associated phenotypes without inducing cell death. The potential of Korean red ginseng-derived components (RGCs) as senolytics in skin aging remains poorly understood. This study investigated whether RGCs exert senolytic activity in senescent primary human dermal fibroblasts and evaluated their potential as anti-aging agents. Cellular senescence was induced by repeated ultraviolet B (UVB) irradiation, and a co-culture model combining UVB-induced senescent cells and normal cells was used to assess the paracrine effects of SASP. Three RGC preparations—total extract, saponin fraction, and non-saponin fraction—were tested. Expression levels of the senescence marker p16, SASP factors (IL-6, IL-8, and IL-1β), and collagen synthesis in fibroblasts were analyzed. RGCs did not show marked senolytic activity. However, clear senomorphic effects were observed. Treatment with RGCs significantly reduced UVB-induced expression of p16, IL-6, IL-8, and IL-1β. Additionally, RGCs enhanced collagen production in fibroblasts. These findings indicate that, although RGCs do not directly eliminate senescent cells, they act as potent senomorphic agents that reduce inflammatory aging signals and limit their spread, supporting their potential as therapeutic candidates for preventing skin aging.

P17. The scratch-itch-quality of life cycle: Characterizing scratching behavior and its impact in chronic pruritus

Ji Hoon Ryoo[1], Nam Gyoung Ha[2], Ji Hyun Lee[3], Chan Ho Na[4], Bark-Lynn Lew[5], Yong Hyun Jang[2]

[1]Department of Education, Yonsei University, Seoul, Korea
[2]Department of Dermatology, School of Medicine, Kyungpook National University, Kyungpook National University Hospital, Daegu, Korea
[3]Department of Dermatology, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul, Korea
[4]Department of Dermatology, College of Medicine, Chosun University, Gwangju, Korea
[5]Department of Dermatology, College of Medicine, Kyung Hee University, Seoul, Korea

Background: Scratching is a central component of the itch-scratch cycle, which exacerbates skin inflammation, impairs barrier function, and contributes to altered skin microbiota in chronic pruritus. Although scratching behavior is clinically significant, its classification and impact on quality of life (QoL) remain underexplored.
Objective: To examine the multidimensional characteristics of scratching behavior in patients with chronic pruritus, assess their relationship with QoL, and investigate the mediating role of sleep disturbance in these associations using structural equation modeling (SEM).
Methods: A multicenter cross-sectional survey was conducted among 206 adult patients with chronic pruritus. Scratching behaviors were analyzed using exploratory and confirmatory factor analyses, which identified four distinct factors (F1–F4). These factors, along with pruritus descriptors (sensory vs. affective), were examined in relation to ItchyQoL scores. SEM using Mplus software tested direct and indirect (mediated by sleep disturbance) effects between scratching behaviors and QoL.
Results: Four behavioral factors were derived: F1: Mild and protective behaviors (e.g., applying moisturizer, ointment), F2 & F3: Moderately intense behaviors, F4: Aggressive or compulsive behaviors (e.g., bleeding, gouging, moxibustion) Among these, only F4 demonstrated a statistically significant indirect effect on QoL via sleep disturbance, indicating that aggressive scratching patterns may worsen pruritus-related QoL by disrupting sleep. In contrast, F1 showed no direct or mediated effect. Additionally, QoL was not significantly mediated by scratching in models involving sensory or affective itch descriptors, though affective pruritus was generally associated with poorer QoL.
Conclusions: Scratching behavior in chronic pruritus patients can be meaningfully categorized into four factors, with aggressive forms (F4) exerting the most detrimental effect on QoL, particularly through sleep disruption. This supports the clinical importance of addressing not only the intensity of pruritus but also the nature of scratching behavior. Interventions aimed at modifying harmful scratching patterns and improving sleep may enhance patient outcomes.

P18. Reduced-depth, contact-cooled high-intensity focused ultrasound for abdominal fat reduction

Sun Woo Son, Soo-Jin Lee, Se Gyoung Kim, Hyun-Young Cha, Young Joon Park
Department of Dermatology, Ajou University School of Medicine, Suwon, Korea

Background: Obesity is a global health concern associated with multiple comorbidities. Beyond its medical impact, demand for aesthetic body contouring has risen, stimulating interest in non invasive fat reduction technologies such as high intensity focused ultrasound (HIFU). Conventional HIFU devices deliver energy deep enough to heat fascia and epidermis, sometimes producing adverse effects. A redesigned HIFU system with a reduced depth focal zone and real time contact cooling may selectively thermolyse adipocytes while sparing surrounding tissues.
Objectives: To assess the efficacy and safety of a reduced depth HIFU system with contact cooling for the reduction of abdominal subcutaneous fat over 12 weeks.
Methods: Pre clinical thermal profiling using the new device confirmed selective subcutaneous heating with lower epidermal temperature rise. Eleven adults received one HIFU session (three pulses of 50 J cm^{-2}; focal depth 9 mm) and were evaluated at baseline and at weeks 1, 4, and 12. Primary endpoints were changes in waist circumference at week 12. Secondary endpoints included waist to hip ratio (WHR), caliper measured and ultrasound measured abdominal fat thickness,body mass index (BMI), and aesthetic outcomes using investigator and subject rated Global Aesthetic Improvement Scales (IGAIS/SGAIS). Adverse events were recorded on every visit.
Results: In vivo and in vitro pre clinical experiments showed that, without cooling, heat increased sufficiently in both the subcutaneous layer and the skin to induce inflammatory proteins linked to hyperpigmentation. Clinically, mean waist circumference declined significantly by week 12. Ultrasound demonstrated a parallel significant reduction in subcutaneous fat thickness, whereas WHR and caliper measurements showed no meaningful change. BMI remained stable, indicating localized rather than systemic fat loss. Both IGAIS and SGAIS scores improved significantly. No pigmentary changes or serious adverse events were reported.
Conclusions: A single session of reduced depth, contact cooled HIFU produced clinically meaningful reductions in abdominal girth and subcutaneous fat thickness without altering overall body weight or inducing pigmentary complications. This technology appears to be a safe, effective adjunct for non invasive management of central obesity and warrants further controlled trials.

P19. Distinct genomic profiling of folliculotropic MF compared to classic MF: Integrated data of whole genome sequencing and single-cell spatial transcriptomics

Myoung Eun Choi, Gyeonghoon Kim, Chong Hyun Won, Sung Eun Chang, Mi Woo Lee, Woo Jin Lee*
Department of Dermatology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea

Folliculotropic mycosis fungoides (FMF) is characterized by its characteristic follicular involvement, distinctive clinical presentation, and aggressive disease progression compared to classical MF. Molecular landscape and transcriptomic features of FMF remain under-recognized. This study aimed to investigate the genomic and transcriptomic landscape of FMF compared to classical MF. We conducted whole-exome sequencing using 9 FMF and 13 classical MF samples, in addition to CosMx Spatial Molecular Imaging using 8 FMF and 8 plaque-MF samples and GeoMx digital spatial profiler comparing 4 advanced FMF and 4 early FMF. We found that tumor mutation burden was higher in FMF, especially advanced FMF compared to classical MF. Mutational signature analysis revealed that advanced FMF presented frequent C>T/G>A transition, while early FMF showed similar signature compared to classical MF. We found 29 upregulated differentially expressed genes (DEGs) and 301 downregulated DEGs in epidermotropic CD4+ T cells from FMF compared to those from classical MF in CosMx analysis. Moreover, advanced FMF showed higher neutrophil degranulation, interleukin-4 and -13 signaling pathway compared to early FMF in GeoMx. Both CCL17 and GNLY were common DEGs upregulated in FMF compared to classical MF and in advanced FMF in comparison to early FMF. In this study, we found that FMF showed distinct genomic and transcriptomic profiles especially in advanced FMF.

대한피부연구학회 발전기금 모금 안내

1991년 KSID가 설립된 후 회원님들의 지속적인 관심과 성원 덕분에 KSID가 이제는 국내외적으로 명실상부한 피부과 연구학회로서 그 위상을 확고히 다지게 되었습니다. 2018년부터 미국, 유럽, 일본 피부연구학회와 함께 세계피부연구학회 (International Societies for Investigative Dermatology)의 정식 회원이 되면서, 명실공히 국제적인 피부연구를 선도하는 학회로 거듭나게 되었습니다. 대한피부연구학회 기금위원회는 작고하신 전남의대 고 김영표 교수님께서 희사하신 발전기금을 시작으로 이사회에서 그 뜻을 받들어 전 회원이 동참하는 발전기금을 모으기로 결의한 후 현재까지 여러 대학교수들과 뜻있는 개원가 회원분들을 중심으로 모금이 진행되었습니다. KSID 발전기금은 KSID 공모 연구비 및 young investigator 연구비, SID 참가비 지원 등에 귀하게 쓰일 계획입니다. 또한 2019년부터 '젊은 연구자들을 양성하기 위한 기금'의 모금을 시작하여 우리나라 젊은 연구자들이 미국, 유럽, 일본 피부연구학회에 참가하는 것을 지원하고, 아시아의 피부과 의사들이 우리나라 피부과 학회에 참가를 지원하여, 우리나라와 아시아의 피부과 발전에 조금이나마 기여하는 기금으로 활용하고자 합니다. KSID가 세계적인 학회로 도약하기 위한 비전에 모두가 관심을 가지고 적극적으로 참여해주시길 부탁드립니다.

■ **납부방식**

1. 분납방식 : 매월 _____원 (기간 __년 __월부터 __년 __월까지)
2. 일시금방식 : _____원

■ **납부계좌**

1. KSID 후원계좌를 통한 납부 (_____)
   우리은행 1005-803-791653 계좌명: 대한피부연구학회
2. 피부과학연구재단 계좌를 통한 납부 (영수증 발급 가능 계좌): 개별 문의 부탁드립니다.

■ **문의**

대한피부연구학회 재무이사 **장용현**, 재무간사 **김지희**
이메일: yhjang@knu.ac.kr, mygirljihee@yuhs.ac

KSID 평생회원

회원번호	성명	재직기관	회원번호	성명	재직기관
LM00001	이 증 훈	충남의대	LM00034	양 준 모	성균관의대
LM00002	윤 태 진	경상의대	LM00035	문 기 찬	울산의대
LM00003	정 진 호	서울의대	LM00036	최 용 범	건국의대
LM00004	은 희 철	서울의대	LM00037	안 규 중	건국의대
LM00005	정 기 양	연세의대	LM00038	신 정 현	인하의대
LM00006	유 희 준	한양의대	LM00039	최 광 성	인하의대
LM00007	김 도 원	경북의대	LM00040	권 오 상	서울의대
LM00008	이 석 종	경북의대	LM00041	노 주 영	이화의대
LM00009	김 규 한	서울의대	LM00042	김 명 화	단국의대
LM00010	이 양 원	건국의대	LM00043	Takashi Takahashi	JAPAN
LM00011	최 혜 영	이화의대	LM00044	박 현 제	부산의대
LM00012	조 상 현	가톨릭의대	LM00045	이 민 걸	연세의대
LM00013	이 은 소	아주의대	LM00046	김 기 호	동아의대
LM00014	강 희 영	아주의대	LM00047	황 규 왕	순천향의대
LM00015	손 성 향	아주의대	LM00048	조 문 균	순천향의대
LM00016	이 동 윤	성균관의대	LM00049	최 응 호	연세원주의대
LM00017	이 경 호	가톨릭의대	LM00050	김 유 찬	아주의대
LM00018	조 광 현	서울의대	LM00051	이 광 훈	연세의대
LM00019	원 종 현	울산의대	LM00052	최 혜 령	서울의대
LM00020	조 소 연	서울의대	LM00053	방 동 식	연세의대
LM00021	이 원 주	경북의대	LM00054	김 성 진	전남의대
LM00022	김 병 수	부산의대	LM00055	장 민 수	고신의대
LM00023	김 태 흥	화이트라인하얀피부과	LM00056	오 상 호	연세의대
LM00024	서 성 준	중앙의대	LM00057	송 해 준	고려의대
LM00025	김 일 환	고려의대	LM00058	김 대 석	연세의대
LM00026	김 수 찬	연세의대	LM00059	이 영	충남의대
LM00027	김 창 덕	충남의대	LM00060	임 명	아이엠피부과
LM00028	김 태 윤	가톨릭의대	LM00061	윤 현 선	서울의대
LM00029	박 영 립	순천향의대	LM00062	황 준 성	한국생명공학연구원
LM00030	이 애 영	동국의대	LM00063	고 주 연	한양의대
LM00031	이 승 호	동국의대	LM00064	유 박 린	경희의대
LM00032	허 창 훈	서울의대	LM00065	나 정 임	서울의대
LM00033	박 경 찬	서울의대	LM00066	최 지 호	울산의대

회원번호	성명	재직기관	회원번호	성명	재직기관
LM00067	이 해 진	연세A&B피부과	LM00094	백 유 상	고려의대
LM00068	김 도 영	연세의대	LM00095	윤 숙 정	전남의대
LM00069	이 지 범	전남의대	LM00096	문 제 호	서울의대
LM00070	노 미 령	연세의대	LM00097	김 희 주	가천의대
LM00071	김 수 연	킴벨피부과	LM00098	김 종 훈	연세의대
LM00072	권 인 호	서울365mc병원	LM00099	김 혜 원	한림의대
LM00073	정 소 영	인제의대	LM00100	배 정 민	힐하우스피부과
LM00074	이 상 은	연세의대	LM00101	김 태 균	연세의대
LM00075	이 승 철	전남의대	LM00102	신 기 혁	부산의대
LM00076	안 효 현	고려의대	LM00103	김 동 현	차의대
LM00077	김 정 수	한양의대	LM00104	신 정 우	차의대
LM00078	이 주 희	연세의대	LM00105	김 정 은	순천향의대
LM00079	손 상 욱	고려의대	LM00106	정 보 영	한림의대
LM00080	장 용 현	경북의대	LM00107	김 대 현	고려의대
LM00081	박 현 선	서울의대	LM00108	심 준 호	성균관의대
LM00082	변 지 원	인하의대	LM00109	박 영 준	아주의대
LM00083	김 정 은	한양의대	LM00110	장 성 은	울산의대
LM00084	고 현 창	부산의대	LM00111	김 경 문	가톨릭의대
LM00085	이 종 희	성균관의대	LM00112	최 수 영	벧엘피부과
LM00086	박 창 욱	연세의대	LM00113	조 수 익	오킴스피부과
LM00087	홍 승 필	연세원주의대	LM00114	김 현 제	서울의대
LM00088	정 기 헌	경희의대	LM00115	홍 종 수	동국의대
LM00089	신 민 경	경희의대	LM00116	신 현 태	인하의대
LM00090	류 영 욱	계명의대	LM00117	진 선 필	서울의대
LM00091	이 우 진	울산의대	LM00118	석 준	중앙의대
LM00092	이 정 수	연세의대	LM00119	황 신 원	연세의대
LM00093	이 시 형	서울의대			

평생회원 및 회비납부 안내

대한피부연구학회에서는 학회의 안정적 재정운영 및 회원관리를 목적으로 평생회원가입 및 평생회비를 독려하고 있습니다.
회비는 30만원으로 평생회원이 되면 매년 내는 연회비(연 2만원)가 무료이며, 학회참가비(등록비)만 내시면 됩니다.
회원 여러분의 적극 동참을 부탁드립니다.

- 문의 및 접수처: 대한피부연구학회 재무이사 장용현(yhjang@knu.ac.kr), 재무간사 김지희(mygirljihee@yuhs.ac)
- 입금계좌: 우리은행 1005-602-939595 대한피부연구학회

Sponsor


ucb
Inspired by patients.
Driven by science.


abbvie


sanofi


NOVARTIS


Lilly


유한양행


종 근 당


L E O


코오롱제약


SINCE 1897


Johnson&Johnson


Pfizer


janssen


동구바이오제약


NEOPHARM
good skin, good life


GALDERMA
EST. 1981


JEIL
제일약품


한국약품

2025 16th
KSID Research Camp

Program and Abstracts

인쇄: 2025년 9월 19일
발행: 2025년 9월 26일

발행인 : 강희영
편집인 : 오상호

발 행: 대한피부연구학회

인 쇄: 나림컨벤스
T. 02-6671-1373, F. 02-6671-1374
E-mail: narim2007@narim.co.kr
Homepage: narimconvens.com