심포지엄 2 (Symposium 2)
건선 / 아토피 (Psoriasis / Atopic Dermatitis)

좌장: **송해준** (고려의대), **서성준** (중앙의대)

April 20 (Wed) 15:30~17:00 Grand Ballroom C (3F) p215

Topic: Atopic dermatitis vs psoriasis; What are similar but different things?

SYP 2-1. Opening & Overview (5min) Chair of the Korean Society for Psoriasis

SYP 2-2. Epidemiology (8min / 8min ; 8min per each speaker of both societies)
차의대 **김동현** / 서울의료원 **김현정**

SYP 2-3. Immunopathogenesis (8min / 8min) 서울의대 **조성진** / 경북의대 **장용현**

SYP 2-4. Skin barrier & microbiome (8min / 8min) 조선의대 **신봉석** / 연세원주의대 **최응호**

SYP 2-5. Treatment (conventional therapy/biologics) (12min / 12min) 인제의대 **박혜진** / 부산의대 **고현창**

SYP 2-6. Q&A (10min)

SYP 2-7. Closing (3min) Chair of the Korean Atopic Dermatitis Association

Symposium 2: Psoriasis / Atopic Dermatitis

◎ Topic: Atopic dermatitis vs psoriasis; What are similar but different things?

◎ Background for the topic: Atopic dermatitis (AD) and psoriasis are common inflammatory skin diseases. Clinical characters of AD and psoriasis are quite different. In AD lesions, scratch marks, exudation, and dry skin in the surrounding area are remarkable. Bacterial, fungal, and viral infections are frequently seen. In contrast, lesional skin of psoriasis is characterized by thick silvery-white scale over well-defined red thickened skin. Some researchers, however, report similar pathological background between the two diseases. IL-17 is reported to be expressed not only in psoriasis lesional skin but also in AD skin. On the other hand, decrease in filaggrin expression has been shown in patients with psoriasis. To explain the clinical differences of these diseases, it has been proposed that Th2/Th22-polarized immune status together with an attenuated Th17 axis may cause insufficient induction of antimicrobial peptides and more severe barrier dysfunction in AD. While skin barrier dysfunction is commonly seen in AD and psoriasis, a Th2-dominant cytokine milieu down-regulates immunity against infections, which may reflect the commonly seen infectious lesions in the skin of AD.

Although Th2 subset is still important, barrier dysfunction and resultant diminished epidermal defense have been drawing more attention since the discovery of loss-of-function mutation of filaggrin in AD patients. With regard to psoriasis, the disease was thought to be induced by dysregulated turnover of keratinocytes. After the therapeutic effect of cyclosporine was discovered, psoriasis had been thought to be mainly mediated by T cells, especially Th1 cells. The concept further evolved when the importance of interleukin (IL)-23, IL-17, and IL-22 in the pathogenesis of psoriasis was discovered.

Taken together, it would be very interesting to discuss the issue for differences between psoriasis and atopic dermatitis.

◎ Program director: Byung Soo Kim, Sang Wook Son (Academic Directors)

◎ Format: Lecture (O), Panel Discussion ( ), Workshop ( ), the others ( )

Symposium 2-2 (SYP 2-2)

Epidemiology of psoriasis and atopic dermatitis

Dong Hyun Kim[1], Hyun Jung Kim[2]

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[2]Department of Dermatology, Seoul Metrocity Medical Center

1. Prevalence

Psoriasis	Atopic dermatitis
· Varied in relation to demographic characteristics (age, geographic region, etc.) in each studies · In children: from 0% (Taiwan) to 2.1% (Italy) · In adults: from 0.91% (U.S.) to 8.5% (Norway), usually 1-3%, In Korea, estimated to be 0.5-1%	· prevalence ranging from 7.2 to 22.6%, - In adults : 1%-3%, - In children : 10%-20%, - primarily a disease of early childhood

2. Age

Psoriasis	Atopic dermatitis
· Incidence - In children: 40.8/100, 000 person-years (U.S.) - In adults: 78.9/100, 000 person-years (U.S.) to 230/100, 000 person-years (Italy) · The incidence of psoriasis is highest in the third decade of life. · It is believed that the bimodal distribution of psoriasis incidence represents two clinical presentations of the disease, type I (early-onset) and type II (late-onset). · Defined as presenting at ≤40 and >40 years of age (or ≤30 and >30 years of age)	· In Korea, according to ISSAC in 1995, the prevalence of AE was 7.3% and 3.9% in age groups of 6-12 years and 12-15 years, respectively. In 2000, the prevalence of AE increased by 10.7% in 6-12 years and 6.1% in 12-15 years. · About 20% of all children develop symptoms of atopic dermatitis at some point in their lives · Half of these develop symptoms within the first year of life with 95% experiencing onset below 5 years of age. · The majority outgrows atopic dermatitis in childhood or early adolescence, but around 25% continue to have eczema into adulthood or experience a relapse of symptoms after some symptom-free years.

3. Associated diseases

Psoriasis	Atopic dermatitis
· Immune-related disease: Psoriatic arthritis, Inflammatory bowel disease, Palmoplantar pustulosis, Celiac disease · Metabolic syndrome: Cardiovascular disease, Diabetes, Hypertension, Obesity · Cancer: Oral and pharyngeal cancer, Lung cancer, Liver cancer, Pancreatic cancer, Kidney cancer, Bladder cancer, Colorectal cancer, Lymphoma, Melanoma, Nonmelanoma skin cancer	· Increasing BMI was significantly associated with AD · The percentage of subjects with both atopic dermatitis and asthma, both asthma and allergic rhinitis, or both atopic dermatitis and allergic rhinitis was 2.5%, 4.7%, and 8.7%, respectively. · The prevalence of comorbid allergic diseases decreased with age

4. Racial difference

Psoriasis	Atopic dermatitis
· Caucasian usually shows higher prevalence of psoriasis than African or Asian. - In Europe & America: usually above 1% (highest in Northern Europe) - In Asia : usually below 1%	· No definite relationship with race · The prevalence of AE is increasing - In Africa, eastern Asia, western Europe and in parts of northern Europe

5. Gender

· No evidence clearly supports gender-based differences in prevalence in both disease

6. Geographic location

Psoriasis	Atopic dermatitis
· No meaningful correlation between absolute latitude and psoriasis prevalence · Humidity also does not appear play a dominant role.	· Urbanised areas tend to have a higher prevalence of AD · In developed regions, such as Hong Kong, Singapore, Korea and Japan, the prevalence has been stabilized

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김동현(Dong Hyun Kim, M.D., Ph.D.)

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김현정(Hyunjung Kim, M.D., Ph.D.)

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Principal researcher, Department of Environmental Health Research

Education:

1993-1997	M.A. in Immune and Cellular Technology. Dept. of Biotechnology Yonsei University
1998-2002	M.D., Yonsei University College of Medicine
2004-2006	M.A., Department of Dermatology, Yonsei University College of Medicine
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2003-2006	Residency, Department of Dermatology, Yongdong Severance Hospital
2007-2008	Research fellowship in Department of Dermatology, Yongdong Severance Hospital
2008-2010	Assistant Clinical Research Professor, Department of Dermatology, Gangnam Severance Hospital, Yonsei University College of Medicine

Faculty Appointment:

2010-present	Director, Department of Dermatology, Seoul Metrocity Medical Center
2010-present	Principal researcher, Department of Environmental Health Research
2012.3-present	Associate professor, Department of Dermatology, Yonsei University College of Medicine
2014.1-2014.12	Visiting research faculty, National Skin Centre, Singapore

Memberships:

2003-present	Korean Dermatological Association, Board Member
2007-present	Korean Society for Skin Barrier Research and Asian-Pacific Barrier Research Society, Director of General
2011-present	Korean Atopic Dermatitis Association, Board Member
2011-present	Korean Academy of Asthma, Allergy and Clinical Immunology
2012-present	Korean Society of Cosmetic Research, Director of Assessment and Risk Management
2015-present	Skin functional food research Society, Director of information
2016-present	Korean Woman medical doctor Assoacation, Director of Intelligence Commitee

Symposium 2-3 (SYP 2-3)

Immunopathogenesis of atopic dermatitis and psoriasis

Yong Hyun Jang[1], Seong Jin Jo[2]

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Atopic dermatitis and psoriasis are common inflammatory skin diseases that share many similar features. They have immune and barrier defects involving genes that encode immune components and structural proteins that regulate differentiation of epidermal cells although they are genetically complex and multifactorial. Each disease is characterized by disrupted terminal differentiation of keratinocytes in lesional epidermis and skin lesions contain immune infiltrates of T cells, dendritic cells, and other types of leukocytes. Atopic dermatitis and psoriasis were believed to be opposing diseases mediated by either of Th2 cells or Th1 cells, respectively. Nowadays, the original hypothesis was modified when new T-cell subsets, the Th17 and T22 cells were introduced. We review similarities and differences between the immunopathogenesis between the diseases and compared them as below;

Atopic dermatitis	Psoriasis
Lesional immune infiltrates	
T_H2/"T22"polarization(Tc22>Th22); lower $T_H1$componentinchronicdisease Attenuated $T_H17$pathway Reduced antimicrobial axis Inflammatory DCs in dermis > epidermis producing CCL17, CCL18, and CCL22	T_H1/$T_H17$polarizationwith$T_H22$present Increased $T_H17$pathway Increased production of antimicrobial agents Inducible nitric oxide synthase, TNF-α, and IL-23 producing inflammatory DCs in dermis
Leukocytes in circulation	
T_H2>$T_H1$andT_H17 Increased IgE levels and eosinophil numbers in circulation IgE autoantibodies correlated with disease activity	Increased T_H1, $T_H17$andT_H22 Normal IgE levels and eosinophil numbers Autoantibodies uncommon

■ CURRICULUM VITAE ■

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Memberships:

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The Korean Society for Psoriasis
The Korean Atopic Dermatitis Association
Korean Hair Research Society
The Korean Society of Skin Barrier Research

■ CURRICULUM VITAE ■

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2002.3-2006.2 Dermatology residency, Seoul National University Hospital, Seoul, Korea

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Memberships:

2006-present Korean Dermatological Association

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2009-present The Korean Society for psoriasis

2009-present The Korean Hair Research Society

2011-present The Korean Society for Skin cancer

2014-present The Korean Society for Aesthetic and Dermatologic Surgery

Symposium 2-4 (SYP 2-4)

Skin barrier and microbiome in psoriasis

Bong Seok Shin, M.D., Ph.D.

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Psoriasis is a common inflammatory skin disease. It is known to be a complex condition with multifactorial mode of inheritance, however the associations between particular pathogenic pathways remain unclear.

Whether psoriasis represents a fundamental disease of the skin or the immune system has been argued for decades. Although the prevailing view had long been that the hyperproliferation and altered differentiation of epidermal keratinocyte-the hallmarks of psoriasis-occur as a result of a genetic defect in the keratinocyte themselves, the pendulum has swung over the past decade to blame the immune system (T lymphocytes). But again, both clinical experience and recent molecular studies support an emerging concept that psoriasis could be driven by a primary defect in epidermal permeability barrier function.

Among psoriasis susceptibility genes, epidermal differentiation complex (*EDC*) genes located within *PSORS4* locus on chromosome 1q21, which encodes numerous proteins required for epidermal differentiation and the formation of the cornified envelope, a structure that is critical for the permeability barrier. Also recently, it has been focused on the family of genes (a part of the *EDC* genes) encoding late cornified envelope proteins (*LCE* proteins). There is evident that *LCE3B/3C* deletion is associated with psoriasis, but not AD. Contrastively, despite a markedly altered filaggrin expression in psoriatic skin, mutation in *FLG* gene are not associated with psoriasis. Therefore, the genetic background underlying the epidermal barrier defect in psoriasis in distinct from that found in atopic dermatitis.

The interplay between epidermis and immune response is also evident sometimes when simply covering up a psoriatic lesion with emollient. Occlusion provides a normal barrier. When barrier function is restored, it may reduce some of the proinflammatory signals and external triggers, such as pollution or cutaneous flora. Therefore, considering psoriasis as a barrier function disorder may open a new perspective in the management of this disorder.

Various microorganisms are associated with the provocation and/or exacerbation of psoriasis. These include bacteria (*streptococcus pyogenes, Staphylococcus aureus*), fungi (*Malassezia, Candida albicans*),

and viruses (papillomaviruses, retroviruses, endogenous retroviruses). Defective defense of the epidermis or an abnormal response of it to bacteria play a role in the pathogenesis of psoriasis.

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신봉석(Bong Seok Shin, M.D., Ph.D.)

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Symposium 2-4 (SYP 2-4)

Skin barrier and microbiome in atopic dermatitis

Eung Ho Choi, M.D., Ph.D.

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Recently, a defect in skin barrier has been formulated as a pathogenesis of atopic dermatitis (AD) development. Congenitally impaired skin barrier as a main cause of AD can increase allergen penetration through stratum corneum (SC) and then induce allergen sensitization easier. Therefore, AD has been considered as a first step of atopic march since it can eventually progress to asthma and allergic rhinitis with aging. So far, the barrier related pathogenesis of AD have been summarized as below. First, loss of function mutation of filaggrin gene is associated with the development of AD presenting early onset, severe symptoms, frequent occurrence of asthma and progression to adult AD, because filaggrin is a key protein of the epidermal differentiation complex of the SC, a major physical barrier. Second, AD skin shows a deficiency of ceramide which mostly consist SC intercellular lipid lamellae. Third, the imbalance between serine proteases (SP) and SP inhibitors caused by their genetic defects was reported in AD. Continued SP activity due to congenital defect of SP inhibitor disrupts barrier integrity and delays barrier recovery. Fourth, tight junction is also congenitally impaired in AD, which also permits easier allergen penetration and sensitization in AD.

Gut microbiome such as *Akkermansia* was not observed in AD patients compared to healthy controls. *Akkermansia* produces short chain fatty acids in the gut mucosa, which contribute to the inhibition of inflammation. Therefore, a reduction of *Akkermansia* in the gut mucosa of infants might result in AD development. No difference in a diversity of skin microbiome was observed between AD patients and control subjects. *Staphylococcus aureus* was more frequently observed in AD patients compared to control subjects, but not significant. On the contrary, *Lactbacillus salivarius* was not observed in the skin of AD patients.

Keywords: Atopic dermatitis, Skin barrier, Atopic march, Microbiome.

■ CURRICULUM VITAE ■

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2003-2005	Dermatology visiting scientist, UC San Francisco, SF, CA, USA

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2006-present	Professor, Dermatology Yonsei University Wonju College of Medicine

Memberships:

1992-present	Korean Dermatological Association
1998-present	Korean Society for Investigative Dermatology
1992-present	Korean Society for Skin Barrier Research
2008-present	Korean Atopic Dermatitis Association

Symposium 2-5 (SYP 2-5)

Treatment (Conventional therapy / biologics)

Hai-Jin Park[1], Hyun-Chang Ko[2]

[1]Department of Dermatology, Inje University, [2]School of Medicine, Pusan National University

I. Emollients

[In psoriasis]

1. Emollients: internationally accepted standard adjunctive therapy in mild psoriasis
2. Keratolytics: Salicylic acid enhances the efficacy of topical agents

[In atopic dermatitis]

1. Emollients: an integral part of treatment (strong evidence that their use can reduce disease severity and need for pharmacologic intervention), should be applied soon after bathing to improve skin hydration
2. Wet-wrap therapy: emollients with or without a topical steroid can be recommended for moderate to severe AD

II. Topical Therapy

[In psoriasis]

1. Steroid: very effective as short-term treatment, long-term use increases risk of side effects (Strength of recommendations: A)
2. Vitamin D Analogs: efficacy is increased by combination with topical steroids, (Strength of recommendations: A)
3. Tazarotene: efficacy is increased by combination with topical steroids and may reduce atrophy due to steroids. (Strength of recommendations: A)
4. Calcineurin inhibitors: Facial and flexural psoriasis, minimally effective for chronic plaque psoriasis (Strength of recommendations: B)

[In atopic dermatitis]

1. Steroids: used on flaring areas as first-line treatment (Strength of recommendations: A), select potency of topical steroids based on severity of AD, consider potential for cutaneous side effects
2. Calcineurin inhibitors: used as steroid-sparing agents (Strength of recommendations: A), proactive use for maintenance, off-label use in those age < 2yr
3. Antimicrobials: bleach baths and intranasal mupirocin for those with moderate to severe AD and clinical infection (Strength of recommendations: B)

III. Phototherapy

[In psoriasis]

- Important therapeutic option for moderate to severe psoriasis
- Those patients in whom topical therapy has failed or widespread psoriasis
- Effective in majority of patients and cost-effective
- Lacks the systemic toxicities and immunosuppressive properties of systemic and biologic treatments

1. PUVA: remission in 70%-90% of patients, CIx: pregnancy, lactation, impaired live function
2. BB-UVB: Goeckerman regimen (coal tar), Ingram regimen (anthralin)
3. NB-UVB (311nm): Effective as a monotherapy, >70% improvement after 4 weeks, useful in pregnancy
4. Excimer laser (308nm): PASI 90: 85% after 7.2 wks, selectively target lesions

[In atopic dermatitis]

- A second-line treatment, after failure of first-line treatment
- Used as maintenance therapy in patients with chronic disease
- NB-UVB is generally the most commonly recommended light treatment considering low risk profile, relative efficacy, availability, and comfort level

IV. Systemic therapy

[In psoriasis]

Cyclosporin A	Methotrexate	Acitretin
Up to 90% of patients achieve clearance	Reduce severity at least 50% in more than 75% of patient	Modestly effective as monotherapy
Nephrotoxicity, HTN, immunosuppression	Hepatotoxicity, myelosuppression	Hepatotoxicity, lipid abnormalities, mucocutaneous toxicity
Intermittent short-course Tx	Long-term use appears to be safe	Can be combined with PUVA and UVB

- Combination therapy: allowing lower-dose, toxicity-sparing regimens of each of the agents. ex) acitretin + UVB or PUVA, methotrexate + UVB
- Rotational therapy: to minimize cumulative dosage and forestall toxicities.
- Sequential therapy: to optimize initial efficacy and then lead to a safe maintenance regimen by the use of specific combinations in a deliberate sequence.

[In atopic dermatitis]

- Systemic immunomodulatory agents are indicated for adult and pediatric AD patients not adequately controlled with optimized topical regimens or phototherapy
- Cyclosporine is effective and recommended as a treatment option for refractory AD to topical treatments
- Other immunomodulatory agents: azathioprine, methotrexate, mycophenolate mofetil, interferon-γ, systemic steroids

- Antihistamines: insufficient evidence to recommend the general use, short-term intermittent use of sedating antihistamines for sleep loss secondary to itch

V. Biologics

[In psoriasis]

1. When to start biologics?
   Lack of efficacy of current systemic agents/ Concern about cumulative dosage (MTX)/ Concern about end organ toxicity/ Co-morbidities
2. Biologics used in Korea for psoriasis
   ① Etanercept: TNF-α blocker, SC injection, PASI 75: 52%
   ② Infliximab: TNF-α blocker, IV infusion, PASI 75: 80%
   ③ Adalimumab: TNF-α blocker, SC injection, PASI 75: 58%
   ④ Ustekinumab: Blocks IL-12 and IL-23 activity, SC injection, PASI 75:69%
3. Biologics targeting IL-23 and IL-17
   Secukinumab (IL-17A, approved), Ixekizumab (IL-17A, phase III), Tildrakizumab (IL-23, phase III), Guselkkumab (IL-23 antibody, phase III)

[In atopic dermatitis]

1. While no biologic therapy is currently approved for the treatment of AD, the number of clinical trials evaluating biological agents targeting mechanisms in AD has increased
2. Biologics with low or weak effect on AD
   : Anti-IgE (Omalizumab), Anti-IL-5 (Mepolizumab), TNF-α blockers (Etanercept, Adalimumab, Infliximab)
3. Biologics in ongoing clinical studies
   : Anti-IL-4R (Dupilumab), Anti-IL-12/23 (Ustekinumab), Anti-IL-13 (Tralokinumab, Lerikizumab), Anti-IL-22 (ILV-094), Anti-IL-31 (BMS-981164), Anti-IL-31R (CIM331), Anti-TSLP (MK-8226, AMG157)

VI. Others

[In psoriasis]

1. Small molecules
   ① PDE4 inhibitor - apremilast
   ② Janus kinase inhibitors - tofacitinib, oral and topical
2. Climatic therapy

[In atopic dermatitis]

1. Allergen-specific immunotherapy: associated with improved symptoms in AD patients
2. Probiotics: not recommended as routine use for established AD because of inconsistent evidence, but associated with reduced risk of eczema when given to pregnant women, breastfeeding women, and infants

■ CURRICULUM VITAE ■

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Training and Fellowship Appointments:

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2006-2011, 2014.9-present	Chairperson, Dermatology, Inje University Ilsanpaik Hospital

Memberships:

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Korean Society of Dermatopathology
American Academy of Dermatology
International Society of Dermatopathology

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고현창(Hyun-Chang Ko, M.D., Ph.D.)

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