2017
The 27th Annual Meeting of the Korean Society for Investigative Dermatology

Program and Abstracts

2017. 3. 24(Fri)~25(Sat)

NEWILHAN Memorial Hall,
Avison BioMedical Research Center (ABMRC),
Yonsei University College of Medicine

[Hosted by] the Korean Society for Investigative Dermatology (KSID)
[Sponsored by] the Korean Dermatological Association (KDA)
Dear colleagues and friends,

On behalf of the organizing committee of the annual meeting of Korean Society for Investigative Dermatology (KSID), it is our great pleasure to invite you to the 27th KSID Annual Meeting on March 24 (Fri)-25 (Sat), 2017.

This meeting has been organized to provide the participants with an excellent scientific program, aiming at communicating leading-edge researches on Antimicrobial peptide, Inflammatory skin diseases & Innate immunity, Rare diseases/genetic regulation, Pigmentation, Appendages & Wound healing, and Epidermal barrier & keratinocyte biology. Our honored guests for this year's session will be Prof. Richard L. Gallo (USA), Prof. Shinichi Sato (Japan), Prof. Akimichi Morita (Japan), Prof. Wen-Hung Chung (Taiwan), Prof. Johann W. Bauer (Austria), and eminent Korean scientists. They will offer new, professional knowledge in their specialized fields.

I would like to appreciate all the members of KSID organizing committee for their great effort in setting up this wonderful meeting. We would like to appreciate all the participants who will graciously share their invaluable knowledge and have an animated discussion. We also hope that all of you will have the chance to enjoy the meeting, experience intellectual interchange. And it will be a great opportunity to foster your friendship between participants.

We anticipate seeing many of you at the 27th KSID meeting in March 2017.

Thank you.

March 2017

Jin Ho Chung
President
Korean Society for Investigative Dermatology
### Time Table

**1st Day, March 24(Fri), 2017**

<table>
<thead>
<tr>
<th>Time</th>
<th><strong>NEWILHAN Memorial Hall</strong> (1st floor, ABMRC)</th>
<th><strong>Pine Room</strong> (Sangnam Institute of Management)</th>
<th><strong>Seminar Room</strong> (1st floor, ABMRC)</th>
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<tbody>
<tr>
<td>07:40~</td>
<td><strong>Registration</strong></td>
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<tr>
<td>08:30~10:00</td>
<td>Free Communications (I) (FC-1 ~ FC-9)</td>
<td>Concurrent Session (I) (HP-1 ~ HP-10)</td>
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<tr>
<td>10:00~10:20</td>
<td><strong>Coffee break</strong></td>
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<tr>
<td>10:20~12:15</td>
<td>Session I. Antimicrobial Peptides</td>
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<td>(Sponsored by AmorePacific Co.)</td>
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<td>12:15~12:20</td>
<td><strong>Group Photo</strong></td>
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<tr>
<td>12:20~13:40</td>
<td>Lunch &amp; Poster viewing</td>
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<td>Poster (68)</td>
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<tr>
<td>13:40~13:50</td>
<td><strong>Opening Ceremony</strong></td>
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<td>(FC-1 ~ FC-18)</td>
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<td>13:50~14:45</td>
<td>KSID Award Lecture</td>
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<td>(HP-1 ~ HP-20)</td>
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<td>14:45~16:45</td>
<td>Session II. Inflammation, Inflammatory Skin</td>
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<td>(PO-1 ~ PO-30)</td>
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<td></td>
<td>Disorders</td>
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<td>16:45~17:05</td>
<td><strong>Coffee break</strong></td>
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<td>17:05~18:50</td>
<td>Session III. Innate &amp; Adaptive Immunity</td>
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<td>19:00~</td>
<td><strong>Welcome Reception</strong> - LOTUS hall, Sangnam</td>
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<td>Institute of Management 1st floor</td>
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<tr>
<td>Time</td>
<td>NEWILHAN Memorial Hall (1st floor, ABMRC)</td>
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<tr>
<td>07:40~</td>
<td>Registration</td>
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<tr>
<td>08:30~10:00</td>
<td>Free Communications (II) (FC-10 ~ FC-18)</td>
<td>Concurrent Session (II) (HP-11 ~ HP-20)</td>
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<tr>
<td>10:00~10:25</td>
<td>Coffee break</td>
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<td>10:25~10:45</td>
<td>UAM Award Lecture</td>
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<td>10:45~12:05</td>
<td>Session IV. Hereditary Disease, Genetic Regulation</td>
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<td>12:05~13:15</td>
<td>Lunch</td>
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<td>13:15~14:55</td>
<td>Session V. Pigment and Pigmentary Disorders</td>
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<td>14:55~15:55</td>
<td>KSID General Assembly</td>
<td>Coffee break &amp; Poster viewing</td>
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<td>15:55~17:10</td>
<td>Session VI. Appendages &amp; Wound healing</td>
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<td>17:10~18:25</td>
<td>Session VII. Keratinocyte Biology &amp; Epidermal Barrier</td>
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<td>18:25~</td>
<td>Closing</td>
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The 27th Annual Meeting of the Korean Society for Investigative Dermatology

Program

March 24(Fri), 2017

07:40- Registration

08:30-10:00 Free Communications (I) (1st floor, ABMRC)
Chair: Il-Hwan Kim (Korea Univ.), Tae Jin Yoon (Gyeongsang National Univ.)

FC-1 Effects of Citron Essential Oils on mediators associated with rosacea in epidermal keratinocytes activated with VD3, TNF α
Hyeon Woo Jeon, Soo Hyeon Bae, Sook Jung Yun, Seung-Chul Lee, Jee-Bum Lee
Department of Dermatology, Chonnam National University Medical School, Gwangju, Korea

FC-2 Psychological stress deteriorates skin barrier function by activation of 11β-hydroxysteroid dehydrogenase 1 as well as endogenous glucocorticoids
Sung Jay Choe¹, Donghye Kim¹, Eun Jung Kim¹, Eung Ho Choi¹
¹Department of Dermatology, Yonsei University Wonju College of Medicine, Wonju, Korea

FC-3 Fibroblast-derived clusterin inhibits melanogenesis
Jiun Lee¹, Misun Kim¹, Tae Jun Park¹, Hee Young Kang¹
¹Department of Dermatology, Ajou University School of Medicine, Suwon, Korea

FC-4 Hippocampal abnormalities and HPA axis alteration in UV irradiated mice
Mira Han¹,²,³, Jung-Soo Bae¹,²,³, Jae-Jun Ban¹,²,³, Chang Yup Shin¹,²,³, Jin Ho Chung¹,²,³
¹Department of Dermatology, Seoul National University College of Medicine, ²Laboratory of Cutaneous Aging and Hair Research, Biomedical Research Institute, Seoul National University Hospital, ³Institute of Human-Environment Interface Biology, Medical Research Center, Seoul National University, Seoul, Korea
FC-5
Pre-elafin is involved in ultraviolet-induced keratinocyte apoptosis
Gwanghoon Kim, Hyangmi Kim\(^1\), Minsoo Noh\(^2\), Ok-Nam Bae\(^3\), Chang-Hoon Lee\(^4\), Ai-Young Lee\(^5\)
\(^1\)Department of Dermatology, Dongguk University Ilsan Hospital, 814 Siksa-dong, Ilsandong-gu, Goyang-si, Gyeonggi-do 410-773, Republic of Korea, \(^2\)College of Pharmacy, Natural products Research Institute, Seoul National University, Seoul 151-742, Republic of Korea, \(^3\)College of Pharmacy, Institute of Pharmaceutical Science and Technology, Hanyang University, Ansan 426-791, Republic of Korea, \(^4\)College of Pharmacy, Dongguk University, Seoul 100-715, Korea

FC-6
SAA1 is induced by UV irradiation and detected by TLR4 to cause skin inflammation
Sangbum Han\(^1,2,3\), Jang-Hee Oh\(^2,3\), Chang-Yup Shin\(^2,3\), Hyun-Sun Yoon\(^2,3,4\), Dong Hun Lee\(^5,6\), Jin Ho Chung\(^1,2,3,5\)
\(^1\)Department of Biomedical Sciences, Seoul National University Graduate School, \(^2\)Department of Dermatology, Seoul National University College of Medicine, \(^3\)Institute of Human-Environment Interface Biology, Medical Research Center, Seoul National University, \(^4\)Department of Dermatology, Seoul National University Boramae Hospital, \(^5\)Institute on Aging, Seoul National University, Seoul, Republic of Korea

FC-7
5-ALA-mediated sonodynamic therapy induced antitumor effect for angiosarcoma
Hiroki Furukawa, Toshiyuki Ozawa, Daisuke Tsuruta
Department of Dermatology, Osaka City University Graduate School of Medicine, Osaka, Japan

FC-8
The mutational origins of sebaceous carcinoma
Jeffrey P. North\(^1\), Kevin McMullen\(^2\), Steve Benz\(^2\), David Solomon\(^1\), Raymond J. Cho\(^2\)
\(^1\)Department of Pathology, University of San Francisco, California, \(^2\)Department of Dermatology, University of San Francisco, California, \(^3\)NantOmics, LLC

FC-9
MITF regulates dynamic melanoma heterogeneity
Loredana Spoerri\(^1\), Crystal A. Tonnessen\(^1\), Kimberley A. Beaumont\(^2\), David S. Hill\(^3\), Russell J. Jurek\(^1\), Sheena M. Daignault\(^1\), Farzana Ahmed\(^1\), Aaron G. Smith\(^4\), Wolfgang Weninger\(^5,6\), Nikola K. Haas\(^7\)
\(^1\)The University of Queensland, The University of Queensland Diamantina Institute, Translational Research Institute, Brisbane, Qld, Australia, \(^2\)The Centenary Institute, Newtown, NSW, Australia, \(^3\)CSIRO Astronomy & Space Sciences, Australia Telescope National Facility, Epping, NSW, Australia, \(^4\)Dermatology Research Centre, Translational Research Institute, School of Medicine, The University of Queensland, Brisbane, Qld, Australia, \(^5\)Discipline of Dermatology, University of Sydney, NSW, Australia, \(^6\)Department of Dermatology, Royal Prince Alfred Hospital, Camperdown, NSW, Australia
08:30-10:00 Concurrent Session (I) (Sangnam Institute of Management, Pine Room)
Chair: Dong-Youn Lee (Sungkyunkwan Univ.), Chang Deok Kim (Chungnam National Univ.)

08:30-10:00  Hot Posters (I)

HP-1  Adiponectin recovers abnormalities of keratinocyte induced by intrinsic and extrinsic stress damage
08:30-08:39
Taewon Jin¹, Min Jeong Kim¹, Won Il Heo¹, Kui Young Park¹, Mi-Kyung Lee², Seung-Phil Hong³, Seong-Jin Kim⁴, Myung Im⁵, Seong Jun Seo¹
¹Department of Dermatology, Chung-Ang University Hospital, Seoul, South Korea,
²Department of Laboratory Medicine, Chung-Ang University Hospital, Seoul, South Korea,
³Department of Dermatology, College of Medicine, Dankook University, Cheonan, South Korea,
⁴Department of Dermatology, Chonnam National University Medical School, Gwangju, South Korea,
⁵Department of Dermatology, Chungnam National University School of Medicine, Daejeon, South Korea

HP-2  Effects of human mesenchymal stem cells co-culture on calcium-induced differentiation of normal human keratinocytes
08:39-08:48
Shyam Kishor Sah¹ and Tae-Yoon Kim¹
¹Laboratory of Dermatology-Immunology, College of Medicine, The Catholic University of Korea, Seoul, Republic of Korea

HP-3  Adiponectin upregulates filaggrin expression via SIRT1-mediated signaling and increase of calcium influx in human normal keratinocytes
08:48-08:57
Taewon Jin¹, Min Jeong Kim¹, Won Il Heo¹, Ju Hee Kim¹, Kui Young Park¹, Seung-Phil Hong³, Seong-Jin Kim⁴, Myung Im⁵, Seong Jun Seo¹
¹Department of Dermatology, Chung-Ang University Hospital, Seoul, South Korea,
³Department of Dermatology, College of Medicine, Dankook University, Cheonan, South Korea,
⁴Department of Dermatology, Chonnam National University Medical School, Gwangju, South Korea,
⁵Department of Dermatology, Chungnam National University School of Medicine, Daejeon, South Korea

HP-4  UV-induced DNA methyltransferase I epigenetically silences tissue inhibitor of metalloproteinase I in the human skin
08:57-09:06
Ha-Young Kim¹,²,³, Min-Kyoung Kim¹,²,³, Mi Hee Shin¹,²,³, Dong Hun Lee¹,²,³, Jin Ho Chung¹,²,³,⁴
¹Department of Biomedical Sciences, Seoul National University Graduate School
²Department of Dermatology, Seoul National University College of Medicine
³Institute of Human-Environment Interface Biology, Medical Research Center, Seoul National University, ⁴Institute on Aging, Seoul National University, Seoul, Republic of Korea
HP-5  Anacardic acid reduces lipogenesis in human differentiated adipocytes via inhibition of histone acetylation
09:06-09:15
Min-Kyoung Kim\textsuperscript{1,2,3}, Eun Ju Kim\textsuperscript{1,2,3}, Yeon Kyung Kim\textsuperscript{1,2,3}, Yu Ri Lee\textsuperscript{1,2,3}, Dong Hun Lee\textsuperscript{1,2,3}, and Jin Ho Chung\textsuperscript{1,2,3,4}
\textsuperscript{1}Department of Dermatology, Seoul National University College of Medicine, \textsuperscript{2}Laboratory of Cutaneous Aging Research, Biomedical Research Institute, Seoul National University Hospital, \textsuperscript{3}Institute of Human-Environment Interface Biology, Seoul National University, \textsuperscript{4}Institute on Aging, Seoul National University, Seoul, Republic of Korea

HP-6  Serum CALML5 as a potential SJS/TEN biomarker
09:15-09:24
Natsumi Hama\textsuperscript{1}, Keiko Nishimura\textsuperscript{2}, Hideaki Kume\textsuperscript{3}, Takeshi Tomonaga\textsuperscript{3}, Hiroshi Shimizu\textsuperscript{2}, Riichiro Abe\textsuperscript{1}
\textsuperscript{1}Division of Dermatology, Niigata University Graduate School of Medical and Dental Sciences, Niigata, Japan
\textsuperscript{2}Department of Dermatology, Hokkaido University Graduate School of Medicine, Hokkaido, Japan
\textsuperscript{3}Laboratory of Proteome Research, National Institute of Biomedical Innovation, Osaka, Japan

HP-7  NK help during protective immunization with protein-vaccine is critical to generate adaptive T effector immunity
09:24-09:33
Taegyun Kim\textsuperscript{1,2,3}, Yong Liu\textsuperscript{1,2}, Christopher J Nirschl\textsuperscript{1,2}, Min-Geol Lee\textsuperscript{3}, Nirosnaha Anandasabapathy\textsuperscript{1,2}
\textsuperscript{1}Brigham and Women’s Hospital, Department of Dermatology, \textsuperscript{2}Harvard Medical School, Boston, MA, \textsuperscript{3}Department of Dermatology, Cutaneous Biology Research Institute, Yonsei University College of Medicine, Seoul, Korea

HP-8  Wound healing promotion using Ar:Air plasma device
09:33-09:42
Ik Jun Moon\textsuperscript{1}, Sang Gyu Hwang\textsuperscript{2}, Hyun Woo Hwang\textsuperscript{2}, Hae Kyeong Yoon\textsuperscript{2}, Hyun Joo Lee\textsuperscript{1}, Woo Jin Lee\textsuperscript{1}, Sung Eun Chang\textsuperscript{1}, Mi Woo Lee\textsuperscript{1}, Ji Ho Choi\textsuperscript{1}, Chong Hyun Won\textsuperscript{1,2}
\textsuperscript{1}Department of Dermatology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea
\textsuperscript{2}Asan Institute for Life Sciences, University of Ulsan College of Medicine, Seoul, Korea

HP-9  \(\beta\)-catenin regulates the expression of cAMP response element-binding protein 1 (CREB) in SCC cells
09:42-09:51
Soo-Yeon Kim, Cho-Ah Lim, Kyung-Cheol Sohn, Myung Im, Young Lee, Young-Joon Seo, Jeung-Hoon Lee, Chang-Deok Kim
Department of Dermatology, School of Medicine, Chungnam National University, Daejeon, Korea

HP-10  Autophagy and aging
09:51-10:00
Hei Sung Kim, Hyeree Kim, Mi Young Youm, Eun Sun Kang, Jeong Deuk Lee
Department of Dermatology, Incheon St. Mary’s Hospital, The Catholic University of Korea, Incheon, Korea
### The 27th Annual Meeting of The Korean Society for Investigative Dermatology

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<tr>
<th>Time</th>
<th>Session / Event</th>
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<tbody>
<tr>
<td>10:00-10:20</td>
<td>Coffee break</td>
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<tr>
<td>10:20-12:15</td>
<td><strong>Session I. Antimicrobial Peptides</strong> <em>(Sponsored by AmorePacific Co.)</em></td>
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<tr>
<td><strong>Chair:</strong></td>
<td>Soo-Chan Kim <em>(Yonsei Univ.)</em>, Kyu Han Kim <em>(Seoul National Univ.)</em></td>
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</tbody>
</table>
| 10:20-10:40 | Correlation between skin barrier function and epidermal antimicrobial peptide expression  
Eung Ho Choi *(Yonsei Univ. Wonju)*                                                                                   50  |
| 10:40-10:45 | Discussion                                                                       |
| 10:45-11:05 | Psoriasin (S100A7) induces abnormal differentiation and regulates nuclear translocation of Jun activator binding (Jab-1) protein  
Eui Dong Son *(AmorePacific Corporation)*                                                                              52  |
| 11:05-11:10 | Discussion                                                                       |
| 11:10-11:30 | TSLP downregulates S100A7 and β-defensin 2 via the JAK2/STAT3/Sin3a signaling pathway  
Sang Wook Son *(Korea Univ.)*                                                                                           54  |
| 11:30-11:35 | Discussion                                                                       |
| 11:35-12:05 | The anti-microbial and pro-microbial parts of skin innate immunity  
Richard L. Gallo *(University of California San Diego, USA)*                                                            56  |
| 12:05-12:15 | Discussion                                                                       |
| 12:15-12:20 | **Group Photo**                                                                  |
| 12:20-13:40 | **Lunch & Poster viewing**                                                       |
| 13:40-13:50 | **Opening Ceremony**                                                            |
| 13:50-14:45 | **KSID Award Lecture**                                                          |
| **Chair:** | Jee Ho Choi *(Univ. of Ulsan)*, Jin Ho Chung *(Seoul National Univ.)*             |
| 13:50-14:15 | **KSID Award Lecture**                                                           |
| Specific immunotherapy in atopic dermatitis  
Kwang Hoon Lee *(Yonsei Univ.)*                                                                                           60  |
| 14:15-14:30 | **KSID Young Investigator Award (I)**                                            |
| Implication of genetic mutations in Korean melanoma patients  
Mi Ryung Roh *(Yonsei Univ.)*                                                                                            64  |
| 14:30-14:45 | **KSID Young Investigator Award (II)**                                           |
| TRPV channels and postburn pruritus  
Hye One Kim *(Hallym Univ.)*                                                                                             66  |
### Session II. Inflammation, Inflammatory Skin Disorders
**Chair:** Tae Yoon Kim (*Catholic Univ.*), Min-Geol Lee (*Yonsei Univ.*)

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<th>Time</th>
<th>Session</th>
<th>Speaker(s)</th>
<th>Location</th>
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<tbody>
<tr>
<td>14:45-15:10</td>
<td><strong>Induction of regulatory T cells is a principal mechanism for phototherapy</strong></td>
<td>Akimichi Morita (<em>Nagoya City University, Japan</em>)</td>
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<td>15:10-15:20</td>
<td><strong>Discussion</strong></td>
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<td>15:20-15:40</td>
<td><strong>PD-1 and PD-L1 in psoriatic inflammation</strong></td>
<td>Eui-Cheol Shin (<em>KAIST</em>)</td>
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<td>15:40-15:45</td>
<td><strong>Discussion</strong></td>
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<td>15:45-16:10</td>
<td><strong>Molecular insights into severe cutaneous adverse reactions</strong></td>
<td>Wen-Hung Chung (<em>Chang Gung Memorial Hospital, Taiwan</em>)</td>
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<td>16:10-16:20</td>
<td><strong>Discussion</strong></td>
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<td>16:20-16:40</td>
<td><strong>The role of air pollution on atopic dermatitis in children</strong></td>
<td>Kangmo Ahn (<em>Sungkyunkwan Univ.</em>)</td>
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<td>16:45-16:50</td>
<td><strong>Discussion</strong></td>
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<td>16:45-17:05</td>
<td><strong>Coffee break</strong></td>
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### Session III. Innate & Adaptive Immunity
**Chair:** Jeung-Hoon Lee (*Chungnam National Univ.*), Akimichi Morita (*Nagoya City Univ., Japan*)

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<tr>
<td>17:05-17:30</td>
<td><strong>Orphan nuclear receptors and regulation of innate immunity</strong></td>
<td>Eun-Kyeong Jo (<em>Chungnam National Univ.</em>)</td>
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<td>17:30-17:35</td>
<td><strong>Discussion</strong></td>
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<td>17:35-17:55</td>
<td><strong>IL-17A producing ILC3s as a new player of atopic dermatitis</strong></td>
<td>Hye Young Kim (<em>Seoul National Univ.</em>)</td>
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<tr>
<td>17:55-18:00</td>
<td><strong>Discussion</strong></td>
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<td>18:00-18:20</td>
<td><strong>Skin resident T cells in mouse and human</strong></td>
<td>Chang Ook Park (<em>Yonsei Univ.</em>)</td>
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<td>18:20-18:25</td>
<td><strong>Discussion</strong></td>
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<tr>
<td>18:25-18:45</td>
<td><strong>CD1 signaling supports the survival and proliferation of epidermal and dermal T cells from human skin</strong></td>
<td>Jung-Im Na (<em>Seoul National Univ.</em>)</td>
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<td>18:45-18:50</td>
<td><strong>Discussion</strong></td>
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The 27th Annual Meeting of The Korean Society for Investigative Dermatology

March 25(Sat), 2017

07:40- Registration

08:30-10:00 Free communications (II)  
Chair: Joo Young Roh (Gachon Univ.), Ki-Ho Kim (Dong-A Univ.)

FC-10  
08:30-08:40  
CCCTC-binding factor controls the homeostatic maintenance and migration of langerhans cells in the skin  
Taegyun Kim\textsuperscript{1,2,3}, Mikyoung Kim\textsuperscript{1}, Sung Hee Kim\textsuperscript{3}, Chae Gyu Park\textsuperscript{2,4}, Yeon-Su Lee\textsuperscript{5}, Min-Geol Lee\textsuperscript{2,3}, Hyoung-Pyo Kim\textsuperscript{1,2}  
\textsuperscript{1}Department of Environmental Medical Biology, Institute of Tropical Medicine, Yonsei University College of Medicine, Seoul, Korea, \textsuperscript{2}Brain Korea21 PLUS Project for Medical Science, Yonsei University College of Medicine, Seoul, Korea, \textsuperscript{3}Department of Dermatology, Yonsei University College of Medicine, Seoul, Korea, \textsuperscript{4}Severance Biomedical Science Institute, Yonsei University College of Medicine, Seoul, Korea, \textsuperscript{5}Cancer Genomics Branch, National Cancer Center, Gyeonggi-do, Korea  

FC-11  
08:40-08:50  
The response to cyclophosphamide therapy is regulated by effector B cells in systemic sclerosis-associated interstitial lung disease  
Satoshi Ebata\textsuperscript{1}, Ayumi Yoshizaki\textsuperscript{1}, Takemichi Fukasawa\textsuperscript{1}, Kouki Nakamura\textsuperscript{1}, Ryosuke Saigusa\textsuperscript{1}, Takashi Taniguchi\textsuperscript{1}, Yoshihide Asano\textsuperscript{1}, Yutaka Kazoe\textsuperscript{2}, Kazuma Mawatari\textsuperscript{1}, Takehiko Kitamori\textsuperscript{2}, Shinichi Sato\textsuperscript{1}  
\textsuperscript{1}Department of Dermatology, The University of Tokyo Graduate School of Medicine, Tokyo, Japan, \textsuperscript{2}Department of Applied Chemistry, The University of Tokyo Graduate School of Engineering, Tokyo, Japan  

FC-12  
08:50-09:00  
The role of innate immunity in scrub typhus  
Chung-Hsing Chang\textsuperscript{1}, Ming-Hsien Tsai\textsuperscript{2}, Rong-Kung Tsai\textsuperscript{1}  
\textsuperscript{1}Skin Institute, China Medical University Hospital, Taichung, Taiwan, \textsuperscript{2}Graduate Institute of Medicine, Kaohsiung Medical University, Kaohsiung, Taiwan  

FC-13  
09:00-09:10  
Effects of AhR Ligands on PBMCs and CD4+T cells from patients with atopic dermatitis and psoriasis  
Jee Hee Son, Min Je Jung, Yong Won Choi, Yong Se Cho, Bo Young Chung, Chon Wook Park, Hye One Kim  
Department of Dermatology, Hallym University, Kangnam Sacred Heart Hospital  

FC-14  
09:10-09:20  
11β-Hydroxysteroid dehydrogenase 1 in the skin has a role in the occurrence of atopic dermatitis  
Noo Ri Lee, Solam Lee, Sung Jay Choe, Donghye Kim, Eunjung Kim, Eung Ho Choi  
Department of Dermatology, Yonsei University Wonju College of Medicine, Wonju, Korea
FC-15  Attempt to establish a hair follicle co-culture model using feeder-free human iPS cells
Manabu Ohyama¹, Aki Tsukashima¹, Momoko Kimishima¹, Yoshihito Yamazaki², Hideyuki Okano²
¹Department of Dermatology, Kyorin University School of Medicine, Tokyo, Japan
²Department of Physiology, Keio University School of Medicine, Tokyo, Japan

FC-16  HMGB1 stimulate hair shaft elongation via prostaglandin E₂
Ji-Hye Hwang¹, Yuri Ahn¹, Zhenlong Zheng¹,² and Do Young Kim¹
¹Department of Dermatology and Cutaneous Biology Research Institute, Yonsei University College of Medicine, Seoul, Korea
²Department of Dermatology, Yanbian University Hospital, Yanji, China

FC-17  CD13, marker for onychofibroblasts within nail matrix onychodermis: Comparison of the nail unit with hair follicle
Ji-Hye Park¹, Dong-Youn Lee¹, Jun-Mo Yang, Kee-Taek Jang², Kyung-Hoon Lee³, Jong Sup Shim⁴
¹Departments of Dermatology, ²Pathology, ³Anatomy and ⁴Orthopedic Surgery, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, South Korea

FC-18  3D in vivo pH imaging analysis of stratum corneum
Masayuki Amagai¹,², Yuki Furuichi¹,², Takeshi Matsui²
¹Department of Dermatology, Keio University School of Medicine, ²Laboratory for Skin Homeostasis, RIKEN Center for Integrative Medical Sciences

08:30-10:00  Concurrent Session (II) (Sangnam Institute of Management, Pine Room)
Chair: Weon Ju Lee (Kyungpook National Univ.), Jee Bum Lee (Chonnam National Univ.)

08:30-10:00  Hot Posters (II)

HP-11  The effect of cilostazol on hair growth: A type of drug repositioning for the treatment of alopecia with the mechanism of vasodilatation
Dong Young Kim¹,², Hye-In Choi¹, Chang-Yup Shin¹, Kyu Han Kim¹,², Ohsang Kwon¹,²
¹Institute of Human-Environment Interface Biology, Medical Research Center, Seoul National University College of Medicine, Seoul, Korea, ²Department of Dermatology, Seoul National University College of Medicine, Seoul, Korea

HP-12  Development of hair loss treatment using deoxycholic acid
Hyyoung-Seok Jang¹, Dong-Youn Lee¹, Hyun-Chul Kwon²
¹Department of Dermatology and ²Cardiology, Samsung Medical Center, Sungkyunkwan University, Seoul, Korea
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<td>HP-13</td>
<td>UV Irradiated human dermal endothelial cells induce pigmentation: The role of vasculature in the development of UV-induced hyperpigmentary disorders</td>
<td>Misun Kim, Tae Jun Park, Hee Young Kang. 1Department of Dermatology, Ajou University School of Medicine, Suwon, Korea, 2Department of Biochemistry and Molecular Biology, Ajou University School of Medicine, Suwon, Korea.</td>
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<td>HP-14</td>
<td>Adiponectin inhibits melanogenesis through AMPK/CREB regulated transcriptional coactivator</td>
<td>Seunghyun Bang, Ik Jun Moon, Woo Jin Lee, Chong Hyun Won, Mi Woo Lee, Jee Ho Choi, Sung Eun Chang. 1Department of Dermatology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea, 2Asan institute for life sciences, University of Ulsan College of Medicine, Seoul, Korea.</td>
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<td>HP-15</td>
<td>HAS3 induces epidermal HA production by hapten stimulation and modulates CHS response</td>
<td>Hitoshi Terui, Kensi Yamasaki, and Setsuya Aiba. Department of Dermatology, Tohoku University Graduate School of Medicine, Sendai, Japan.</td>
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<td>HP-16</td>
<td>CCCTC-binding factor is essential to the maintenance and quiescence of hematopoietic stem cells in mice</td>
<td>Taegyun Kim, Sueun Kim, Mikyoung Kim, Bobae Yang, Min-Geol Lee, Hyoung-Pyo Kim. BK21 Project for Medical Science, Yonsei University College of Medicine, Seoul, Korea, 2Department of Environmental Medical Biology, Yonsei University College of Medicine, Seoul, Korea, 3Department of Dermatology, Cutaneous Biology Research Institute, Yonsei University College of Medicine, Seoul, Korea.</td>
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<td>HP-17</td>
<td>IWR-1, an inhibitor for Wnt/β-catenin signaling, reduces collagen synthesis in skin fibroblasts</td>
<td>So-Ra Choi, Ming-Wei Zhou, Jeong-Min Ha, Kyung-Cheol Sohn, Myung Im, Young Lee, Young-Joon Seo, Chang-Deok Kim, Jeung-Hoon Lee. 1Department of Dermatology, School of Medicine, Chungnam National University, Daejeon, Korea, 2Department of Medical Science, School of Medicine, Chungnam National University, Daejeon, Korea.</td>
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<td>HP-18</td>
<td>CHSY1 may be a major regulator of GAG chain length on decorin and biglycan in intrinsically aged and photoaged human skin</td>
<td>Hanon Lee, Jang-Hee Oh, Min Kyeong Shin, Jiyeong Lim, Yeon Kyung Kim, Soyun Cho, and Jin Ho Chung. 1Department of Dermatology, Seoul National University College of Medicine, Seoul, Korea, 2Institute of Human-Environment Interface Biology, Medical Research Center, Seoul National University, Seoul, Korea, 3Biomedical Research Institute, Seoul National University Hospital, Seoul, Korea, 4Department of Biomedical Sciences, Seoul National University Graduate School, Seoul, Korea, 5Department of Dermatology, Seoul National University Boramae Hospital, Seoul, Korea.</td>
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The 27th Annual Meeting of The Korean Society for Investigative Dermatology

HP-19
09:42-09:51
The mortality rate of Stevens-Johnson syndrome and toxic epidermal necrolysis increases in dialysis patients
Miyuki Kato¹, Yoko Kano¹, Tetsuo Shiohara¹ and Manabu Ohyama¹
¹Department of Dermatology, School of Medicine, Kyorin University, Tokyo Japan

HP-20
09:51-10:00
Palmitic acid and inflammation in SZ95 sebocyte
Chong Won Choi, Eun Ju Kim, Eun Young Seo, Jin Ho Chung
Department of Dermatology, Seoul National University College of Medicine, Institute of Human-Environment Interface Biology, Medical Research Center, Seoul National University, Seoul, Republic of Korea

10:00-10:25 Coffee break

10:25-10:45 UAM Award Lecture Chair: Seung Chul Lee (Chonnam National Univ.)

10:25-10:45 The role of S100A8 and S100A9 in human skin
Young Lee (Chungnam National Univ.)

10:45-12:05 Session IV. Hereditary Disease, Genetic Regulation Chair: Jun Mo Yang (Sungkyunkwan Univ.), Seong Jun Seo (Chung-Ang Univ.)

10:45-11:15 Stem cells in dermatology
Johann W. Bauer (Paracelsus Medical University, Salzburg, Austria)

11:15-11:25 Discussion

11:25-11:55 Epigenetic downregulation of transcription factors, Fli1 and KLF5, in scleroderma
Shinichi Sato (University of Tokyo, Japan)

11:55-12:05 Discussion

12:05-13:15 Lunch & Poster viewing

13:15-14:55 Session V. Pigment and Pigmentary Disorders Chair: Kyoung Chan Park (Seoul National Univ.), You Chan Kim (Ajou Univ.)

13:15-13:35 Role of autophagy in relation to melanogenesis and other skin disorders
Sung Eun Chang (Univ. of Ulsan)

13:35-13:40 Discussion
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<td>Eun-Gyung Cho (AmorePacific Corporation)</td>
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<td>The role of cutaneous vasculature in the skin pigmentation</td>
<td>Hee Young Kang (Ajou Univ.)</td>
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<td>The effects of hydroporation on melasma with anti-aging cocktail via enhancing microenvironment of the skin</td>
<td>Jung Won Shin (Seoul National Univ.)</td>
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<td>Study on the hair follicle-inducing genes identified from spheroid cultivation of human dermal papilla cells</td>
<td>Young Kwan Sung (Kyungpook National Univ.)</td>
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<td>Hair loss in hephaestin knockout mouse with iron deficiency</td>
<td>Sang Ho Oh (Yonsei Univ.)</td>
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<td>Development of cell-penetrating asymmetric interfering RNA targeting connective tissue growth factor (CTGF) for anti-scar therapeutics</td>
<td>Dong-Ki Lee (Sungkyunkwan Univ.)</td>
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<td><strong>Session VII. Keratinocyte Biology &amp; Epidermal Barrier</strong></td>
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<td>Chair: Kee-Yang Chung (Yonsei Univ.), Gwang Seong Choi (Inha Univ.)</td>
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<td>Se Kyoo Jeong (Seowon Univ.)</td>
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<td>Targeted deletion of Crif1 in mouse skin epidermis impairs skin homeostasis and hair morphogenesis</td>
<td>Chang Deok Kim (Chungnam National Univ.)</td>
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18:00-18:20  Ceramide with long-chain fatty acids and skin barrier  
Seung Phil Hong (Dankook Univ.) ................................................................. 148
18:20-18:25  Discussion

18:25-  Closing
All accepted abstracts including Free Communications (FC) and Hot Poster (HP) should exhibit Poster presentation.

PO-1 Serum HMGB1 induced TRIM21 expression on monocytes from Behcet’s disease patients
Yuri Ahn1, Ji-Hye Hwang1, Zhenlong Zheng1,2, Do Young Kim1
1Department of Dermatology and Cutaneous Biology Research Institute, Yonsei University College of Medicine, Seoul, Korea, 2Department of Dermatology, Yanbian University Hospital, Yanji, China

PO-2 Vitamin D and extracellular calcium regulate inflammation on cultured sebocytes
Jun Hong Park, Dong Hyuk Eun, Yong Hyun Jang, Seok-Jong Lee, Do Won Kim, Weon Ju Lee
Department of Dermatology, School of Medicine, Kyungpook National University, Daegu, Korea

PO-3 T-cell-immunoglobulin- and mucin-domain-containing molecule-3 (TIM-3) expression in the patients with psoriasis vulgaris
Soo-Eun Jung, Hyun Soo Lee, Young Joon Park, Mi-Jin Park, Eun-So Lee
Department of Dermatology, Ajou University School of Medicine, Suwon, South Korea

PO-4 Clinical and histopathological differences between men and women with moderate to severe psoriasis
Young Joon Park1, Soo-Eun Jung1, Ji Young Yang1, Mi-Jin Park1, Eun-So Lee1
1Department of Dermatology, Ajou University School of Medicine, Suwon, Korea

PO-5 Increased expression of TRPV3 and TRPV4 channel in keratinocytes under Th2 inflammation
Woo-II Kim1,2, Kihyuk Shin1, Bo-Yeon Kim1, Gun-WOOK Kim1, Hoon-SoO Kim1, Byung-Soo Kim1, Moon-Bum Kim1, Hyun-Chang Ko1
1Department of Dermatology, School of Medicine, Pusan National University, Busan, Korea
2Department of Dermatology, Pusan National University Yangsan hospital, Yangsan, Korea

PO-6 Genetic Polymorphism of Thymic Stromal Lymphopoietin(TSLP) in Korean Atopic Dermatitis(AD) and Allergic March(AM) patients
Eun Jung Ko1, Won Il Heo1, Joon Seok1, Hyun Jung Kwon1, Ga Ram Ahn1, Taewon Jin1, Ju Hee Kim1, Min Jeong Kim1, Kui Young Park1, Mi-Kyung Lee2, Seong Jun Seo1
1Department of Dermatology, Chung-Ang University Hospital, Seoul, South Korea
2Department of Laboratory Medicine, Chung-Ang University Hospital, Seoul, South Korea

PO-7 Epidermal growth factor relieves inflammatory signals in S. aureus treated human epidermal keratinocytes and atopic dermatitis-like skin lesions in Nc/Nga mice
Sun Young Choi1, Hyun Ji Kang1, Woo Jin Lee1, Chong Hyun Won1, Mi Woo Lee1, Jee Ho Choi1, Sung Eun Chang1, Sang Hyun Cho1
1Department of Dermatology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea
2Department of Dermatology, Incheon St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Incheon, Korea
PO-8 Striae distensae due to topical glucocorticoids is relatively rare in atopic dermatitis than psoriasis patients
Sung Jay Choe1, Donghye Kim1, Eun Jung Kim1, Eung Ho Choi1
1Department of Dermatology, Yonsei University Wonju College of Medicine, Wonju, Korea .......... 159

PO-9 Association of COL6A6 and CDKAL1 polymorphisms with early-onset atopic dermatitis in Korean population
Won Il Heo1, Kui Young Park1, Mi-Kyung Lee2, Taewon Jin1, Min Jeong Kim1, Ju Hee Kim1, Seong Jun Seo1
1Department of Dermatology, Chung-Ang University Hospital, Seoul, Korea
2Department of Laboratory Medicine, Chung-Ang University Hospital, Seoul, Korea .......... 160

PO-10 ΔNP63α transcriptional regulator is modified with O-GlcNAc in HaCaT keratinocytes
Kyung-Cheol Sohn1, Jeung-Hoon Lee1, Chang-Deok Kim1
1Department of Dermatology, College of Medicine, Chungnam National University, Daejeon, Korea
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PO-11 Inhibition of collagen production by ICG-001, a small molecule inhibitor for Wnt/β-catenin signaling, in skin fibroblasts
Kyung-II Kim1, Do-Sun Jeong1, Eui Chang Jung2, Jeung-Hoon Lee3,4, Chang Deok Kim3, Tae-Jin Yoon1
1Department of Dermatology and Institute of Health Sciences, School of Medicine, Gyeongsang National University & Hospital, Jinju, Korea
2Department of Dermatology, School of Medicine, Chungnam National University, Daejeon, Korea
3Department of Dermatology, School of Medicine, Chungnam National University, Daejeon, South Korea
4Skin Med Company, Daejeon, South Korea ................................................................. 162

PO-12 NecroX-5 inhibits poly(I:C)-induced inflammatory reaction of keratinocytes
Jung Soo Kim, Ji Hyun Lee, Yeong Ho Kim, Hyun-Min Seo, Chul Whan Bang, Jun Young Lee, Young Min Park
Department of Dermatology, Seoul St. Mary’s Hospital, College of Medicine, The Catholic University of Korea, Seoul, Korea ................................................................. 163

PO-13 A cell model for Th2 allergic inflammation in keratinocytes
Min Song Suh1, Na Hee Kim1, Jee-Young Choi1, Mei Shan Piao1, Sook Jung Yun1, Jee-Bum Lee1, Seung-Chul Lee1
Departments of 1Dermatology, Chonnam National University Medical School, Gwangju, Korea .... 164

PO-14 A functional role of GDA as melanogenic stimulator
Ik Jun Moon, Young Jae Kim, Tai Kyung Noh, Woo Jin Lee, Chong Hyun Won, Mi Woo Lee, Jee Ho Choi, Sung Eun Chang
Department of Dermatology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea ................................................................. 165
PO-15 The role of TGF-β3 in melanogenesis and senescence  
Heun Joo Lee, Ho Jeong Shin, Woo Jin Lee, Chong Hyun Won, Sung Eun Chang, Mi Woo Lee, and Jee Ho Choi  
Department of Dermatology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea  
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PO-16 Clusterin is secreted from endothelial cells and regulates pigmentation  
Misun Kim1, Jiun Lee1, Tae Jun Park2, Hee Young Kang3  
1Department of Dermatology, Ajou University School of Medicine, Suwon, Korea, 2Department of Biochemistry and Molecular Biology, Ajou University School of Medicine, Suwon, Korea  
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PO-17 Isolation of NCSCs from hair follicle bulge and differentiation induction into melanocyte precursors  
Tae-Hoon Kim, Ho-Jin Kim, Jeong-Wan Seo, Ki-Ho Kim  
Department of Dermatology, College of Medicine, Dong-A University, Busan, Republic of Korea  
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PO-18 Double-stranded RNA induces inflammation via the NF-κB pathway and inflammasome activation in the outer root sheath cells of hair follicles  
Jung-Min Shin1, Dae-Kyoung Choi1, Kyung-Cheol Sohn1, Soo-Yeon Kim1, Ji-Young Kim1, Young Ho Lee2, Myung Im1, Young-Joon Seo2, Chang Deok Kim1, Jeung-Hoon Lee1, Young Lee1  
1Department of Dermatology, School of Medicine, Chungnam National University, Daejeon, Korea  
2Department of Anatomy, School of Medicine, Chungnam National University, Daejeon, Korea  
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PO-19 Gasdermin C is induced by ultraviolet and contributes to MMP-1 expression via activation of ERK and JNK pathways  
Kusumaningrum Novi1,2,3, Dong Hun Lee1,2,3, Hyun-Sun Yoon2,3,4, Yeon Kyung Kim1,2,3, Chi-Hyun Park1,2,3, and Jin Ho Chung1,2,3  
1Department of Biomedical Sciences, Seoul National University College of Medicine;  
2Department of Dermatology, Seoul National University College of Medicine;  
3Institute of Human-Environment Interface Biology, Medical Research Center, Seoul National University;  
4Department of Dermatology, Seoul National University Boramae Hospital, Seoul, Republic of Korea  
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PO-20 Epithelial precursor cell-conditioned media ameliorates UV irradiation-induced extracellular matrix damage in human skin equivalents  
Ha-Yeon Kim1, Su-Ji Sohn1, A-Ram Kim1, Seung-Hwa Baek1, Chang-Jin Lee1, Dong Hyun Kim1,2, Tae-Aug Kim1  
1Skin Biology Research Center, Department of Biochemistry, School of Medicine, CHA University,  
2Department of Dermatology, CHA Bundang Medical Center, Seongnam-Si, Gyeonggi-Do, Republic of Korea  
171
PO-21 Preventive effect of Cacao extract on UVB-induced skin wrinkle formation via inhibition of DNA methylation

A-Ram Kim, Jina Choi, Jihwan Lee, SeungHwa Baek, Dong Hyun Kim and Tae-Aug Kim

Skin Biology Research Center, Department of Biochemistry, School of Medicine, CHA University,
Department of Dermatology, CHA Bundang Medical Center, Seongnam-Si, Gyeonggi-Do, Republic of Korea

PO-22 PD-1 expression in cutaneous extranodal NK/T-cell lymphoma: its effect on clinical characteristics

Young Jae Kim, Chong Hyun Won, Sung Eun Chang, Mi Woo Lee, Jee Ho Choi, Woo Jin Lee

Department of Dermatology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea

PO-23 Beneficial effects of silybum marianum extract and silymarin on regulation of decorin and biglycan in human dermal fibroblasts

Min Kyeong Shin, Jiyeong Lim, Hanon Lee, Jang-Hee Oh, Soyun Cho, and Jin Ho Chung

Department of Dermatology, Seoul National University College of Medicine, Seoul, Korea,
Institute of Human-Environment Interface Biology, Medical Research Center, Seoul National University, Seoul, Korea,
Biomedical Research Institute, Seoul National University Hospital, Seoul, Korea,
Department of Biomedical Sciences, Seoul National University Graduate School, Seoul, Korea,
Department of Dermatology, Seoul National University Boramae Hospital, Seoul, Korea

PO-24 Plant oils have antioxidant activity in UVB-irradiated NHEKs by upregulating detoxifying enzymes

Hye Rin You, Jee-Young Choi, Mei Shan Piao, Egyung Park, Sook Jung Yun, Jee-Bum Lee and Seung-Chul Lee

Departments of Dermatology, Chonnam National University Medical School, Gwangju, Korea

PO-25 Reductions in matrix metalloproteinase and collagen transcription by decreasing signal transduction through the Transforming growth factor-β/Smad pathway in normal senescing human dermal fibroblasts

Young Il Kim, Mu-Hyoung Lee, Hye-Jin Ahn and Min Kyung Shin

Medical Science Research Institute, Kyung Hee University Medical Center, Seoul, Korea

Department of Dermatology, College of Medicine, Kyung Hee University, Seoul

PO-26 A case of vascular Ehlers-Danlos syndrome with a novel mutation in COL3A1

Kosuke Shido, Kaname Kojima, Yoichi Suzuki, Masao Nagasaki, Kenshi Yamasaki, Setsuya Aiba

Department of Dermatology, Tohoku University Graduate School of Medicine, Sendai, Japan

Graduate School of Medicine, Tohoku University, Sendai, Japan

Tohoku Medical Megabank Organization, Tohoku University, Sendai, Japan
PO-27 Extracellular superoxide dismutase (SOD3) inhibits Propionibacteria acnes-induced inflammation in vitro
Cuong Thach Nguyen, Kim Jeong-Ho, Kim Tae-Yoon
Department of Dermatology, College of Medicine, The Catholic University of Korea, Seoul 137-040, South Korea

PO-28 Adiponectin signaling regulates lipid production in human sebocytes
Yu-Ra Jung1, So-Ra Choi1,2, Jin-Hyup Lee1, Kyung-Cheol Sohn1, Young Lee1, Young-Joon Seo1, Chang-Deok Kim1, Jeung-Hoon Lee1, Seung-Phil Hong2, Seong-Jun Seo1, Seong-Jin Kim1, Myung Im1
1Department of Dermatology, College of Medicine, Chungnam National University, Daejeon, Korea
2Department of Dermatology, College of Medicine, Dankook University, Cheonan, Korea

PO-29 LRG1 is involved in skin aging by upregulation of matrix metalloproteinase-1 and downregulation of type 1 collagen in human dermal fibroblasts
So Yun Ahn1, Dong Hun Lee2, Jin Ho Chung2, and Seung-Taek Lee1
1Department of Biochemistry, College of Life Science and Biotechnology, Yonsei University, Seoul, Republic of Korea, 2Department of Dermatology, Seoul National University College of Medicine, Seoul, Republic of Korea

PO-30 A B cell subset that produces IL-10 suppresses contact dermatitis
Masahiro Kamata1,2, Kathleen M. Candando7, Evgueni Kountikov7, Ayumi Yoshizaki1,2, Tomomitsu Miyagaki1,2, Jacquelyn M. Lykken7, Jonathan C. Poe7, Shinichi Sato1, and Thomas F. Tedder2
1Department of Dermatology, School of Medicine, The University of Tokyo, Tokyo, Japan
2Department of Immunology, Duke University Medical Center, Durham, North Carolina, USA
The 27th Annual Meeting of the Korean Society for Investigative Dermatology

2017. 3. 24(Fri)
The 27th Annual Meeting of the Korean Society for Investigative Dermatology

Free Communications (I)

(FC-1 ~ FC-9)
Background: Citron is well-known for its abundant antioxidative ingredients such as vitamin C, polyphenol compounds, and limonoids.

Objectives: We aimed to evaluate effects on rosacea mediators by citron essential oils on the activated keratinocytes in vitro.

Methods: We used normal human epidermal keratinocytes (NHEKs) derived from neonatal foreskin. NHEKs were stimulated with VD3, TNFα to induce rosacea mediators; Kallikrein 5 (KLK5), LL-37, vascular endothelial growth factor (VEGF), transient receptor potential vanilloid 1 (TRPV1), IL-8. These mediators were analyzed after treatment of citron seed and unripe citron oils, and also EGCG as a control. The analysis was performed by semi-quantitative RT-PCR, immunocytofluorescence and ELISA.

Results: mRNA and protein levels of KLK5, LL-37, VEGF, TRPV1, and IL-8 of activated keratinocytes were suppressed by citron seed and unripe citron oils.

Conclusion: These results show that citron essential oils have suppressive effects on rosacea mediators on activated epidermal keratinocytes, which indicate that this agent may be a valuable adjuvant treatment candidate for rosacea.
**FC-2**

**Psychological stress deteriorates skin barrier function by activation of 11β-hydroxysteroid dehydrogenase 1 as well as endogenous glucocorticoids**

Sung Jay Choe¹, Donghye Kim¹, Eun Jung Kim¹, Eung Ho Choi¹

¹Department of Dermatology, Yonsei University Wonju College of Medicine, Wonju, Korea

**Background:** Psychological stress (PS) increases endogenous glucocorticoids (GC) by the activation of hypothalamic-pituitary-adrenal axis. Negative effects of PS on skin barrier function have been known as a result of interaction between endogenous GC and GC receptor in peripheral tissues. However, endogenous GC can exert its activity when cortisone (inactive form) is converted into cortisol (active form) by 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1) in peripheral tissues.

**Objective:** We performed this study to elucidate the effect of 11β-HSD1 on skin barrier function under PS.

**Methods:** 25 medical students were enrolled. All measurements were repeated under PS and normal state. Salivary cortisol at 8AM and midnight, mRNA and protein expression of 11β-HSD1 from oral mucosa cells, skin barrier functions were assessed. Furthermore, using insomniac PS murine model, we compared the changes in skin barrier function and the expression of 11β-HSD1 in the epidermis among the control group, PS group and the group treated with topical 11β-HSD1 inhibitor under PS.

**Results:** In PS status, cortisol level in the saliva and corneocytes, as well as basal transepidermal water loss (TEWL), and 11β-HSD1 of oral mucosa (both mRNA and protein expression) significantly increased, and SC integrity significantly decreased compared to normal. In addition, the cortisol level in the corneocytes was positively correlated with basal TEWL and 11β-HSD1 in oral mucosa and negatively correlated with SC integrity. In animal studies, epidermal expression of 11β-HSD1 was increased in the PS group compared to the control group and was decreased in the group treated with topical 11β-HSD1 inhibitor compared to the PS group. Furthermore, the expression of 11β-HSD1 in the epidermis showed a positive correlation with basal TEWL and a negative correlation with SC integrity and barrier recovery.

**Conclusion:** PS deteriorates skin barrier function by the activation of 11β-HSD1 as well as endogenous GC.
FC-3

**Fibroblast-derived clusterin inhibits melanogenesis**

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**Background:** Clusterin (CLU), also known as apolipoprotein J, appears to be extremely sensitive biosensor of oxidative injury in human tissues. CLU expression is highly induced during many types of oxidative stress including UV exposure.

**Objective:** We analyzed the role of CLU in the regulation of skin pigmentation in line with the epidermal/dermal cross-talk between fibroblasts and melanocytes.

**Method:** CLU expressions were investigated with cultured skin cells, UV irradiated fibroblasts, and acutely UV irradiated in vivo skin. Fibroblast was infected with CLU lenti-virus or shRNA. Normal human melanocytes were treated with conditioned medium derived from the fibroblasts and the melanogenesis was analyzed. The pigmentation was also assessed using the ex vivo skin and the artificial skin. The TGF β signaling pathway was analyzed.

**Results:** CLU mRNA and protein were highly expressed in fibroblasts, scarcely expressed in keratinocytes, and not in melanocytes. CLU expression was increased in the UV irradiated fibroblasts and the acutely UV irradiated in vivo skin. The melanin contents and tyrosinase activity were significantly reduced in the melanocytes treated with conditioned medium from CLU overexpressed fibroblasts. The mRNA and protein expression levels of melanogenesis-associated proteins, microphthalmia-associated transcription factor (MITF) and tyrosinase were significantly down-regulated. An inhibitory role of CLU on pigmentation was also demonstrated on ex vivo skin treated with a conditioned medium from fibroblasts. CLU suppressed PAX3 and MITF expressions via TGF β/Smad signaling activation.

**Conclusion:** UV irradiation induces CLU in the skin and the fibroblasts derived-CLU inhibits melanogenesis via the cross-talk between fibroblasts and melanocytes.
FC-4

Hippocampal abnormalities and HPA axis alteration in UV irradiated mice

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Background: Adult hippocampal neurogenesis is suppressed by stress. Stressful stimuli triggers Hypothalamic-pituitary-adrenal (HPA) axis and increasing corticosterone in the blood. The increased corticosterone in the blood inhibits hippocampal neurogenesis via glucocorticoid receptor activation and neurotropic signaling pathway regulation. Recently, it is reported that UV stimuli could activation both central and cutaneous HPA axis.

Objective: To prove our hypothesis that repeated UV stimuli could activate HPA axis and increase corticosterone and lead to hippocampus abnormalities.

Methods: We used the UV irradiated mouse model to illuminate possible molecular mechanism by which UV exposure affects hippocampal neurogenesis and synaptogenic factors. 2 weeks after UV irradiation skin, hormone, and brain were analyzed for biological changes.

Results: We observed that the decrease of number of immature neuron (DCX) and synaptic plasticity markers (NMDAR2a and PSD-95) in hippocampus by UV irradiation. Hormonal analysis revealed that plasma level of glucocorticoid hormone (CORT) was significantly increased in UV irradiated group. Furthermore, we observed central Hypothalamic-pituitary-adrenal (HPA) axis and cutaneous HPA axis activation. Immunohistochemistry results revealed that glucocorticoid receptor (GR), the receptor of CORT, was translocated into nucleus by UV irradiation in hippocampal dentate gyrus (DG) region. Moreover, brain-derived neurotropic factor (BDNF) and phosphorylated-ERK, downstream molecule of BDNF, were down-regulated in the hippocampus.

Conclusion: Taken together, we propose that repeated UV exposure is one of stressor which can affect hippocampal abnormalities possibly by HPA axis activation.
Pre-elafin is involved in ultraviolet-induced keratinocyte apoptosis

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Pre-elafin is known to control keratinocyte integrity via cornified envelope (CE) formation and inhibition of desquamation, but its role in ultraviolet (UV)-induced keratinocyte apoptosis has not been hitherto addressed, thus it was examined in volunteer skin samples and primary cultured normal human keratinocytes irradiated with phototoxic doses of UVA/NB-UVB and in keratinocytes with pre-elafin overexpression/knockdown under low/high-calcium conditions. Phototoxic UV doses increased pre-elafin mRNA and protein expression, inversely proportional to keratinocyte survival. Pre-elafin overexpression under low calcium conditions, which was localized to the cytoplasm in contrast to high calcium conditions, increased keratinocyte apoptosis, whereas knockdown inhibited UV-induced apoptosis. Pre-elafin was colocalized with, but not bound to cleaved caspase-3. Pre-elafin reduced cystatin-A expression, which was bound to pro-caspase-3. In conclusion, UV phototoxicity-induced pre-elafin in the inside of keratinocytes before CE formation could be involved in UV-induced keratinocyte apoptosis via cystatin-A downregulation resulting in pro-caspase-3 activation.

Keywords: UV, Keratinocyte apoptosis, Pre-elafin, Cellular location, Cystatin-A, Pro-caspase-3
Background: Cutaneous inflammation induced by UV irradiation has been well documented clinically. However, despite many previous efforts, it is not fully understood which molecules mediate this inflammatory response. Our previous study showed that SAA1 induced by UV irradiation in epidermal keratinocytes can affect dermal fibroblasts.

Objective: The main objectives of this study are to investigate whether SAA1 mediates UV-induced proinflammatory cytokines in human skin and to elucidate its mechanism of action.

Methods: Normal human dermal fibroblasts (NHDFs) were treated with recombinant human SAA1 (rhSAA1) and proinflammatory cytokine levels were analyzed by qRT-PCR and ELISA. Next, keratinocyte conditioned media (KCM) with or without UV irradiation and SAA1 siRNA transfection were treated NHDFs and proinflammatory cytokine levels were analyzed. NHDFs were treated with rhSAA1 after TLR4 knockdown or BAY 11-7082 (NF-κB inhibitor) and proinflammatory cytokine levels were analyzed.

Results: NHDFs treated with rhSAA1 showed increased proinflammatory cytokines in mRNA and protein levels. UV-irradiated KCM induced proinflammatory cytokines in NHDFs but this was inhibited by knockdown of SAA1. Also, knockdown of TLR4 or blocking NF-κB signaling pathway inhibited rhSAA1-induced increases of proinflammatory cytokines in NHDFs.

Conclusion: The results of this study suggest that SAA1 can be a potential mediator for UV irradiation-induced cutaneous inflammation.
**FC-7**

**5-ALA-mediated sonodynamic therapy induced antitumor effect for angiosarcoma**

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**Background:** Angiosarcoma, a highly malignant vascular tumor arising from vascular endothelial cells, is a lethal disease for which complete cure is rarely seen. Effective treatment options are limited for patients with angiosarcoma. Therefore we hypothesized that sonodynamic therapy (SDT) may be value in the treatment of angiosarcoma that is resistant to conventional therapy.

**Objective:** We investigated the effect of SDT for angiosarcoma by low frequency ultrasound with 5-aminolevulinic acid (5-ALA), a precursor of protoporphyrin IX (PpIX) in heme synthetic process.

**Method:** We used *in vitro* tumor cell model by inoculating human angiosarcoma cell line ISO-HAS-B. The tumor was sonicated by 1-MHz ultrasound 3 hours following administration of 5-ALA. The cytotoxicity was investigated 24h after 5-ALA-mediated sonodynamic action.

**Result:** The number of tumor cells was significantly decreased in ALA (+) US (+) group compared with control group (ALA (-) US (-)), non-5-ALA administrated (ALA (-) US (+)) group or non-sonicated (ALA (+) US (-)) group.

**Conclusion:** The results suggest the therapeutic effect of 1-MHz ultrasound for the angiosarcoma in 5-ALA administrated group by inducing apoptotic change of tumor cells. This study elucidated the feasibility of therapeutic use of 1 MHz, relatively low frequency, ultrasound in sonodynamic therapy using 5-ALA as a sonosensitizer precursor. The utilization of this frequency will contribute to the development of SDT for angiosarcoma and the spread of this technique in dermatological area.
Sebaceous carcinomas (SeCAs) represent cutaneous malignancies that are capable of leading to incurable metastases. SeCAs develop with higher prevalence in the context of the DNA repair deficiency Muir-Torre syndrome, but can also arise de novo on sun-exposed skin, particularly of the face. To dissect their genetic origins, we exome-sequenced 25 SeCAs not associated with known Muir-Torre syndrome. We discovered that the mutational profile of SCAs notably mirrors that of squamous cell carcinomas, including HRAS, FAT1, or diverse NOTCH receptor mutations in 87% of cases, while lacking mutations characterizing basal cell cancers. These similarities in somatic aberration suggest that SCAs and squamous malignancies may share a cell of origin and possible responses to targeted and immunotherapies. Surprisingly, we also identified novel, recurrent inactivating mutations in the UBR5 ubiquitin ligase in 32% of our cases. Loss-of-function UBR5 mutations are usually seen in the context of activating NOTCH mutations in hematologic cancers, rather than the inactivating NOTCH aberrations we previously identified in epithelial tumors. As UBR5 has long been associated with key DNA damage response pathways, including ATM, these findings therefore also delineate a subset of cancers that may be amenable to synthetic lethal oncotherapeutic strategies.
FC-9

MITF regulates dynamic melanoma heterogeneity

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Background: Differential tumor cell behavior caused by environmental conditions, termed dynamic heterogeneity, is a prime source for drug resistance.

Objective: As distinct proliferative and invasive capabilities reflect variable drug sensitivities, identifying and characterizing these different responses is crucial to design effective therapies.

Methods: We utilize real-time cell cycle imaging (FUCCI) in 3D in vitro and in vivo to study melanoma heterogeneity.

Results: Mouse xenograft tumors generated from cell lines with high microphthalmia-associated transcription factor (MITF) level displayed a homogeneous distribution of cycling cells throughout. In contrast, tumors generated from cell lines with low MITF levels were composed of clusters of cycling cells and clusters of G1-arrested cells. The proliferating areas were in close proximity to blood vessels, presumably characterized by oxygen/nutrient availability. Melanoma spheroids recapitulated the in vivo cycling behavior, considering that here oxygen and nutrients are supplied by diffusion. MITF was undetectable within the hypoxic G1-arrested spheroid core, indicating hypoxia-induced MITF downregulation. Modulation of MITF expression impacted spheroid density, with overexpression giving rise to less compacted structures and vice versa. We showed that MITF protects from cell cycle arrest induced by oxygen/nutrient deprivation. High MITF levels prevent cell cycle arrest by reducing the cell-intrinsic propensity to arrest in response to low oxygen/nutrient and concurrently by allowing sufficient supply of oxygen/nutrients to cells. The latter is achieved through decreased cell-cell adhesion resulting in the generation of looser, ‘spongier’ tumors that allows more efficient oxygen/nutrient diffusion.

Conclusion: These data that MITF is a potent regulator of dynamic heterogeneity, which in turn impacts on drug sensitivity.

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The 27th Annual Meeting of the Korean Society for Investigative Dermatology

Hot Posters (I)
(HP-1 ~ HP-10)
HP-1

Adiponectin recovers abnormalities of keratinocyte induced by intrinsic and extrinsic stress damage

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Background: The human skin is one of the most composite and calling defense organ which is exposed to intrinsic and extrinsic environment. The antimicrobial peptide is an essential element of the unique innate immune system established by the keratinocytes. Abnormal regulation of the antimicrobial peptide caused by intensive and accumulative intrinsic or extrinsic stress damage may jeopardize the innate immune system of the skin which leads to immune related diseases. Adiponectin has been suggested as a positive factor on functional recovery due to its well-established global effects against energy metabolism-related regulation or inflammatory signaling pathways. However, there are few studies on adiponectin related with keratinocytes except of wound healing.

Objective: The object of this study was aimed to reveal the effects of adiponectin against UVB or H2O2 stress damage in keratinocytes.

Methods: Normal human keratinocytes were exposed to 100 μM H2O2 or continuously exposed to low energy of UVB with indicated intervals presence or absence of adiponectin treatment. Experimented cells were used for mRNA, protein and cytological analysis.

Results: Adiponectin reduced senescence associated beta-galactosidase (SA-β-gal) positive ratio and expression of p16INK4a and histone H2A.X which represents premature senescence. Additionally, adiponectin increased SIRT1 and FoxO activity and also up-regulated Manganese superoxide (MnSOD) which provided results of ROS scavenging. Also, adiponectin reduced abnormal increase of human beta-defensin 2 (hBD2) which was induced by both stress factors. In addition, the present clinical study showed higher expression of hBD2 in sun-exposed skin of elderly group which correlates with the in-vitro results. Adiponectin suppressed AP-1 transcription factor activities via p38/JNK pathway suppression in damaged keratinocytes which reverses the abnormal expression of hBD2.

Conclusion: These results assure the possibility of adiponectin as an anti-stress agent that recovers innate immune barrier in damaged keratinocytes by intrinsic or extrinsic stress factors.
HP-2

Effects of human mesenchymal stem cells co-culture on calcium-induced differentiation of normal human keratinocytes

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Background: The influence of mesenchymal stem cells (MSCs) on keratinocytes in altered microenvironments is poorly understood. High Ca²⁺ environment to keratinocytes can disrupt normal skin barrier function due to abnormal/premature differentiation of keratinocytes.

Objective: we aimed to co-culture umbilical cord blood-derived MSCs with normal human epidermal keratinocytes (NHEK) to evaluate their paracrine effect in the presence of high extracellular calcium (Ca²⁺) concentration.

Methods: we co-cultured MSCs and keratinocytes for 20 hours before Ca²⁺ treatment to let the medium conditioned with the secretome from either MSCs or keratinocytes. Then, we induced differentiation in keratinocyte with Ca²⁺ (1.5 mM) for further 24 hours and harvested the keratinocytes for gene and protein analysis.

Results: Surprisingly, we found that MSCs suppress both proliferation and differentiation of keratinocytes under a high Ca²⁺ environment in TGF β1-dependent manner. Furthermore, we determined that MSCs can regulate the mitogen-activated protein kinases (MAPKs), phosphatidylinositol 3-kinase (PI3K) / protein kinase B (AKT), and protein kinase C (PKC α) pathways in Ca²⁺-induced differentiated keratinocytes. Knockdown of TGF β1 from MSCs results in decreased suppression of differentiation with significantly increased proliferation of keratinocytes compared with control MSCs. MSCs-derived TGF β1 further induced growth inhibition of keratinocyte in high extracellular Ca²⁺ environment as analyzed by a decrease in DNA synthesis, accumulation of phosphorylated retinoblastoma protein (pRb), cdc2, and increased mRNA level of p21, and independent of TGF β1/SMAD pathway.

Conclusion: We found that MSCs-derived TGF β1 is a critical regulator of keratinocyte function, and involves multiple proximal signaling cascades. This study could have significant implication in wound management studies.
Adiponectin upregulates filagrin expression via SIRT1-mediated signaling and increase of calcium influx in human normal keratinocytes

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Backgrounds: Filagrin (FLG) is the major component of the epidermal granular layer and binds to and condenses the keratin cytoskeleton. Filagrin thus contributes to cell compaction and serves as a natural moisturizing factor by promoting unfolding and degradation into hygroscopic amino acids. Loss or downregulation of FLG has been shown to result in a weak stratum corneum, which causes water loss and increases the possibility of skin barrier-related seizure. Adiponectin (Acrp30) contributes to the functional recovery of somatic cells, including human normal epidermal keratinocytes (NHEKs).

Objects: We aim to reveal the possibility of adiponectin to regulate Filagrin

Methods: NHEKs were treated with Acrp30 and the levels of FLG were examined. SIRT-targeting siRNA and ARNT-targeting siRNA were used to identify the role of various signal transduction pathway components. Also, calcium influx was measured on NHEKs to cross talk its relations with differentiation and filagrin expression.

Results: Acrp30 upregulated SIRT1 and ARNT expression in NHEKs, resulting in increased FLG expression. Treatment with both SIRT1-targeting siRNA and ARNT-targeting siRNA blocked Acrp30 stimulation and silenced FLG expression. Also, acrp30 increased calcium influx in NHEKs which is a trigger of differentiation.

Conclusion: Adiponectin upregulates FLG expression through a SIRT1-mediated pathway and increase of calcium influx. Our results suggest that Acrp30 is a promising agent for skin barrier permeability improvement.

Keywords: Keratinocyte, SIRT1, Filagrin, ARNT, Adiponectin
**HP-4**

**UV-induced DNA methyltransferase I epigenetically silences tissue inhibitor of metalloproteinase I in the human skin**

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**Background:** Ultraviolet (UV) has been reported to influence the activities of epigenetic regulation by affecting the expression of genome regulators such as DNA methyltransferase I (DNMT1). DNMT1 is a “gene silencer,” that is responsible for the maintenance of DNA methylation and contribution to de novo methylation. Implications of DNMT1’s involvement in the expression of UV-induced proteins have been previously reported. In UV-irradiated skin, the levels of matrix metalloproteinases (MMPs) has been reported to elevate, and the levels of tissue inhibitor of metalloproteinases (TIMPs), an inhibitor of MMPs, to decrease.

**Objective:** In this study, we examined the role of DNMT1 in the suppression of TIMP1 in UV-irradiated human skin.

**Methods:** The expression of DNA methylation-related proteins and TIMP1 were analyzed in UV-irradiated human skin in vivo and in human dermal fibroblasts in vitro. To analyze the relationship between TIMP1 and DNMT1, we performed the inhibition, knockdown, and overexpression of DNMT1. Lastly, to confirm the increase in methylation on the CpG island residing in the TIMP1 promoter region, methylation-specific PCR was performed.

**Results:** In acutely UV-irradiated human skin, we observed an increase in the expression of DNMT1 in a time-dependent manner in vivo. De novo methyltransferases, DNMT3a and DNMT3b, however, showed minor changes. Congruent outcomes were seen in UV-irradiated human dermal fibroblasts in vitro. The expression of TIMP1 decreased as a result of UV irradiation. With the decrease of DNMT1 expression, TIMP1 increased. However, the overexpression of DNMT1 led to reduced levels of TIMP1. In addition to these findings, methylation-specific PCR is being performed to confirm the increase in methylation on the CpG island residing in the TIMP1 promoter region.

**Conclusion:** UV-induced expression of DNMT1 suggests increased methylation, and thus, the silencing of TIMP1, in the human skin.
**HP-5**

**Anacardic acid reduces lipogenesis in human differentiated adipocytes via inhibition of histone acetylation**

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**Background:** Here we investigated effects of anacardic acid (AA) on the regulation of lipogenesis in differentiated human adipocytes, and elucidated possible epigenetic mechanisms via p300 histone acetyltransferase activity.

**Objective:** To investigate the role of histone acetylation in lipogenesis regulation, we evaluated triglyceride (TG) contents, expression of key lipogenic enzyme acetyl-CoA carboxylase (ACC) and sterol regulatory element binding protein 1c (SREBP-1c) in primary cultured adipocytes isolated from subcutaneous adipose tissues.

**Results:** Treatment of AA or knockdown of p300 by using transient transfection of p300 siRNA led to significant reduction of TG contents, SREBP-1c and ACC expression, indicating that p300 mediates SREBP-1, ACC expression, and corollary lipid production. While p300 overexpression by p300WT was associated with significantly enhanced activity of SREBP1-908luc promoter, the SREBP1-908luc promoter activity was significantly reduced in the presence of p300\(^{Δ}\)HAT. In addition, we performed a promoter assay using HEK293T cells treated with AA or TSA. While the SREBP1-908luc promoter activity was significantly decreased by AA, but significantly increased by TSA treatment.

**Conclusion:** These findings suggest that histone acetyltransferase activity of p300, not a p300 expression per se, is critical for the transcriptional regulation of SREBP-1 and p300HAT inhibitors such as AA could be employed as anti-obesity modalities.
**Background:** Several biomarkers have been reported about diagnosis of Stevens-Johnson syndrome (SJS)/toxic epidermal necrolysis (TEN). For example, granulysin might be a candidate for early-diagnostic marker, however other disease, such as drug induced hypersensitivity syndrome, also show high serum levels of granulysin.

**Objective:** The aim of this study is to identify beneficial biomarker for diagnosis of SJS/TEN and prediction of disease severity.

**Methods:** Peripheral blood mononuclear cells (PBMCs) were obtained from SJS/TEN patients (n=4) or non-severe drug eruption e.g. maculopapular exanthema and erythema multiforme (MPE/EM) patients (n=4). PBMCs were exposed to causative drugs and then cultured for 5 days to allow proliferation of drug-specific T cells. The cells were stimulated by reexposure with same causative drugs for 1 day in medium without serum, and then supernatant was collected. Sera of SJS/TEN (n=7) were collected.

**Results:** First, the proteins in SJS/TEN and MPE/EM supernatant were identified by mass spectrometry analysis. In each samples, 200~500 proteins were identified. Then the proteins that found in SJS/TEN but not in MPE/EM were ed (n=47) as biomarker candidates and quantitated using ed reaction monitoring with stable synthetic isotope-labeled peptides as an internal control. The SRM results revealed that significant differences were observed in 22 proteins which have never been reported to be involved in SJS/TEN. Among them, we focused on calmodulin-like protein 5 (CALML5) which has been reported to contribute to keratinocyte differentiation. We measured their serum levels using ELISA. All of normal control levels were not detected (n=5) (cut off; 0.156ng/ml), whereas CALML5 level from SJS/TEN patients were significantly elevated (n=7; 0.58÷0.36ng/ml) (P < 0.03). In addition, CALML5 level showed a tendency to relate with disease severity such as area of skin erosion.

**Conclusion:** Serum CALML5 might be a candidate of biomarker for diagnosis of SJS/TEN and prediction of disease severity.
Dendritic cells (DCs) are critical regulators of adaptive immunity by directing T cells to antigen-specific immunity or immune tolerance. We previously demonstrated that Flt3L-dependent lymphoid-resident classical DCs are required for protective immunization using a protein-immunization model with a TLR-adjuvant. Although TLR-based licensing has been well described during immunogenic programming of DCs, it remains unclear which cellular components and additional cues are provided to DC to distinguish danger signals and self-antigens at the time of priming. Here, we demonstrate that NK cells are a critical innate immune component to facilitate protective immunization. We successfully depleted NK cell populations in multiple organs by using anti-Asialo-GM-1 antibody. Depletion of NK cells during vaccine priming led to significantly decreased antigen-specific effector responses, without diminishing cognate CD4+ T cell recall-proliferation, indicating that NK help was primarily required for effector activity, over memory. As early as 5 hrs after vaccine/adjuvant footpad injection, NK cells had highly activated phenotypes. Mechanistically, NK-driven IFN-γ augmented in vitro CD4+ T cell migration toward DCs, and this effect was mediated by CXCL10. Accordingly, a population of antigen-specific effector T cells was precisely confined to the CXCR3+ cells, and NK cells were required for IFN-γ production from CXCR3+CD4+ T cells. Our results suggest that innate help may differentially impact T effector activity and clonal expansion and provide a clinical rationale for generating sufficient NK cell activity during vaccine development.
Wound healing promotion using Ar:Air plasma device

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Plasma technology is becoming widely implemented in the field of medicine. Some of its established medical uses include instrument disinfection and tooth blanching. As treatment with Ag:Air plasma has been recently shown to promote wound healing via induction of IL-6 and TGF-beta, well-controlled in vivo experiments using murine models have been carried out in our institution. Full-thickness wounds made on the back of the mice were treated with Ar:Air plasma. In comparison to the control, plasma-treated wounds showed more rapid rate of healing, supported by the immunohistological findings of increased collagen I, TGF-beta, alpha-SMA and K16. The mRNA levels of TGF-β, pro-collagen 1a1, and collagen 3 in fibroblasts tested to reveal that TGF-β expression was significantly increased compared to control. In vitro experiments using HaCat cells demonstrated that treatment with plasma increased both cell proliferation and cell migration. Furthermore, fibroblasts treated with plasma showed increased migration. These findings suggest that Ar:Air plasma is a promising novel therapy to promote cutaneous wound healing. Future studies assessing the possible harms and longterm efficacy in animal models and eventually humans are warranted.
HP-9

β-catenin regulates the expression of cAMP response element-binding protein 1 (CREB) in SCC cells

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It has been demonstrated that β-catenin signaling is essential for sustaining the cancer stem cell phenotype, because that ablation of the β-catenin gene results in the loss of cancer stem cells and complete tumor regression. However, the precise action mechanism and downstream effectors of β-catenin signaling remain to be elucidated. In this study, we attempted to find the β-catenin-regulated downstream effector in squamous cell carcinoma (SCC) cells. To identify the β-catenin-regulated genes in SCC cells, we overexpressed β-catenin using recombinant adenovirus in SCC13 cells, then screened the putative downstream genes. We found that CREB (cAMP response element binding protein 1) is upregulated by β-catenin in human SCC13 cell line. When β-catenin was overexpressed, the expression levels for CREB was significantly increased. To investigate the potential effect of CREB on cancer cell behavior, we overexpressed CREB using recombinant adenovirus in SCC13 cells. As a result, overexpression of CREB led to the marked increase of clonogenic activity. These results suggest that CREB is a β-catenin-regulated transcription factor that promotes cancer characteristics in SCC cells, providing new insight into the molecular mechanism underlying the regulation of cancer stemness by β-catenin in SCC.

Keywords: CREB, β-catenin, Squamous cell carcinoma
Background: Autophagy is an intracellular degradative system that is believed to be involved in the aging process. However, the contribution of autophagy to age-related changes in the human skin is yet unclear.

Objectives: In this study, we aimed to examine the relationship between autophagy and skin aging.

Methods: We compared the levels of autophagy in human skin fibroblasts from the photo-protected areas of the young and old population.

Results: Transmission electron microscopy analyses revealed the presence of the autophagosomes and autophagolysosome in both the young and old population. Western blot analysis did not show statistically significant difference in the amount of LC3-II, a form associated with autophagic vacuolar membranes. Both young and aged dermal fibroblasts were affected by inhibition of autophagic activity. The amount of type I pro-collagen was increased in the aged dermal fibroblasts compared with that in young fibroblasts.

Conclusion: Our findings suggest that the autophagy pathway is intact in aged dermal fibroblasts. The balance between the production of damaged/degenerated intracellular products and autophagy should be crucial in the maintenance of cellular homeostasis.
Session I.

Antimicrobial Peptides

(Sponsored by AmorePacific Co.)
CURRICULUM VITAE

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Scientific Interest:
Skin barrier, Atopic dermatitis, Aging, Diabetes, Stem cell
Correlation between skin barrier function and epidermal antimicrobial peptide expression

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The interaction between innate immunity and skin barrier has been elucidated by the various studies that antimicrobial peptides (AMPs) are stored along with precursor lipids in the lamellar bodies, which are secreted into the interspace between stratum corneum (SC) and stratum granulosum, and then co-localized with mature lipids within the SC intercellular lamellae. Acute disruption of skin barrier upregulates AMP expression in the epidermis. In the disrupted epidermis, AMP expression is blocked by occlusion, which is similar to permeability barrier recovery. Conversely, the AMP expression can regulate the permeability barrier recovery as well as wound healing.

Atopic dermatitis (AD) is a representative disease which the fundamental pathogenesis is attributable to a congenitally impaired skin barrier. Incomplete SC lipids and decreased AMP expression can permit the continuous occurrence of secondary skin infection in AD skin, which then results in rapid aggravation of AD. In addition, topical glucocorticoids and topical calcineurin inhibitors as the major drugs in AD treatment, and psychological stress known as an aggravating factor in AD and psoriasis can decrease barrier function and AMP expression in the epidermis. Recently, the epidermis of type 2 diabetes or chronic hyperglycemia patients showed simultaneous decrease of barrier function and AMP expression. Rosacea is recently known as a skin problem caused by hyper-activated AMP expression such as cathelicidin due to inherently overstimulated endoplasmic reticulum (ER) stress, as well as impaired barrier function. In this way, psoriasis shows a similar pathogenesis with rosacea. Low-dose UV light exposure can enhance skin barrier function by simultaneously increasing the production of lipids and AMPs in the epidermis through the activation of vitamin D receptor.

In conclusion, skin barrier function is correlated with epidermal AMP expression. From this result we can expect to develop novel therapeutic modalities enhancing both the skin barrier function and innate immunity.

Keywords: Skin barrier, Antimicrobial peptide, Innate immunity, Stratum corneum, Atopic dermatitis, Diabetes
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Research Experience:
1995-1997 Researcher (Hair Regrowth)
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Psoriasisin (S100A7) induce abnormal differentiation by enhancing IL-6 and nuclear translocation of jun activator binding (Jab-1) protein

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Psoriasisin (S100A7), a member of the S100 protein family, is a well-known antimicrobial peptide and a signalling molecule which regulates cellular function and is highly expressed in hyperproliferative skin conditions such as atopic dermatitis (AD) and psoriasis with disrupted skin barrier function. However, its role in epidermal differentiation remains unknown. We examined the effect of secreted and overexpressed S100A7 on epidermal differentiation in normal human keratinocytes (NHKs) and on a reconstituted human epidermis model.

When NHKs were exposed to disruptive stimuli such as Staphylococcus aureus, ultraviolet irradiation and retinoic acid, the secretion of S100A7 into the culture medium and the translocation of it to nucleus increased, and the expression of epidermal differentiation markers decreased.

The recombinant S100A7 and overexpressed S100A7 significantly inhibited epidermal differentiation by reducing the expression of keratin 1, keratin 10, involucrin and loricrin and by increasing the expression of IL-6, one of proinflammatory cytokines. We verified that the MyD88-IκB/NF-κB signal cascade was activated via RAGE after S100A7 treatment, and S100A7 regulated nuclear translocation of Jun activator binding protein (Jab-1) known as S100A7 binding protein. Finally, we confirmed that S100A7 is a negative regulator of epidermal differentiation using a reconstituted human epidermis model.

This study suggests that S100A7-related signaling molecules could be potent targets for recovering skin barrier function in AD and psoriasis where S100A7 is accumulated excessively.
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1989-1995  Korea University college of Medicine, Seoul, Korea
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**TSLP downregulates S100A7 and ß-defensin 2 via the JAK2/STAT3/Sin3a signaling pathway**

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**Background:** Elevated T-helper type 2 (Th2) cytokines in atopic skin, such as interleukin (IL)-4 and IL-13, were thought to be responsible for an impaired expression of antimicrobial proteins (AMPs) which may contribute to the increased susceptibility to skin infections in patients with atopic dermatitis (AD).

**Objective:** We investigated JAK2/STAT3 signaling as the potential pathway involved in the regulation of AMPs expression by TSLP.

**Methods:** In this study, the relationship between thymic stromal lymphopoietin (TSLP) and AMPs, and the involved molecular pathway was defined in normal human epidermal keratinocytes (NHEKs) and human skin equivalent model (HSEM).

**Results:** Stimulation of NHEKs with TSLP decreased both mRNA and protein levels of S100A7 and human beta defensin (hBD) 2 in a dose-dependent manner, and the regulation was JAK2/STAT3-dependent. TSLP decreased the AMPs expression, even in the presence of IL-17, which is their strong inducer. STAT3 directly regulated the S100A7 and hBD2 promoters in NHEKs. Immunohistochemically, lesional atopic skin stained more intensely with phospho-STAT3 compared to healthy control. Our results reveal that upregulated TSLP may contribute to the deficiency of AMPs in AD, including S100A7 and hBD2, by JAK2/STAT3-dependent mechanism, and that STAT3/Sin3A might directly control the transcriptional activity of the AMP promoters in NHEKs.

**Conclusion:** Taken together, a novel role of the JAK2/STAT3 signaling pathway in TSLP-mediated immune response in NHEKs might give us clues to understanding the pathological signal transductions in AD.
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1980 A.B. University of Chicago, Chicago, IL
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1997 Dermatology Foundation Award
1998 American Skin Association Research Development Award
2006 William Montagna Award, Society of Investigative Dermatology
2007 CE.R.I.E.S. Dermatology Research Award
2009 Stiefel Award, Dermatology Foundation
2009 Oclasson Lectureship, Pacific Dermatology Association
2009 Mertz award and Stiftungsprofessur Goethe University Frankfurt
2011 Presidential Citation, American Academy of Dermatology
2011 Rene’ Touraine Award, European Society of Dermatological Research
2012 Marion B. Sulzberger Award, American Academy of Dermatology
2013 Arthur Rook Oration, British Association of Dermatologists
2014  Dohi Memorial Lectureship, Japanese Dermatology Society
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**Professional Societies:**

Society for Investigative Dermatology
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The anti-microbial and pro-microbial parts of skin innate immunity

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A revolution in understanding of skin immunity has taken place with recognition that epithelial and other cell types, not only bone marrow derived cells, play critical roles in immunity. Antimicrobial peptides (AMPs) are produced by keratinocytes, leukocytes and dermal adipocytes to provide essential elements of host defense. Dysregulation of some AMPs may drive the pathogenesis of diseases such atopic dermatitis (too little AMP production) and Rosacea and Psoriasis (too much AMP production). The mechanism through which the cathelicidin AMP LL37 enables keratinocyte cytokine and growth factor production will be discussed. Furthermore, it became obvious that human AMPs are not indiscriminate antibiotics but rather have evolved to permit a defined community of microbes to exist within us. High-throughput functional analysis of the action of this skin microbiome has revealed surprising functions and previously unknown molecules that are critical components of human epithelial immunity. Animal and human data will be presented to show how AMPs made by the microbiome limit the growth of pathogens and influence epithelial growth. Therefore, we now recognize that specific members of the skin microbiome provide the first layer of skin immunity. We propose that the skin immune system is the product of coordinated interactions of cells in a superorganism. Recognizing this dictates that we respect both human and microbial elements of immunity when treating infections, cancers and inflammatory diseases.
The 27th Annual Meeting of the Korean Society for Investigative Dermatology

KSID Award Lecture
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2002-2005 Director, Medical Science Research Affairs, Yonsei University
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2003-2007 President, Korean Atopic Dermatitis Association
2006-2009 Chairman of Scientific Committee, Organizing Committee for 18th Asian Dermatological Congress, Seoul
2007-2011 Chairman, Department of Dermatology, Yonsei University College of Medicine
2007-2011 Director, Cutaneous Biology Research Institute, Yonsei University College of Medicine
2009-2011 Chairman, Board of Directors, Korean Dermatological Association
2009-2011 Organizing Committee Member, 22nd World Congress of Dermatology, Seoul
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2013-2014  President, Korean Society for Investigative Dermatology
2014-2016  Director, Institute for Immunology and Immunological disease, Yonsei University College of Medicine
2014-present Advisory Committee Member, Seoul Atopy and Asthma Information Center
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Awards:
1989  Yuhan SP Fellowship, Korean Dermatological Association
1990  Fogarty International Research Fellowship, NIH, U.S.A.
1990  American Society for Dermatologic Surgery Research Grant, U.S.A.
1998  Dong-A Scientific Prize, Korean Dermatological Association
1999  1st Wu Ahm Award, Korean Society for Investigative Dermatology
2003  Scientific Prize, Korean Society for Investigative Dermatology
2003  Prize for Excellent Professor for Research, Yonsei University
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Professional societies:
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Allergen specific immunotherapy in atopic dermatitis

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Atopic dermatitis (AD) is a chronic inflammatory skin disease with intractable pruritus. Allergen can penetrates through damaged skin barrier and bind an epidermal dendritic cell and activate Th2 polarization. Therefore, it is important for extrinsic AD patients to avoid possible exacerbating factors. And one of the most frequently noted allergens for AD exacerbation is a house dust mite (HDM). Allergen specific immunotherapy (SIT) using house dust mite (HDM) extracts has been performed mainly with patients of asthma and allergic rhinitis. Randomized controlled trials of SIT in AD revealed significant improvement of clinical symptoms and also, positive result was shown by a following meta-analysis study of these trials. SIT is the only disease-specific treatment modality that suppresses allergic responses for a long-period of time. SIT aims to induce allergen-specific tolerance otherwise known as allergen vaccination through acquiring immune tolerance with induction of allergen-specific regulatory T cells (Tregs). We assessed the clinical efficacy of SIT in AD patients who were treated with SIT for at least 3 years. Improvement after SIT therapy was observed in 88.4% patients. Among these patients, 63.5% achieved excellent, near-complete or complete clinical remission. We confirmed the usefulness of long-term HDM SIT as a disease-modifying therapy for AD.

Although subcutaneous immunotherapy (SCIT) is a clinically effective treatment in AD, there was no mouse model to understand the mechanism of HDM immunotherapy in AD. We establish a mouse model of SCIT in HDM induced AD. Female NC/Nga mice were treated with Dermatophagoides farinae (D. farinae) body extract ointment for 4 weeks to induce AD-like skin lesion. Then we separated the mice into 2 groups, control group (induction of AD only) and immunotherapy (IT) group. Both groups were continuously treated with D.farinae body extract ointment for 4 weeks, however, only immunotherapy (IT) group was treated with 8 injections of D.farinae (100 μg; subcutaneous) twice a week along with D.farinae body extract ointment treatment. We observed that AD-like skin lesions of IT group were milder than control group. In mice sera, the level of total IgE was decreased in IT group than control group at 3 weeks from the beginning of immunotherapy. The level of D.farinae-specific IgE was also up-regulated in control group compared to IT group. Increased level of IL-10 in IT group was observed at 2 weeks from the beginning of immunotherapy than control group. In mice splenocyte, expression of IL-4 in CD4+ T cells was decreased in IT group than control group using flow cytometry. Population of Foxp3 in CD4+CD25+ T cells was also increased in IT group than control group, and expression of IL-10 in CD4+CD25+Foxp3+ regulatory T cells and NK cells was increased in IT group. Lastly, IT group
made a strong contrast with control group at the relative mRNA levels of IL-10, IL-17 and IFN-gamma. Using this model, it will be possible to find novel way to potentiate the effects of allergen immunotherapy.

SIT can be divided into two major groups depending on the route of administration: sublingual (SLIT) and SCIT methods. However, frequent visit over 3 years results to the low compliance and the effects of SLIT in AD is controversial. Therefore we develop HDM allergen loaded microneedle patch and to confirm the stability and allergenicity of house dust mite allergen in the hyaluronic acid (HA) microneedle. Also, safety of the allergen loaded microneedle was investigated in mouse model. Protein concentrations were maintained in *D. farinae*-loaded HA microneedle patches. Inhibition ELISA analysis revealed that there was no decrease of allergenicity after loading *D. farinae* into the HA microneedle patch. Also, 4-week application of *D. farinae*-loaded HA microneedle patches does not showed any erythema, bleeding, or infection. However, 4-week application of high dose allergen-loaded patches (10 μg/patch) increased the total IgE level. We found that the allergenicity of *D. farinae* is maintained after loaded into the HA microneedle patches. There were no adverse effects, such as bleeding or infection, and high dose allergen-loaded HA microneedle patches were able to sensitize BALB/c mice. Therefore, we suggest that *D. farinae*-loaded HA microneedle patches successfully delivered allergen into the skin and this patched might be useful in the diagnosis and the treatment of the allergic diseases.
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Implication of genetic mutations in Korean melanoma patients

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Recent efforts in genomic research have enabled characterization of molecular mechanisms underlying many types of cancers, ushering novel approaches for diagnosis and therapeutics. Melanoma arise through progressive accumulation of genetic and epigenetic alterations that disrupt homeostatic pathways, resulting in uncontrolled tumor cell proliferation followed by invasion and lymphatic or hematogenous dissemination of the tumor cells to distant sites. Melanoma in Koreans differs to Caucasian in many aspects. The most common type of melanoma in Koreans is acral melanoma which consists up to 50%. With this difference, the oncogene mutation status is significantly different. The incidence of BRAF and NRAS mutation in Korean melanoma patients is much lower compared to Caucasian patients. This could be explained by the different proportions of common histologic subtypes of melanoma between the two races. We also found that UV-induced TERT promoter mutation frequencies vary depending on melanoma subtype. However, we showed that the negative impact of PTEN promoter methylation on survival is apparently preserved between Caucasian and Korean melanoma cohorts. This effect on survival also suggests that a core biologic mechanism may by universal. In a genetic aspect, acral melanoma is still not fully understood. Therefore, additional studies are warranted to find out the fundamental genetic drivers of acral melanoma.
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The Korean Atopic Dermatitis Association, Director of planning
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TRPV channels and post-burn pruritus

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Background: Post-burn pruritus is a common distressing sequela of burn wounds. Empirical antipruritic treatment often fails to have a satisfactory outcome because the mechanism has not been fully elucidated.

Objective: The aim of this study was to evaluate the manifestation of transient receptor potential (TRP) channels and other related receptors in post-burn pruritus.

Methods: Sixty-five burn patients with \( n = 40 \) or without \( n = 25 \) pruritus were investigated, including skin biopsies. Keratinocytes and fibroblasts from those samples were separated. Immunohistochemical staining for TRPV3 and TRPA1; and immunofluorescence staining for TSLP, TSLPR, loricrin, involucrin, \( \alpha \)-SMA, and TGF-\( \beta \), were performed on samples of burn scars and normal skin. Real-time PCR and western blotting of TRPV3, TRPA1, PAR2, NK1R, TSLP, and TSLPR were done. We also measured intracellular \( \text{Ca}^{2+} \) levels in keratinocytes from scars with or without pruritus, following TRPV3 activation and blocking, and measured the effects of PAR2 agonist on TRPV3 function. Expressions of TSLP after TRPV3 activation in keratinocytes were measured by western blotting and real-time PCR.

Results: In immunohistochemical and immunofluorescence staining, TRPV3, TSLP, and TSLPR stained more intensely the epidermis of the burn scars of post-burn-pruritus patients, than that of non-pruritic-burn patients. Real time-PCR showed that mRNA of TRPV3 and TSLP were significantly more abundant in keratinocytes from pruritic burn scars than in keratinocytes from non-pruritic burn scars. In addition, mRNA and protein levels of PAR2, NK1R, TSLP, and TSLPR were also significantly increased in pruritic burn scars. With TRPV3 activation, intracellular \( \text{Ca}^{2+} \) concentrations were more significantly increased in keratinocytes from pruritic burn scars than in those from non-pruritic ones. In keratinocytes from pruritic burn scars, PAR2 activation markedly potentiated opening of TRPV3 channels. TRPV3 activation itself resulted in little increase of \( \text{Ca}^{2+} \) influx with PAR2 inhibition in keratinocytes. In keratinocytes from all samples, PLC-\( \beta \), PKA, PKCs, and PKD inhibitor markedly reduced intracellular \( \text{Ca}^{2+} \) level by TRPV3 activation, as well as by PAR2 activation. TRPV3 activation also increased mRNA and protein expression of TSLP in keratinocytes.

Conclusions: In conclusion, we confirmed that TRPV3 of keratinocytes and PAR2, NK1R, TSLP, and TSLPR were highly expressed in pruritic burn scars. In addition, it seemed that PAR2 sensitized TRPV3 channels with PKA, PKC, PKD signaling pathways. It also seemed that TRPV3 activation induced TSLP expression.
Session II.
Inflammation, Inflammatory Skin Disorders
CURRICULUM VITAE

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Research fellow, Laboratory of Immunology, Aichi Cancer Center
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1988, 1987 Summer student, Laboratory of Biochemistry,
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Session II. Inflammation, Inflammatory Skin Disorders

Editorial Board of Journal:
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- 2015.1 Journal of Investigative Dermatology (JID) (Associate editor)
- 2008.5-2013.5 Journal of Dermatological Science (JDS) (Chief-in-editor)
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- 2011.2 Psoriasis: Targets and Therapy (Honorary editorial board)
- 2008.12. The Kaohsiung Journal of Medical Sciences (Editorial Board member)
- 2008.8 The Open Microbiology Journal (Editorial board member)
- 2007.4 The Open Dermatology Journal (Editorial board member)
- 2005-2007 Journal of Dermatological Science (Section editor)
- 2002 Der Hautarzt (Editorial international advisory board)
- 2000-2004 Photodermatology, Photomedicine, Photoimmunology

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Societies:
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- Society of Investigative Dermatology
- Japanese Society of Immunology
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Induction of regulatory T cells is a principal mechanism for phototherapy

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Natural sunlight has beneficial effects for various skin conditions and immunoregulatory functions. Phototherapy utilizes the beneficial effects and immunoregulatory functions of natural sunlight. Phototherapy is used for refractory skin disease when topical steroid treatment is not effective. Ultraviolet light (UV) phototherapy using narrowband UVA (311–313 nm) is a well-established treatment for refractory skin disease, such as psoriasis. UV phototherapy has two primary modes of action: apoptosis induction and immune suppression. Narrowband UVB depletes pathogenic T cells by inducing apoptosis and induces regulatory T cells. UVB, psoralen and UVA (PUVA), and UVA-1 (340-400 nm) are useful treatments of refractory skin diseases, and can be used in conjunction with topical steroids. Selective wavelength phototherapies are used to minimize the carcinogenic risks of UV exposure. UVA-1 effectively penetrates the dermal layers, and is thus superior to UVB, which is mainly absorbed by the epidermis. Excimer light (308 nm) therapy effectively targets affected skin without undue exposure of other areas and increases the levels of T regulatory cells. Fewer treatments and a lower cumulative UVB dose are other advantages of excimer light; the greater carcinogenic risk is ameliorated by the reduced number of treatments needed.

We previously evaluated the effects of bath-PUVA therapy on Th17/Treg balance in peripheral blood obtained from patients with psoriasis. Bath-PUVA therapy significantly reduces the number of Th17 cells. Treg function is significantly increased and Treg function is restored to almost normal levels. The Treg Functional Ratio is inversely correlated with the Psoriasis Area and Severity Index score (r= -0.407 p=0.084). These findings confirm that Treg are dysfunctional in psoriasis patients, and that bath-PUVA therapy restores Treg function in most patients. Furthermore, activated Treg (aTreg) are significantly increased in the early sessions of bath-PUVA therapy and later diminished afterward. The psoriasis lesions improved concomitantly with the increase in aTreg. Bath-PUVA therapy resolves the Th17 and Treg imbalance in patients with psoriasis and induces aTreg. Treg are induced in exposed skin by the clustering a certain dendritic population.

Based on these mechanism analyses, phototherapy effects have led to several improvements in the design and protocols, providing several options to patients with skin disease.
CURRICULUM VITAE

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Education:
1990-1996  M.D., Yonsei University College of Medicine, Seoul, Korea
1996-2001  Ph.D. (in Microbiology & Immunology),
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Career:
1999-2002  Medical Scientist, Department of Microbiology,
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2002-2007  Research Fellow, Immunology Section, Liver Diseases Branch, National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), National Institutes of Health (NIH), Bethesda, MD, USA
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2007-2013  Assistant Professor, Graduate School of Medical Science and Engineering, KAIST, Daejeon, Korea
2013-present Associate Professor, Graduate School of Medical Science and Engineering, KAIST, Daejeon, Korea

Academic Activities:
2011-2012  Chair, Vaccine Interest Group, The Korean Association of Immunologists, Korea
2014-present Chair, Academic Committee, The Korean Vaccine Society, Korea
2010-present Editorial Board, Frontiers in Microbiology
2011-present Editorial Board, PLoS One
2012-present Editorial Board, Clinical and Experimental Medicine
2013-present Associate Editor, Immune Network
2016-present Associate Editor, Clinical and Experimental Vaccine Research
Immunological Methods

**Society Membership:**

- The Korean Association of Immunologists
- The Korean Society of Virology
- The Korean Association for the Study of the Liver
- The Korean Society for Biochemistry and Molecular Biology
- The Korean Society for Molecular and Cellular Biology
- The American Association of Immunologists
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PD-1 and PD-L1 in psoriatic inflammation

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**Background:** Psoriasis is one of the most common chronic inflammatory diseases in the skin. Recently, IL-17-producing T cells have been shown to play a critical role in psoriatic inflammation. Programmed cell death-1 (PD-1) is a co-inhibitory receptor expressed on T cells in various chronic inflammatory diseases; however, the expression and function of PD-1 during psoriatic inflammation have not previously been characterized.

**Objective:** We examined PD-1 expression on IL-17A-producing T cells from Imiquimod (IMQ)-treated mice and psoriasis patients. Additionally, we investigated the therapeutic effect of recombinant programmed cell death ligand-1 (PD-L1) protein on IMQ-induced psoriatic inflammation.

**Methods:** PD-1 expression on IL-17A-producing γδ T cells from IMQ-treated mice was examined using multi-color flow cytometric analysis. In the psoriatic skin of patients, PD-1 and IL-17A expression was analyzed using immunofluorescence. The therapeutic effect of PD-L1-Fc fusion protein (PD-L1-Fc) was assessed in IMQ-treated mice ex vivo and in vivo.

**Results:** During IMQ-induced psoriatic inflammation, PD-1 is overexpressed on CD27-Vγ1γδ T cells. Further, PD-1 expression on IL-17A⁺ T cells was confirmed in psoriatic skin tissues from patients and IMQ-treated mice. In the CD27-Vγ1γδ T cell population, Vγ4⁺γδ T cells with Vγ6 mRNA expression showed a high level of PD-1 expression. Further, these PD-1hiVγ4⁺ (Vγ6⁺) γδ T cells were specialized for anti-CD3-induced IL-17A production, which was inhibited by PD-L1-Fc protein treatment. In IMQ-treated mice, PD-L1-Fc protein reduced psoriatic inflammation when given alone and enhanced the therapeutic effect of anti-p40 when given in combination.

**Conclusion:** PD-1 is overexpressed in IL-17A-producing T cells in both IMQ-treated mice and psoriasis patients. Moreover, recombinant PD-L1-Fc protein alleviates psoriatic inflammation in IMQ-treated mice.
CURRICULUM VITAE

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Education:
1990-1997 School of Medicine, Chung Shan Medical University (M.D.)
2003-2008 Taiwan International Graduate Program, Molecular Medicine, Academia Sinica and National Yang Ming University (Ph.D.)

Fulltime Employment:
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2009-2010 Assistant Professor, school of medicine, Chang Gung University, Taiwan
2009-present Physician scientist, Chang Gung Memorial Hospital, Taiwan
2011-present Associate Professor, Chang Gung Memorial Hospital& school of medicine, Chang Gung University, Taiwan
2011-present Director of drug hypersensitivity clinical and research center, Chang Gung Memorial Hospital, Taiwan
2015-present Professor, Chang Gung Memorial Hospital& school of medicine, Chang Gung University, Taiwan
2015-present Director of department of dermatology, Chang Gung Memorial Hospital, Taipei, Taiwan
2015-present Director of Whole-Genome Research Core Laboratory of Human Diseases

Honors:
2005 Chen-yuan Lee Young Scientist Medical Research Award
2006 The 2006 Top 10 Rising Stars in Taiwan
2006 Prize of Taiwan Skin Education Research & Development Foundation
2009 Chang Gung Memorial Hospital President Wang’s Golden award
2009 The 7th Y.Z. Hsu Scientific Paper award
2009 Young Investigator Award (Ta-You Wu Memorial Award), Taiwan
2009 The 47th Ten Outstanding Young Persons, Taiwan
2011 The International League of Dermatological Societies (ILDS) Young Dermatologist
International Achievement Award.
2015  Outstanding Research Award of Ministry of Science and Technology, Taiwan

**Focus of Interest:**
Adverse drug reactions and drug hypersensitivity
Pharmacogenomics
Clinical trials, Epidemiology, Immunology
Molecular insights into severe cutaneous adverse reactions

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The clinical presentations of drug hypersensitivity may vary from mild maculopapular exanthema to severe, life-threatening severe cutaneous adverse drug reactions (SCAR), including Stevens-Johnson syndrome (SJS) and toxic epidermal necrosis (TEN) and drug reaction with eosinophilia and systemic symptoms (DRESS). In the ensuing decade, a number of important pharmacogenomic associations with SCAR have been found in different populations. Some specific HLA alleles are strongly associated with drug-induced hypersensitivity reactions, such as HLA-B*15:02 for carbamazepine-induced SJS/TEN, HLA-B*58:01 for allopurinol hypersensitivity, HLA-B*57:01 for abacvir hypersensitivity and HLA-B*13:01 for dapsone hypersensitivity. In contrast, there are many other drugs induced hypersensitivity reactions showing not a highly HLA restricted manner.

Preferential T cell receptor (TCR) clonotypes are also identified as a crucial role for SCARs. Activation of specific T cells expressing preferential TCR which interacts with drug-specific HLA allele and culprit drugs is involved in the immune mechanism.

Though the HLA and TCR play critical roles in drug-induced SCAR, evidences also show drug metabolism may also contribute to the pathogenesis of SCAR. We recently identified CYP2C variants, including CYP2C9*3, known to reduce CYP2C9 enzymatic activity and decrease drug clearance of phenytoin, as an important genetic factor associated with phenytoin-induced SCAR. Furthermore, in allopurinol-SCAR, renal impairment decreased the excretion of plasma oxypurinol (a metabolite of allopurinol) was found to be correlated with the clinical severity and prognosis. A further study showed coexistence of HLA-B*58:01 and renal impairment increased the risk and predictive accuracy of allopurinol hypersensitivity.
CURRICULUM VITAE

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Education and Training:
1989   Seoul National University College of Medicine
1990-1994  Residency, Department of Pediatrics, Seoul National University Children’s Hospital
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1997-1999  Clinical Fellow, Division of Pulmonology and Allergy, Department of Pediatrics, Samsung Medical Center, Seoul, Korea
1999-2000  Research Fellow, Division of Allergy, National Children’s Medical Research Center, Tokyo, Japan
2002-2005  Seoul National University, Post-graduate Doctoral Course

Current and Past Professional Positions:
2000-2005  Assistant Professor, Department of Pediatrics, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea
2005-2006  Visiting Scholar in Division of Pediatric Allergy and Immunology and the Jaffe Food Allergy Institute, Mount Sinai School of Medicine, New York, USA
2005-2011  Associate Professor, Department of Pediatrics, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea
2011-  Professor, Department of Pediatrics, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea
2011-  Director, Environmental Health Center for Atopic Diseases, Samsung Medical Center, Seoul, Korea

Society Memberships:
Korean Academy of Pediatric Allergy and Respiratory Disease (KAPARD)
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Korean Society of Pediatrics
The role of air pollution on atopic dermatitis in children

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The pathogenesis of atopic dermatitis (AD) is attributed to both genetic and environmental factors. With regard to a genetic basis, the genes encoding filagrin (FLG) has been most consistently replicated in more than 20 different studies. The loss-of-function mutation in FLG gene has been frequently reported in European populations, and the other responsible genes remain to be elucidated. In terms of environmental factors, contact with endotoxin in utero or during the first year of life, \textit{i.e.} hygiene hypothesis, has a protective effect on the development of asthma, hay fever and allergic sensitization, although this effect is not evident in AD. In contrast, altered distribution in gut flora during infancy seems to influence the development of AD. Moreover, recent evidences suggest that air pollution might cause AD in a susceptible individual. For example, the exposure to a variety of indoor and outdoor air pollutants such as environmental tobacco smoke, volatile organic compounds, nitrogen dioxide, and particulate matter has been reported to develop AD.

In a prospective study to evaluate the clinical effects of outdoor air pollution on skin symptoms in children with AD, 22 patients were followed on a regular basis for 18 months. Analysis with a generalized linear mixed model revealed that a 1-ppb increase in benzene concentration was associated with a 27.38% increase in AD symptoms. A 1-ppb elevation in total VOC concentration was related to a 25.86% increase in AD symptoms on the following day. Although the impact was small, an increased PM\(_{10}\) concentration by 1 \(\mu g/m^3\) was significantly associated with a 0.44% increase in AD symptoms on the following day. Indoor air pollutants also increase the risk of AD aggravation in children and toluene in the indoor environment might act as an aggravating factor.

In a cross-sectional study, a water-damaged home was a significant risk factor of moderate to severe AD (adjusted odds ratio 14.52, 95% confidence interval 1.75-121.13, \(P=.0025\)). A causal relationship between air pollution and aggravation of AD symptoms was directly investigated by a provocation test. Exposure to formaldehyde and NO\(_2\) increased transepidermal water loss (TEWL) in patients with AD, while exposure to room air did not. In another study of adult patients with AD, the exposure to VOCs increased TEWL and dermal blood flow was enhanced by prior exposure to Der p 1. In children with AD, exposure to airborne formaldehyde increased TEWL and skin pH.

There is growing evidence that air pollution might act as an important environmental risk factor in development or aggravation of AD. However, a limited number of studies have been done and many issues still remain to be elucidated. Further research is needed to examine the role of air pollutants on AD. It will help to expand our understanding and establish a better strategy for primary, secondary, and tertiary prevention of AD.
Session III.

Innate & Adaptive Immunity
CURRICULUM VITAE

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1991.2. M.D., College of Medicine, Chungnam National University (CNU), Korea
1996.2. Ph.D., Department of Microbiology, College of Medicine, CNU, Korea

Current and Past Professional Positions:
1997-2003 Full-time instructor (1997-1999) and Assistant Professor (1999-2003), Dept. of Microbiology, College of Medicine, CNU
2002 Visiting Scientist, Tokyo Medical and Dental University
2003-2004 Research Associate, Imperial College London, U. K.
2004-2008 Associate Professor, Dept. of Microbiology, College of Medicine, CNU
2007-2016 Director, Medical Research Center (ISNRC), CNU
2008-present Professor, Dept. of Microbiology, College of Medicine, CNU

Awards:
2006 Research Award for Young Medical Scientist of Societies for Korean Basic Medical Sciences
2008 Eui-Dang Research Award, Korean Association of Medical Doctors
2008 Award of Excellent Papers in Science and Technology, Korean Association of Scientists
2010 Award of KUN-IL, Korean Association of Woman Medical Doctors
2012 Macrogen Woman Scientist Award, KSBMB
2012 Pfeizer’s Research Award for Basic Medicine
2015 Wunsch Medical Award

Society Memberships:
The Korean Society for Microbiology (Board member)
The Korean Society for Microbiology and Biotechnology
The Korean Association of Immunologists (Board member)
Korean Society for Molecular and Cellular Biology (Board member)
Korean Society for Biochemistry and Molecular Biology (Board member)
American Association of Immunologists
Orphan nuclear receptors and regulation of innate immunity

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Background: Orphan nuclear receptors (NRs) are a subset of NR superfamily which ligands and functions have not been fully characterized. The atypical orphan NR, small heterodimer partner (SHP, NR0B2), is a well-known corepressor of numerous other NRs. Estrogen-related receptor \( \alpha \) (ERR\( \alpha \); NR3B1) is the first identified orphan NR that plays an important role in regulation of energy metabolism and mitochondrial biogenesis. Emerging evidence has accumulated that several orphan NRs play critical roles in the regulation of innate immunity to prevent harmful inflammatory responses in the host. However, functional studies regarding the specific roles of SHP and ERR\( \alpha \) in the regulation of the immune and inflammatory responses are in their infancy. Objective: We aimed to characterize the roles of SHP and ERR\( \alpha \) in the regulation of NLRP3 inflammasome activation and toll-like receptor (TLR)-induced inflammatory responses, respectively.

Methods: A variety of cell and molecular biological methods, as well as in vivo experiments, were performed to characterize the functions of SHP and ERR\( \alpha \), and to elucidate the mechanisms by which these orphan NRs regulate innate immune responses.

Results: SHP was found to inhibit NLRP3 inflammasome activation through an interaction with NLRP3 and mediating its translocation into mitochondria. SHP deficiency resulted in an increased secretion of IL-1\( \beta \) and IL-18, and excessive pathologic responses typically observed in mouse models of kidney tubular necrosis, colitis, and peritoneal gout. In addition, ERR\( \alpha \) was a novel regulator of the TLR-induced inflammatory response, with the unique capacity to modulate Tnfaip3/A20 transcriptional induction and p65 acetylation through metabolic reprogramming via enhancement of mitochondrial biogenesis and function.

Conclusion: Both SHP and ERR\( \alpha \) were suggested new regulators in innate and inflammatory responses. Unveiling the new and existing functions of orphan NRs could accelerate develop and improve novel strategies against human inflammatory diseases.
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Education:
1997-2001   B.S in Biology, Ewha Womans University
2001-2003   M.S in Genetics, Seoul National University
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Professional Experiences:
2006-2011   Research fellow, Boston Children’s Hospital/Harvard Medical School
2011-2013   Research associate, Boston Children’s Hospital/Harvard Medical School
2014-present Associate professor, Seoul National University College of Medicine, Department of Biomedical science and Medical science

Awards:
2014       L’Oréal-UNESCO Awards for Women in Science
2010       Houseofficer development award (Children’s Hospital Boston)
Atopic dermatitis (AD) is generally characterized as a Th2-mediated inflammatory skin disease. During past several years, it has become clear that innate lymphoid cells (ILCs) play important roles in tissue homeostasis and inflammation. ILCs lack antigen-specific receptors which expressed by T and B cells, but ILCs are activated by innate cytokines. In the skin, ILCs can sense danger signals derived from keratinocyte and/or skin epithelial cells. Group 2 Innate lymphoid cells(ILC2s) is known to induce AD by producing type 2 cytokine. However, several recent studies have been reported that IL-17A is also increased in patients with AD as well as murine AD model. To address the roles of ILC3s in the development of AD, we used house dust mite(HDM)-induced AD models using NC/Nga mice. Interestingly, both type 2 and type 3 cytokines were increased in the skin-draining lymph nodes as well as skins of AD mice compared with control mice. Moreover, neutralizing IL-17A delayed the development of AD and adoptive transfer of ILC3s into the recipient NC/Nga mice accelerated the development of AD compared with the PBS-injected mice. Taken together, our results suggest that IL-17A producing ILC3s play critical roles in the pathogenesis of AD by orchestrating the production of both type 2 and type 3 cytokines.

Keyword: Innate lymphoid cell, ILC3, Atopic dermatitis, NC/Nga, House dust mite
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Education and Training:
- 2002: M.D., Yonsei University College of Medicine, Seoul, Korea
- 2002-2003: Intern, Severance Hospital, Yonsei University College of Medicine, Seoul, Korea
- 2003-2007: Resident, Severance Hospital, Yonsei University College of Medicine, Seoul, Korea
- 2004-2006: M.S., Graduate School of Medicine, Yonsei University, Seoul, Korea
- 2010-2015: Ph.D., Graduate School of Medicine, Yonsei University, Seoul, Korea

Current and Past Professional Positions:
- 2007-2009: Chief Dermatologist, Department of Dermatology, The Armed Forces Gangneung Hospital, Gangneung, Korea
- 2009-2010: Chief Dermatologist, Department of Dermatology, The Armed Forces Byukjae Hospital, Goyang, Korea
- 2010-2011: Instructor, Department of Dermatology, Yonsei University College of Medicine, Seoul, Korea
- 2011-2012: Clinical Research Assistant Professor, Department of Dermatology, Yonsei University College of Medicine, Seoul, Korea
- 2012-2016: Postdoctoral Research Fellow, Department of Dermatology, Brigham and Women’s hospital, Harvard Medical School, Boston, MA, USA
- 2014-2016: Clinical Research Assistant Professor, Department of Dermatology, Yonsei University College of Medicine, Seoul, Korea
- 2016-present: Assistant Professor, Department of Dermatology, Yonsei University College of Medicine, Seoul, Korea
Awards:
1999  The Se-Wha Award, Department of Biochemistry, Yonsei University College of Medicine
2002  The Severance Award, MD degree awarded with highest honor, Yonsei University College of Medicine
2002  32th GSK Award, Highest honor in the Korean Medical Examination
2010  Graduate School of Yonsei University Paper/Thesis Award, Yonsei University
2011  3rd LG Young Investigator Award, the Korean Medical Association
2012  1st “Soodang” Young Research Scholars’ Award, Yonsei University College of Medicine
2014  Kligman Travel Fellowship Award, 73th Annual SID meeting

Society Memberships:
The Korean Medical Association
The Korean Association of Dermatology
The Korean Society for Investigative Dermatology
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**Skin resident T cells in mouse and human**

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Skin resident T cells are virtually entirely memory and express both cutaneous lymphocyte antigen (CLA) and CCR4 (skin homing markers). The discovery that there are twice as many T cells in skin ($2.0 \times 10^{10}$) as are in blood ($1.1 \times 10^{10}$), and the fact that they are all skin homing memory T cells is striking and leads to the literally paradigm shifting into tissue resident memory T (T\textsubscript{RM}) cells. Such T\textsubscript{RM} cells have been studied most carefully in murine models of viral infection, which have focused on CD8+ T cells that produce IFNg on activation. *Candida albicans* is a dimorphic fungus to which humans are exposed early in life; by adulthood, it is part of the mycobiome of skin and other tissues. We adapted a highly reproducible model of skin infection with *C. albicans*. Prior to *C. albicans* infection, IL-17 producing cells in murine skin were composed entirely of dermal gd T cells. At 30 days after *C. albicans* infection, however, CD4 ab T cells become the predominant producers of IL-17, replacing dermal gd T cells. By intravital microscopy, these cells resided in the papillary dermis in previously infected mice and were sessile. These TRM made frequent contacts with CD11c+ dermal dendritic cells (DCs). Next, we confirmed that normal human skin CD4 T cells also produced significant IL-17 when incubated with heat-killed *C. albicans*. These studies demonstrate that *C. albicans* infection of skin preferentially generates CD4+ IL-17 producing T\textsubscript{RM}, which mediate durable protective immunity.
CURRICULUM VITAE

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Education and Training:
2003 M.D., Collage of Medicine, Seoul National University, Seoul, Korea
2003-2004 Internship in Seoul National University Hospital, Seoul, Korea
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2007 M.S. in Medical Science, Graduate School, College of Medicine, Seoul National
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2011 Ph. D. in Medical Science, Graduate School, College of Medicine, Seoul National
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Current and Past Professional Positions:
2008-2010 Fellowship in Department of Dermatology, Seoul National University Bundang
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CD1 signaling supports the survival and proliferation of epidermal and dermal T cells from human skin

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**Background:** The skin surface of a healthy adult contains approximately 10 billion nonrecirculating resident memory T cells (TRM), 10% in the epidermis and 90% in the dermis, in close proximity to CD1a-expressing Langerhans cells and CD1b-expressing dermal dendritic cells, respectively. Human CD1 molecules (CD1a,b,c,d) are specialized for the presentation of lipid antigens. CD1a, CD1b and CD1c are unique to humans and not expressed in mice. Human CD1-restricted T cells are incompletely characterized but a subset recognize mycobacterial antigens and a larger number are autoreactive and respond to self lipids. CD1a, CD1c and CD1d-autoreactive T cells are common in peripheral blood but CD1b-autoreactive T cells are rare and their function is unknown.

**Objective:** We aimed to characterize epidermal and dermal skin resident T cells and compare their reactivity to CD1a and CD1b molecules.

**Methods:** T cells from the epidermis and dermis of human skin were isolated and analyzed using Flow cytometry, TCR sequencing and stimulated with CD1a- and CD1b-transfected K652 cells to test CD1 reactivity.

**Results:** In the epidermis, 88% of the T cells were CD69 positive, a marker of resident memory T cells. In the dermis 79% of T cells were Trm. Epidermal T cells had potent effector functions but proliferated poorly and the potent effector function of epidermal T cells declined rapidly once these cells were removed from the skin microenvironment. In contrast, dermal T cells survived well in culture. Co-culture of epidermal T cells with CD1a transfected K562 cells significantly increased the survival of epidermal T cell. Ki-67 positivity of epidermal T cells was also significantly increased with CD1a stimulation. In dermal T cells, although CD1a stimulation also enhanced the survival, CD1b stimulation was more effective.

**Conclusion:** CD1 signaling supports the survival of skin resident T cells in vitro. Trm in epidermis are enriched for CD1a reactivity whereas Trm in dermis are enriched for CD1b reactivity.
The 27th Annual Meeting of the Korean Society for Investigative Dermatology

2017. 3. 25(Sat)
Free Communications (II)

(FC-10 ~ FC-18)
Langerhans cells (LCs) are skin-resident dendritic cells (DCs) that orchestrate skin immunity. CCCTC-binding factor (CTCF) is a highly conserved DNA-binding protein that regulates higher order chromatin organization and is involved in various gene regulation processes. A possible role for CTCF in DC/LC homeostasis and function is largely unclear yet. To this end, we generated DC- and LC-specific CTCF-ablated mice using a conditional gene deletion mouse system. DC-specific CTCF deletion led to a reduced pool of systemic DCs, with LCs most severely affected. Decreases in epidermal LC number were specifically associated with self-turnover defects. Interestingly, CTCF-deficient LCs demonstrated impaired migration out of the epidermis. Whole-transcriptome analyses revealed that genes that promoted cell adhesion were highly expressed, but CCR7 was down-regulated in CTCF-depleted LCs. Repeated epicutaneous sensitization to protein antigen was attenuated in LC-specific CTCF-deficient mice, indicating that molecular targeting of CTCF activity in LCs can be a useful therapeutic strategy to dampen cutaneous allergic sensitization. Our results show that CTCF positively regulates the homeostatic pool and the efficient emigration of LCs, which are required for modulating the functional immune network of the skin.
FC-11

The response to cyclophosphamide therapy is regulated by effector B cells in systemic sclerosis-associated interstitial lung disease

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Background: Only the cyclophosphamide (CYC) treatment was found to be effective in stabilizing or improving lung function of systemic sclerosis-associated interstitial lung disease (SSc-ILD) in randomized clinical trials, which contained abundant numbers of patients. However, there are not a few non-responders to the CYC treatment. We need to know the difference between the responders and the non-responders.

Objective: We hypothesized that cytokine-producing effector B cells determine the effectiveness of CYC treatment in SSc-ILD patients. In this study, we assessed the role of interleukin (IL)-10-producing regulatory B cells and IL-6-producing pathogenic B cells in the responders or non-responders of CYC treatment.

Methods: We cultured human lung endothelial cells in a microchannel. We loaded B cells from responders or non-responders to CYC treatment into the microchannel. After B cells which adhered to lung endothelial cells had produced cytokines, we measured IL-6 and IL-10 production by our original micro fluidic-ELISA system. Similarly, we assessed B cells from topoisomerase (topo) I and complete Freund’s adjuvant-induced SSc model mice.

Results: In SSc-ILD patients, the number of B cells adhering to lung endothelial cells significantly increased compared with healthy controls. In addition, the serum levels of anti-lung endothelial cell antibodies increased. The B cells adhering to lung endothelial cells from CYC-responders produced higher amount of IL-10 and lower amount of IL-6 than those from CYC-non-responders. In mouse study, topo I-specific B cells had higher numbers of B cells which can adhere to lung endothelial cells than non-specific conventional B cells. Topo I-specific B cells also showed higher production of IL-6 and lower production of IL-10 than conventional B cells.

Conclusion: These results suggested that the effectiveness of CYC treatment to SSc-ILD patients is associated with the cytokine profile of B cells which interact with lung endothelial cells.
The role of innate immunity in scrub typhus

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Background: Scrub typhus is caused by the obligate intracellular bacterium Orientia tsutsugamushi (OT), transmitted via the bite of infected mite vectors. It is an important travel-associated zoonosis returning from Asia. Severe complications in patients are mainly caused by cytokine storm; nevertheless, the molecular mechanism for the occurrence remains obscure.

Objective: We investigate the interactive regulation of cytokines and micro-RNA (miR) in human macrophages infected with OT.

Methods: Using low and high dose of OT to infect macrophage cell line and patients’ peripheral blood monocyte, and examining cytokines and miR.

Results: During low dose infection, macrophages produce high levels of IL-10 through extracellular signal-regulated kinase (ERK) activation, which inhibits proinflammatory cytokine production and facilitates pathogen replication. Increasing levels of pathogen results in reduced levels of IL-10, and macrophages begin to generate high levels of proinflammatory cytokines through NF-κB activation. However, during a high dose infection, macrophages produce high levels of miR-155 to slow the proinflammatory response. The ERK/IL-10 axis suppresses the NF-κB / TNFα axis via activation of signal transducer and activator of transcription 3 (STAT3). Both IL-10 and miR-155 inhibit the NF-κB signaling pathway. Furthermore, IL-10 is a potent inhibitor of miR-155. Patients susceptible to a cytokine storm, peripheral blood mononuclear cells showed significantly lower IL-10 and miR-155 responses to OT challenge.

Conclusion: IL-10 and miR-155 cross-regulate the proinflammatory cytokines in OT-infected macrophages to prevent cytokine storm. IL-10 and miR155 may serve as biomarkers to predict immune response of patients in acute phase, and imply the potential for immune-regulatory treatment.
**FC-13**

**Effects of AhR Ligands on PBMCs and CD4⁺T cells from patients with atopic dermatitis and psoriasis**

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**Background:** Aryl hydrocarbon receptor (AhR) is a ligand-activated transcription factor, important for xenobiotic metabolism, which binds to various endogenous and exogenous ligands in the skin. Aim: To evaluate the role of the AhR and its ligands in chronic inflammatory skin disease such as psoriasis (PS) and atopic dermatitis (AD).

**Methods:** PS and AD patients and healthy volunteers were recruited. The clinical severities were assessed. Blood samples were taken from the three groups for isolation of peripheral blood mononuclear cells (PBMCs) and CD4⁺T cells. Each sample of isolated PBMCs and CD4⁺T cells was treated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and 6-for-myldolo[3,2-b] carbasole (FICZ) at various concentrations and durations. Changes in each group were analyzed using a fluorescence-activated cell sorter (FACS), enzyme-linked immunosorbent assay, enzyme-linked immunospecific assay (ELISA), and real-time polymerase chain reaction (PCR). The activation of AhR ligands in PBMCs was confirmed with AhR-targeted specific small interference (si) RNA, suppressing the expression of AhR.

**Results:** In PBMCs, after treatment with TCDD and FICZ, the expression of AhR-related factors (AhR, Aryl hydrocarbon receptor nuclear translocator; ARNT, Cytochrome P450, family 1, member A1;CYP1A1), and IL-22 increased in PS patients and AD patients. Regarding the expression of IL-17, it increased in PS patients but remained stable in AD patients. In the CD4⁺T cells, compared to the control group, CCR10’ cells (marker for Th22 cells) and CD161⁺CCR10’ cells (marker for Th17 and Th22 cells) increased significantly in PS patients, and CCR10’ cells significantly increased in AD patients of the TCDD- treated group. Compared to the control group, after treatment with TCDD, the IL-4 level increased in CD4⁺ T cells of AD patients. Moreover, the levels of IFN-γ, IL-17, and IL-22 were significantly higher in PS patients, and the levels of IL-13, IL-17, and IL-22 were significantly higher in AD patients, compared to those of the healthy controls.

**Conclusions:** PBMCs of AD and PS patients presented higher expressed AhR and related factors and more interleukin (IL)-17 and IL-22. The CD4⁺T cells of AD and PS groups tended to show features of Th17 cells or Th22 cells. These results suggest that AD and PS patients may be affected by AhR ligands and that activation of AhR may contribute to the development of disease in AD and PS.
**FC-14**

**11β-Hydroxysteroid dehydrogenase 1 in the skin has a role in the occurrence of atopic dermatitis**

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**Background:** 11β-hydroxysteroid dehydrogenase 1 (11β-HSD1) is an enzyme that converts inactive cortisol into cortisol, an active form. It is expressed in various tissues including the skin, and its expression level is reported to be elevated with aging. Atopic dermatitis (AD) is a chronically relapsing, pruritic inflammatory skin disease, and its prevalence is the highest in infants, is relatively very low in the elderly.

**Objective:** To evaluate if 11β-HSD1 expression in the skin has a role not only in skin aging but also in the occurrence of AD.

**Methods:** First, we have compared three groups of mice; young (8 weeks old) mice irradiated with sham light for 8 weeks, young mice irradiated with ultra violet (UV) light for 8 weeks, and old (56 weeks old) mice irradiated with sham light for 8 weeks. Second, cortisol level in the stratum corneum (SC) was compared among normal young, normal aged, diabetes mellitus (DM) patient group, and AD group. Third, we have performed an experiment comparing the old (62 weeks old) and young (8 weeks old) mice in the occurrence of AD following repeated oxazolone (Ox) challenges. We have also used topical 11β-HSD1 inhibitor and vehicle in each aged and young group.

**Results:** Young mice group irradiated with UV light showed impaired skin permeability barrier function and increased 11β-HSD1 expression in the skin comparable with the old mice group. The SC cortisol level of normal young human group was significantly lower than AD and DM group. AD developed more rapidly and seriously in the young mouse group, and 11β-HSD1 inhibitor applied group after multiple Ox challenges.

**Conclusion:** 11β-HSD1 expression in the skin is increased in aged skin and may be responsible for the lower prevalence of AD in the elderly. Novel agents modulating the 11β-HSD1 activity could be used for AD treatment.
FC-15

Attempt to establish a hair follicle co-culture model using feeder-free human iPS cells

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Background: A co-culture model using hair follicle (HF) epithelial and mesenchymal cells provide a tool for the investigation of HF biology and drug discovery for HF disorders. However, limitation in human HF materials represents a major hurdle for preparation. Human induced pluripotent stem cells (hiPSCs)-derived cells can provide materials for the development of such model. However, the maintenance of hiPSCs usually requires feeders of which preparation is costly and laborious.

Objective: To establish a co-culture model mimicking HF using hiPSCs maintained under feeder-free condition.

Methods: hiPSCs maintained without feeders were induced into keratinocytes (KCs) and dermal papilla (DP) substituting cells using retinoic acid and BMP4 or mesenchymal stem cell medium and previously-established dermal papilla activation medium respectively. Resultant cell populations were assessed for KC or DP marker expression. Subsequently, two cell populations were combined and co-cultured. The expression levels of HF markers were compared to those of co-culture model adopting normal human KCs and DP cells.

Results: hiPSCs maintained without feeders were successfully induced into KC or DP-like cells as confirmed by their distinct morphology and high expression of KC (keratin 14 [KRT14], p63) or DP (ALPL, WIF1 and WNT5A) markers. When co-cultured, hiPSC-derived KCs combined with hiPSC-derived DP substitute cells expressed HF-related markers including LEF1, MSX2, TRPS1 and KRT17 at similar to higher levels compared to control KCs co-cultured with normal DP cells.

Conclusion: The study is still preliminary, however, these findings suggested that hiPSCs maintained under a feeder-free condition hold promise as an easily accessible and a useful cell source for the development of co-culture model reproducing at least some aspects of HF epithelial-mesenchymal interactions.
Background: High-mobility group box 1 (HMGB1) is a ubiquitous nuclear protein that is released from the nuclei of cells after tissue damage. HMGB1 can be a plausible candidate to explain the mechanism of trauma-induced hair growth.

Objective: To elucidate the effect of HMGB1 on hair growth and their action mechanism.

Methods: MTT assay was conducted to check the effect of HMGB1 on hDPCs proliferation. RT-PCR and Western blot analysis demonstrated expressions of PGE\textsubscript{2} metabolic enzymes in HMGB1-treated hDPCs. Hair growth was measured using ex-vivo hair organ culture. The Ki67 expression was measured by immunofluorescence.

Results: HMGB1 enhanced hair shaft elongation in ex vivo hair organ culture. We confirmed that the mRNA and protein expression levels of mPGES-1 was significantly increased after HMGB1 treatment on hDPCs. HMGB1 also stimulated PGE\textsubscript{2} production from hDPCs. HMGB1-induced PGE\textsubscript{2} production was abrogated with RAGE antagonist.

Conclusion: Our results suggest that HMGB1 plays a role in the promotion of hair growth via PGE\textsubscript{2} production from hDPCs. These mechanism can explain a paradoxical phenomenon, trauma induced hair growth and HGMB1 may serve as an additional therapeutic target for the treatment of alopecia.
FC-17

CD13, marker for onychofibroblasts within nail matrix onychodermis: Comparison of the nail unit with hair follicle

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Background: The nail is similar to the hair. In the past, we demonstrated the presence of special mesenchyme containing onychofibroblasts and named it onychodermis.

Objective: To further characterize the onychodermis by immunohistochemistry.

Methods: Immunohistochemistry for CD10 and CD13 was performed in the nail unit (polydactyly and adult cadaver samples) and nevus sebaceous samples.

Results: While CD10 was expressed in the mesenchyme below the nail matrix and nail bed, CD13 was expressed mainly in the mesenchyme containing onychofibroblasts below the nail matrix. CD10 was expressed only in the dermal sheath of terminal hair follicles, but it was expressed in the dermal sheath and follicular dermal papilla of primitive hair follicles of nevus sebaceous. CD13 was expressed in the dermal sheath as well as dermal papilla of terminal hair follicles. On the contrary, both were not detected in the dermis, except around blood vessels and eccrine structures or basement membranes of epithelial-dermal junctions.

Conclusion: The findings of CD13 immunohistochemistry provide another evidence of the similarity between the nail and hair. In addition, our results suggest that CD13 seems to be a marker for onychofibroblasts within nail matrix onychodermis, which may be a counterpart of follicular dermal papilla.
FC-18

3D in vivo pH imaging analysis of stratum corneum

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Background: Enhanced cutaneous sensitization by skin barrier dysfunction has been extensively investigated as an initial important step for the onset of allergic disorders. The regulation of pH is an important element in various biological functions. The pH of stratum corneum (SC) is considered to be acidic in general while its precise pH distribution and dynamic changes in different environments within SC layers remain to be clarified.

Objective: The purpose of this study is to produce a transgenic mouse as a tool to visualize in vivo pH in SC and dissect a role of pH in SC homeostasis.

Methods: We generated a ratiometric pH biosensor with pH sensitive fluorescent protein, VenusH148G, and pH insensitive protein, mCherry, expressed specifically in the uppermost layer of stratum granulosum (SG1) using knock-in construct of SASPase gene by modified CRISPR/Cas9 system.

Results: VenusH148G-mCherry protein was stably expressed and pH was able to be monitored in both SG1 layer and entire SC layers without losing signals in ear skin. Confocal microscopic analysis of living pH imaging demonstrated that SC has at least two distinct middle-acidic and upper-neutral layers with cross section view, rather than gradual pH changes across the layers. When ear skin was exposed to phosphate buffers with various pH (pH 5.4, 6.6 and 7.4) by topical application, the upper layers revealed dynamic changes of pH, while the middle layers was not affected by the applied buffer and kept its acidic pH. These findings indicated the dynamic and complex nature of SC in terms of pH regulation.

Conclusion: The 3D in vivo pH imaging mice will provide a valuable tool to dissect the homeostatic mechanisms of SC by pH regulation. Uncovering the physiological mechanisms of SC homeostasis will lead us to develop a novel body surface tactics to conquer allergic disorders.
Hot Posters (II)

(HP-11 ~ HP-20)
The effect of cilostazol on hair growth: A type of drug repositioning for the treatment of alopecia with the mechanism of vasodilatation

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**Background:** Cilostazol, a phosphodiesterase3 (PDE3) inhibitor, increases the intracellular cyclic adenosine monophosphate (cAMP) level in vascular smooth muscle cells causing vasodilation and is widely used for the supportive treatment of chronic peripheral vascular diseases. Topical application of cilostazol is reported to improve local blood flow in rabbit skin and enhance wound healing.

**Objective:** Herein, we introduce the promotive effect of cilostazol on hair growth for the first time.

**Methods:** To validate the effects of cilostazol on hair growth, we treated cilostazol to human dermal papilla (DP) cells and to outer root sheath (ORS) cells, and performed \textit{ex vivo} hair follicle organ culture. Also, we demonstrated the effect of cilostazol on C57BL/6 mice model.

**Results:** As a result, we confirmed that the mRNA levels of PDE3A and PDE3B were highly expressed in human DP cells, but almost absent in ORS cells. Cilostazol significantly enhanced the viability of DP cells and increased phosphorylated extracellular signal-regulated kinase (ERK) levels proven by western blot analysis. Additionally, cilostazol promoted hair shaft elongation with increased proliferation of matrix keratinocytes in hair follicle organ culture. Furthermore, cilostazol treatment accelerated the anagen hair induction when topically applied on 7-week-old C57BL/6 mice.

**Conclusion:** Our results show that cilostazol promoted hair growth and may serve as an alternative therapeutic target for the treatment of alopecia.
HP-12
Development of hair loss treatment using deoxycholic acid

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Background: There have been reports that the increase in nuclear β-catenin plays an important role in the development of hair follicles. Previously, we found that deoxycholic acid (DCA) increases the level of β-catenin in the nucleus through increased uPAR (uro plasminogen activator receptor).

Objective: To investigate the effect of DCA on hair growth in vivo

Methods: We applied DCA solution in mouse model and compared its effect with that of minoxidil. In addition, we treated DCA in the testosterone-induced androgenic alopecia mouse model and compared its effect with those of minoxidil and finasteride.

Results: In the phase-transition animal model, DCA (1% w/v) showed a hair growth rate of 70-80% of minoxidil (2% w/v). Horizontal cross sections of the skin tissue did not show the increase of the number of hair follicles in minoxidil (2% w/v) group, but in DCA (1% w/v) group, the number of hair follicles was increased by 30% compared to the control group. In the testosterone-induced androgenic alopecia animal model, minoxidil was not effective, but finasteride (100ug) effectively blocked androgenic alopecia. DCA (100ug) had a similar effect to that of finasteride (100ug), and the number of hair follicles was more than 40% higher than that of the control group.

Conclusion: Our results suggest that DCA seems to be useful for the treatment of hair loss.
HP-13

UV Irradiated human dermal endothelial cells induce pigmentation: The role of vasculature in the development of UV-induced hyperpigmentary disorders

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Background: We previously reported that normal human dermal endothelial cells (HDMECs) have an inhibitory role in the regulation of skin pigmentation. However, UV induced hyperpigmented skins of melasma and solar lentigo were characterized by increased vasculature.

Objective: In this study, we investigated the role of UV-irradiated HDMECs in the skin pigmentation and identified secreted factors from the HDMECs.

Method: HDMECs were irradiated with UVB. Normal human melanocytes were treated with conditioned media obtained from the UV irradiated HDMECs and the pigmentation was measured. RNA sequencing analysis was performed to identify the secreted factors from the UV irradiated HDMECs.

Results: UV-irradiated endothelial cells showed stimulatory action on skin pigmentation. The mRNA and protein expression levels of melanogenesis-associated proteins, microphthalmia-associated transcription factor (MITF) and tyrosinase were increased in the conditioned medium from UV irradiated HDMECs compared to non-irradiated HDMECs. Consistently, ex vivo skin pigmentation was significantly induced. RNA sequencing analysis showed differential expressions of melanogenic and anti-melanogenic factors in the UV irradiated HDMECs compared with non-irradiated cells.

Conclusion: UV-irradiated endothelial cells have a stimulatory role in the skin pigmentation, which may play a role in the development of UV-induced hyperpigmentary disorders.
**HP-14**

**Adiponectin inhibits melanogenesis through AMPK/ CREB regulated transcriptional coactivator**

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**Background:** Several reports have demonstrated the dermatologic beneficial effect of adiponectin, an important adipokine which lowers obesity and diabetes by increasing insulin sensitivity via AMP-activated protein kinase (AMPK) pathway. In our recent microarray data, adiponectin decreased in the lesion in comparison with non-lesional skin of melasma on the face.

**Objective:** As AMPK is a key mediator of adiponectin signaling, we investigated the effect of adiponectin and AICAR, a cell permeable activator of AMPK, in melanogenesis.

**Methods:** To investigate adiponectin expression in melasma lesional skin and serum, we performed quantitative RT-PCR, immunohistochemistry and serum analysis using blood sample. We tested cell viability and melanin contents in Mel-ab cells and normal human melanocytes cultured with adiponectin and AICAR. The expression level of melanogenesis associated genes and protein was determined using qRT-PCR and western blot. To identify adiponectin and AICAR signal pathway, western blot and MITF promoter assay was performed.

**Results:** We showed that adiponectin and AICAR reduced melanin content in normal human and mouse melanocytes by inducing the inhibitory phosphorylation of CREB regulated transcription coactivators (CRTCs) via enhancing AMPK activity. This anti-melanogenic effect of adiponectin correlated with downregulation of MITF, tyrosinase, tyrosinase-related protein-1 (TRP-1) and dopachrometautomerase (DCT) expression, which was resulted from decreased transcriptional activity of CREB.

**Conclusion:** These data emphasize the depigmenting effect of adiponectin is mediated through the activation of AMPK and subsequently suppressing a novel CRTC/CREB pathway in melanocytes and suggest a clinical strategy for using adiponectin and analogues in the treatment of hyperpigmentation disorders.
Spongiosis is a well-known hallmark of eczematous dermatitis, but its pathogenesis has not been fully understood. We previously demonstrated that the augmented production of hyaluronan (HA) and the decreased E-cadherin expression by keratinocytes stimulated with IL-4/IL-13 or IFN-γ cause spongiosis in acute eczema and that hyaluronan synthase 3 (HAS3) is responsible for augmented HA production by keratinocytes. In this study, to examine the role of HA synthesis in contact hypersensitivity (CHS), we used murine 2,4-dinitro-1-fluorobenzene (DNFB)-induced CHS model. We first demonstrated that HA accumulated in the epidermis after hapten challenge, while HA deposition did not significantly alter in the dermis. We next examined HAS1, HAS2, and HAS3 gene expression in the epidermis and dermis in the elicitation phase of CHS and found that only HAS3 mRNA was significantly upregulated in the epidermis, while there was no significant change of either HAS gene expression in the dermis. When we used HAS3 null mice (HAS3 KO), HA did not accumulate in the epidermis in the elicitation phase. These studies confirmed our previous results. Then, we compared ear swelling in the elicitation phase between WT mice and HAS3 KO mice, which revealed that HAS3 KO mice significantly reduced ear swelling. Moreover, HAS3 KO recipients of sensitized lymphocytes from WT mice also showed significantly reduced ear swelling, which indicates that HAS3 plays a crucial role in the elicitation phase. These studies demonstrated that HAS3 is indispensable for HA production by keratinocytes in the elicitation phase and contributes to the histogenesis of CHS.
HP-16

CCCTC-binding factor is essential to the maintenance and quiescence of hematopoietic stem cells in mice

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Hematopoiesis involves a series of lineage differentiation programs initiated in hematopoietic stem cells (HSCs) found in bone marrow (BM). To ensure lifelong hematopoiesis, various molecular mechanisms are needed to maintain HSC pools. CCCTC-binding factor (CTCF) is a DNA-binding, zinc-finger protein that regulates the expression of its target gene by organizing higher order chromatin structures. Currently, the role of CTCF in controlling HSC homeostasis is unknown.

Using a tamoxifen-inducible CTCF conditional knockout mouse system, we aimed to determine whether CTCF regulates the homeostatic maintenance of HSCs. In adult mice, acute systemic CTCF ablation led to severe BM failure and rapid shrinkage of multiple c-Kit⁺ progenitor populations, including Sca-1⁺ HSCs. Similarly, hematopoietic system-confined CTCF depletion elicited an acute loss of HSCs and highly increased mortality. Mixed BM chimeras reconstituted together with supporting BM demonstrated that CTCF deficiency-mediated HSC depletion is a cell-autonomous effect. Although myeloid progenitor cells were severely reduced after ablating Ctf, common lymphoid progenitors and their progenies were less affected by the lack of CTCF. Whole transcriptome microarray and cell cycle analyses indicated that CTCF deficiency results in enhanced expression of the cell cycle-promoting program, and CTCF-depleted HSCs express higher levels of reactive oxygen species (ROS). Importantly, in vivo treatment with an antioxidant partially rescued c-Kit⁺ cell populations and their quiescence. Altogether, our results suggest that CTCF is indispensable for maintaining adult HSC pools, likely by regulating ROS-dependent HSC quiescence.
IWR-1, an inhibitor for Wnt/β-catenin signaling, reduces collagen synthesis in skin fibroblasts

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Hyperproliferation of dermal fibroblasts result in the formation of keloid. Many researches suggest that aberrant activation of Wnt/β-catenin signaling is involved in the pathogenesis of keloid. In this study, we investigated the effects of IWR-1, a small molecule inhibitor for Wnt/β-catenin signaling, on production of collagen and matrix metalloproteinase (MMP) in dermal fibroblasts. We cultured human normal skin- and keloid-derived fibroblasts, then treated with IWR-1. The effects of IWR-1 on collagen and MMP production were determined by Western blot, ELISA and zymography. IWR-1 significantly suppressed the proliferation and migration of both the normal and keloid fibroblasts. IWR-1 also inhibited the production and secretion of type I collagen from the fibroblasts. In addition, IWR-1 significantly increased the expression of MMPs, such as MMP-1, MMP-3 and MMP-13, along with the increase of gelatinase activity. These results suggest that inhibitory effect of IWR-1 on collagen production may be related with the increased MMP activity. This study provides the possible action mechanism of IWR-1 on regulation of collagen expression, on which to base further investigation for preventing skin fibrotic diseases such as keloid.

Keywords: Collagen, Fibroblasts, IWR-1, Keloid, Matrix metalloproteinase
HP-18

CHSY1 may be a major regulator of GAG chain length on decorin and biglycan in intrinsically aged and photoaged human skin

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Background: Change of glycosaminoglycans (GAGs) and proteoglycans in skin aging processes has not been well elucidated. Recently, reduction of decorin and biglycan in intrinsically aged and photoaged skin was reported, and the shortened GAG chain length on decorin was observed in intrinsically aged skin. However, its mechanism or GAG length change in photoaged skin has not been revealed yet.

Objective: To investigate the GAG chain length change in photoaged skin and the regulation of GAG chain-synthesizing enzymes.

Methods: Skin samples of buttock and forearm skin were obtained from young (20–40 yr, n=6) and aged volunteers (70–78 yr, n=9). Detection of decorin and biglycan protein levels and sizes were examined with or without treatment with chondroitinase ABC by Western blot. SiRNA-mediated mRNA downregulation of GAG chain synthesizing enzymes were detected by realtime RT-PCR.

Results: By Western blot analysis of dermal protein samples, detected decorin and biglycan sizes were decreased in intrinsically aged buttock skin, compared with young buttock skin. In contrast, their sizes were increased in aged forearm skin, compared to each individual’s buttock skin. Using chondroitinase ABC, all decorin and biglycan bands were detected only at the sizes of their core proteins in both buttock and forearm samples, suggesting the increase of GAG chain length. By realtime RT-PCR, mRNA expression levels of chondroitin sulfate synthase (CHSY) 1 and 3 were reduced in intrinsically aged skin, while increased in photoaged skin. In dermal fibroblasts, only siRNA-mediated downregulation of CHSY1 resulted in significant reduction of GAG chain length on decorin and biglycan.

Conclusion: Taken together, GAG chain length of decorin and biglycan is differentially regulated in intrinsically aged and photoaged skin, and the regulation of CHSY1 expression may be a key regulator of the GAG chain length changes of decorin and biglycan in aged skin.
Background: Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) represent life-threatening drug eruptions. A severity-of-illness score for toxic epidermal necrolysis is a widely-used scale to predict mortality in SJS/TEN cases which adopts renal failure (blood urea nitrogen higher than 28mg/dl) as a risk factor. However, the influence of dialysis on the prognosis of SJS/TEN has not been elucidated.

Objective: To assess the impact of dialysis on the mortality rate in SJS/TEN cases.

Methods: A retrospective chart review was conducted on 370 SJS (n=257) /TEN (n=111) patients admitted to 212 Japanese medical institutions from 2005 to 2007. The mortality rates in SJS/TEN cases with or without the history of dialysis were respectively obtained.

Results: In SJS/TEN cohort, 4.9% (18/370) cases were receiving dialysis. The mortality rate was 7.8% (29/370) in all cases while that in hemodialysis cases was 24.1% (7/29). Therefore, dialysis cases demonstrated significantly higher incidence of lethal outcome than non-dialysis cases (p<0.01). Interestingly, the development of mucosal involvement subsequent to the emergence of cutaneous manifestation was more delayed in dialysis cases.

Conclusion: The medical history of dialysis may be a risk factor of poor prognosis in SJS/TEN at least in Japan. The delay of intensive treatment due to late-detection of mucous membrane involvement may adversely affect the prognosis.
Background: Although the relationship between the acne and diet has been controversial, recent studies suggested an alleged relationship between the acne and specific diet. Recently, high fat diet was suggested as an aggravating factor in acne pathogenesis.

Objective: To evaluate the effects of palmitic acid on the lipid production and inflammation in sebocytes and to elucidate the relation between high fatty acid diet and the development and aggravation of acne.

Methods: The SZ95 sebocytes were supplemented with palmitic acid to mimic the increased level of serum free fatty acids in high fat diet. The effects of palmitic acid on the lipid production were analyzed by evaluating the intracellular lipids. In addition, the secretion of proinflammatory cytokines including IL-6 and IL-8 were also analyzed by using RT-PCR and ELISA. We also investigate the role of TLRs in palmitic acid induced inflammation in SZ95 sebocytes.

Results: The SZ95 sebocytes supplemented with palmitic acid showed increased cytoplasmic lipid accumulation and intracellular triglycerides normalized to the protein contents. In addition, the results of RT-PCR and ELISA showed increased mRNA expression and secretion of IL-6 and IL-8. The Western blot revealed that the changes in SZ95 sebocytes after palmitic acid treatment were regulated by NF-κB and MAPK signaling pathway. As for IL-8, treatment of anti-TLR2 antibody and anti-TLR4 antibody decreased the production of IL-8, while the secretion of IL-6 was significantly decreased in anti-TLR2 antibody treated SZ95 sebocytes.

Conclusion: Palmitic acid treatment on SZ95 sebocytes increased the lipid accumulation and the production of proinflammatory cytokines. We also found that TLR2 and TLR4 are required in palmitic acid induced IL-6 and IL-8 production in SZ95 sebocytes. We suggest that the palmitic acid can initiate or aggravate the acne in the group of patients with high serum free fatty acids induced by high fat diet.
The 27th Annual Meeting of the Korean Society for Investigative Dermatology

UAM Award Lecture
CURRICULUM VITAE

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The role of S100A8 and S100A9 in human skin

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Background: S100A8 and S100A9 are members of the S100 protein family that exist as homodimers and heterodimers in neutrophils, monocytes, and macrophages. Recent studies have shown the pivotal roles of S100A8 and S100A9 in the propagation of inflammation and neoplastic disorders; however, their roles in cutaneous inflammatory disorders and squamous cell carcinoma (SCC) are not well defined.

Objective: This study aims to investigate the role of S100 in cutaneous inflammatory and neoplastic disorders.

Methods: We performed immunohistochemical staining to detect S100A8 and S100A9 expression in psoriasis and SCC. Furthermore, we constructed a recombinant adenovirus over-expressing S100A8 or S100A9 to investigate the role of S100A8 and S100A9 in keratinocytes and SCC cell differentiation.

Results: We found that the upregulated production of S100A8 and S100A9 by psoriatic epidermal keratinocytes activated adjacent keratinocytes to produce several cytokines. Moreover, S100A8 and S100A9 themselves function as pro-angiogenic and chemotactic factors, generating a psoriatic milieu in skin. In cutaneous SCCs, S100A8 and S100A9 are highly upregulated and play important roles in the aggressiveness of SCC by enhancing proliferation and migration in vitro. In addition, SCID mice injected with S100A8-overexpressing and/or S100A9-overexpressing SCC12 cells show marked induction of tumor growth.

Conclusion: This study provides new insight into the key role of S100A8 and/or S100A9 in cutaneous inflammatory and neoplastic disorders, and suggests that inhibiting S100A8 and/or S100A9 may be a novel therapy targeting various molecular events involved in cutaneous skin disorders.
Session IV.
Hereditary Disease, Genetic Regulation
CURRICULUM VITAE

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- 1981-1988 Study of medicine, Leopold Franzens University of Innsbruck, Austria
- 1989 Institute of Molecular Biology, Austrian Academy of Sciences, Salzburg
- 1989-1992 Department of Pharmacology, University of Innsbruck, Austria
- 1993-1994 Department of Dermatology, Emory University School of Medicine, Atlanta, USA
- 1998 Department of Dermatology, Thomas Jefferson University, Philadelphia, USA
- 2005-2014 Principal Investigator Division of Experimental
- 2007-2009 Master of Business Administration in Health Care Management, Department of Economics, Paris Lodron University, Salzburg
- 2011 Associate Professor of Dermatology, Paracelsus Medical University Salzburg

Current and Past Professional Positions:
- 2001 Associate Professor of Medical Molecular Biology, Division of Genetics and Cell Biology, Paris Lodron University, Salzburg
- 2001-present Professor of Medical Molecular Biology, Department of Cell Biology, Faculty of Natural Sciences, University of Salzburg
- 2006-present Director eb house Austria, Salzburg
- 2008-2014 Endowed Professor for Therapy of Genodermatoses
- 2009-2012 Lead coordinator of the EU-Interreg IV project “Therapie für Schmetterlingskinder-therapy for epidermolysis bullosa”.
- 2014-present Professor and Chairman, Department of Dermatology, Paracelsus Medical University, Salzburg

Honors and Awards:
- 1993 Erwin Schrödinger grant of the Austrian science fung, FWF
- 1994 Hans-Wendt Annual Award Salzburg Medical Society
- 1998 Josef Kyrle foreign grant of the ÖGDV
- 2008 Scientist of the year PMU Salzburg
- 2012 Ferdinand von Hebra award of the ÖGDV
Board Membership:

Austrian Society of Dermatology and Venerology:
  2001-present Chairman of the Working group for Human Genetics and Molecular Therapy
  2008-2010 General Secretary

Paracelsus Medical University:
  Member of Habilitationskommission, Universitätskollegium

2009-2014 European Society of Dermatological Research

Foundation Rene Touraine

2011-2014 Chairman of the Genodermatoses Committee
The clinical use of stem cells has been becoming increasingly popular in dermatology. Besides cosmetic indications, in which mainly adipose tissue-derived cells are used, keratinocyte-, mesenchymal-derived and hematopoetic stem cells are investigated in clinical studies. Mesenchymal stem cells have immunomodulatory properties that suggest their application in autoimmune diseases and in genetic diseases that have an inflammatory component. Indeed such studies have been reported in the genetic blistering skin disease epidermolysis bullosa (EB). Surprisingly, also allogenic hematopoetic stell cells seem to home to a small extend to the skin. This property has been the basis for one clinical study in dystrophic EB. Keratinocyte stem cells can be applied as transplanted sheets to correct epidermal based diseases. Clinical studies in EB patients suggest that this approach is clinically feasible. So far 5 patients suffering from dystrophic EB have received such transplantations. Furthermore, we have transplanted 3 patients suffering from junctional EB on up to 80% of skin surface. All procedures have been clinically successful so far. Thus a larger study with 12 patients with dystrophic EB is currently underway.
CURRICULUM VITAE

Shinichi Sato, M.D., Ph.D.

Professor and Chairman, Department of Dermatology,
Graduate School of Medicine and Faculty of Medicine,
The University of Tokyo

Education:
1989 M.D., The University of Tokyo, Tokyo, Japan
1994 Ph.D., The University of Tokyo, Tokyo, Japan

University Appointments:
1989-1994 Assistant, Department of Dermatology, The University of Tokyo, Tokyo
1994-1997 Research Associate, Department of Immunology, Duke University Medical Center, Durham, NC, USA
1997-2002 Assistant Professor, Department of Dermatology, Kanazawa University School of Medicine, Kanazawa, JAPAN
2002-2004 Associate Professor, Department of Dermatology, Kanazawa University, Graduate School of Medical Science, Kanazawa, JAPAN
2004-2009 Professor and Chairman, Department of Dermatology, Nagasaki University Graduate School of Biomedical Sciences, Nagasaki, JAPAN
2009- Professor and Chairman, Department of Dermatology, Graduate School of Medicine and Faculty of Medicine, The University of Tokyo, Tokyo, JAPAN
2015- Vice President, The University of Tokyo Hospital

Awards:
2003 Japanese Society of Investigative Dermatology (JSID) Award
2005 Abbott Japan-Medical Award on Rheumatic Disease

Major Research Interests:
1. Scleroderma
2. Autoimmunity and B lymphocyte
3. Cell adhesion molecule and inflammation
Epigenetic downregulation of transcription factors, Fli1 and KLF5, in scleroderma

Shinichi Sato, M.D., Ph.D.

Department of Dermatology, Graduate School of Medicine and Faculty of Medicine, The University of Tokyo, Tokyo, Japan

Systemic sclerosis (SSc) is a multisystem autoimmune disease characterized by initial vasculopathy and resultant skin and lung fibrosis. Although the pathogenesis of SSc remains unknown, our data have demonstrated that epigenetic suppression of transcription factor Fli1 is involved in activation of fibroblasts and endothelial cells in SSc. In murine models, Fli1 haploinsufficiency results in increased skin expression of type I collagen, but fibrosis does not occur partly because expression of CTGF, which plays a critical role to establish and maintain tissue fibrosis, remains unchanged. Since our pilot studies have revealed that transcription factor KLF5 is a potent repressor of CTGF gene and its expression is epigenetically suppressed in SSc dermal fibroblasts, we generated mice with heterozygous deletions of Fli1 and KLF5. Notably, Klf5<sup>−/−</sup>;Fli1<sup>−/−</sup> mice spontaneously exhibited cardinal features of SSc, including peripheral vasculopathy, skin and lung fibrosis, autoimmunity and inflammation. Dermal vascular abnormalities, including stenosis of arterioles and bushy capillaries, occurred around the age of 1-2 months before the development of dermal fibrosis with ultrastructural changes of collagen fibrils similar to those seen in SSc. Lung fibrosis with B cell lymphoid aggregates and vascular changes characteristic of pulmonary arterial hypertension appeared around the age of 2-3 months and progressed along with disease duration. Serum IL-6 levels and IL-6 mRNA levels in skin and lung were elevated around the age of 1-2 months and splenic B cells secreted a greater IL-6 amount in response to LPS and/or anti-CD40 antibody. Importantly, expression of CD19, a critical signal transduction molecule of B cells, was increased by 15%, which was also observed in SSc. Furthermore, anti-nuclear antibodies were detected. These studies underscore the concept of epigenetic reprogramming underlying pathogenic changes in SSc and implicate the Fli1 and KLF5 pathways as central mediators of this disease.
The 27th Annual Meeting of the Korean Society for Investigative Dermatology

Session V.
Pigment and Pigmentary Disorders
CURRICULUM VITAE

Sung Eun Chang, M.D., Ph.D.
Professor, Director of Department of Dermatology,
University of Ulsan College of Medicine, Asan Medical Center, Seoul, Korea

Education:
1989-1995 Seoul National University College of Medicine (MD), Seoul, Korea
1997-2001 MS and PhD Department of Dermatology, The Graduate School
University of Ulsan College of Medicine, Seoul, Korea

Training and Fellowship Appointments:
1996-2000 Dermatology residency, University of Ulsan College of Medicine, Asan Medical Center
2000 Board Certified by the Korean Board of Dermatology
2000-2003 Clinical fellow
2007-2009 Research fellow, Harvard medical school, Boston, MA, USA

Faculty Appointment:
2003-2005 Clinical professor, Department of Dermatology, University of Ulsan College of Medicine, Asan Medical Center
2005-2010 Assistant professor, Department of Dermatology, University of Ulsan College of Medicine, Asan Medical Center
2010-2015 Associate professor, Department of Dermatology, University of Ulsan College of Medicine, Asan Medical Center
2016-present Professor, Department of Dermatology, University of Ulsan College of Medicine, Asan Medical Center

Memberships:
The Korean Medical Association
The Korean Society of Dermatologists
The Korean Society of Investigative dermatology
The Korean Society of Cosmetic Dermatology and pigmenitary research, secretary general
The Korean Society of Contact Dermatitis and Allergy
The Korean Society of Skin Barrier, secretary
The Korean Society of Skin Laser
Board member of KEIT
Role of autophagy in squamous cell carcinomas and melanocytes

Sung Eun Chang, M.D., Ph.D.

Department of Dermatology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea

Autophagy is the central cellular mechanism for delivering organelles to lysosome for degradation and recycling of the components. In tumor, autophagic cellular survival versus death have been an issue for long time. As such, whether autophagy is related to melanogenesis versus degradation of melanin has been controversial. We have studied roles of autophagy in squamous cell carcinoma (SCC) and hyperpigmentation.

In head and neck SCC cells, silencing of β-catenin augmented autophagic cell death through increase of AMPK signaling. The autophagic vacuoles and the expression of type II light chain 3 (LC3) were increased due to activated AMPK and subsequent suppression of mTOR. Secondly, in skin SCC cells, we activated autophagic pathways by serum starvation in well differentiated SCC13 and undifferentiated ATF3-overexpressing SCC13 (ATF3-SCC13) cell lines. ATF3-SCC13 cells demonstrated high proliferative capacity and low p53 and lower autophagy levels in comparison with SCC13 cells under basal conditions. Through autophagic stimulation, ATF3-SCC13 cells demonstrated further more growth inhibition and senescence. Thus, autophagy activation may be an antitumor approach for advanced cutaneous SCC.

In melanocytes, autophagy is not a mere parallel phenomenon with melanogenesis but we found a mechanistic link between microtubule-associated protein LC3 activation and melanogenesis. LC3 was higher in pigment-rich melanocytic nevi of sun-exposed skin. Rapamycin-induced autophagy increased the melanin, tyrosinase activity, MITF, pre-melanosome protein and tyrosinase.; Silencing of LC3, but not beclin-1 or ATG5, decreased melanin, tyrosinase activity and markedly inhibited MITF expression even in the presence of rapamycin. LC3 knockdown also suppressed α-MSH-mediated melanogenesis by attenuating cAMP response element-binding protein (CREB) phosphorylation/MITF expression via decreased ERK activity. Overexpression of constitutively active ERK reversed the effect of LC3 knockdown demonstrating that LC3 contributes to melanogenesis by increasing ERK-dependent MITF expression.
CURRICULUM VITAE

Eun-Gyung Cho, Ph.D.

Team Leader, Biomics Research Team, Basic Research & Innovation Division, R&D Unit, AmorePacific Corporation

Education:
1998.3-2002.2 Ph.D., School of Biological Sciences, Seoul National University, Seoul, Korea (Mentor: Prof. Dongeun Park)
1996.3-1998.2 M.S., Dep. of Life Science, GIST, Kwangju, Korea (Mentor: Prof. Dongeun Park)
1992.3-1996.2 B.S., Seoul National University, Seoul, Korea

Employments:
2012.1-Present  Team Leader, Biomics Research Team, Basic Research & Innovation Division, R&D Unit, AmorePacific Corporation
2010.12-2011.12 Leader, Biomics Research Team, Basic Research & Innovation Division, R&D Unit, AmorePacific Corporation
2005.5-2010.10 Postdoctoral Fellow at Sanford-Burnham Medical Research Institute, San Diego, USA (Mentor: Prof. Stuart Lipton)
2002.3-2005.3 Postdoctoral Fellow at National Creative Research Initiative Center for Cell Death, Korea University (Mentor: Prof. Eui-Ju Choi)
2001.3-2002.2 Guest Researcher (pre-doctor) at LCML, NIH, USA (Mentor: Dr. Moon Gyo Kim)

Society Membership:
International Society for Extracellular Vesicles
Background: Skin melanocytes are activated by exposure to UV radiation to secrete melanin-containing melanosomes to protect the skin from UV-induced damage. Despite the continuous renewal of the epidermis, the turnover rate of melanocytes is very slow, and they survive for long periods. However, the mechanisms underlying the survival of melanocytes exposed to UV radiation are not known.

Objective: We investigated the role of melanocyte-derived extracellular vesicles (EVs) in melanocyte survival.

Methods: EVs were isolated from the cultured supernatants of human primary melanocytes and analyzed by dynamic light scattering. Proteomic and network analyses were performed to find proteins enriched in EVs. The change of fibronectin protein levels in EVs after UVB treatment was verified using EV biogenesis inhibitors. The effect of fibronectin-containing EVs on melanocyte survival was assessed through apoptosis assay. Fibronectin expression around melanocytes was examined in the hyperpigmented lesions of melasma patients.

Results: Network analysis of the melanocyte EV proteome identified the extracellular matrix component fibronectin at a central node, and the release of fibronectin-containing extracellular vesicles was increased after exposure of melanocytes to UVB radiation. Using an anti-fibronectin neutralizing antibody and specific inhibitors of extracellular vesicle secretion, we demonstrated that EVs enriched in fibronectin were involved in melanocyte survival after UVB radiation. Furthermore, we observed that in the hyperpigmented lesions of patients with melasma, the extracellular space around melanocytes contained more fibronectin compared with normal skin, suggesting that fibronectin is involved in maintaining melanocytes in pathological conditions.

Conclusion: Collectively, our findings suggest that melanocytes secrete fibronectin-containing EVs to increase their survival after UVB radiation. These data provide important insight into how constantly stimulated melanocytes can be maintained in pathological conditions such as melasma.
CURRICULUM VITAE

Hee Young Kang, M.D., Ph.D.
Professor in Department of Dermatology,
Ajou University School of Medicine

Education and Training:
1994 M.D. in Medicine, Ajou University School of Medicine
2003 Ph.D. in Dermatology, Ajou University Graduate School

Professional Experience:
1995-1997 Research fellow in Department of Life Science, POSTECH, Korea
2008.5-2009.8 Visiting scholar (Enseignant-chercheur) in Department of Dermatology, University of Nice, France
2013.9-present Professor in Department of Dermatology, Ajou University School of Medicine

Awards:
2014 John Pawelek Award from the Asian Society for Pigment Cell Research

Major Interest:
Pigmentary disorders and melanocyte biology
The role of cutaneous vasculature in the skin pigmentation

Hee Young Kang, M.D., Ph.D.

Department of Dermatology, Ajou University School of Medicine, Suwon, Korea

We previously demonstrated that UV induced hyperpigmentary skins such as melasma and solar lentigo are characterized by increased vasculature suggesting the role of cutaneous vasculature in the development of hyperpigmentation. In this study we report that endothelial cells play a role in the regulation of pigmentation. Our results demonstrate that endothelial cells have a negative effect on pigmentation. However, their effect differs after they are irradiated with UV, as they then positively influence the induction of melanogenesis. To investigate the molecular mechanism responsible for regulation of pigmentation by endothelial cells, we analyzed by RNA sequencing at which levels HDMECs and UV irradiated cells express various known secreted factors such as KITL, ET-1, and TGF β1 that influence melanocyte function differentially. These findings suggest that under normal physiological conditions, vascular endothelial cells maintain low levels of pigment production. Contrary to this, the UV irradiated endothelial cells play a stimulating role on pigmentation which may play a role in the development of hyperpigmentary disorders.
CURRICULUM VITAE

Jung-Won Shin, M.D., Ph.D.
Clinical Assistant Professor, Department of Dermatology
Seoul National University Bundang Hospital

Education:
2017  Ph.D., postgraduate School, College of Medicine, Seoul National University, Seoul, Korea
2012  M.S., Postgraduate School, College of Medicine, Seoul National University, Seoul, Korea
2006  M.D., Collage of Medicine, Seoul National University, Seoul, Korea

Professional Training and Employment:
2006-2007  Internship in Seoul National University Hospital, Seoul, Korea
2007-2010  Resident in Department of Dermatology, Seoul National University Hospital, Seoul, Korea
2011-2014  Fellowship in Department of Dermatology, Seoul National University Bundang Hospital, Seongnam-si, Korea
2015-  Clinical assistant professor in Department of Dermatology, Seoul National University Bundang Hospital, Seongnam-si, Korea

Society Memberships:
Korean Dermatological Association
Korean Pigment Cell Society
Korean Hair Research Society
Korean Society for Dermatopathology
Korean Society for Aesthetic and Dermatologic Surgery
Korean Association of Anti-aging Medicine
The effects of hydroporation with anti-aging cocktail on melasma via enhancing microenvironment of the skin

Jung-Won Shin, Je-Byeong Chae, Kyoung-Chan Park

Department of Dermatology, Seoul National University Bundang Hospital

Melasma is a common hypermelanotic skin condition usually presenting with brown-colored macules and patches on sun-exposed areas. Although melasma is considered as the one of the epidermal pigmentation disorders, it has characteristic dermal histological features in common. Dermal extracellular matrix abnormalities, such as solar elastosis, or basement membrane disruption are frequently observed in lesional melasma skin compared with perilesional skin. Furthermore, melasma skin lesion shows increased number of blood vessels, vessel density, and vessel size. From these histological findings, melasma can be regarded as one of the phenotypes of photodamaged skin, rather than just one of the epidermal pigmentation disorders. Therefore, therapeutic approaches for melasma have to focus on improving the damaged and photo-aged skin condition, not just removing the epidermal pigmentation.

We evaluated the effect of hydroporation with GHR formulation composed of copper-GHK, oligo-hyaluronic acid, Rhodiola extract, tranexamic acid, and b-glucan on melasma. Results showed that MI and EI were significantly decreased. Skin biopsy showed that collagen fibers are increased in upper dermis after treatment. Immunohistochemical staining revealed that increased collagen type IV and procollagen in the basement membrane area and upper dermis, respectively. Furthermore, the number of p63 positive cells is increased along the basement membrane. These results suggested that hydroporation with GHR formulation may have depigmenting and erythema decreasing effects by enhancing the microenvironment of skin.
Session VI.
Appendages & Wound Healing
CURRICULUM VITAE

Young Kwan Sung, Ph.D.

Professor, Department of Immunology,
Kyungpook National University School of Medicine

Education and Training:
1992 B.S. Department of Genetic Engineering, Kyungpook National University, Korea
1996 Ph.D. Department of Biology, Imperial College, UK
1996 Postdoc, Cambridge University, UK
1996-2001 Postdoc, Johns Hopkins University School of Medicine, USA

Current and Past Professional Positions:
2001-2004 Research Professor, Kyungpook National University Biomolecular Engineering Center
2004-2006 Assistant professor, Kyungpook National University Hospital Bio-Medical Research Institute
2006-2008 Assistant professor, Department of Immunology, Kyungpook National University School of Medicine
2008-2013 Associate professor, Department of Immunology, Kyungpook National University School of Medicine
2013-present Professor, Department of Immunology, Kyungpook National University School of Medicine

Society Memberships:
Korean Society for Molecular and Cellular Biology
Korean Society for Biochemistry and Molecular Biology
Korean Hair Research Society
Tissue Engineering and Regenerative Medicine International Society
Society for Investigative Dermatology
Study on the hair follicle-inducing genes identified from spheroid cultivation of human dermal papilla cells

Young Kwan Sung, Ph.D.

Department of Immunology, Kyungpook National University School of Medicine, Daegu, Korea

Recent studies have shown that the hair-inducing capacity (trichogenicity) of dermal papilla (DP) cells is restored when 3D culture rather than 2D culture is employed. However, little is known about why there is a restoration in trichogenicity when dissociated DP cells are prompted to form spherical structures. With the hope of identifying trichogenic genes of human DP cells, we used proteome profiler array as well as Affymetrix gene array. We found that a number of genes and secretory proteins including activin A, secreted frizzled-related protein 2 (SFRP2), and alkaline phosphatase (ALP) were upregulated in the DP spheres compared with 2D-cultured DP cells. We then explored the role of activin A, SFRP2, and ALP in trichogenicity of human DP spheres by adopting small interfering RNA (siRNA)-mediated gene knockdown approach and in vivo hair reconstitution assay. We observed that human DP spheres with knock-down of those genes are severely impaired in hair follicle induction when combined with mouse epidermal cells. Our data strongly suggest that activin A, SFRP2, and ALP affect hair inductive potency of human DP cells.
CURRICULUM VITAE

Sang Ho Oh, M.D., Ph.D.

Department of Dermatology and Cutaneous Biology Research Institute, Severance Hospital, Yonsei University College of Medicine

Education and Training:
1999 M.D., Yonsei University College of Medicine
2000-2004 Severance Hospital, Yonsei University Health System, Seoul, Korea
2010 Ph.D., Yonsei University College of Medicine

Current and Past Professional Positions:
2007-2009 Research Fellow, Department of Dermatology, Yonsei University College of Medicine
2009-2010 Clinical Professor, Department of Dermatology, Yonsei University College of Medicine
2010-2016 Assistant Professor, Department of Dermatology, Yonsei University College of Medicine
2014-2016 Visiting Professor, Department of Dermatology, University of Pennsylvania, Philadelphia, PA
2016-present Associate Professor, Department of Dermatology, Yonsei University College of Medicine

Awards:
2010 Clinical Research Award (Asia-Pacific Foundation of La Roche-Posay)
2010 Academic Award for Excellence (Yonsei University College of Medicine)
2013 Young Investigator Award (Yonsei University College of Medicine)

Society Memberships:
Korean Society of Investigative Dermatology
Korean Dermatological Association
Korean Society of Photomedicine
Korean Pigment Cell Research
Korean Society of Dermatopathology
Korean Society of Vitiligo (Board member)
Hair loss in hephaestin knockout mouse with iron deficiency

Sang Ho Oh¹,³, Yujie Mao¹, Stephen Prouty¹, Zai-Xin Yang¹, Xuegang Xu¹, Arben Nace¹, Ying Zheng¹, Jacob Beer¹, Heather Rosengard¹, Tzevete Dentchev¹, Josh Dunaief², George Cotsarelis¹

1Department of Dermatology and 2Department of Ophthalmology, Perelman School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania, USA, 3Department of Dermatology and Cutaneous Biology Research Institute, Severance Hospital, Yonsei University College of Medicine, Seoul, Korea

Background: Iron deficiency is known to be associated with hair loss and hair shaft abnormalities in humans. Hephaestin, a copper-dependent ferroxidase converts ferrous iron to ferric iron and it cooperates with ferroportin to allow iron to reach the circulation. Here we report that the hephaestin knockout (heph K/O) mouse develops hair loss associated with iron deficiency.

Objective: to elucidate the possible pathogenesis of hair loss in iron deficiency through heph K/O mouse model.

Methods: Using heph K/O mouse model, we investigated whether hair loss in this mouse is associated with iron deficiency via blood test, iron supplement experiment and foster mother exchange experiment. In addition, related molecules, which could induce hair loss were examined through q-PCR, western blotting, in situ zymogram and immunostainings.

Results: The heph K/O mouse displayed hair loss beginning between postnatal day (P) 16 and 35 during catagen and telogen, progressing in a cephalocaudal direction. The head and tail were spared. Hair loss within a litter was an all or none phenomenon. Young mothers, who gave birth to pups at less than 100 days old more often had litters with the hair loss phenotype compared to older mothers. Mice with hair loss showed decreased hemoglobin/hematocrit and MCV levels and low iron content in skin and liver. Iron supplementation using injection of iron dextran (0.5 mg/gm body weight) rescued the hair loss phenotype. Skin from mice that developed hair loss displayed smaller hair bulbs prior to the onset of hair loss at P10 and after its onset, broken and distorted hair shafts in hair orifices and dilated cysts. Proliferation of hair matrix cells, measured by quantitation of Ki67 staining, did not appear affected by iron deficiency and there was no significant increase in apoptotic cells. The heph K/O mouse showed thin hair shafts and various kinds of hair shaft abnormalities such as trichoschisis and trichorrhexis nodosa. Decreased hair shaft differentiation markers and increased protease activity were observed.

Conclusion: This study suggests that iron deficiency might cause hair loss by affecting hair differentiation and imbalance between protease and protease inhibitor.
CURRICULUM VITAE

Dong-ki Lee, Ph.D.
Professor, Global Research Laboratory of RNAi Medicine, Department of Chemistry, Sungkyunkwan University, Korea

Educational Background:
1994.8-1999.1 Ph.D. in Molecular Biology, Section of Biochemistry, Molecular and Cell Biology, Cornell University (Advisor: John T. Lis)
1989.2-1993.2 B.S. in Chemistry, Department of Chemistry, Korea Advanced Institute of Science and Technology (KAIST)

Professional Career:
2010.02-present Founder and CEO, OliX Pharmaceuticals
2008.10-present Principal Investigator, Global Research Laboratory for RNAi Medicine (in collaboration with Prof. Chiang J. Li, Harvard Medical School)
2008.3-present Professor, Department of Chemistry, Sungkyunkwan University (SKKU) (tenured)
2004.3-2008.2 Assistant Professor, Department of Chemistry, POSTECH
2003.5-2004.3 Post-doc. Department of Chemistry, KAIST
2001.5-2003.5 Senior Research Scientist and Group leader (Microarray and Functional Genomics), Toolgen Inc.
1999.5-2001.4 Post-doc. Center for Calcium and Learning, Pohang University of Science and Technology (POSTECH)
1999.1-1999.5 Post-doc. Section of Biochemistry, Molecular and Cell Biology, Cornell University

Honors and Awards:
2016.10 Korea Daily Creative Management award: as CEO of OliX Pharmaceuticals
2012.12 Sungkyun Family Award, Education and Research
2010.8 “Outstanding Achievements in Basic Science Research” awarded by Korean Ministry of Education, Science and Technology
1994-1995 Sage graduate fellow, Cornell University
Professional Activities:
2012-present  Editorial Board, Molecular Therapy-Nucleic Acids
2011-present  Asian Editor, Nucleic Acid Therapeutics (formerly Oligonucleotides)
2010-present  Editorial Board, Recent Patents on Anti-infective Drug Discovery
2010-2015    Editorial Board, BMB Reports
2009-present  Founding Member and Vice President, Korean Nucleic Acids Society

Research Interest
Mechanism of RNA polymerase II transcription
RNA aptamers for diagnostics and therapeutics
RNAi therapeutics
Nucleic acid-triggered antiviral innate immune responses
Development of cell-penetrating asymmetric interfering RNA targeting connective tissue growth factor (CTGF) for anti-scar therapeutics

Jihye Hwang¹, Chanil Chang¹, Ji Hyun Kim¹, Chang Taek Oh², Ha Neul Lee³, Changki Lee³, Donghoon Oh³, Changjin Lee³, Beomjoon Kim², Sun Woo Hong¹, and Dong-ki Lee⁴

¹OliX Pharmaceuticals, Inc., Seoul, Korea,  
²Department of Dermatology, Chung Ang University Medical Center, Seoul, Korea,  
³Hugel Inc., Chuncheon, Korea,  
⁴Department of Chemistry, Sungkyunkwan University, Suwon, Korea

Connective tissue growth factor (CTGF) is a multifunctional matricellular protein, playing a role as a central mediator in tissue remodeling and fibrosis. A number of reports have demonstrated the pivotal roles of CTGF in the progression of fibrosis, suggesting CTGF as a promising therapeutic target for the treatment of fibrotic disorders including hypertrophic scars and keloids. In this study, we present the development of a novel interfering RNA molecule which efficiently inhibits the expression of CTGF via RNA interference mechanism both in vitro and in vivo. Chemical modifications were introduced to the asymmetric interfering RNA (asiRNA) backbone structure. The resulting RNA molecule, termed cell-penetrating asiRNA (cp-asiRNA), entered into cells and triggered RNAi-mediated gene silencing without delivery vehicles. The gene silencing activity of cp-asiRNA targeting CTGF (cp-asiCTGF) was examined both in vitro and in vivo. Furthermore, the administration of cp-asiCTGF in the rat skin excision wound model efficiently reduced the induction of CTGF as well as collagens during the wound healing process. These results suggest that the cp-asiCTGF molecule could be developed into novel anti-fibrotic therapeutics such as anti-scar drugs.
Session VII.
Keratinocyte Biology & Epidermal Barrier
Sekyoo Jeong, Ph.D.

Assistant Professor, Seowon University, Department of Cosmetic Science

Education and Training:
2007 Ph.D. Department of Medical Science, Yonsei University College of Medicine, Korea
1999 M.S. Biochemical Engineering, School of Chemical Engineering, Seoul National University, Korea
1997 B.A. Department of Chemical Technology, Seoul National University, Korea

Employments:
2016- Assistant Professor, Seowon University, Department of Cosmetic Science
2013-2016 Adjunct Professor, Department of Pharmaceutics, Chungbuk National University College of Pharmacy
2011, 2013 Visiting Researcher, Department of Dermatology, University of California San Francisco, CA, USA
2006-2016 Research Director, CRID Center, NeoPharm Co., Ltd.
2002-2003 Visiting Researcher, Central Research Laboratories, Yongdong Severance Hospital, Yonsei University College of Medicine
1999-2006 Senior Researcher, Aekyung Corporation Central Research Laboratories

Major Interests:
Development of new cosmetic ingredients and efficacy evaluation
Skin barrier function homeostasis
Sphingolipid derivative developments
Role of autophagy activators on epidermal homeostasis

Sekyoo Jeong¹, Kyungsook Yoo², Jiyeon Park², Sungwoo Kim², Jongmi Lim², Hyejin Song⁴, Chaejin Lim³, Keedon Park², Hyunjung Kim⁴

¹Department of Cosmetic Science, Seowon University, Cheongju, South Korea,
²CRID Center, NeoPharm Co., Ltd., Daejeon, South Korea,
³Incospharm Corporation, Daejeon, South Korea,
⁴Department of Dermatology, Seoul Medical Center, Seoul, South Korea

**Background:** Even with recently increased interests on autophagy process in various cellular responses, little has been reported about the roles of autophagy in skin homeostasis, including skin barrier function, inflammation, and aging.

**Objective:** In this study, we investigated the potential involvement of autophagy on skin homeostasis using newly developed autophagy activators.

**Methods:** *In vitro* screening system, assessing the change of autophagic flux in cultured human epidermal keratinocytes (NHEK) and dermal fibroblast (hDF), was developed for identifying new small molecules with autophagy activating effects. With the newly developed autophagy activating compounds, *in vitro* and *in vivo* activities on keratinocytes differentiation, inflammation and collagen synthesis were investigated. Clinical efficacy was also observed.

**Results:** Several compounds were identified as having autophagy activating effects on culture human skin cells. Experiments with reconstituted 3D skin model showed that autophagy activators can stimulates the expression of collagen proteins in dermal fibroblasts. Poly (I:C) and cytokines cocktail-induced *in vitro* atopic dermatitis model also showed a significant anti-inflammatory activity from the autophagy activators.

**Conclusion:** While there are a few reports about the potential involvement of autophagy in epidermal differentiation, little have been reported about the other potential roles of autophagy in skin homeostasis. In this study, we observed that autophagy is also involved in epidermal and dermal functions, including skin inflammation and dermal aging. There results also suggest that autophagy activator might be a new potential target for skin inflammation and anti-aging, which implies a possibility of developing new strategy for anti-aging products for diseased skin or sensitive skin.
CURRICULUM VITAE

Chang Deok Kim, Ph.D.

Associate Professor, Department of Dermatology, School of Medicine, Chungnam National University, Daejeon, Korea

Education:
1986.3-1990.2 Department of Zoology, School of Natural Sciences, Seoul National University, Seoul, Korea (BS)
1990.3-1992.2 Department of Molecular Biology, School of Natural Sciences, Seoul National University, Seoul, Korea (MS)
1992.3-2002.8 School of Biological Sciences, Seoul National University, Seoul, Korea (PhD)

Career:
2003.9-2006.2 Department of Dermatology, School of Medicine, Chungnam National University, Daejeon, Korea (Postdoctoral researcher)
2006.3-Present Department of Dermatology, School of Medicine, Chungnam National University, Daejeon, Korea (Associate professor)
2010.10-2012.3 Department of Dermatology, University of Pennsylvania School of Medicine, Philadelphia, PA (Visiting Scholar)
Targeted deletion of Crif1 in mouse epidermis impairs skin homeostasis and hair morphogenesis

Jung-Min Shin¹, Dae-Kyoung Choi¹, Kyung-Cheol Sohn¹, Ji-Young Kim¹, Myung Im¹, Young Lee¹, Young-Joon Seo¹, Minho Shong², Jeung-Hoon Lee¹, Chang Deok Kim¹

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Background: The epidermis, which consists mainly of keratinocytes, acts as a physical barrier to infections by regulating keratinocyte proliferation and differentiation. Hair follicles undergo continuous cycling to produce new one. Therefore, optimum supply of energy from the mitochondria is essential for maintaining skin homeostasis and hair growth. CRIF1 is a mitochondrial protein that regulates mitoribosome-mediated synthesis and insertion of mitochondrial oxidative phosphorylation polypeptides into the mitochondrial membrane in mammals. Recent studies reveal that conditional knockout (cKO) of Crif1 in specific tissues of mice induced mitochondrial dysfunction.

Objective: To determine whether the mitochondrial function of keratinocytes affects skin homeostasis and hair morphogenesis.

Method: We generated epidermis-specific Crif1 cKO mice.

Results: Deletion of Crif1 in epidermis resulted in impaired mitochondrial function and Crif1 cKO mice died within a week. Keratinocyte proliferation and differentiation were markedly inhibited in Crif1 cKO mice. Furthermore, hair follicle morphogenesis of Crif1 cKO mice was disrupted by down-regulation of Wnt/β-catenin signaling.

Conclusion: These results demonstrate that mitochondrial function in keratinocytes is essential for maintaining epidermal homeostasis and hair follicle morphogenesis.
CURRICULUM VITAE

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Ceramide with long-chain fatty acids and skin barrier

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Ceramides (CERs) are well-known as an essential component for maintaining healthy permeability barrier functions, which contains fatty acids (FAs) of various chain lengths. According to carbon chain length, FAs with >C20 are called very long-chain FAs (VLCFAs), and those with >C24 are referred to as ultra-long-chain FAs (ULCFAs). Unlike CERs in other tissues, CERs in epidermis contain extremely long FAs (>C36). It has been recently found that the chain length and the amount of ceramides are important factors for the maintenance of homeostasis in skin barrier. Interestingly, ceramides (CERs) with very long-chain fatty acids (FAs) are decreased in the stratum corneum (SC) of patients with atopic dermatitis and psoriasis as well as aged skin.

Among various lipid synthetic enzymes, fatty acid elongases (ELOVs) and ceramide synthases (CERSs) have critical roles in the formation of long-chain ceramides. Although the molecular mechanisms of this shift in chronic inflammatory skin diseases and aging skin are poorly understood, it has been suggested that the inflammatory cytokines, especially IFN-γ, TNF-α, and IL-4, are related to decreased expression of ELOVs and CERSs. This talk will review the changes of fatty acid chain length and provide a chance to think of another strategy for reinforcing skin barrier function.

Keywords: Long chain fatty acid, Ceramides, Elongases, Atopic dermatitis, Psoriasis, aging, skin barrier
The 27th Annual Meeting of the Korean Society for Investigative Dermatology

Posters (68)

FC-1 ~ FC-18
HP-1 ~ HP-20
PO-1 ~ PO-30
PO-1

Serum HMGB1 induced TRIM21 expression on monocytes from Behcet’s disease patients

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Background: High motility group box 1 (HMGB1) is a non-histone nuclear protein that is implicated in chronic inflammatory and autoimmune disease. In recent studies, enhanced of HMGB1 expression has been found in serum of Behcet’s disease (BD). Previously study, tripartite motif containing 21 (TRIM21), an E3 ligase protein, upregulated in the monocyte of BD patients and facilitated Th1/Th17 differentiation of naïve T cell through secretion of proinflammatory cytokine.

Objective: The purpose of this study was to determine the role of TRIM21 in the regulation of enhanced HMGB1 in serum from BD patients.

Methods: HMGB1 of serum was measured using western blot and ELISA. THP-1 cells were stimulated with recombinant HMGB1 to examine the induction of TRIM21 expression and confirmed using blocker antibody of HMGB1 receptor. Functional analyses using small interfering RNA performed to examine the pathological role of TRIM21 upon HMGB1 stimulation.

Results: HMGB1 expression was increased in serum of BD patients compared to healthy control. HMGB1 significantly induced TRIM21 expression in concentration dependent manner via HMGB1 binding to TLR4 and expression of IRF8, a representative ubiquitination target of TRIM21, decreased in THP-1 cells. Knock-down of TRIM21 using siRNA prevented NF-kB activation as a result decreased secretion of proinflammatory cytokine.

Conclusion: From our results suggest that increased of TRIM21 expression by serum HMGB1 promote inflammatory response may be exacerbated behcet’s disease.
**PO-2**

**Vitamin D and extracellular calcium regulate inflammation on cultured sebocytes**

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**Background:** Sebum production in sebaceous glands leads to accumulation of lipid droplets and excretion into the skin surface in a holocrine manner. Excessive sebum production and changes in sebum component are supposed to be one of the pathogenic factors for acne. Extracellular calcium levels regulate growth and differentiation of keratinocytes and sebocytes. In addition, sebocytes were identified as bioactive vitamin D-responsive target cells. Both vitamin D and calcium act as promoters of epithelial cell functions. Inflammatory cytokines also play an important role in the pathophysiology of acne. Inflammatory cytokines can be produced by sebocytes.

**Objective:** The purpose of this study was to investigate regulation of inflammation on cultured sebocyte of vitamin D and extracellular calcium.

**Methods:** We investigated changes in the expression of inflammatory biomarkers and lipogenesis after the treatment of cultured sebocytes with vitamin D (10-10 to 10-6 M) or calcium (0.25, 0.5, 1 and 1.25 mM). Real time-polymerase chain reaction and enzyme-linked immunosorbent assay were done to measure changes in the expression of inflammatory biomarkers including IL-1ß, IL-6, IL-8 and TNF-α after treatment with vitamin D or calcium. Real time-polymerase chain reaction was done for the expression of antimicrobial peptides, including psoriasin, hBD2 and LL37, and sebocyte differentiation markers, including MC1R and MC5R, after treatment with vitamin D or calcium.

**Results:** Vitamin D and calcium decreased the expression of IL-1ß, IL-6, IL-8 and TNF-α in cultured sebocytes. Calcium increased the expression of psoriasin, hBD2 and LL37 in cultured sebocytes. In addition, calcium increased the expression of MC1R and MC5R.

**Conclusion:** Vitamin D and calcium have a potential to regulate the expression of inflammatory biomarkers, antimicrobial peptides, differentiation markers in cultured sebocytes.
**PO-3**

**T-cell-immunoglobulin- and mucin-domain-containing molecule-3 (TIM-3) expression in the patients with psoriasis vulgaris**

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**Background:** Psoriasis vulgaris is a chronic immune-mediated inflammatory skin disease. T-cell-immunoglobulin- and mucin-domain-containing molecule-3 (TIM-3) is usually expressed on the surface of Th1 or Th17 cells, and binding with galectin-9, it serves as a negative regulator of binding cells.

**Objective:** The goal of this study was to investigate expression of TIM-3 on the T cells in the patients with psoriasis vulgaris.

**Methods:** Patients with psoriasis vulgaris (n=10) and healthy control (n=13) were involved. The expression of TIM-3 as well as CD3, CD4, CD8, CD11b and CD56 in peripheral blood mononuclear cells (PBMC) was assessed by flow cytometry.

**Results:** The percentage of CD3⁺T cells and CD4⁺T cells in PBMC was significantly lower in patients with psoriasis compared to control. There was no significant difference in TIM-3 expression in PBMC from patients with psoriasis and healthy control. Even in the subsets of T cells, which was TIM-3⁺CD3⁺, TIM-3⁺CD4⁺, TIM-3⁺CD8⁺, TIM-3⁺CD11b⁺, and TIM-3⁺CD56⁺, there also has no significant difference.

**Conclusion:** The expression of TIM-3 was not significantly difference in PBMC from psoriasis and control.
PO-4

Clinical and histopathological differences between men and women with moderate to severe psoriasis

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Background: Psoriasis is a chronic inflammatory skin disorder characterized by epidermal hyperplasia, vascular proliferation and inflammatory infiltrates. Recent aim of the authors is to fabricate a database for psoriasis patients for development of further studies. Recently, during the registration of psoriasis patients, we noticed that the extent of involvement was larger in male patients than in female patients. Thus, we hypothesized that estrogen, through its one of various pathways, might affect the severity of the disease.

Objective: To investigate whether sexual difference affects the severity of psoriasis.

Materials and methods: Epidemiologic and clinical data and assessments for severity of extent and activity of psoriasis were collected from 497 patients. 20 male patients and 20 female patients with body surface area (BSA) >10% and psoriasis activity index (PASI) >10 (Group 1 and 2, respectively) and 10 male and 10 female patients with BSA < 10% and PASI < 10 (Group 3 and 4, respectively) were chosen from the registry. We investigated the expression of potential markers of estrogen pathway including estrogen α / β receptor, IP-10, MCP-1, RANTES, IFN-γ, IL-22 and AP-1 by immunohistochemistry. We also checked serum levels of estrogen, progesterone and testosterone using radioimmunoassay kits from 5 patients of each group.

Results: Of the 497 patients, 298 were male and 199 were female. Age and mean age of onset did not differ between the sexes. BSA and PASI score was higher in men compared to women. No other clinical history including hypertension, diabetes, cardiovascular disease and cancer did not show significant difference. On hematoxylin and eosin stain of the 60 patients, there was no significant difference between the 4 groups. The serum levels of sex hormones and immunohistochemical studies of the 4 groups abled the authors to discern the difference between them.

Conclusion: Many previous studies suggest estrogen as a benefactor in psoriasis, but only few studies have studied differences between the sexes. Our study not only shows the relationship between serum estrogen and the severity of psoriasis, but also shows various expression of molecules related to estrogen in skin tissue, thereby aiding future studies that should be done to specify the exact roles of estrogen in psoriasis.
PO-5

Increased expression of TRPV3 and TRPV4 channel in keratinocytes under Th2 inflammation

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Background: Pruritus in the patients with atopic dermatitis is frequently affected by environmental temperature change. Transient receptor potential (TRP) channel which is a nonselective cation channel play a central role on sensory response to noxious physical and chemical stimuli. Populations of non-neuronal cells in the skin express many different types of TRP channels and TRP channels are involved in various key cutaneous functions including pruritus, differentiation, and inflammatory process.

Objectives: To evaluate the molecular expression of TRP channels in keratinocytes under Th2 inflammation.

Methods: Primary epidermal keratinocytes were cultured with IL-4, IL-13, IL-17A, and IFN-\(\gamma\). Expression of TRPV1, TRPV3, TRPV4 and TRPA1 on keratinocytes was analyzed by quantitative real-time PCR, and western blot and flow cytometry.

Results: Real-time PCR and western blotting analysis revealed elevated expression of TRPV3, TRPV4 and TRPA1 on keratinocytes cultured with IL-4, IL-13, IL-17A. Especially, TRPV3 and TRPV4 expression is significantly increased under influence of Th2 inflammatory cytokines such as IL-4 and IL-13. Flow cytometry analysis confirmed increased expression of TRPV3 and TRPV4 in keratinocytes cultured with IL-4 and IL-13.

Conclusion: Expression of TRPV3 and TRPV4 channels are significantly increased on keratinocytes under Th2 inflammation. TRPV3 (warm $>32^\circ\text{C}$) and TRPV4 (warm $>28^\circ\text{C}$) may be more relevant TRP channels in atopic dermatitis than TRPV1 (warm $>43^\circ\text{C}$).

Key word: Atopic dermatitis, Th2, TRPV
PO-6

Genetic Polymorphism of Thymic Stromal Lymphopoietin (TSLP) in Korean Atopic Dermatitis (AD) and Allergic March (AM) patients

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Background: TSLP promotes the differentiation of naïve T cells into type 2 helper T cells, a cell type associated with pathogenesis in atopic dermatitis and allergic march. Studies of the TSLP polymorphism showed different single nucleotide polymorphisms (SNPs) pattern according to ethnic and atopic disease. However, there is little study of TSLP polymorphism in Korean atopic dermatitis.

Objective: This study aimed to investigate whether the polymorphisms in the TSLP gene are associated with atopic dermatitis and allergic march in the Korean population.

Method: First, we screened TSLP polymorphism using whole exom sequencing (WES) methods in 20 AD patients. Second, we analyzed four SNPs in three group patients made up 79 AD patients, 74 AM patients and 130 controls and then examined the association between SNP and lab findings in Korean atopic patients.

Results: Total nine TSLP variants detected in WES. No associations between normal control and AD were observed in sanger sequencing. Interestingly, there were statically significant associations between normal control and AM in two SNP (rs2289276 and rs2289278). Compared with AM patients with the CC genotype of SNP rs2289276, those with the TT or CT genotype had a significantly decreased risk of AM. Also, the GG of CG genotypes combined of SNP rs2289278 may be significantly negatively associated with AM.

Conclusion: Our results suggest that two TSLP polymorphisms are possibly correlated with the susceptibility to allergic march.

Key words: Allergic march, Atopic dermatitis, Genetic polymorphism, Sanger sequence, TSLP
PO-7

Epidermal growth factor relieves inflammatory signals in *S. aureus* treated human epidermal keratinocytes and atopic dermatitis-like skin lesions in Nc/Nga mice

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**Background:** Atopic dermatitis (AD) is a very common inflammatory and skin barrier-defected skin disease necessitating various topicals in addition to systemic treatments, but topical treatments are still unsatisfactory. Above facilitation of wound healing, epidermal growth factor (EGF) was shown to inhibit inflammation and EGF receptor inhibitors increased *Staphylococcus aureus* (*S. aureus*), a potential role of EGF in chronic skin inflammatory disorders including AD has not been clarified.

**Objective:** We investigated the effect of EGF on inflammatory cytokines and antimicrobial peptides (AMPs) *in vitro* and *in vivo*.

**Methods:** Human epidermal keratinocytes were treated with heat-inactivated *S. aureus* (HKSA) and recombinant human EGF (rhEGF). The expression of inflammatory cytokines and AMPs was assessed using RT-qPCR and ELISA assay. Intracellular signaling pathways such as NFκB and p38 were investigated using RT-qPCR. *In vivo* study, we investigated the effects of topical EGF or pimecrolimus (Elidel®) cream for 24 hours on 2,4-dinitrochlorobenzene (DNCB)-induced AD-like skin lesions in Nc/Nga mouse model.

**Results:** HKSA-induced IL-6 and NFκB expression and mRNA levels of p38 were decreased by EGF. Intriguingly, EGF increased the expression of human β defensin-2 (hBD-2). Both EGF and pimecrolimus groups showed significantly decreased infiltrates of inflammatory cells and CD3⁺ T cells and decreased expression of thymic stromal lymphopoietin (TSLP) compared with untreated controls at 3 hr and 24 hr. TSLP expression was much more reduced in EGF group than in pimecrolimus group. In both EGF and pimecrolimus groups, murine β defensin-3 (mBD-3) was increased compared to the controls.

**Conclusion:** EGF contributed to relieve inflammatory signals induced by *S. aureus* and AD-like skin lesions in Nc/Nga mice. Considering that AD is characterized by decreased AMPs with defective innate immune function leading to uncontrolled inflammation, EGF may be a potential topical treatment option in AD.
PO-8

Striae distensae due to topical glucocorticoids is relatively rare in atopic dermatitis than psoriasis patients

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**Background:** Atopic dermatitis (AD) and psoriasis (PSO) are chronic inflammatory dermatosis, which have been commonly treated with topical glucocorticoids (GCs). Although long-term use of topical GCs may induce striae distensae (SD), patients with AD have been felted to develop less SD than patients with PSO. AD is characterized by infiltration of eosinophils and fibrosis in chronic lesions. The association of tissue eosinophilia with inflammatory fibrotic lesions of asthma is well known as well.

**Objective:** This study was designed to elucidate whether patients with AD have less SD, and the development of more fibrosis of the skin, and tissue eosinophilia than patients with PSO.

**Methods:** Using the Korea Health Claims Database (KHCD) from 2009 to 2013, we compared the proportion of patients who had SD among all patients diagnosed with AD or PSO, respectively. For skin fibrosis and tissue eosinophilia, we compared the histopathology of both lesional and non-lesional skin of patients with AD and PSO. The degree of fibrosis of the skin was analyzed and tissue eosinophils were counted. RNA-sequencing and microarrays were performed to identify differentially expressed genes in 6 AD patients versus 5 PSO patients.

**Results:** We confirmed that AD patients have significantly less SD than PSO patients from KHCD. Besides, the degree of fibrosis in the lesional to non-lesional skin was significantly higher in AD than PSO. Tissue eosinophils were also significantly higher in AD patients. Among the genes, periostin (POSTN: a systemic biomarker of eosinophilic fibrosis gene in asthma), MMP12, and MMP 28 were increased while TGFA, IL1B, IL8, CXCL10 and HRH2 were decreased in the AD patients than PSO.

**Conclusion:** We found that patients with AD develop less SD compared to PSO despite topical GC treatments, which could be due to the formation of skin fibrosis from tissue eosinophilia.
**PO-9**

**Association of COL6A6 and CDKAL1 polymorphisms with early-onset atopic dermatitis in Korean population**

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**Background:** The prevalence of atopic dermatitis (AD) has increased over the last 10 years. AD tends to run in families that passed down from generation to generation and commonly starts in childhood. The prevalence of atopic dermatitis is as high as 20% in children. Thus, early diagnosis and treatment of atopic dermatitis are important, and understanding of its genetic basis is needed to facilitate early diagnosis.

**Methods:** To identify family-specific candidate genetic variants of early-onset atopic dermatitis in Koreans, we carried out whole-exome sequencing of three separate families with AD. Additional validation was performed using Sanger sequencing.

**Results:** 5 family-specific variants predicted protein damage by functional prediction program were identified as common or rare variants shared among the three families, and detected AD only. To validate these 5 variants, 112 AD and 61 control subjects under 3 years old were enrolled in the study. COL6A6 (rs16830494 and rs59021909, rs200963433) and ERBB2 (rs1058808), CDKAL1 (rs77152992) were sequenced by Sanger sequencing. Three variants of the COL6A6 gene appeared in all three families and were in close proximity to atopic dermatitis-related loci on chromosome 3q21. The homozygous frequency for the rs16830494 minor allele (AA) and the rs59021909 (TT) allele and the rs200963433 heterozygous (CT) frequency were all higher in AD cases compared to controls in a population-based case-control study. ERBB2 (rs1058808) were no association. Whereas, TT and CT genotypes combined of CDKAL1 (rs77152992) were significantly decreased risk for AD and associated with total eosinophil.

**Conclusion:** Our study suggests that COL6A6 variants may be risk factors for AD. CDKAL1 (rs77152992) may be able to have beneficial role by decreasing the risk of AD. We also observed significant association between CDKAL1 variant and eosinophil. This study provides a genetic basis for early-onset AD diagnosis in Korean patients and the development of new therapies.
p63 controls the expression of specific genes involved in epithelial tissue integrity and homeostasis, cell survival, as well as migration and EMT-related features of cancer cells. The TP63 gene encodes two main isoforms through the use of alternative promoters. The TAp63 transcripts are generated from a promoter upstream of exon 1 and encode p63 isoforms that contain the canonical N-terminal transactivating domain (TAD). The usage of an alternative promoter within intron 3 leads to the production of truncated ΔNp63 isoforms partially lacking the TAD. Alternative splicing occurring at the 3’ end of the TP63 RNA generates at least three C-terminal variants (α, β, and γ) for both TAp63 and ΔNp63. ΔNp63α is predominantly expressed in epithelial stem cells and undifferentiated basal keratinocytes, where it acts as a critical proliferative factor. Previous reports showed that the p63 could be post-translationally modified (PTM) with phosphorylation, acetylation, sumorylation etc, which influence the molecular and cellular function of p63.

O-linked β-D-N-acetylglucosamine (O-GlcNAc) modification (O-GlcNAcylation) onto serine and threonine residues of proteins is an important PTM, which is involved in many crucial biological processes including transcription, translation, proteasomal degradation, and signal transduction. O-GlcNAcylation is, 1) a monosaccharide modification onto hydroxyl groups of Ser/Thr residues, which is not elongated to complex sugar structures; 2) almost exclusively on proteins localized in the nucleus, cytoplasm, and mitochondria; 3) reversible and highly dynamic, which is controlled by two enzymes: O-GlcNAc transferase (OGT) (which catalyzes the addition of O-GlcNAc to Ser/Thr residues) and β-D-N-acetylglucosaminidase (O-GlcNAcase) (which removes O-GlcNAc). Aberrant O-GlcNAcylation of protein is directly linked to the pathological progression of chronic diseases including diabetes, cancer, and neurodegenerative disorders.

Here we showed that endogenously expressed ΔNp63α was O-GlcNAcylated in HaCaT keratinocytes. Though the extensive mutagenesis analysis, we identified that the 528th serine residue of ΔNp63α is susceptible to O-GlcNAcylation. Currently we have been focused on the molecular and cellular analysis for validating the functional significance of the O-GlcNAcylation on ΔNp63α 528S during the maintenance of stemness of keratinocytes, cellular proliferation and/or differentiation, which will unveil O-GlcNAcylation dependent functional modification of ΔNp63α.
PO-11

Inhibition of collagen production by ICG-001, a small molecule inhibitor for Wnt/β -catenin signaling, in skin fibroblasts

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Background: Wnt/β -catenin signaling is important in development and differentiation of melanocytes.

Objective: In this study, we evaluated the effects of ICG-001, a small molecule inhibitor for Wnt/β -catenin signaling, on collagen expression in human normal skin- and keloid-derived fibroblasts.

Methods: Fibroblasts were treated with Wnt/β -catenin signaling inhibitor, ICG-001, and then collagen expression level were checked.

Results: To evaluate the effect of ICG-001 on type I collagen synthesis, we performed Western blot and RT-PCR. Treatment of normal fibroblasts with ICG-001 resulted in dramatic decrease of type I collagens in the dose- and time-dependent manners. Also, the secretion of procollagen was significantly reduced by ICG-001. The gel contraction was occurred in a time-dependent manner in control groups, while ICG-001 treatment resulted in significant inhibition of gel contraction. Moreover, ICG-001 markedly inhibited the type I collagen synthesis in keloid fibroblasts. Next, we wondered if inhibition of Wnt/β -catenin signaling affects TGF-β signaling. Pretreatment with ICG-001, however, failed to inhibit TGF-β-induced phosphorylation of Smad2/3. We first confirmed whether ICG-001 actually inhibited Wnt/β -catenin signaling in dermal fibroblasts. Cells were transduced with TOPflash reporter adenovirus, then treated with ICG-001. Also, overexpression of β -catenin markedly diminished the inhibitory potential of ICG-001 on type I collagen production.

Conclusions: In summary, we demonstrated that ICG-001 inhibited the collagen production in normal and keloid fibroblasts. Our data suggest that ICG-001 can be applied as therapeutics for fibrotic skin diseases.
PO-12

NecroX-5 inhibits poly(I:C)-induced inflammatory reaction of keratinocytes

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**Background:** NecroX-5 is a cell permeable necrosis inhibitor with cytoprotective effects. It can inhibit mast cell degranulation and the development of a novel antiallergic agent that suppresses IgE-dependent mast cells signaling. However, the anti-inflammatory effect of NecroX-5 on keratinocytes has not yet been fully elucidated.

**Objective:** In this study, we aimed to examine whether NecroX-5 has the potential to inhibit the inflammatory response of keratinocyte induced by double-stranded RNAs mimicking poly (I: C).

**Methods:** Induction of inflammatory response of normal human keratinocytes was induced by poly (I: C) treatment. The therapeutic effect of NecroX-5 on the early apoptosis and inflammatory responses of keratinocytes was measured by flow cytometry for annexin V and RT-PCR for proinflammatory and inflammatory cytokines, respectively.

**Results:** WST-1 assay showed that administration of 10 μM NecroX-5 resulted in a decreased cell viability. NecroX-5 increased annexin V in human keratinocytes and inhibited the poly (I: C) -induced mRNA expression of IL-1β, IL-6, IL-8 and TNF-α as increased annexin V in human keratinocytes.

**Conclusion:** These results suggest that NecroX-5 might be a potential candidate which regulates cutaneous inflammatory diseases such as psoriasis.
PO-13

A cell model for Th2 allergic inflammation in keratinocytes

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Background: Reactive oxygen species (ROS) plays an important role to activate the Th2 type allergic inflammation in asthma or rhinitis.

Purpose: This study was aimed to evaluate whether ROS is implicated in the pathogenesis of Th2 inflammation in the skin. Compound 48/80-treated HaCaT cells were tested for a new cell model.

Materials and Methods: In compound 48/80-treated HaCaT cells, ROS production, PI3K-HIF-1α activation, and expression levels of biomarkers for Th2 inflammation in the skin were evaluated. The role of ROS on Th2 inflammation in the skin were verified in the compound 48/80-treated HaCaT cells, which were treated with N-acetyl cysteine (NAC).

Results: Compound 48/80 induced the ROS production, PI3K-HIF-1α, as it modulated biomarkers for Th2 inflammation in the skin: upregulation of PAR2 (pro-Th2 inflammatory marker), TSLP and IL-33 (Th2 inflammatory markers), and NGF and CGRP (neurogenic inflammatory markers); and downregulation of filaggrin (barrier function marker). The compound 48/80-induced modulation of those biomarkers could be suppressed by antioxidant of NAC, indicating that ROS plays a crucial role during Th2 type allergic inflammation in keratinocyte.

Conclusion: We propose a new cell model for Th2-type allergic inflammation in KCs
A functional role of GDA as melanogenic stimulator

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Background: Guanine deaminase (GDA)—a ubiquitous enzyme—causes the hydrolytic deamination of guanine and mediates the initial stage of purine metabolism, and is thus involved in xanthine and tyrosine metabolism. Previous studies in Korea have shown that GDA is upregulated in melasma lesional skin, as compared to normal adjacent skin. Xanthine metabolism is reportedly involved in darker Fitzpatrick skin phototypes. Although GDA is an important enzyme in many signaling pathways in neuron and cancer biology, the effects of GDA on melanogenesis remain unclear.

Methods: We examined the expression status of GDA in hyperpigmentation disorders and its related mechanisms in a co-culture model of normal human melanocytes (NHMs) with keratinocytes using western blot and quantitative real-time PCR.

Results: Our results showed that ultraviolet B (UVB) irradiation induced the mRNA and protein expression of GDA in monocultures of NHMs and normal human keratinocytes (NHKs), respectively. In a co-culture model of NHM, GDA expression was upregulated in a dose-dependent manner by UVB exposure. Following co-stimulation with stem cell factor (SCF) and endothelin-1 (ET-1), the melanin content of co-culture increased up to 20%, and GDA and tyrosinase expression increased simultaneously. Treatment with GDA siRNA reduced GDA mRNA expression and melanin content in the co-culture model. In Mel-ab cells, L-750,667—a ive antagonist of the dopamine receptor—suppressed GDA expression. Attenuation of GDA activity by GDA inhibitors led to dendrite reduction and shortening and focal swelling in NHMs at 3 days after treatment. Western blot and quantitative real-time PCR of hyperpigmented skin lesions also showed increased GDA expression, as compared to normal adjacent skin.

Conclusion: GDA is involved in melanogenesis and dendrite formation and its modulation may thus serve as an alternative treatment for skin pigmentary disorders.

Key words: Guanine deaminase; Melanogenesis; Dendrite; Hyperpigmentation disorders
PO-15

The role of TGF-β3 in melanogenesis and senescence

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Background: Transforming growth factor-β (TGF-β) is the cytokine that regulating physiological and pathological effects, such as proliferation, differentiation, apoptosis, angiogenesis and metastasis. TGF-β family has three isoforms: TGF-β1, β2, and β3. These homologous forms show different activities in certain cell types and systems. Contrary to TGF-β1 or β2, TGF-β3 is expressed in fetal developing skin and known to decrease the scar formation.

Objective: The present study aims to investigate the effect of TGF-β3 in B16F10 melanoma cells, normal human melanocyte (NHM), keratinocyte, and fibroblast cells.

Methods: Melanin contents were measured and expression of proteins related to proliferation and differentiation was determined.

Results: TGF-β3 inhibited the melanin production, tyrosinase activity in B16F10 cells. TGF-β3 decreased the melanin in NHM and NHM-keratinocyte co-culture. TGF-β3 also decreased tyrosinase activity in NHM-keratinocyte co-culture. We then assessed the impact of TGF-β3 on the proliferation and senescence. TGF-β3 slightly decreased the proliferation of keratinocyte and increased the proliferation of fibroblast. TGF-β3 increased the expression of involucrin in keratinocyte. Because Smads are known to play important roles in TGF-β signaling, we confirmed the expression of Smad in keratinocyte and fibroblast. TGF-β3 induced the expression of phosphorylated form of Smad 2 or 3 in both keratinocyte and fibroblast.

Conclusion: Altogether, our findings suggest that TGF-β3 suppress melanogenesis and reverse senescence.
**PO-16**

**Clusterin is secreted from endothelial cells and regulates pigmentation**

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**Background:** Cutaneous vasculatures play a role in regulating skin pigmentation.

**Objective:** To identify novel factors secreted from endothelial cells controlling pigmentation and to determine the paracrine role of endothelial cells-derived clusterin on melanogenesis

**Method:** We analyzed the RNA seq to identify novel factors secreted from endothelial cells controlling pigmentation. The endogenous expression of clusterin in HDMECs was investigated using real-time PCR and western blot. HDMECs were infected with clusterin-lentivirus or shRNA. Normal human melanocytes were treated with a conditioned medium obtained from the HDMECs and the pigmentation was analyzed.

**Results:** Transcripts for clusterin were highly expressed in HDMECs with levels being 1.31-fold higher than transcripts for TGF β 1. The melanin contents and tyrosinase activity were significantly reduced in the presence of clusterin overexpressed HDMECs. The mRNA and protein expression levels of melanogenesis-associated proteins, microphthalmia-associated transcription factor (MITF), and tyrosinase were also significantly down-regulated. Consistently, clusterin downregulation in the HDMECs was associated with increased melanogenesis in melanocytes.

**Conclusion:** Clusterin is secreted from endothelial cells and inhibits melanogenesis.
Background: Neural crest stem cells (NCSC) can migrate and differentiate into multipotent lineages of various functional cells including Schwann cell and melanocytes. In the bulge of adult human follicle, various kinds of stem cells are present such as pluripotent epithelial stem cells, melanocyte stem cells and NCSCs. BMP4 is capable of inducing MITF expression in NCSCs and postulated melanocyte stem cells, and α-MSH subsequently promotes differentiation of MITF expressing cells along the melanocyte lineage.

Objective: The aim of this work is to ascertain whether the NCSCs could be isolated from human hair follicle bulge and induced to differentiate along the melanocyte lineage by BMP-4 and α-MSH. And we try to ascertain the NCSCs could be utilized as the cell sources for better repigmenting therapy in vitiligo treatment in terms of survival and stability of melanocytes.

Methods and Results: We obtained a population of cells that expressed markers of NCSC, SOX10 during the emigration culture of hair follicle bulge portions from adult human scalp. First, an increased proliferation of SOX10 positive cells were observed in bFGF-added condition, compared to control. Second, the postulated melanocyte precursor cells expressed MITF after treatment of BMP-4 and α-MSH. Third, the emigrated hair bulge cells spontaneously differentiated into Schwann cell progenitors expressing SOX2 after prolonged cultivation, without any MITF expression detected.

Conclusion: The data showed that bFGF promotes the proliferation and survivals of NCSCs. And BMP-4 and α-MSH are capable of inducing MITF expression and promoting a further differentiation along the melanocyte lineage from NCSC.
PO-18

Double-stranded RNA induces inflammation via the NF-κB pathway and inflammasome activation in the outer root sheath cells of hair follicles

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Alopecia areata (AA), a chronic, relapsing, hair-loss disorder, is considered to be a T cell-mediated autoimmune disease. It affects approximately 1.7% of the population, but its precise pathogenesis remains to be elucidated. Despite the recent attention focused on the roles of inflammasomes in the pathogenesis of autoinflammatory diseases, little is known about inflammasome activation in AA. Thus, in this study, we investigated the pattern of NLRP3 inflammasome activation in the outer root sheath (ORS) cells of hair follicles. We found that interleukin (IL)-1β and caspase-1 expression was increased in hair follicle remnants and inflammatory cells of AA tissue specimens. After stimulation of ORS cells with the double-stranded (ds)RNA mimic polyinosinic:polycytidylic acid (poly[I:C]), the activation of caspase-1 and secretion of IL-1β were enhanced. Moreover, NLRP3 knockdown decreased this poly(I:C)-induced IL-1β production. Finally, we found that high-mobility group box 1 (HMGB1) translocated from the nucleus to the cytosol and was secreted into the extracellular space by inflammasome activation. Taken together, these findings suggest that ORS cells are important immunocompetent cells that induce NLRP3 inflammasomes. In addition, dsRNA-induced IL-1β and HMGB1 secretion from ORS cells may contribute to clarifying the pathogenesis and therapeutic targets of AA.
PO-19

Gasdermin C is induced by ultraviolet and contributes to MMP-1 expression via activation of ERK and JNK pathways

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Background: Ultraviolet (UV) irradiation contributes various skin diseases including photoaging, cancer and inflammation. UV is known to regulate the expression of a wide variety of genes including matrix metalloproteinases (MMPs). Gasdermin C (GSDMC) belongs to Gasdermin family and is known to be expressed in epithelial cells and metastatic melanoma cells. However, the functions of GSDMC in the skin have not been reported yet.

Objective: In this study, we investigated the regulation of GSDMC expression by UV and the role of GSDMC in the expressions of various MMPs induced by UV in human skin tissues and human skin keratinocytes.

Methods: Human skin, normal human epidermal keratinocytes (NHEK) and an immortalized human keratinocyte cell line (HaCaT cell) were irradiated with UV. Knockdown or overexpression of GSDMC was performed to study the effect of GSDMC. mRNA and protein levels of many targets were analyzed by quantitative real-time polymerase chain reaction (qRT-PCR) and Western blotting, respectively. GSDMC expression in human skin was also analyzed by immunohistochemical staining.

Results: UV irradiation increased GSDMC expression at the mRNA and protein levels in human skin, NHEK and HaCaT cells. Knockdown of GSDMC reduced the MMP-1 expression and the ERK and JNK activities induced by UV. Furthermore, overexpression of GSDMC increased the MMP-1 expression and the activities of ERK and JNK.

Conclusion: Our results show that GSDMC is increased by UV irradiation, which contributes to the increase in MMP-1 expression via activation of ERK and JNK pathways. Therefore, we suggest that GSDMC plays an important role in UV-induced MMP-1 expression.
PO-20

Epithelial precursor cell-conditioned media ameliorates UV irradiation-induced extracellular matrix damage in human skin equivalents

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**Background:** Human skins protect their physiological and structural integrities from various insults such as UVB-irradiation (UV-IR). UV-IR induces multiple downstream signaling pathways that regulate expression of genes. Although human stem cell conditioned mediums show the regenerative effects on photo-aged skin, the molecular mechanism of epithelial precursor cell-conditioned media (EPCs-CM) action on photo-aged skin is not elucidated yet.

**Objective:** We examine whether EPCs-CM supplementation attenuates UVB-mediated skin photo-aging and regulate UVB-induced ERK signaling on dermal fibroblasts and 3-dimentional human skin equivalents (3D HSE).

**Methods:** 3D HSE has been used with the aim of analyzing regenerative photo-protection after UV exposure. EPCs-CM treatment is carried out with various concentrations on dermal fibroblasts (DFs). Protein expressions of MMP1 and procollagen-I in skin aging are evaluated by Western blot, and Immunohistochemistry (IHC). Modulation of ERK signaling by EPCs-CM treatment are examined in DFs and 3D HSE exposed to UVB-IR.

**Results:** Our current studies show that UVB-IR up-regulates the expression of MMP-1 and decreases the level of procollagen-I in primary human dermal fibroblasts respectfully. However, EPCs-CM treatment recovers the expression of MMP-1 and procollagen-1 regulated by UVB-IR. Furthermore, EPCs-CM delays UVB-mediated senescence status and inhibits UVB-induced ERK phosphorylation in 3D HSE.

**Conclusion:** Therefore, our studies demonstrate that EPCs-CM may protect skin damages via inhibiting UVB-mediated ERK activation, suggesting that EPCs-CM can be useful material for repair and regeneration of photo-aged skin.
PO-21

Preventive effect of Cacao extract on UVB-induced skin wrinkle formation via inhibition of DNA methylation

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Background: Cacao beans contain various bioactive phytochemicals that can attenuate or delay the onset of disease condition. However, the effect of cacao powder (CP) on UVB-induced wrinkle formation and the molecular mechanisms responsible has not previously been explored.

Objective: We aim to study the protective effect of CP on UVB-induced wrinkle formation by regulating DNA methylation and gene expression.

Methods: We use pattern matched gene analysis of transcriptome and DNA methylome in mouse skin tissues. Gene expression and gene promoter methylation profiles are obtained from RNA and DNA samples that were extracted from human skin cell lines. Protein expression patterns are confirmed with immune-staining analysis and western blot.

Results: Transcriptome analysis revealed that 853 genes are down- or up-regulated with CP supplementation, compared with UVB-irradiated mouse skin controls. CP elicited anti-wrinkle effects via inhibition of UVB-induced MMP-1 expression in a skin equivalent model and human dermal fibroblasts (HDFs). Inhibition of UVB-induced AP-1 via CP supplemtations is likely to affect the expression of MMP-1. In addition, pattern-matched analysis of transcriptome and DNA methylome provide many new signature molecules regulated by CP and UVB. CP specifically modulated the expression of gene coding for the human profilin1 (PFN1) by suppression of their DNA methylation. Furthermore, 5’-(3’,4’-Dihydroxyphenyl)- γ -valerolactone (DHPV), a major in vivo metabolite of CP, showed effects similar to CP supplementation such as inhibition of PFN1 gene promoter methylation and upregulation of PFN1 expression in human keratinocyte cell.

Conclusion: These results suggest that cacao extract may offer a protective effect against the photoaging process by epigenetic regulation of signature molecules, leading to an overall reduction in wrinkle formation.
**PO-22**

**PD-1 expression in cutaneous extranodal NK/T-cell lymphoma: its effect on clinical characteristics**

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**Background:** Recent studies have evaluated the expression of programmed death-1 (PD-1) and its prognostic value in malignant T-cell lymphomas.

**Objectives:** This study investigated whether the positivity of PD-1 was associated with the clinical characteristics of cutaneous extranodal NK/T-cell lymphoma (ENKTL) and evaluated its effects on survival outcomes.

**Methods:** Forty-one patients with cutaneous ENKTL were included. Clinical features and survival outcomes were analyzed according to the positivity of PD-1.

**Results:** There was no significant difference between primary cutaneous ENKTL and secondary cutaneous ENKTL in the expression of PD-1. The degree of disease dissemination was not affected by the positivity of PD-1. Higher positivity for PD-1 was associated with lesions presenting erythematous to purpuric patches that are mainly composed of small tumor cells. Cutaneous ENKTL presenting nodular lesions had a significantly lower number of PD-1-positive infiltrating cells than those with other clinical morphologies. There was no significant effect of PD-1 expression on outcomes such as overall and progression-free survival.

**Limitations:** The present study used a retrospective design and had a small sample size.

**Conclusion:** Higher PD-1 positivity is associated with small-cell-predominant cutaneous ENKTL. However, PD-1 expression has no prognostic value in cutaneous ENKTL.

Key words: Programmed death-1; Skin; Natural killer/T-cell lymphoma; Lymphoma; Survival; morphology
PO-23

Beneficial effects of *silybum marianum* extract and silymarin on regulation of decorin and biglycan in human dermal fibroblasts

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**Background:** *Silybum marianum* (milk thistle) extract (SME) is well known to have a beneficial effect on improvement of liver health; however, its effect on skin aging has been rarely studied. Major active component of SME is known as silymarin, which contains silibinin, isosilibinin, silicristin, silidianin, and others. In skin aging, loss of collagen and increase of their degrading enzyme, matrix metalloproteinase-1 (MMP-1), and reduction of collagen-supporting proteoglycans, including decorin and biglycan, are notable characteristics.

**Objective:** To investigate the effects of SME on the matrix protein regulation in primary cultured human dermal fibroblasts (HDFs).

**Methods:** Detection of decorin and biglycan protein levels and sizes were examined with or without treatment with chondroitinase ABC by Western blot. Inhibitory effect test on granzyme B and neutrophil elastase was performed by incubation of recombinant human biglycan with active granzyme B or neutrophil elastase, and the inhibition of biglycan degradation was detected by Western blot.

**Results:** Treatment with SME increased expressions of procollagen, decorin, and biglycan in HDFs, while reduced MMP-1 expression. Furthermore, molecular sizes of decorin and biglycan were also elevated, suggesting that longer dermatan sulfate chain synthesis was induced by treatment with SME. Treatment with silymarin also showed similar results in HDFs. In addition, we also found that both SME and silymarin have direct inhibitory effects on granzyme B and neutrophil elastase, which are enzymes degrading proteoglycans, using recombinant human biglycan and decorin. Degradation of recombinant human biglycan and decorin by recombinant human granzyme B or neutrophil elastase were blocked by addition of SME or silymarin.

**Conclusion:** Taken together, because SME and silymarin can increase the production of matrix proteins and even inhibit the degradation of biglycan and decorin, they can be new considerable candidates for anti-aging molecules.
**PO-24**

**Plant oils have antioxidant activity in UVB-irradiated NHEKs by upregulating detoxifying enzymes**

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**Background:** Plant-derived extracts including essential oils have long been used as important ingredients of cosmetics. In recent years, there has been a demand for safe cosmeceuticals with excellent antiaging activity.

**Aim:** This study aimed to develop candidate plant extracts with excellent antioxidant and detoxifying activities in keratinocytes with a view to protecting photoaging of the skin.

**Methods:** The ROS-scavenging activities of candidate plant essential oils were determined in ultraviolet B (UVB)-irradiated normal human epidermal keratinocytes (NHEKs). Their antioxidant and detoxifying activities were determined by measuring enzymatic antioxidants, phase 2 enzymes, and total polyphenol content.

**Results:** In DCF-DA and confocal microscopy studies, the plant essential oils including Lavender oil, Lemongrass oil, Rosemary oil, Chamomile oil, and Peppermint oil showed excellent ROS-scavenging activity in UVB-irradiated NHEKs. In RT-PCR studies, all these oils upregulated the expression of enzymatic antioxidants (CuSOD, GPx II, and Prx I) and phase 2 detoxifying enzymes (HO-1, NQO-1, GSTpi, GSTA4, and GCLM) via Nrf2 in NHEKs.

**Conclusions:** All of the five essential oils can be regarded as good candidate natural products with potential for use as new cosmeceuticals.
PO-25

Reductions in matrix metalloproteinase and collagen transcription by decreasing signal transduction through the Transforming growth factor-β/Smad pathway in normal senescing human dermal fibroblasts

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Background: Transforming growth factor-β (TGF-β) plays an important role in regulating cell growth, differentiation, and biosynthesis. Smads are important intracellular components of the TGF-β signal transduction pathway. The TGF-β/Smad pathway is the major regulator of biosynthetic processes in the extracellular matrix (ECM), including the synthesis of collagen type I and type III by skin fibroblasts. However, the mechanism controlling the expression of matrix metalloproteinases (MMPs) and collagen in human dermal fibroblasts is not well understood.

Objective: We investigated whether TGF-β/Smad signaling could regulate transcription of MMPs and collagen and whether NF-κ B and mitogen-activated protein kinase (MAPK) signaling pathways were involved in the aging of normal human dermal fibroblasts.

Methods: Normal human dermal fibroblasts were isolated from tissue removed after the circumcision of two 13- and 14-year-old males. The expression of mRNA and protein was quantified by real-time polymerase chain reaction and immunoblot analysis from passage numbers 5 to 15.

Results: TGF-β 1, TGF-β 3, and TGF-β receptor type I (TGF β RI) transcript levels decreased with increasing passage numbers. TGF-β 2, TGF β RII, and TGF β RIII mRNA levels increased in passage 10 but decreased in passage 15. Smad2, 3, 4, and 7 decreased with increasing passage numbers. MMP-1 accumulated with increasing passage numbers, and MMP-2, tissue inhibitor of metalloproteinase (TIMP)-1, and TIMP-2 all increased in passage 10 but decreased in passage 15. Collagens type I and II decreased with increasing passage numbers. The Smad3, NF-κ B, Iκ B α , p38, extracellular signal-regulated kinase (ERK), AKT, and c-Jun N-terminal kinase (JNK) proteins became phosphorylated with increasing passage numbers.

Conclusion: These results suggest that the reduction of MMP and collagen type I and III expression in aging human dermal fibroblasts is regulated by reduced expression of TGF-β/Smad and TGF-β receptors and by the lower TGF-β receptor binding capacity of fibroblasts, suggesting active roles for NF-κ B and the MAPK signaling pathway.

Key words: Transforming growth factor-β, Smad, matrix metalloproteinases, Collagen
A case of vascular Ehlers–Danlos syndrome with a novel mutation in COL3A1

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The vascular type of Ehlers–Danlos syndromes (vascular EDS) is a rare autosomal dominantly inherited connective tissue disorder that is characterized by easy bruising, thin skin with visible veins, characteristic face, and rupture of arteries, uterus, or intestines. The diagnosis is confirmed by abnormal diameter, contour, and shape of collagen fibrils/fibers observed in situ or in vitro or by the identification of a pathogenic mutation in the gene for type III procollagen (COL3A1). The majority of identified pathogenic variants results in single amino-acid substitutions for glycine in the Gly-X-Y repeat of the triple helical region of the type III procollagen molecule. The next common variants occur at splice sites, that result in exon skipping. We reported a case of vascular EDS with a characteristic ‘Madonna’ face, fragile uterus, and easy bruising in addition to a past history of cavernous sinus fistula. To confirm the diagnosis, we demonstrated decreased production of type 3 collagen by cultured fibroblasts and its diameter variation and identified a novel mutation of the splice site of COL3A1 gene (g.IVS14+2T>G). To determine the significance of the identified mutation, we next examined whether this mutation is included in the pathogenic mutations of COL3A1 reported in several databases or in the single nucleotide variations (SNVs) in the whole genome sequences of 2,049 healthy Japanese individuals reported by Tohoku medical megabank organization (ToMMo) cohort study. This mutation identified in this case is not included in either database, suggesting that it is an extremely rare SNV of COL3A1 and is not included in the list of the pathogenic COL3A1 mutations. These results demonstrated that our case is vascular EDS with a novel mutation of COL3A1.
Background: Propionibacteria acnes (P. acnes) is a well-known commensal bacterium and plays an important role in pathogenesis of acne vulgaris. During the infection, P. acnes induces the inflammation in human skin and contribute to chronic inflammatory disease.

Objective: In this study, we tried to investigate the effect of SOD3, an antioxidant enzyme, on P. acnes/peptidoglycan (PGN)-induced inflammation in vitro.

Methods: Western blot, RT-PCR methods were used to analyse gene and protein expressions. Intracellular lipid accumulation was determined by Oild red O staining.

Results: Our data indicates that SOD3 downregulated the Toll-like receptor (TLR)-2 expression at mRNA and protein levels in P. acnes/peptidoglycan (PGN) treated HaCaT, keratinocyte cell line cells and SB95, sebocyte cell line. Moreover, we found that SOD3 suppressed the expressions of p-NF-κB and p-p38 in P. acnes/PGN treated both cell lines, whereas did not have any effects on expressions of p-JNK and p-ERK. SOD3 also exhibited a role of anti-inflammation via reducing expressions of inflammasome related proteins (NLRP3, ASC, caspase-1) as well as inhibition of cytokine expressions (TNF-α, IL-1β, IL-6, IL-8) in vitro. In addition, SOD3 reduced the lipid accumulation in sebocytes during the P. acnes infection.

Conclusion: With these findings, we believe that this study will provide valuable information for developing the effective clinical applications against P. acnes associated acne vulgaris.
Adiponectin signaling regulates lipid production in human sebocytes

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Adiponectin plays important roles in metabolic function, inflammation and multiple biological activities in various tissues. However, evidence for adiponectin signaling in sebaceous glands is lacking, and its role remains to be clarified. This study investigated the role of adiponectin in lipid production in sebaceous glands in an experimental study of human sebocytes. We demonstrated that human sebaceous glands invivo and sebocytes invitro express adiponectin receptor and that adiponectin increased cell proliferation. Moreover, based on a lipogenesis study using Oil Red O, Nile red staining and thin layer chromatography, adiponectin strongly upregulated lipid production in sebocytes. In three-dimensional culture of sebocytes, lipid synthesis was markedly enhanced in sebocytes treated with adiponectin. This study suggested that adiponectin plays a significant role in human sebaceous gland biology. Adiponectin signaling is a promising target in the clinical management of barrier disorders in which sebum production is decreased, such as in atopic dermatitis and aged skin.

Keywords: Adiponectin, Sebocytes, Lipid, Sebum, Barrier
PO-29

LRG1 is involved in skin aging by upregulation of matrix metalloproteinase-1 and downregulation of type 1 collagen in human dermal fibroblasts

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Background: Intrinsic and extrinsic factors involved in skin aging often reduce collagen synthesis and increase expression of matrix metalloproteinases (MMPs). Compared with the extrinsic skin aging mainly by photo-damage, the mechanism of intrinsic skin aging relatively remains unclear.

Objective: Genes involved in the intrinsic skin aging were screened from skin tissues of young and old mouse model. The role of a screened gene in extracellular matrix integrity of skin and intrinsic skin aging was characterized.

Methods: Differentially expressed genes (DEGs) were analyzed by RNA sequencing of dorsal skin tissues from 3-month-old (young) and 24-month-old (old) SKH1 hairless mice. Expression of potential target genes were analyzed by RT-PCR, western blotting and immunohistochemistry.

Results: The gene encoding leucine-rich alpha-2-glycoprotein 1 (LRG1) was found to be significantly down-regulated in skin tissues of the old group. Although LRG1 is known as a positive regulator of transforming growth factor-beta signaling pathway, its function and detailed mechanism in skin aging has not been studied well. Lrg1 mRNA level using RT-PCR and LRG1 protein level by western blotting and immunohistochemistry markedly decreased in the old group compared with the young group. In addition, treatment of recombinant human LRG1 (rhLRG1) increased synthesis of type I collagen and decreased secretion of MMP-1 in human dermal fibroblasts. Moreover, LRG1-induced type I collagen up-regulation and MMP-1 down-regulation involve activation of TGF-beta signaling pathway.

Conclusion: Our findings indicate that LRG1 has a potential to prevent skin aging by up-regulation of type I collagen and down-regulation of MMP-1. Therefore, LRG1 may play an important role in retardation of intrinsic skin aging.
PO-30

A B cell subset that produces IL-10 suppresses contact dermatitis

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Background: B cells secrete antigen-specific antibodies during immune responses to neutralize pathogens and foreign antigens. Despite this well-characterized pro-inflammatory B cell function, a rare B cell subset (B10 cells) in both humans and mice negatively regulates inflammation and autoimmunity by producing the inhibitory cytokine interleukin (IL)-10. It has been reported that B10 cells have suppressive effects on autoimmune diseases in mouse models. Nonetheless, therapeutic effect on contact dermatitis has not been reported. Furthermore, several signaling pathways are involved in B10 cell development, including the Toll-like receptor, CD40 and B cell antigen receptor (BCR) pathways. Many studies have suggested that both BCR specificity and signaling strength are involved in B10 cell development. However it remains unknown whether BCR specificity is essential for B10 cell function.

Objective: To investigate whether B10 cells suppress contact dermatitis using a contact hypersensitivity (CHS) model mouse. If so, we assess whether antigen-specific BCR signals are essential for B10 effector cell-mediated regulation of immune responses in a CHS mouse model.

Methods: B10 cells are so rare as to make experiments difficult. Thereby, we established regulatory B cells expanding culture system using NIH-3T3-CD154/BLYS cells and exogenous IL-4 and IL-21, which allowed splenic B cells to expand about 25,000-fold. Then we characterized cultured B cells. Cultured B10 cells were adoptively transferred to CHS model mice. Then, to examine whether antigen specificity is essential or not, cultured B10 cells with or without oxazolone-specific BCR were adoptively transferred to oxazolone CHS model mice, and evaluated the effect of those on CHS inflammation.

Results: Cultured B cells demonstrated the competence to produce IL-10. Adoptively transferred cultured B10 cells suppressed inflammation in a CHS model. Cultured B10 cells with oxazolone-specific BCR could suppress oxazolone CHS inflammation, whereas cultured B10 cells without oxazolone-specific BCR could not suppress oxazolone CHS inflammation.

Conclusion: B10 cells have suppressive effect on contact dermatitis. Furthermore, antigen specificity is required for B10 cells to suppress contact dermatitis.
Bylaws of KSID

Chapter 1. General Provisions

• Article 1 Name of Association. The name of this General Incorporated Association shall be the Korean Society for Investigative Dermatology (hereinafter, the “Society”).

• Article 2 Objectives. The objectives of the Society shall be to promote basic and clinic research in dermatology and to make contributions to the advancement of dermatology. The Society is a non-profit organization.

• Article 3 Activities. The Society shall carry out the following activities to accomplish the purposes set forth in Article 2:
  1. Holding academic conferences and other meetings
  2. Publishing a Society Newsletter and a Society Journal
  3. Maintaining exchanges and partnerships with related domestic and overseas academic organizations
  4. Other activities to accomplish the aims of the Society

Chapter 2. Society Members

• Article 4 Membership eligibility and admissions. The Society shall consist of members who engage in and/or are interested in dermatological research and agree to the purposes of the Society and have passed the prescribed entrance formalities after their applications are reviewed and approved by Board of Directors.

• Article 5 Membership categories. The Society shall have the following members:
  1. Regular Members: Researchers, physicians and students in the dermatologic field or related fields, who reside in Korea and agree to the purposes of the Society
  2. Overseas Members: Researchers in the dermatologic field or related fields in a foreign country, who agree to the purposes of the Society
  3. Honorary Members: Persons who are nominated by the Board of Directors due to remarkable achievements in dermatologic research and great contributions for the advancement of the Society
  4. Organization Members: Organizations that agree with the purposes of the Society

• Article 6 Duties. The Society members shall observe the by-laws, various regulations and decisions of the Society. Regular members, overseas members and organization members shall pay their membership fee and other burden charges.
• **Article 7 Rights.** All members have the right to receive the official journal of the Society, *Annals of Dermatology*. Regular members have the right to vote, be eligible for election and have other prescribed voting rights.

• **Article 8 Expulsion.** When a member does not observe the duties imposed by the Society, damages the reputation of the Society, and/or has not paid the membership fee for more than 3 years without proper reasons, such member may be expelled from the Society by a resolution adopted by the Board of Directors at a General Assembly.

### Chapter 3. Officers

• **Article 9 Composition.** The Society shall have 1 President, 1 President-Elect, fewer than 50 members of Board of Directors, and 2 Auditors as its Officers.

• **Article 10 Appointment**

  1. The President and President-Elect shall be elected by a resolution of the Board of Directors and approved by the General Assembly.
  2. The Auditors shall be selected by the General Assembly.
  3. The Executive Directors shall be selected by the President. Directors shall be selected by the Committee for Recommendation of Directors; one-third of the Directors may be replaced per term upon approval by the General Assembly.

• **Article 11 Term of Office of Directors.** The term of office of all officers shall be 2 years, and officers may be reappointed with the exception of the President. The Present-Elect shall be selected 2 years prior to the assumption of his/her presidency. The term of office of Directors shall be 2 years, and Directors can be reappointed up to 4 times. The terms served as the President and Executive Directors shall not be included in the terms of reappointment for Directors. The term of office of a Director who has been selected to fill a vacancy shall expire when the term of office of his/her predecessor expires.

• **Article 12 Responsibilities**

  1. The President shall represent the Society, oversee overall businesses of the Society and preside over the General Assembly.
  2. The President-Elect shall take the office of the President in case the President is absent or the term of office of the incumbent President expires.
  3. The President shall oversee overall businesses of the Board of Directors and chair the meetings of the Board of Directors and the Executive Board of Directors.
  4. The Directors shall be members of the Board of Directors and review major matters related to the operation of the Society.
  5. The Executive Directors shall be members of the Board of Directors and Executive Board of Directors and execute overall businesses of the Society.
  6. Secretaries may be appointed in order to assist the Executive Directors to implement their duties. The Secretaries can attend the meetings of Board of Directors and Executive Board of Directors.
Chapter 4. Meetings

- **Article 13 Divisions.** The Society shall hold General Assemblies, Board of Directors meetings, and Executive Board of Directors meetings.

- **Article 14 General Assembly**
  1. The President shall convene a regular General Assembly once a year. However, an extraordinary session of the General Assembly shall be convened by the request of more than 1/5 of regular members or by the request of the Board of Directors.
  2. A General Assembly shall be formally established when 1/3 of regular members are present and shall adopt resolutions by a majority vote. However, when a General Assembly cannot be formally established, a General Assembly shall be valid when 1/3 of the regular members who have registered their attendance at the General Assembly are present, and resolutions shall be adopted by a majority vote.
  3. The General Assembly shall adopt resolutions regarding the following matters.
     (i) Election of Auditors
     (ii) Approval of the President, President-Elect, Directors, and Executive Directors
     (iii) Formulation of budget and settlement of accounts
     (iv) Approval of the amendments to the by-laws of the Society
     (v) Other matters presented by the Board of Directors

- **Article 15 Board of Directors**
  1. The Board of Directors shall consist of the President, President-Elect, Directors and Executive Directors and can be attended by the Auditors and Secretaries.
  2. The Board of Directors shall be established by the presence of the majority of the Directors, and resolutions adopted by a majority vote.
  3. A regular meeting of the Board of Directors shall be convened by the President two times a year. However, an extraordinary meeting can be convened by the President as often as needed and by the request of more than 1/3 of the Directors.
  4. The Board of Directors shall elect President and President-Elect, and review, resolve or approve overall matters necessary for the operation of the Society.

- **Article 16 Executive Board of Directors**
  1. The Executive Board of Directors shall consist of the President, President-Elect, General Secretary, Director of Academic Affairs, Director of Publications, Director of Financial Affairs, Director of Information, Director of International Affairs, and a few Directors without Portfolio. It shall review overall matters necessary for the operation of the Society and shall execute the appropriate businesses.
  2. Each of the Executive Directors shall fulfill the following affairs of the Society.
     (i) General Secretary: Managing Society businesses, affairs related to Society meetings, and
various matters related to Society member friendship and other affairs
(ii) Director of Academic Affairs: Academic conferences and academic matters
(iii) Director of Publications: Businesses related to Society Newsletter, Society Journal and other publications
(iv) Director of Financial Affairs: Businesses related to finances and accounting of the Society
(v) Director of Information: Businesses related to information, communication and website management and operation
(vi) Director of International Affairs: Businesses related to international communication

3. Each Executive Director may form the Operation Committee, Scientific Committee, Publication Committee, Financial Committee, Information Committee and International Committee, each consisting of several regular members.

Chapter 5. Finances

• Article 17 Source of Revenue. The financial resources of the Society shall consist of the membership fee, entrance fee, donations and other earnings.
• Article 18 Financial Year. The financial year of the Society shall commence on the date of the annual General Assembly and end on the date of the next annual General Assembly.
• Article 19 Audit. The financial accounts of the Society for the previous financial year shall be reported to the annual General Assembly after they have been settled and audited by the Auditors.


• Article 20 Amendment of By-Laws of Society. These by-laws shall be amended only after a review by the Board of Directors and approval by the General Assembly.
• Article 21 Governing Law. Any matter not set forth in the By-Laws of the Society shall be governed by other relevant laws and ordinances and general and common practices.
• Article 22 Promulgation of Amendments
  1. These by-laws shall be put into effect as of the promulgation date (March 23, 1991).
  2. The amendments to these by-laws shall be put into effect as of March 21, 1992.
  3. The amendments to these by-laws shall be put into effect as of March 20, 1993.
  4. The amendments to these by-laws shall be put into effect as of March 16, 1996.
  5. The amendments to these by-laws shall be put into effect as of March 16, 2002.
  6. The amendments to these by-laws shall be put into effect as of March 25, 2006.
  7. The amendments to these by-laws shall be put into effect as of Oct. 17, 2009.
  8. The amendments to these by-laws shall be put into effect as of March 29, 2015.
대한피부연구학회 회칙

제 1 장 총칙

제 1 조 (명칭) 본 학회는 대한피부연구학회(The Korean Society of Investigative Dermatology)라 한다.
제 2 조 (목적) 본 학회는 피부과학의 기초 및 임상 연구를 촉진하여 피부과학 발전에 기여함을 목적으로 하는 비영리단체다.
제 3 조 (사업) 본 학회는 제2조의 목적 달성을 위하여 다음과 같은 사업을 수행한다.
1. 학술대회 및 강연회 개최
2. 학회보 및 학회지의 발행
3. 국내, 국외의 관계 학술단체와의 교류 및 제휴
4. 기타 본 학회 목적 달성에 필요한 사업

제 2 장 회원

제 4 조 (자격) 본 학회의 회원은 피부과학의 연구에 종사하거나 피부과학 연구에 관심을 가지고 본 학회의 취지에 찬동하는 자로서 소정의 입회 수속을 받고 상임이사회의 심의 및 추천을 받은 후 이사회의 의결을 거친 자로 한다.
제 5 조 (구분) 본 학회의 회원은 다음과 같이 구분한다.
1. 정회원: 피부과학 또는 관련분야의 연구자로서 본 학회 목적에 찬동하는 자로 한다.
2. 국외회원: 외국에서 피부과학 또는 관련분야에 종사하는 자로서 본 학회 목적에 찬동하는 자는 국외회원이 될 수 있다.
3. 명예회원: 피부과학 연구 업적이 탁월하고 본 학회 발전에 공헌이 지대한 자는 명예회원이 될 수 있다.
4. 단체회원: 본 학회의 목적에 찬동하는 연구소는 단체회원이 될 수 있다.
제 6 조 (의무) 회원은 본 학회의 회칙, 제규정 및 결의사항을 준수하여야 하고, 정회원, 국외회원 및 단체회원은 회비 및 기타의 부담금을 납부할 의무가 있다.
제 7 조 (권리) 모든 회원은 본 학회에서 발간하는 학회지를 배부 받을 권리가 있으며 정회원은 선거권, 피선거권 및 기타 소정의 의결권을 가진다.
제 8 조 (제명) 본 학회의 의무를 준수하지 않거나, 본 학회의 명예를 훼손하거나, 정당한 이유없이 3년 이상 회비를 납부하지 않은 회원은 이사회의 의결을 거쳐 총회의 인준을 받아 제명할 수 있다.
제 3 장 임 원

제9조 (구성) 본 학회는 회장(President) 1명, 차기회장(President-elect) 1명, 이사(Board of Directors) 50명 미만, 감사(Auditors) 2명의 임원을 둔다.

제10조 (선임)
1. 회장과 차기회장은 이사회에서 투표로 선출하여 총회의 인준을 받는다.
2. 감사는 총회에서 선출한다.
3. 상임이사는 회장이 선임한다. 이사라는 이사추천회를 구성하여 선임하고, 임기당 1/3을 교체할 수 있으며, 총회의 인준을 받는다.

제11조 (임기) 임원의 임기는 2년으로 하며 연임 할 수 있다. 회장의 임기는 2년이며 연임할 수 없다. 차기회장은 취임 2년 전에 선출한다. 이사의 임기는 2년이며 4회까지만 할 수 있다. 단, 회장 및 상임이사의 임기는 이사의 연임기간에 포함되지 않는다. 전임자의 유고로 인해 보선된 임원의 임기는 전임자의 잔여 임기기간으로 한다.

제12조 (직무)
1. 회장은 본 학회를 대표하여 업무를 총괄하고 총회의 의장이 된다.
2. 차기회장은 회장 유고 시 그 직무를 대행하여 현 회장의 임기 후 회장직을 맡는다.
3. 회장은 이사회의 업무를 총괄하고 이사회 및 상임이사회의 의장이 된다.
4. 이사는 이사회의 구성원이 되며 본 학회의 운영의 주요한 사항을 심의한다.
5. 상임이사는 이사회 및 상임이사회의 구성원이 되며 본 학회의 제반업무를 집행한다.
6. 상임이사의 제반 업무를 보좌하기 위하여 간사를 둘 수 있다. 간사는 이사회 및 상임이사회에 참석할 수 있다.
7. 감사는 본 학회의 재산 상황과 사업과 관련된 사항을 감시하고 이를 총회에 보고한다.

제 4 장 회 의

제13조 (구분) 본 학회에는 총회, 이사회, 상임이사회를 둔다.

제14조 (총회)
1. 정기총회는 년 1회 회장이 소집한다. 단, 정회원 5분의 1 이상의 요구나 이사회의 요청이 있으면 임시총회를 소집하여야 한다.
2. 총회는 정회원의 3분의 1 출석으로 성립하고 재적인원 과반수로 의결한다. 단, 총회가 성립되지 않을 때는 총회에 참가 등록한 정회원의 3분의 1 출석으로 성립하고 재적인원 과반수로 의결한다.
3. 총회는 다음과 같은 사항을 의결한다.
   (1) 감사 선출
   (2) 회장, 차기회장, 이사, 상임이사 인준
   (3) 예산과 결산
   (4) 회칙 개정의 인준
   (5) 기타 이사회에서 제출한 사항
제 15조 (이사회)
1. 이사회는 회장, 차기회장, 이사와 상임이사로 구성하고 감사 및 간사가 참석할 수 있다.
2. 이사회는 이사, 과반수 충족으로 성립하고 재직인원 과반수로 의결한다.
3. 정기이사회는 년 2회 회장이 소집한다. 단, 임시 이사회는 회장 수시로 소집할 수 있으며, 이사 3분의 1이상의 요구가 있을 때 소집하여야 한다.
4. 이사회는 회장, 차기회장을 선출하며, 본 학회의 운영에 필요한 제반사항을 심의, 의결 또는 인준한다.

제 16조 (상임이사회)
1. 상임이사회는 회장, 차기회장, 총무이사, 학술이사, 간행이사, 재무이사, 정보이사, 국제협력이사, 및 약간 명의 무임소 이사로 구성하며 본 학회의 운영에 필요한 제반사항을 심의하고 업무를 집행한다.
2. 각 상임이사는 다음과 같이 회무를 분담한다
   1) 총무이사 : 본회의 관리, 회무 및 회원 상호간의 친목 등에 관한 업무 총괄
   2) 학술이사 : 학술대회 및 학술 등에 관한 업무
   3) 간행이사 : 학회보, 학회지 및 기타 간행 등에 관한 업무
   4) 재무이사 : 재정 및 회계 등에 관한 업무
   5) 정보이사 : 정보, 통신 및 홈페이지 관리와 운영에 관한 업무
   6) 국제협력이사 : 국제적 교류에 대한 업무
3. 각 상임이사는 정회원 약간명씩으로 구성된 운영위원회, 학술위원회, 간행위원회, 재무위원회, 정보위원회, 국제협력위원회를 구성할 수 있다.

제 5 장 재정

제 17조 (재원) 본 학회의 재원은 회비, 입회비, 찬조금 및 기타 수입금으로 하며 남은 양여금은 회원에게 배분하지 않는다.

제 18조 (회계 연도) 본 학회의 회계 연도는 매년 정기총회에서 다음 정기총회일까지로 한다.

제 19조 (감사) 본 학회의 수지결산은 감사의 감사를 거쳐 차기 정기총회에 보고한다.

제 6 장 부칙

제 20조 본 회칙의 개정은 이사회의 심의를 거쳐 총회의 인준을 받아야 한다.

제 21조 본 회칙은 규정되지 않은 절차는 일반 관례에 준한다.

제 22조
1. 본 회칙은 공포일(1991년 3월 23일)부터 시행한다.
2. 본 회칙은 1992년 3월 21일부터 개정 시행한다.
3. 본 회칙은 1993년 3월 20일부터 개정 시행한다.
4. 본 회칙은 1996년 3월 16일부터 개정 시행한다.
5. 본 회칙은 2002년 3월 16일부터 개정 시행한다.
6. 본 회칙은 2006년 3월 25일부터 개정 시행한다.
7. 본 회칙은 2009년 10월 17일부터 개정 시행한다.
8. 본 회칙은 2015년 3월 29일부터 개정 시행한다.
About KSID

- Founded in 1991

**Purpose**
- To achieve scientific excellence in dermatological research, to enhance communication amongst researchers, and to support career development of cutaneous biologists from academia and industry, both domestic and overseas

**Activities**
- Annual scientific meeting (spring)
- Annual research camp (fall)
- Publishing official journal: Annals of Dermatology

**Annual Scientific Meeting**
- Held in March or April of every year
- 2-day meeting
- Official language is English since 2009
- Program: invited lectures, research presentations (free communications and posters)
- Academic award: Uam Award since 1999
  - endowment by late Professor Young Pio Kim (1926-2013)
  - awarded to researcher with highest achievement each year

**Annual Research Camp**
- 1st camp held in 2009
- Two-day long program in the autumn
- At a remote location
- In casual attire
- With beer and barbeque
- Purpose: to foster exchange of ideas, encourage collaboration, build friendship and mentorship among more established researchers and young researchers in investigative dermatology and cutaneous biology
- Active, lively discussion is an integral part
Official Journals of KSID

- Journal of Korean Society for Investigative Dermatology
  - 1994 - 2009
  - Issued 4 times annually
- Annals of Dermatology
  - Published jointly with Korean Dermatological Association since 2010
  - Issued bimonthly
  - Indexed in SCI-E
  - 2014 impact factor 1.393
  - Contents: reviews, original articles, short communications in the field of dermatological research
대한피부연구학회 연혁

1991. 2. 2  발기인대회
강남성모병원에서 발기인 50인이 참석
1991. 3. 23  창립총회 및 제1차 학술대회 (르네상스호텔)
특강연자: Sadao Imamura (Kyoto Univ.)
박상대 (서울대 자연과학대)
일반연제 10권
제1대 회장단 취임
회장: 김영표, 이사장: 이정복
1991. 11. 23  제1차 심포지엄
주제: Interleukin, 발표: 6연제
강남성모병원
1992. 3. 21  제2차 학술대회 (르네상스호텔)
특강연자: Jouni Uitto (Thomas Jefferson Univ.)
교육강연 2연제, 심포지엄 황피부과학 6연제, 심포지엄 PCR 6연제,
포스터 19연제
제2차 총회에서 회칙개정
평의원제를 폐지하고 이사장, 상임이사제도를 신설
1992. 11. 7  제2차 심포지엄 (가톨릭의대 대학원 강의실)
주제: 피부세포배양, 발표: 6연제
가톨릭의대 대학원 강의실 130여명 참석
대한피부연구학회보 창간
1993. 3. 20  제3차 학술대회 (르네상스호텔)
특강연자: Irvin H. Epstein (USCF)
교육강연 6연제, 구연 13연제, 포스터 14연제
제2대 회장단 취임
회장: 이유신, 이사장: 윤재일
1993. 11. 6  제3차 심포지엄 (가톨릭의대 대학원 강의실)
주제: 분자생물학, 발표: 9연제
국외 초청연사: Peter Steinert (NIH)
정수일 (NIH)
1994. 3. 19  제4차 학술대회 (소피텔 앨버서더호텔)
특강연자: K. Nishioka (Tokyo Medical and Dental Univ.)
교육강연 4연제, 구연 15연제, 포스터 13연제
1994. 11. 5
제4차 심포지엄 (가톨릭의대 마리아홈)
주제: 광의학, 발표: 8연제
대한피부연구학회지 창간호 발간

1995. 3. 18
제5차 학술대회 (세라톤 위키힐호텔)
특강연자: Roger Allen (University Hospital Nottingham)
교육강연 5연제, 구연 14연제, 포스터 14연제
제3대 회장단 취임
회장: 허원, 이사장: 노병인

1995. 11. 25
제5차 심포지엄 (가톨릭의대 마리아홈)
주제: Apoptosis, 발표: 5연제
국외 초청연자: Kouchi Ikai (Kyoto 대학)

1996. 3. 16
제6차 학술대회 (서울중앙병원)
특강연자: Motomu Manabe (Juntendo Univ.)
교육강연 4연제, 구연 14연제, 심포지엄 (유전질환) 6연제, 포스터 8연제

1996. 11. 23
제6차 심포지엄 (호텔롯데)
주제: 노화와 광노화, 발표: 13연제
국외 초청연자: John J. Voorhees (Univ. of Michigan)
Masamitsu Ichihashi (Kobe Univ.)

1997. 3. 4
대한피부연구학회가 대한의학회 준회원으로 인준받음

1997. 3. 15
제7차 학술대회 (서울중앙병원)
특강연자: C. E. Orfanos (Free Univ.)
심포지엄 모발 특강연자: S. Arase (Tokushima Univ.) / R. Tsuboi (Juntendo Univ.)
/ A. G. Messenger (Royal Hallamshire Hospital) / 김정철 (경북대)
구연 14연제, 포스터 10연제
제4대 회장단 취임
회장: 이성낙, 이사장: 방동식

1997. 11. 15
제7차 심포지엄 (가톨릭의대 의과학연구원)
주제: 피부과학 연구에서 새로운 연구장비의 활용, 발표: 7연제
국외 초청연자 (SID sponsored lecturer): David A. Norris (Univ. of Colorado)
Warren W. Piette (Univ. of Iowa)

1998. 3. 14
제8차 학술대회 (가톨릭의대 의과학연구원)
특강연자: Kazuhiko Takehara (Kanazawa Univ.)
교육강연 3연제, 자유연제 15연제, 포스터 20연제
심포지엄 Wound healing, 5연제

1998. 9. 24
제8차 심포지엄
주제: Bullous dermatoses
특강연자: Grant J. Anhalt (Johns Hopkins Univ.)
발표: 5연제
1999. 3. 13
제9차 학술대회 (서울대학교병원 임상의학연구소)
특강연자: Alfred T. Lane (Stanford Univ.) / 이광훈 (연세의대) / Ressell P. Hall III (Duke Univ.)
자유연제 18연제, 포스터 21연제
심포지엄 피부질환 연구를 위한 동물실험기법 5연제
제1회 우암학술상 시상: 이광훈 (연세의대)
제5대 회장단 취임
회장: 고재경, 이사장: 온희철
대한피부연구학회지 년 4회 발행
1999년부터 년 2회에서 4회로 발행함

2000. 3. 17
제10차 학술대회 (서울대학교병원 임상의학연구소)
특강연자: Luis Diaz (North Carolina Univ.) / Irene Leigh (St.Bartholomew's and Royal London School) / Yasuo Kitjma (Gifu Univ.)
자유연제 18연제, 포스터 14연제, 심포지엄 자기면역질환 7연제
피부생물학 연수교육 12 강의
제2회 우암학술상 시상: 조광현 (서울의대)
재11차 학술대회

2001. 3. 17
특강연자: Masamitsu Ichihashi (고베 대학, 일본) / Ralf Paus (Hamburg Univ., Germany)
자유연제 16연제, 포스터 24연제, 심포지엄 피부생물학 연수교육 12 강의
제3회 우암학술상 시상: 김도원 (경북의대)
제5대 회장단 취임
회장: 이정복, 이사장: 이광훈

2002. 3. 15
제12차 학술대회 (가톨릭대학교 의과대학 의과학 연구원)
특강연자: Mark Udey (NIH NCI), Kunihiko Tamaki (Tokyo Univ.) / Thomas Luger (Westfälische Wilhelms Univ.), Setsuya Aiba (Tohoku Univ.)
자유연제 15연제, 포스터 27연제, 심포지엄 Dendritic Cell 7연제
피부생물학 연구교육 11강의
제4회 우암학술상 시상: 김수찬 (연세의대)

2003. 3. 28
제13차 학술대회 (강남성모병원 의과학연구원)
특강연자: Michael J. Detmar, M.D. / 미국 Harvard 의과대학 피부과 교수 / Stephen I. Katz, M.D., Ph.D. / 미국 NIAMS NIH원장 / Enno Christophers, M.D. / 독일 Kiel대학 피부과 교수
자유연제 18연제, 포스터 35연제, 심포지엄 6연제, 피부생물학 연수교육 8강의
제5회 우암학술상 시상: 서성준 (중앙의대)

2004. 3. 26
제14차 학술대회 (서울아산병원 동관 6층 대강당)
특강연자: Thomas S. Kupper, M.D. / 미국 Harvard 의과대학 피부과 교수 / Joost J. Oppenheim, M.D. / 미국 NIH Laboratory of Molecular
Immunoregulation

2005. 3. 25

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<th>제목</th>
<th>연재</th>
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<td>제15차 학술대회 (서울아산병원 동관 6층 대강당)</td>
<td>20연재</td>
<td>26연재</td>
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<tr>
<td>특강연자: Akira Ito, Ph.D. / 일본 Department of Biochemistry and Molecular Biology, Tokyo University of Pharmacy and Life Science 교수</td>
<td>James Varani, Ph.D. / 미국 University of Michigan의대 병리학 교수</td>
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<td>자유연재 19연재, 포스터연재 28연재, 심포지엄 Extracellular matrix 7연재</td>
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2006. 3. 24

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<td>제16차 학술대회 (서울대학교병원 임상의학연구소 1층 강당)</td>
<td>19연재</td>
<td>19연재</td>
<td>62연재</td>
<td>7강의</td>
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<tr>
<td>특강연자: Jackie Bickenbach, Ph.D. / 미국 Anatomy &amp; Cell Biology, Dermatology, Molecular Biology, The University of Iowa 교수</td>
<td>Emi Nishimura, M.D., Ph.D. / 일본 Department of Dermatology and Creative Research Institute Sousei, Hokkaido University Graduate School of Medicine 교수</td>
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<td>자유연재 19연재, 포스터연재 62연재, 심포지엄 Epidermal Stem Cell 7연재</td>
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2007. 3. 23

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<td>19연재</td>
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<td>특강연자: Prof. George Cotsarelis, M.D. / 미국 Dept. of Dermatology, University of Pennsylvania School of Medicine 교수</td>
<td>Prof. Kotaro Yoshimura, M.D. / 일본 Department of Plastic Surgery, University of Tokyo School of Medicine 교수</td>
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<td>자유연재 19연재, 포스터연재 36연재, 심포지엄 Stem Cell 5연재</td>
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2008. 3. 21

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<td>제18차 학술대회 (서울대학교병원 임상의학연구소 1층 강당)</td>
<td>10연재</td>
<td>40연재</td>
<td>1연재</td>
<td>6강의</td>
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<tr>
<td>특강연자: Alain Mauviel, Ph.D. / 프랑스 Research Director 2nd class, DR2, INSERM 교수</td>
<td>Prof. Hiroshi Shimizu, M.D. / 일본 Dept. of Dermatology Hokkaido University Graduated School of Medicine 교수</td>
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<td>자유연재 10연재, 포스터연재 40연재, 1st KSID-JSID Joint Symposium 6연재</td>
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2009. 3. 19–21

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<td>제19차 학술대회 (서울대학교병원 임상의학연구소 1층 강당)</td>
<td>10연재</td>
<td>40연재</td>
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<tr>
<td>특강연자: Tan Suat Hoon (National Skin Center, SIN)</td>
<td>Richard Clark (The State Univ. of New York Stony Brook, USA)</td>
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<tr>
<td>John McGrath (King’s College, St. Thomas’s Hospital, London, GBR)</td>
<td>Dong Youn Lee (Sungkyunkwan Univ., KOR)</td>
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<tr>
<td>Yohanes Widodo (Gadjah Mada Univ., INA)</td>
<td>Amrinder Jit Kanwar (Postgraduate Institute of Medical Education and Research-Chandigarh, IND)</td>
<td></td>
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</table>
Li-Fang Wang (National Taiwan Univ., Hospital, TPE)
Phan Hong Hai (Hospital of Dermato-Venereology, Ho Chi Minh City, VIE)
Zhou Chen (Peking Univ., People’s Hospital, CHN)
Belen Dofitas (St. Luke’s Medical Center, PHI)

자유연제 10연제, 포스터연제 40연제, Symposium I: Aging 4연제
Symposium II: Animal Medels in Dermatologic Research 3연제
제11회 우암학술상 시상: 박경찬 (서울의대)

2009. 10.31~11. 1 제1회 Research CAMP
Cutaneous Biology (Epidermis, Dermis, Adipose Tissue, Skin Appendage), Skin Diseases (FMF, Acne, vitiligo, Atopic dermatitis, Psoriasis, etc)

2010. 4. 2~3 제20차 학술대회 (서울대학교병원 임상의학연구소 1층 장당)
특강연자: Shigetoshi Sano (Kochi Univ. Japan)
Chun-Di HE (China Medical Univ., China)
Hironobu Ihn (Kumamoto Univ., Japan)
자유연제 8연제, 포스터구연 6연제, 포스터연제 40연제, Symposium I: EMT 3연제
Plenary Lecture 2연제
제12회 우암학술상 시상: 유욱 (연세의대)

2010. 8. 27~28 제2회 Research CAMP
Melanogenesis & Pigmentary disorders, Inflammation & Autoimmunity

2011. 3. 25~26 제21차 학술대회 (서울대학교병원 임상의학연구소 1층 장당)
특강연자: Monja Ständer (Münster Univ, Germany)
Kenji Takamori (Juntendo Univ, Japan)
Hideya Ando (Doshisha Univ, Japan)
Shiou-Hwa Jee (National Taiwan Univ, Taiwan)
자유연제 6연제, 포스터구연 6연제, 포스터연제 31연제, Symposium I : Itch 3연제,
Symposium II: Melanogenesis 3연제, Plenary Lecture 2연제
제13회 우암학술상 시상: 이석종 (경북대)

2012. 3. 23~24 제22차 학술대회 (가톨릭의대 성마리아홈)
특강연자: Chung-Hsing Chang (Kaohsiung, Taiwan)
Manabu Ohyama (Tokyo, Japan)
Jürgen Schauber (Munich, Germany)
Alain Mauviel (Orsay, France)
Tomotaka Mabuchi (Kanagawa, Japan)
Liangdan Sun (Anhui, China)
자유연제 9연제, 포스터연제 36연제(포스터구연7연제), Symposium I: Angiogenesis 3연제,
Symposium II: Hair 3연제, Plenary Lecture 1연제
제14회 우암학술상 시상: 김창덕(충남의대)

2012. 10. 26~27 제4회 KSID 추계심포지엄 및 Research Camp 2012 (부산대학교병원)
Mesenchymal Stem Cells, Dendritic Cells, Research Communication
The 27th Annual Meeting of the Korean Society for Investigative Dermatology

2013. 3. 22-23  제23차 학술대회 (가톨릭의대 성마리아홀)
특강연자: Keiji Iwatsuki (Okayama University, Japan)
          Sam Hwang (Medical College of Wisconsin, USA)
          Kenji Kabashima (Kyoto University, Japan)
          Yoshiki Tokura (Hamamatsu University, Japan)
          Masayuki Amagai (Keio University, Japan)
          Chikako Nighigori (Kobe University, Japan)
          Shigaku Ikeda (Juntendo University, Japan)
          Jianzhong Zhang (Peking University, China)
자유연제 5연제, 포스터연제 36연제(포스터구연 5연제), Invited Lectures 1-6:
15연제, 제15회 우암학술상 시상: 최응호(연세의대)
2013. 11. 1-2.  제5회 KSID 추계심포지엄 및 Research Camp 2013 (경북대학교병원 10층 대강당)
Research Communication & Special Lectures
2014. 3. 28-29  제24차 학술대회 (세브란스병원 에비슨의생명연구센터 유일한홀)
특강연자: Keiichi Yamanaka (Mie University, Japan)
          Shinichi Sato (University of Tokyo, Japan)
          Alice Pentland (University of Rochester, USA)
          Nick Reynolds (Newcastle University, U.K.)
          Satoshi Hirakawa (Hamamatsu University, Japan)
자유연제 8연제, 포스터연제 51연제(포스터구연 8연제), Invited Lectures 1-4:
13연제, 제16회 우암학술상 시상: 이승철(전남의대)
2014. 11  제6회 KSID 추계심포지엄 및 Research Camp 2014 (전남대학교병원 명학회관 강당)
Research Communication & Special Lectures
2015. 3.27-28  제25차 학술대회 (세브란스병원 에비슨의생명연구센터 유일한홀)
특강연자: Toshifumi Nomura (Hokkaido University, Japan)
          Masashi Akiyama (Nagoya University, Japan)
          Cheng Ming Chuong (University of Southern California, USA)
          Emi Nishimura (Tokyo Medical and Dental University, Japan)
          Jun Muto (Aichi Medical University, Japan)
          Michel Gilliet (University of Lausanne CHUV, Switzerland)
자유연제 6연제, 포스터 49연제 (포스터구연 9연제), Invited Lectures 1-4, 제 17회 우암학술상 시상: 정기양(연세의대)
2015. 10. 30-31  제7회 KSID Research Camp 2015 (충청남도 공주시 동학산장)
2016. 3.25-26  제26차 학술대회 (세브란스병원 에비슨의생명연구센터 유일한홀)
특강연자: Mauro Picardo (San Gallicano Dermatologic Institute, Italy)
          Anthony Oro (Stanford University, USA)
          Rei Ogawa (Nippon Medical School, Japan)
          Fu-Tong Liu (Academia Sinica, Taiwan)
          Setsuya Aiba (Tohoku University, Japan)
          Mei Yu (Estee Lauder Co.)
자유연제 12연제, 포스터 46연제 (포스터구연 14연제),
KSID Academic Achievement Award: 이준호(충남의대)
KSID Young Investigator Award: 이동훈(서울의대), 임명(충남의대)
18th UAM Research Award: 이재주(경북의대)
제27차 학술대회 (세브란스병원 애비슨의 생명연구센터 유일한홀)
특강연사: Richard L. Gallo (University of California San Diego, USA)
Akimichi Morita (Nagoya City University, Japan)
Wen-Hung Chung (Chang Gung Memorial Hospital, Taiwan)
Johann W. Bauer (Paracelsus Medical University, Salzburg, Austria)
Shinichi Sato (University of Tokyo, Japan)
자유연제 18연제, 핫포스터 20연제 포함 포스터 총 68연제
KSID Award Lecture: 이광훈(연세의대)
KSID Young Investigator Award: 노미령(연세의대), 김혜원(한림의대)
19th UAM Research Award: 이영(충남의대)
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, 학회 참가비(등록비)만 내시면 됩니다. 회원 여러분의 적극 동참을 부탁드립니다.

- 문의 및 접수처: 대한피부연구학회 재무이사 이주희 (ksid2015@gmail.com),
- 입금계좌: 우리은행 1002-653-61878 이주희(대한피부연구학회)
대한피부연구학회 발전기금 모금 안내

1991년 KSID가 설립된 후 회원님들의 지속적인 관심과 성원 덕분에 KSID가 이제는 국내외적으로 명실상부한 피부과 연구학회로서 그 위상을 확고히 다지게 되었습니다. 그러나 현재 KSID가 지금까지의 성과에 안주하지 않고 미국의 SID, 일본의 JSID 및 유럽의 ESDR과 더불어 세계적인 피부연구학회로서의 위상을 높이기 위한 새로운 노력을 더욱 경주해야 할 중요한 시기입니다.

이에 작년에 작고하신 고 김영표 교수님이 발전기금을 희사 하셨고 그 뜻을 후학들이 받아들이 전 회원이 동참하는 발전기금을 모으기로 이사회에서 결의한 바 있습니다.

KSID 발전기금은 2023년 세계피부연구학회 한국 유치, KSID 전용 사무실 및 학술재단설립을 위한 중장기적인 비전을 이루는데 꼭 필요한 기금입니다. 또한 과부과학 연구를 활성화하기 위한 우수논문 학술품 제정, KSID 공모연구비 및 young investigator 연구비 제정, SID 참가비 지원 등에 귀하게 쓰일 계획입니다. 향후 여러분께서 보내 주신 소중한 발전기금이 어떤 용도로 사용되었는지 구체적인 내용을 보고 드리겠습니다. KSID가 세계적인 학회로 도약하기 위한 비전에 회원 모두가 관심을 가지고 적극적으로 참여해 주시길 부탁드립니다.

기금위원회 회장 이승철

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입금계좌: 국민은행 771301-01-609105 [예금주: 이승철(KSID 발전기금)]
### KSID Lifetime Member (평생회원)

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## Board Members

(2016. 3.)

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대한피부연구학회 임원명단
(2017년 3월 현재)

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(2015.4 ~ 2017.3)

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| 간 행 이사 | 강희영(아주야의대) |
| 정 보 이사 | 손상욱(고려의대) |
| 무임소이사 | 이지범(전남의대), 이원주(경북의대), 이동훈(성균관의대), 김창덕(충남의대) |
| 국제 관계 | 조소연(서울의대) |
| 학 술 간 사 | 김효영(연세의대) |
| 국제관계간사 | 김해성(가톨릭의대) |

감 사
(2015.4 ~ 2017.3)

| 노주영 (가천의대) |
| 김유찬 (아주야의대) |

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*기본 1구좌: 20만원

### ♠ 발전기금 모금방법 ♠

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