

Brief Communication



The Risk of Psoriasis in Patients With Allergic Diseases: A Nationwide Population-based Cohort Study

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ABSTRACT

The spectrum of allergic diseases includes atopic dermatitis (AD), allergic rhinitis (AR), and asthma. To date, the association between allergic diseases and psoriasis has not yet been completely evaluated. This study was conducted to determine the risk of psoriasis in patients with allergic diseases. A health screening database, a sub-dataset of the Korean National Health Insurance Service database, was used. All 9,718,722 subjects who underwent health examination in 2009 at age over 20 were included. Subjects with allergic diseases including AD (n = 35,685), AR (n = 1,362,713), asthma (n = 279,451) and control subjects without all three allergic diseases (n = 8,210,042), without AD (n = 9,683,037), without AR (n = 8,356,009) and without asthma group (n = 9,439,271) were analyzed. The subjects were tracked using their medical records during the 8-year period from 2010 to 2017 to identify those who developed psoriasis. Multivariate Cox regression models were used to assess the risk of psoriasis. The incidence probability of psoriasis was analyzed through the Kaplan–Meier method. The incidence of psoriasis per 1,000 person-years was 9.57, 3.78, and 4.28 in the AD, AR, and asthma groups, respectively. The AD group exhibited a significantly increased risk of developing psoriasis compared to subjects without AD (hazard ratio [HR], 3.18; 95% confidence interval [95% CI], 3.05–3.31; $P < 0.001$) after adjustment for confounding factors. The risk of psoriasis was significantly increased in the AR group compared to subjects without AR (HR, 1.32; 95% CI, 1.31–1.34; $P < 0.001$) and asthma group compared to subjects without asthma (HR, 1.30; 95% CI, 1.27–1.33; $P < 0.001$). Allergic diseases, particularly AD, may be a risk factor for psoriasis.

Keywords: Psoriasis; dermatitis, atopic; rhinitis, allergic; asthma; cohort studies; population; incidence; risk factors

INTRODUCTION

The spectrum of allergic diseases includes atopic dermatitis (AD), allergic rhinitis (AR), asthma, and eosinophilic esophagitis,¹ which involve a type 2-dominant immune response.² AD can develop into asthma and/or AR, described as an ‘atopic cluster.’^{2,3}

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There are no financial or other issues that might lead to conflict of interest.

Recently, psoriasis has been considered a systemic disease that can lead to many comorbidities. In the pathogenesis of psoriasis, not only a T-helper (Th)-1 but also a Th17/Th22 is important.⁴ Lifestyles, such as alcohol, smoking, and obesity, are independently associated with the risk of psoriasis.^{5,6}

AD and psoriasis are major skin diseases, but there are a limited number of comparative studies focusing on genetics, prevalence, hospitalization rate, comorbidities and socio-economic burden. Patients with psoriasis have an increased prevalence of asthma (risk ratio [RR] 3.89; 95% confidence interval [CI], 1.18–1.40) and AR (RR, 1.25; 95% CI, 1.18–1.33).⁷ However, no cohort study has assessed the risk of psoriasis in patients with AD or other allergic diseases. According to Rocken *et al.*,⁸ 11% of patients with psoriasis have AD; however, due to the different age of onset, this corresponds to less than 0.01% of the population.

The risk of psoriasis in patients with AD, AR, or asthma has not been investigated in a large cohort study. Therefore, we evaluated the risk of psoriasis in patients with allergic diseases using a Korean National Health Insurance Service (KNHIS) database.

MATERIALS AND METHODS

Data source

The KNHIS database is managed by the Korean government and covers the almost entire Korean population. The KNHIS is a computerized database of healthcare-related data on all types of claims and diagnoses are based on the International Statistical Classification of Disease and Related Health Conditions, 10th revision (ICD-10) code. In addition, the KNHIS provides free regular health and cancer-screening examinations. All medical care needs are monitored throughout the life cycle and linked to the patient's identification number. Thus, the healthcare records are not duplicated or omitted. This study was approved by the Institutional Review Board of the Korean National Institute for Bioethics Policy (NHIS-2018-1-224) and the Institutional Review Board of the Catholic University of Korea (19ZESI0239).

Study population

Of the 10,505,818 subjects who underwent a health examination in 2009, those aged < 20 years (n = 15,327) and those with missing data were excluded from this study. Finally, 9,718,722 subjects were included in this study. All subjects were at ≥ 20 years of age, had visited a clinic or hospital at least 3 times during the study period, and had the ICD-10 diagnostic code for AD (L20), AR (J301–304), or asthma (J45–46). Subjects with allergic diseases including AD (n = 35,685), AR (n = 1,362,713), asthma (n = 279,451) and control subjects without all three allergic diseases (n = 8,210,042), without AD (n = 9,683,037), without AR (n = 8,356,009) and without asthma group (n = 9,439,271) were analyzed. The subjects were tracked using their medical records during the 8-year period from 2010 to 2017 to identify those who developed psoriasis (L40). We excluded the patients whom diagnosed with psoriasis prior to enrollment. The study was initiated after a 1-year washout period from 2008 to 2009 to reduce the confounding effect of previously diagnosed psoriasis.

Clinical, laboratory, and anthropometric measurements

Information on age, gender, and household income (dichotomized as ≤ 20% vs. >20% of the median) was obtained from the medical check-up program of the KNHIS. Information on smoking, alcohol status, and physical activity was extracted from questionnaires

completed during the check-ups. Smoking status was classified as non-smoker, ex-smoker, or current smoker, and alcohol consumption was classified as abstinence (no alcoholic drinks consumed within the past year), moderate drinking (< 30 g pure alcohol per day), or heavy drinking ($\geq$ 30 g of pure alcohol per day). The history of comorbidities including hypertension, dyslipidemia, and diabetes mellitus was evaluated based on the ICD-10 codes and prescribed medications. The history of stroke or heart disease was assessed using self-reported questionnaires. Blood samples for measurement of fasting glucose, lipid, and creatinine levels were analyzed. Body mass index (BMI), calculated as the weight (kg) divided by the square of the height (m), was measured during the checkups.

Statistical analysis

Baseline demographic characteristics are demonstrated as mean $\pm$ standard deviation or as number (%). Controls and subjects with AD, AR, and asthma were compared by Student's *t* test (**Table 1**). The incidence probability of psoriasis according to the presence or absence of allergic diseases was analyzed through the Kaplan–Meier method. The log-rank test was used to analyze differences between the study and control groups. Multivariate Cox regression models were used to assess the risk of psoriasis after adjusting for age, gender, smoking, drinking, physical activity, income level, and BMI. The post hoc method was used to identify the tendency of psoriasis risk according to the number of allergic diseases, and the Bonferroni method was used for the post-hoc method. Type 3 test was conducted to determine whether there is significant HR in any of the 3 categories of the ‘number of allergic diseases’ variable, excluding the reference category. The data were analyzed using SAS version 9.4 (SAS Institute, Cary, NC, USA).

RESULTS

Baseline characteristics of the study population

Subjects with AD, AR, and asthma were investigated. We analyzed subjects with allergic diseases, including AD (*n* = 35,685), AR (*n* = 1,362,713), and asthma (*n* = 279,451), as well as control subjects without all 3 allergic diseases (*n* = 8,210,042), without AD (*n* = 9,683,037), without AR (*n* = 8,356,009) and without asthma (*n* = 9,439,271). The baseline and clinical characteristics of the study population are summarized in **Table 1**.

Table 1. Demographic characteristics of the control and allergic diseases groups

Characteristics	Atopic dermatitis			Allergic rhinitis			Asthma		
	No (<i>n</i> = 9,683,037)	Yes (<i>n</i> = 35,685)	<i>P</i>	No (<i>n</i> = 8,356,009)	Yes (<i>n</i> = 1,362,713)	<i>P</i>	No (<i>n</i> = 9,439,271)	(<i>n</i> = 279,451)	<i>P</i>
Male	5,296,782 (54.70)	16,857 (47.24)	< 0.0001	4,723,823 (56.53)	589,816 (43.28)	< 0.0001	5,198,879 (55.08)	114,760 (41.07)	< 0.0001
Smoking			< 0.0001			< 0.0001			< 0.0001
Non-smoker	5,770,260 (59.59)	23,125 (64.80)		4,857,523 (58.13)	935,862 (68.68)		5,598,223 (59.31)	195,162 (69.84)	
Ex-smoker	1,381,834 (14.27)	5,238 (14.68)		1,190,071 (14.24)	197,001 (14.46)		1,347,935 (14.28)	39,137 (14.00)	
Current smoker	2,530,943 (26.14)	7,322 (20.52)		2,308,415 (27.63)	229,850 (16.87)		2,493,113 (26.41)	45,152 (16.16)	
Drinking status			< 0.0001			< 0.0001			< 0.0001
Abstinence	4,984,335 (51.47)	21,210 (59.44)		4,187,656 (50.12)	817,889 (60.02)		4,813,691 (51.00)	191,854 (68.65)	
Moderate drinking	4,035,309 (41.67)	12,705 (35.60)		3,565,291 (42.67)	482,723 (35.42)		3,971,616 (42.08)	76,398 (27.34)	
Heavy drinking	663,393 (6.85)	1,770 (4.96)		603,062 (7.22)	62,101 (4.56)		653,964 (6.93)	11,199 (4.01)	
Low income	2,553,789 (26.37)	10,010 (28.05)	< 0.0001	2,194,407 (26.26)	369,392 (27.11)	< 0.0001	2,486,265 (26.34)	77,534 (27.75)	< 0.0001

Values are mean $\pm$ standard deviations or number (%) by independent *t*-test (continuous variables) and as geometric means and 95% confidence intervals for skewed distributions. Low income is defined as a household income $\leq$ 20% of the median.

Table 2. Incidence of psoriasis based on the presence of allergic diseases (atopic dermatitis, allergic rhinitis, and asthma)

Characteristics	Number	Psoriasis	Person-years	IR (per 1,000 person-years)	HR (95% CI) Model 1	P value	HR (95% CI) Model 2	P value
Atopic dermatitis						< 0.0001*		< 0.0001*
No	9,683,037	204,611	69,581,807.21	2.94	1 (Ref.)		1 (Ref.)	
Yes	35,685	2,368	247,341.55	9.57	3.18 (3.05–3.31)	< 0.0001	3.18 (3.05–3.31)	< 0.0001
Allergic rhinitis						< 0.0001*		< 0.0001*
No	8,356,009	170,105	60,066,307.59	2.83	1 (Ref.)		1 (Ref.)	
Yes	1,362,713	36,874	9,762,841.18	3.78	1.32 (1.30–1.33)	< 0.0001	1.32 (1.31–1.34)	< 0.0001
Asthma						< 0.0001*		< 0.0001*
No	9,439,271	198,639	67,878,116.72	2.93	1 (Ref.)		1 (Ref.)	
Yes	279,451	8,340	1,951,032.04	4.28	1.30 (1.27–1.33)	< 0.0001	1.30 (1.27–1.33)	< 0.0001
Number of allergic diseases						< 0.0001*		< 0.0001*
0	8,210,042	165,147	59,058,419.20	2.80	1 (Ref.)		1 (Ref.)	
1	1,341,938	36,240	9,596,852.81	3.78	1.33 (1.31–1.34)	< 0.0001	1.33 (1.32–1.35)	< 0.0001
2	164,315	5,434	1,157,268.31	4.70	1.55 (1.51–1.60)	< 0.0001	1.55 (1.51–1.59)	< 0.0001
3	2,427	158	16,608.45	9.51	3.16 (2.71–3.70)	< 0.0001	3.16 (2.70–3.69)	< 0.0001

Multivariate Cox regression models were used to assess the risk of psoriasis.

Model 1: adjusted for age and gender; Model 2: adjusted for age, gender, smoking, drinking, physical activity, income level, and body mass index.

IR, incidence rate; HR, hazard ratio; CI, confidence interval

*Type 3 test: conducted to determine whether there is significant HR in any of the 3 categories of the 'number of allergic diseases' variable, excluding the reference category.

Incidence and risk of psoriasis among patients with allergic diseases

Table 2 shows the incidence and HR of psoriasis according to the presence of allergic disease. The incidences of psoriasis were 9.57 and 2.94 per 1,000 person-years in the AD group and the control group without AD, respectively as well as 3.78 and 2.83 per 1,000 person-years in the AR group and the control group without AR, respectively. Furthermore, the incidences of psoriasis were 4.28 and 2.80 per 1,000 person-years in the asthma group and the control group without asthma, respectively.

After adjusting for age, sex, smoking, drinking, physical activity, income level, and BMI, the AD group showed a significantly increased risk of developing psoriasis (HR, 3.18; 95% CI, 3.05–3.31; $P < 0.001$) compared to the control subjects without AD. After adjusting for confounding factors, the AR group (HR, 1.32; 95% CI, 1.31–1.34; $P < 0.001$) and the asthma group (HR, 1.30; 95% CI, 1.27–1.33; $P < 0.001$) also exhibited a significantly increased risk of psoriasis. A Kaplan–Meier analysis showed that the risk of psoriasis increased with increasing duration of AD compared to the control groups without AD (**Figure A**) or AR compared to the control groups without AR (**Figure B**) or asthma compared to the control groups without asthma (**Figure C**) ($P < 0.001$ for all).

We investigated the risk of psoriasis based on the number of allergic diseases (**Table 2**). The HRs of psoriasis in the presence of 1, 2, and 3 allergic diseases were 1.33 (95% CI, 1.32–1.35; $P < 0.001$), 1.55 (95% CI, 1.51–1.59; $P < 0.001$), and 3.16 (95% CI, 2.70–3.69; $P < 0.001$), respectively. The risk of psoriasis tended to increase with the number of accompanying allergic diseases.

DISCUSSION

In this nationwide population-based cohort study, AD, AR, or asthma significantly increased the risk of psoriasis. Notably, patients with AD had a higher risk of psoriasis than did those with AR or asthma. The risk of psoriasis increased as the number of associated allergic diseases increased.

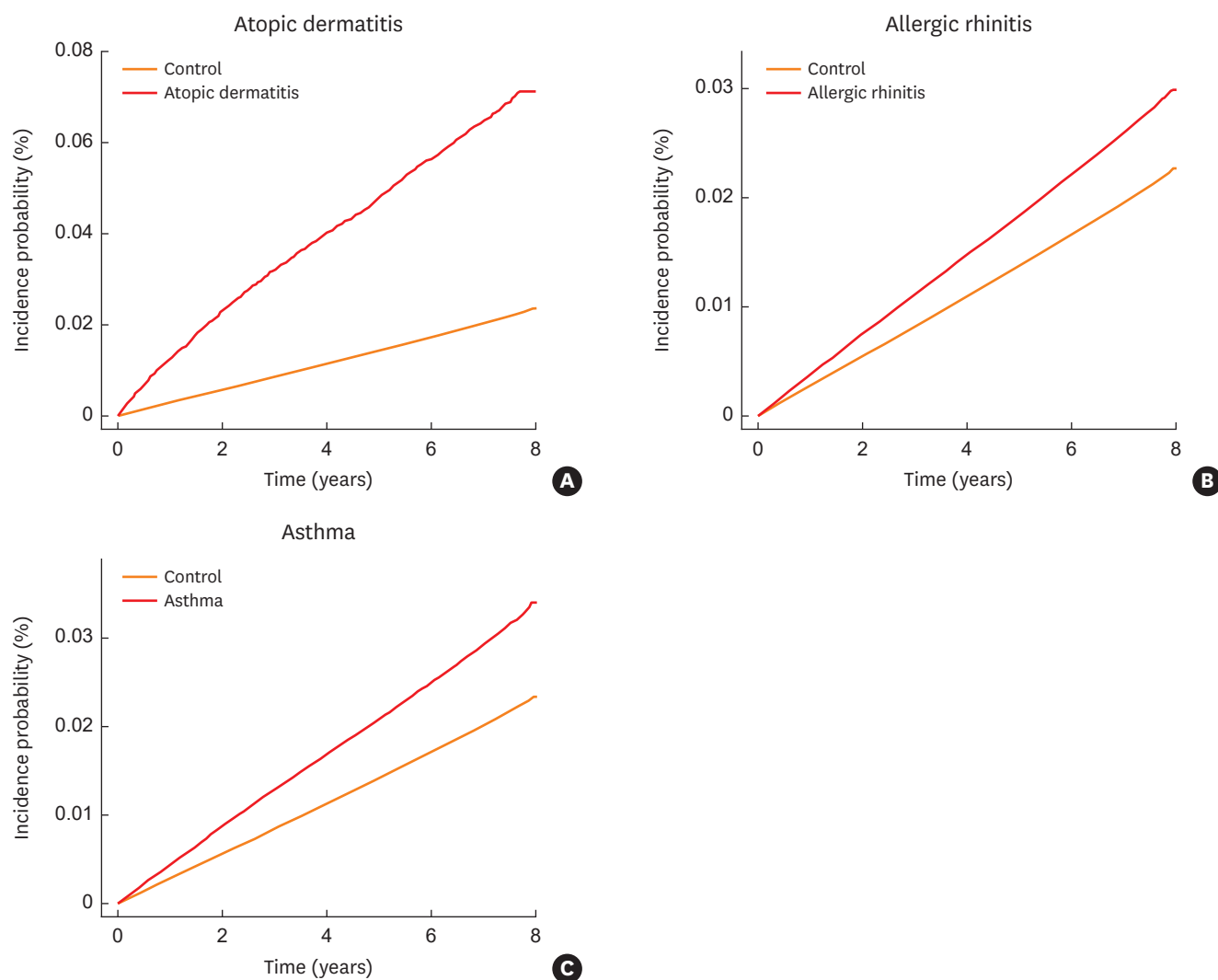


Figure. Risk of psoriasis in patients with atopic dermatitis, allergic rhinitis, or asthma. Kaplan-Meier plots of the incidence of psoriasis. The incidence of psoriasis was significantly higher in patients with atopic dermatitis compared to control groups without atopic dermatitis (A), allergic rhinitis compared to control groups without allergic rhinitis (B), and asthma compared to control groups without asthma (C) ($P < 0.001$, respectively). The log-rank test was used to analyze differences between the study and control groups.

This study is the unique to show an increased likelihood of psoriasis in patients with AD, AR, or asthma. Psoriasis is a chronic inflammatory disease which manifests in the skin, nails, and joints.⁶ AD is a chronic relapsing skin inflammation characterized by disrupted skin barrier function and immunoglobulin E sensitization to environmental allergens.^{9,10} Also, AD is associated with AR and asthma.^{2,11}

Psoriasis and AD are immunological disorders with interactions between hereditary and environmental factors.¹² Psoriasis is induced and maintained by Th1 cells, and AD by the Th2-dominant immune response. However, the traditional Th1/Th2 paradigm was altered by the discovery of Th17 and Th22 cells. Indeed, Th17-related cytokines, such as interleukin-17A and interleukin-17 receptor A, which has recently been considered key molecules in psoriasis and allergic diseases.^{13,14} Guttman-Yassky *et al.*¹⁵ reported that activation of the Th17/IL-23 pathway is a mainstream in the mechanism of chronic AD and psoriasis. Eyerich *et al.*¹² suggested that

colonization of atopic eczema lesions by *Staphylococcus aureus* induces an innate immune reaction by activating Th17 and Th1 cells. Also, IL-17 reportedly plays a crucial role in AD and psoriasis.

Some AD phenotypes have high IL-17 expression and histologic features of psoriasis. Noda *et al.*¹⁶ proposed that, in particular, the phenotype of Asian AD is contributed by TH17, including IL17A, IL19, S100A12, and IL-22; increased hyperplasia; and parakeratosis. The Th17 phenotype, which is more common in South Korean AD patients, may affect the development of psoriasis.

Psoriasis and AD share several pathomechanisms with each other. Choy *et al.* detected gene expression related to neutrophilic chemoattractants in AD and psoriasis lesions.¹⁷ Cookson *et al.*¹⁸ revealed that the chromosomal loci 1q21, 17q25, and 20p are linked to AD, and these regions correspond closely with psoriasis loci. Variants containing CARD14 are found in both diseases, and induce a keratinocyte response through the nuclear factor-kappa B (NF- κ B) pathway.¹⁹ Interleukin (IL)-1 and tumor necrosis factor (TNF)- α are major proinflammatory cytokines in AD and psoriasis, suggesting that the 'inflammatory skin march' increases the risk of comorbidities such as obesity, hypertension, dyslipidemia, DM, and cardiovascular diseases. IL-1/TNF- α , which controls and maintains chronic inflammation, may mediate the association between AD and psoriasis.²⁰

The cytokines produced during type 2 or type 17 inflammation further exacerbate inflammation in the epidermis and dermis.²¹ Thus, chronic inflammatory status may contribute to both psoriasis and atopic disease. Environmental factors, such as smoking and stress, also affect both diseases.²² Thus, inflammatory, immune, genetic, and environmental factors might contribute to the link between AD and psoriasis.

Asthma is a chronic airway inflammatory disorder.²³ In this study, the risk of psoriasis was increased in patients with asthma. Few studies reported the association of asthma and psoriasis and these results were inconclusive. In a report of Sweden, an association between psoriasis and asthma in women, but not in men, was observed.²⁴ However, the sample size was small, and the analysis only considered hospitalization rates. In Taiwan, patients with psoriasis increased approximately 40% risk of asthma.²⁵ A significantly increased risk of asthma in patients with psoriasis was observed in a Danish study. Interestingly, the increased risk was dominantly of nonatopic asthma and was found for late-onset asthma (≥ 18 years).²⁶ In children with asthma aged 6 months to 14 years, the incidence of psoriasis was 3.94 (95% CI, 2.16–7.17).²⁷ Finally, in a meta-analysis, patients with psoriasis, particularly older patients, had a higher risk of asthma.²⁸ Asthma was formerly considered a Th2-driven disease. However, Th17 cells and neutrophils are now known to contribute to inflammation in asthma. Asthma, especially the IL-17-high subtype, is related with dysfunction of bronchial epithelium, up-regulation of antimicrobial reaction, and activation of IL-1 β , IL-6, IL-8, and β -defensin, similar to the immunophenotype of psoriasis.²⁹ Such shared immunological mechanisms support an association between the 2 diseases.

The strength of this study lies in its population-based cohort design and large sample size. However, this study also has several limitations. First, the patients were identified based only on the ICD-10 codes. Therefore, the role of immunoglobulin E was not analyzed. Secondly, we did not have data on the severity of allergic diseases. Thirdly, the lack of correction for potential confounding geographic or ethnic variables limits the generalizability of our results to other populations.

In conclusion, patients with AD, AR, or asthma showed increased risk of psoriasis compared with controls. This suggests that these diseases share some immune and inflammatory features with each other. Moreover, the incidence of psoriasis was higher in patients with AD than with AR or asthma. Further research is required to explore the shared pathogenesis of psoriasis and allergic diseases, particularly AD.

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