

Original Article



Association Between Obstructive Sleep Apnea and Atopic Dermatitis in the Korean Adult Population: Results From the 2019–2022 Korea National Health and Nutrition Examination Survey

Jung Min Lee ,¹ Wanhyung Lee ^{2*}

¹Department of Dermatology, College of Medicine, Chung-Ang University, Seoul, Korea

²Department of Preventive Medicine, College of Medicine, Chung-Ang University, Seoul, Korea

OPEN ACCESS

Received: Mar 1, 2025

Revised: Jul 24, 2025

Accepted: Sep 1, 2025

Published online: Oct 31, 2025

Correspondence to

Wanhyung Lee, MD, PhD

Department of Preventive Medicine, College of Medicine, Chung-Ang University, 84 Heukseok-ro, Dongjak-gu, Seoul 06974, Korea.

Tel: +82-2-820-5571

Fax: +82-0504-374-7000

Email: wanhyung@gmail.com

Copyright © 2025 The Korean Academy of Asthma, Allergy and Clinical Immunology · The Korean Academy of Pediatric Allergy and Respiratory Disease

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<https://creativecommons.org/licenses/by-nc/4.0/>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ORCID iDs

Jung Min Lee

<https://orcid.org/0000-0002-4670-2521>

Wanhyung Lee

<https://orcid.org/0000-0001-6408-7668>

Disclosure

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

ABSTRACT

Purpose: To examine the association between the risk of obstructive sleep apnea (OSA) and atopic dermatitis (AD), in addition to the dose-response relationship between the risk of OSA and the prevalence of AD.

Methods: This study analyzed data from 15,095 participants aged ≥ 40 years who were part of the 2019–2022 Korea National Health and Nutrition Examination Survey (KNHANES). AD was assessed using self-reported questionnaires, and the risk of OSA was determined using the STOP-BANG score. Demographic distributions were analyzed by using χ^2 tests, whereas adjusted odds ratios (ORs) and 95% confidence intervals (CIs) were estimated using multivariable logistic regression analysis. The weighted prevalence of AD and 95% CIs were calculated to account for the complex sampling design of KNHANES.

Results: Multivariable logistic regression revealed a significant association between OSA risk and AD, with an OR of 2.159 (95% CI, 1.456–3.202). The weighted prevalence of AD increased with higher OSA risk, from 1.43% in the low-risk group to 1.74% in the intermediate-risk group, and 2.17% in the high-risk group (P value for trend = 0.013).

Conclusions: OSA risk was significantly associated with AD prevalence, having a dose-response relationship. Given the observed associations, incorporating OSA management into clinical strategies may warrant further investigation as a potential avenue for mitigating AD-related risk.

Keywords: Dermatitis, atopic; sleep apnea, obstructive; prevalence; risk

INTRODUCTION

Atopic dermatitis (AD) is a chronic recurrent, and multifactorial inflammatory skin disease characterized by pruritus, with clinical heterogeneity depending on onset age, lesion morphology, lesion distribution, disease severity, and long-term persistence.^{1,2} In AD, impaired epidermal barrier function could lead to increased transepidermal water loss

and greater susceptibility to microbes and allergens.³⁻⁶ Notably, impaired antimicrobial defense mechanisms, particularly against pathogens such as *Staphylococcus aureus*, are a hallmark of AD.⁷ A key feature of AD is the upregulation of Th2 cytokines and chemokines, which drive tissue inflammation and suppress the expression of proteins critical for epidermal differentiation and lipid synthesis.⁸ Several factors aggravate AD, including age, environmental conditions, lifestyles, cosmetic use, dietary habits, stresses, and particularly sleep disturbances.^{9,12}

Sleep disturbances are a common and impactful consequence of AD, exacerbating disease severity and impairing quality of life. These disruptions are bidirectionally linked to the disease, with nocturnal pruritus and cytokine-driven inflammation disrupting sleep architecture, and poor sleep quality exacerbating the systemic inflammatory burden.^{13,14} This interplay extends beyond dermatological outcomes, affecting broader immunological and neuroendocrine homeostasis.^{15,16} Notably, the potential influence of obstructive sleep apnea (OSA), a prevalent sleep disorder characterized by intermittent hypoxia and fragmented sleep, on AD remains underexplored. OSA triggers systemic inflammation, oxidative stress, and sympathetic activation, all of which may contribute to AD immunopathogenesis.^{17,18}

Although previous studies have highlighted the association between sleep disorders and immune dysfunction, few have systematically examined the relationship between OSA and AD, particularly in adults. Moreover, the STOP-BANG questionnaire, a widely validated tool for accessing OSA risk, offers an opportunity to explore this relationship in a large-scale population-based context. This 8-item tool (yes/no questions) includes 4 self-reported factors (STOP: snoring, tiredness, observed apnea, and high blood pressure) and 4 demographic parameters (BANG: body mass index [BMI], age, neck circumference, and gender).¹⁹

Our study addresses notable gaps in the literature. While many studies on AD focus on children, we investigated adults aged ≥ 40 years, addressing the lack of data on adult patients with AD. Additionally, we evaluated OSA risk using the STOP-BANG questionnaire, emphasizing its association with AD in adults. Moreover, by utilizing nationally representative survey data, our study provides a comprehensive analysis of various risk factors contributing to this relationship.

MATERIALS AND METHODS

Data collection and study participants

This study utilized data from the Korea National Health and Nutrition Examination Survey (KNHANES) conducted between 2019 and 2022. The KNHANES employs a carefully designed sampling method and size calculation to ensure that its findings accurately reflect the entire Korean population, making the results serve as a representative of Korea's overall population for each survey year.²⁰ The survey was conducted over 4 years with participant numbers distributed as follows: 8,110 in 2019, 7,359 in 2020, 7,090 in 2021, and 6,265 in 2022. Individuals aged below 40 years, those without STOP-BANG scores, and those with missing data were excluded from the analysis. The study focused on individuals aged ≥ 40 years, incorporating a comprehensive range of variables, including sex, age, educational level, household income, current smoking status, high-risk drinking behavior, BMI, occupation, aerobic physical activity, risk of OSA, and history of AD (**Fig. 1**).

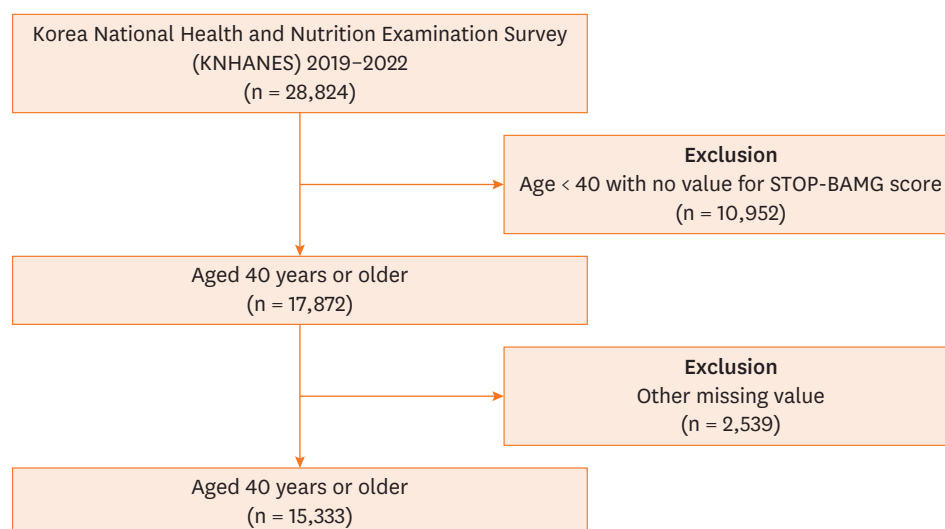


Fig. 1. Flow chart of the study design.

OSA risk and STOP-BANG questionnaire

The risk of OSA was assessed using the STOP-BANG questionnaire, a validated tool for identifying individuals at high-risk for OSA. The questionnaire consists of 8 items: snoring, tiredness or daytime sleepiness, observed apneas or choking, hypertension, obesity (BMI > 35 kg/m²), age (> 50 years), neck circumference (> 40 cm), and sex (male). Participants were categorized into 2 groups: low-risk for OSA (“No”) and high-risk for OSA (“Yes”). Individuals were classified as high-risk (“Yes”) for OSA if they met at least one of the following criteria: (a) answered “yes” to 5 or more questions, or (b) answered “yes” to 2 or more of the “STOP” questions and met at least one of the following conditions: BMI > 35 kg/m², neck circumference > 40 cm, or male sex.^{21–23} Those who did not meet these criteria were classified as low-risk (“No”) for OSA.

Assessment of AD

The presence or absence of AD was determined using a single question from the health interview: “Have you ever been diagnosed with AD by a physician?” Participants who responded “yes” were categorized into the AD group. However, the assessment of AD using self-reported data has several limitations. It relies on the memory of the participants, which introduces recall bias, particularly for mild or past episodes of AD. Additionally, the diagnosis is subjective, as individuals may interpret symptoms and severity differently. The lack of clinical validation by healthcare professionals for self-reported AD further increases the risk of misclassification. Despite these limitations, self-reported AD has demonstrated acceptable validity in epidemiological studies. It strongly correlates with clinical diagnoses and is linked to outcomes such as asthma, allergic rhinitis, and mental health disorders, making it a valuable tool for understanding the prevalence and impact of AD.^{24,25}

Covariates

The analysis considered a range of covariates, including age, categorized into 3 groups (40–49, 50–59, and ≥ 60 years); sex (male or female); educational attainment grouped as elementary school or lower, middle school, high school, or college and above; household income divided into quartiles (1st, 2nd, 3rd, and 4th quartiles); current smoking status

classified as yes or no; high-risk drinking behavior, defined as consuming an average of ≥ 7 drinks per occasion for men and ≥ 5 drinks for women, at least twice per week²⁶; occupational status categorized as none or working; BMI categorized based on the Asian population-specific cutoff values into $< 18.5 \text{ kg/m}^2$, $18.5\text{--}25 \text{ kg/m}^2$, and $\geq 25 \text{ kg/m}^2$ based on the cutoff values recommended for the Asian population²⁷; and aerobic physical activity, defined as performing at least 150 minutes of moderate-intensity aerobic activity per week, at least 75 minutes of vigorous-intensity aerobic activity per week, or a combination of both intensities (where 1 minute of vigorous activity equals 2 minutes of moderate activity) with equal time allocated to each activity.²⁸

Statistical analysis

A χ^2 test was performed to examine the distribution of demographic characteristics among participants with AD. The association between the risk for OSA and AD was assessed using multivariate logistic regression analysis, which yielded adjusted odds ratios (aORs) and 95% confidence intervals (CIs). The analysis accounted for covariates including sex, age, educational level, household income, smoking status, high-risk drinking behavior, BMI, occupation, and aerobic physical activity. We conducted further analyses using weighted prevalence to explore the association between OSA risk and AD, incorporating a dose-response relationship. OSA risk was categorized into low, intermediate, or high groups, according to the total STOP-BANG score, to reflect varying levels of OSA severity. The weighted prevalence and 95% CIs of AD were calculated using a sampling clustering weight that accounted for the complex sampling design of the KNHANES. All statistical analyses were performed using SAS version 9.4 (SAS Institute, Cary, NC, USA). A 2-tailed *P* value < 0.05 was considered statistically significant.

RESULTS

The KNHANES 2019–2022 dataset included 28,824 participants, of whom 17,872 were individuals aged ≥ 40 years with available STOP-BANG scores. After excluding cases with missing data, a total of 15,333 individuals were included in the final analysis. **Table 1** summarizes the general characteristics of participants with AD. The overall prevalence of AD in the study population aged ≥ 40 years was 1.55%. Prevalence varied by age group, with 2.11% in those under 49 years, 1.42% in individuals aged 50–59 years, and 1.36% in those aged ≥ 60 years. Current smoking was significantly associated with a higher prevalence of AD, as was the risk for OSA. However, no significant associations were found between AD prevalence and factors such as sex, educational level, household income, high-risk drinking behavior, BMI, occupation, and aerobic physical activity.

Multivariable logistic regression analysis revealed a significant association between OSA risk and AD (**Table 2**). After adjusting for the covariates, individuals at risk for OSA had a significantly higher likelihood of having AD (aOR, 2.159, 95% CI, 1.456–3.202).

Fig. 2 illustrates the weighted prevalence of AD by OSA risk category. A total STOP-BANG score of 0–2 was classified as low-risk for OSA, 3–4 as intermediate-risk, and 5–8 as high-risk. The weighted prevalence of AD increased with OSA risk, from 1.43 in the low-risk group to 1.74 in the intermediate-risk group and 2.17 in the high-risk group. The trend was statistically significant, with a *P* value of 0.013, indicating a clear association between higher OSA risk and increased prevalence of AD.

Table 1. Characteristics of the study participants by AD

Characteristics	AD		P value
	No	Yes	
Total	15,095 (98.45)	238 (1.55)	
Sex			0.382
Male	6,549 (98.35)	110 (1.65)	
Female	8,546 (98.52)	128 (1.48)	
Age (yr)			0.005
≤ 49	3,529 (97.89)	76 (2.11)	
50–59	3,807 (98.58)	55 (1.42)	
≥ 60	7,759 (98.64)	107 (1.36)	
Educational level			0.239
Elementary school or lower	3,597 (98.49)	55 (1.51)	
Middle school	1,875 (98.79)	23 (1.21)	
High school	4,866 (98.50)	74 (1.50)	
College or higher	4,757 (98.22)	86 (1.78)	
Household income			0.308
1st quartile	3,344 (98.24)	60 (1.76)	
2nd quartile	3,805 (98.45)	60 (1.55)	
3rd quartile	3,819 (98.55)	56 (1.45)	
4th quartile	4,127 (98.52)	62 (1.48)	
Current smoker			0.0008
No	12,849 (98.59)	184 (1.41)	
Yes	2,246 (97.65)	54 (2.35)	
High-risk drinker			0.537
No	13,506 (98.47)	210 (1.53)	
Yes	1,589 (98.27)	28 (1.73)	
BMI (kg/m ²)			0.778
< 18.5	402 (98.77)	5 (1.23)	
18.5–25	9,047 (98.44)	143 (1.56)	
≥ 25	5,646 (98.43)	90 (1.57)	
Occupation			0.12
None	8,935 (98.58)	129 (1.42)	
Working	6,160 (98.26)	109 (1.74)	
Aerobic physical activity			0.169
Insufficient	9,224 (98.56)	135 (1.44)	
Sufficient	5,871 (98.28)	103 (1.72)	
The risk for OSA (accessed by STOP-BANG score)			< 0.0001
No	13,765 (98.58)	198 (1.42)	
Yes	1,330 (97.08)	40 (2.92)	

Values are presented as number (%).

AD, atopic dermatitis; BMI, body mass index; OSA, obstructive sleep apnea.

Table 2. Association of the risk for obstructive sleep apnea with atopic dermatitis

The risk for OSA (accessed by STOP-BANG score)	Crude OR	95% CI	aOR*	95% CI
No	1.000		1.000	
Yes	2.092	1.482–2.951	2.159	1.456–3.202

OSA, obstructive sleep apnea; OR, odds ratio; CI, confidence interval; aOR, adjusted odds ratio.

*Adjusted by sex, age, educational level, household income, smoking status, high-risk drinking behavior, body mass index, occupation, and aerobic physical activity.

DISCUSSION

This study identified a significant association between OSA risk and AD, demonstrating a dose-dependent relationship between OSA risk level and the weighted prevalence of AD. Clinically, individuals at high-risk for OSA are more likely to exhibit an increased prevalence of AD. These findings provide epidemiological insights into how OSA may influence the risk of AD.

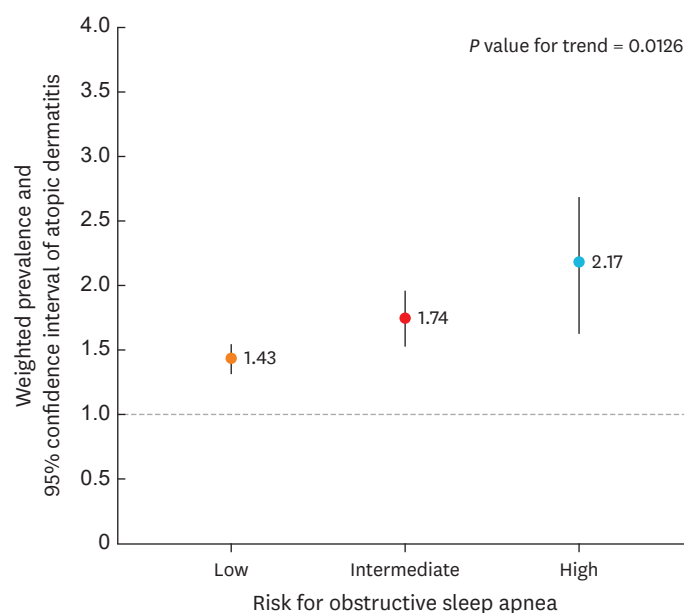


Fig. 2. The weighted prevalence of atopic dermatitis by obstructive sleep apnea (low to high).

The mechanisms underlying the association between OSA and AD are complex. However, various plausible explanations have been proposed. Obesity, a well-known determinant of OSA, has been implicated as a significant risk factor for AD.²⁹ Obesity can contribute to OSA pathogenesis by exerting mechanical stress on the airway and by disrupting upper airway neuromuscular control through systemic inflammation.³⁰ Moreover, recent population-based studies have shown that it is significantly related with higher prevalence and severity of AD in children and adolescents.³¹⁻³³ In obesity, reduced levels of adiponectin and PPAR- γ contribute to the overproduction of pro-inflammatory cytokines and chemokines, activating immune cells and driving systemic inflammation.³⁴ This chronic inflammatory state, further amplified by leptin-mediated signaling, is closely linked to the immunopathological features of AD. Specifically, it promotes the upregulation of Th2 cytokines and chemokines, hallmark features of AD, while suppressing regulatory T-cell responses.³⁵ Additionally, obesity impairs innate antimicrobial defense mechanisms and increases susceptibility to pathogens such as *S. aureus*, thereby heightening the risk of AD.³⁶

Hypertension, another factor contributing to OSA, has been shown to have a significant linear relationship with elevated interleukin (IL)-6 levels, which can induce inflammation.³⁷ In a mouse model of AD, *S. aureus*-induced severe barrier dysfunction led to the sustained expression of pro-inflammatory cytokines such as IL-1 β , IL-6, and tumor necrosis factor- α , exacerbating skin inflammation.³⁸ Therefore, an increase in pro-inflammatory cytokines may lead to hypertension, which acts as a key component of OSA, thereby influencing the risk of OSA and contributing to AD inflammation. These observations suggest that pro-inflammatory cytokines may mediate the complex interplay among hypertension, OSA, and AD.

Additionally, OSA induces hypoxemia, sleep fragmentation, and sympathetic nervous system activation, which can contribute to daytime fatigue, cognitive impairment, and psychological stress, including depression and anxiety.^{39,40} Stress is known to aggravate AD.^{12,41} Acute stress induces a rapid release of cortisol and adrenaline (or noradrenaline), which stimulates the production of pro-inflammatory cytokines, promoting cellular immune responses

and inflammation.^{42,43} Simultaneously, stress impairs the epidermal barrier function and shifts immune balance toward a Th2-dominant response, thereby potentially exacerbating the pathophysiology of AD.^{44,45} Therefore, addressing sleep disorders, such as OSA, and reducing the associated stress may help alleviate AD symptoms.⁴⁶

The observed association between OSA and AD aligns with previous studies.^{47,48} In our analysis of adults aged ≥ 40 years, the prevalence of AD was found to decrease significantly with age, while current smokers had a significantly higher prevalence of AD than nonsmokers. Although high-risk drinking behavior, elevated BMI, employment, and optimal aerobic physical activity were linked to an increased prevalence of AD, these associations were not statistically significant. No significant correlations were found between AD and sex, educational level, or household income.

This study highlights important clinical implications for healthcare professionals, particularly dermatologists and sleep specialists, who encounter patients with AD. Recognizing OSA as a potential comorbidity and contributing factor to AD is particularly crucial for patients with persistent or severe symptoms or those experiencing sleep disturbances. Addressing the underlying OSA, in addition to skin-directed therapies, could be an essential approach. Effective management of OSA in patients with AD could improve treatment outcomes, enhance overall well-being, and potentially reduce the reliance on topical corticosteroids and other AD-specific medications. Integrating these clinical implications into practice will enable healthcare professionals to optimize care for patients with AD and improve treatment outcomes.

This study has notable strengths that contribute to the robustness and reliability of its findings. The use of data from the KNHANES, a nationally representative survey, provided a large sample of Korean adults, enhancing the statistical power of the analysis and improving the generalizability of the results to the broader Korean population. The population-based design of the KNHANES minimizes selection bias and ensures an accurate representation of the overall Korean adult population, enhancing the external validity of the findings. Moreover, the inclusion of objective OSA measures, such as apnea-hypopnea index scores derived from polysomnography, provided a more precise and reliable assessment of OSA risk level compared to self-reported measures. To strengthen the validity of the analysis, the study adjusted for various potential confounding factors, including age, sex, BMI, smoking status, and alcohol consumption, thereby reducing the likelihood of spurious associations. Additionally, the observed dose-response relationship between OSA risk level and AD prevalence provides compelling evidence supporting the potential causal relationship between these conditions.

Despite its strength, the study has some limitations. First, OSA risk and AD diagnoses were based on self-reported questionnaires and STOP-BANG scores, which, although useful, cannot fully substitute clinical diagnostic methods and may present limitations regarding diagnostic accuracy. While the STOP-BANG score is not the gold standard for diagnosing OSA, it has demonstrated high sensitivity, making it a valuable screening tool for assessing OSA risk in population-based studies.^{23,49} A STOP-BANG score of 3 or higher showed sensitivities of 84%, 93%, and 100% for identifying individuals at risk for mild, moderate, and severe OSA, respectively.⁵⁰ Secondly, this study did not assess disease progression, severity, or patient outcomes, limiting the ability to evaluate the relationship between the severity of AD and OSA risk and to derive more detailed findings. Thirdly,

the cross-sectional design precludes establishing a causal relationship between OSA and AD, making it unclear whether OSA is a risk factor for AD or vice versa. Finally, the study population was limited to individuals aged ≥ 40 years, which may not adequately reflect the association between OSA risk and AD in younger populations, where AD prevalence is typically higher. This could lead to an underestimation of the association. Despite these limitations, this study remains significant for its systematic analysis of the relationship between AD and OSA risk utilizing a nationally representative, large-scale dataset. Further research is warranted to elucidate the underlying mechanisms and long-term relationships among these conditions.

In conclusion, this study provides robust evidence of a significant association between OSA risk and AD in Korean adults, emphasizing the importance of considering OSA in the comprehensive management of AD. The clear dose-response relationship observed between OSA risk level and AD prevalence suggests that the need for further investigation into the potential impact of OSA on AD.

These findings, with substantial clinical implications, highlight the need for increased awareness among healthcare professionals, early detection, and management of OSA in patients with AD, and a holistic approach to AD care that addresses both skin-related issues and underlying comorbidities. Further research is required to elucidate the mechanisms linking OSA and AD and to evaluate the long-term impact of OSA treatment on AD outcomes. Such investigations could focus on exploring the roles of obesity, psychological stress, inflammatory mediators, oxidative stress, and sleep disruption in the interplay between these conditions. Additionally, prospective randomized controlled trials are essential to assess the effectiveness of various interventions, such as CPAP therapy and lifestyle modifications, in improving AD outcomes in patients with OSA. By integrating these findings into clinical practice and conducting further research, healthcare professionals can contribute to a more comprehensive understanding of AD and improve the quality of life for individuals affected by this chronic condition.

REFERENCES

1. Eichenfield LF, Tom WL, Chamlin SL, Feldman SR, Hanifin JM, Simpson EL, et al. Guidelines of care for the management of atopic dermatitis: section 1. diagnosis and assessment of atopic dermatitis. *J Am Acad Dermatol* 2014;70:338-51. [PUBMED](#) | [CROSSREF](#)
2. Chovatiya R, Silverberg JI. DESCRIBE-AD: a novel classification framework for atopic dermatitis. *J Am Acad Dermatol* 2022;87:541-50. [PUBMED](#) | [CROSSREF](#)
3. Kim BE, Leung DY. Epidermal barrier in atopic dermatitis. *Allergy Asthma Immunol Res* 2012;4:12-6. [PUBMED](#) | [CROSSREF](#)
4. Katsarou S, Makris M, Vakirlis E, Gregoriou S. The role of tight junctions in atopic dermatitis: a systematic review. *J Clin Med* 2023;12:1538. [PUBMED](#) | [CROSSREF](#)
5. Bhattacharya N, Sato WJ, Kelly A, Ganguli-Indra G, Indra AK. Epidermal lipids: key mediators of atopic dermatitis pathogenesis. *Trends Mol Med* 2019;25:551-62. [PUBMED](#) | [CROSSREF](#)
6. Gunasegaran J, Teh YY, Lim CK, Ng SF. Review on prevalence, risk factors, and research advancements on the use of medical gloves concerning hand dermatitis among health care workers. *Saf Health Work* 2024;15:129-38. [PUBMED](#) | [CROSSREF](#)
7. Hata TR, Gallo RL. Antimicrobial peptides, skin infections, and atopic dermatitis. *Semin Cutan Med Surg* 2008;27:144-50. [PUBMED](#) | [CROSSREF](#)
8. Brandt EB, Sivaprasad U. Th2 cytokines and atopic dermatitis. *J Clin Cell Immunol* 2011;2:110. [PUBMED](#) | [CROSSREF](#)

9. Solomon I, Ilie MA, Draghici C, Voiculescu VM, Căruntu C, Boda D, et al. The impact of lifestyle factors on evolution of atopic dermatitis: an alternative approach. *Exp Ther Med* 2019;17:1078-84. [PUBMED](#) | [CROSSREF](#)
10. Marcelino J, Giménez-Arnau AM. Impact of cosmetics and cleansers in atopic dermatitis—how to advise patients. *Curr Treat Options Allergy* 2024;11:62-76. [CROSSREF](#)
11. Molla A. Dietary patterns and their impact on atopic dermatitis: a comprehensive review. *Open Dermatol J* 2024;18:e18743722306189. [CROSSREF](#)
12. Yanagida N, Saito R, Kamegashira A, Morioka S, Tanaka A. Changes in patient-perceived aggravating factors during the course of atopic dermatitis. *J Cutan Immunol Allergy* 2023;6:219-24. [CROSSREF](#)
13. Chang YS, Chiang BL. Sleep disorders and atopic dermatitis: a 2-way street? *J Allergy Clin Immunol* 2018;142:1033-40. [PUBMED](#) | [CROSSREF](#)
14. Mann C, Gorai S, Staubach-Renz P, Goldust M. Sleep disorders in dermatology - a comprehensive review. *J Dtsch Dermatol Ges* 2023;21:577-84. [CROSSREF](#)
15. Hsiao YH, Chen YT, Tseng CM, Wu LA, Lin WC, Su VY, et al. Sleep disorders and increased risk of autoimmune diseases in individuals without sleep apnea. *Sleep* 2015;38:581-6. [PUBMED](#) | [CROSSREF](#)
16. Smith PC, Mong JA. Neuroendocrine control of sleep. *Curr Top Behav Neurosci* 2019;43:353-78. [PUBMED](#) | [CROSSREF](#)
17. Turkiewicz S, Sochal M, Ditmer M, Gabryelska A. Evaluation of oxidative stress in obstructive sleep apnea and its comorbidities using serum concentrations of oxidized guanine species. *Pol Arch Intern Med* 2023;133:16542. [PUBMED](#) | [CROSSREF](#)
18. Somers VK, Dyken ME, Clary MP, Abboud FM. Sympathetic neural mechanisms in obstructive sleep apnea. *J Clin Invest* 1995;96:1897-904. [PUBMED](#) | [CROSSREF](#)
19. Shahid A, Wilkinson K, Marcu S, Shapiro CM. STOP, THAT and one hundred other sleep scales. New York (NY): Springer; 2012.
20. Kweon S, Kim Y, Jang MJ, Kim Y, Kim K, Choi S, et al. Data resource profile: the Korea National Health and Nutrition Examination Survey (KNHANES). *Int J Epidemiol* 2014;43:69-77. [PUBMED](#) | [CROSSREF](#)
21. Kim K, Kim HJ, Lee S, Lee W. Association and interaction effect of shift work and the risk for obstructive sleep apnea on diabetes mellitus in middle-aged Korean workers. *Sleep Breath* 2024;28:2721-8. [PUBMED](#) | [CROSSREF](#)
22. Shahid A, Wilkinson K, Marcu S, Shapiro CM. STOP-BANG questionnaire. In: STOP, THAT and one hundred other sleep scales. New York (NY): Springer; 2011. 371-83.
23. Chung F, Abdullah HR, Liao P. STOP-BANG questionnaire: a practical approach to screen for obstructive sleep apnea. *Chest* 2016;149:631-8. [PUBMED](#) | [CROSSREF](#)
24. Silverberg JI, Patel N, Immaneni S, Rusniak B, Silverberg NB, Debashis R, et al. Assessment of atopic dermatitis using self-report and caregiver report: a multicentre validation study. *Br J Dermatol* 2015;173:1400-4. [PUBMED](#) | [CROSSREF](#)
25. Drucker AM, Wang AR, Li WQ, Severson E, Block JK, Qureshi AA. The burden of atopic dermatitis: summary of a report for the National Eczema Association. *J Invest Dermatol* 2017;137:26-30. [PUBMED](#) | [CROSSREF](#)
26. Hwang S. Estimation of high-risk drinkers and drinking behavior in Korea - focusing on Korean National Health and Nutrition Examination Survey (KNHANES) and Korean Statistical Information Service data -. *J Environ Health Sci* 2020;46:65-77. [CROSSREF](#)
27. World Health Organization. The Asia-Pacific perspective: redefining obesity and its treatment. Sydney: Health Communications Australia Pty Limited; 2000.
28. Hootman JM. 2008 Physical Activity Guidelines for Americans: an opportunity for athletic trainers. *J Athl Train* 2009;44:5-6. [PUBMED](#) | [CROSSREF](#)
29. Hersoug LG, Linneberg A. The link between the epidemics of obesity and allergic diseases: does obesity induce decreased immune tolerance? *Allergy* 2007;62:1205-13. [PUBMED](#) | [CROSSREF](#)
30. Kirkness JP, Patil SP. Pathogenesis of obstructive sleep apnea in obesity. In: Dixon AE, Clerisme-Beaty EM, editors. Obesity and lung disease: a guide to management. New York (NY): Springer; 2012. 71-97.
31. Lim JH, Jang YS, Kim DB, Jang SY, Park EC. Association between body mass index and atopic dermatitis among adolescents: findings from a national cross-sectional study in Korea. *PLoS One* 2024;19:e0307140. [PUBMED](#) | [CROSSREF](#)
32. Manjunath J, Silverberg JI. Association of obesity in early childhood with atopic dermatitis in late childhood and adolescence. *J Am Acad Dermatol* 2022;87:426-7. [PUBMED](#) | [CROSSREF](#)
33. Sendrea AM, Cristea S, Salavastru CM. Association Between increased body mass index (BMI) and atopic dermatitis in children attending a tertiary referral center: a case-control study. *Cureus* 2024;16:e60770. [PUBMED](#) | [CROSSREF](#)

34. Yang S, Zhu T, Wakefield JS, Mauro TM, Elias PM, Man MQ. Link between obesity and atopic dermatitis: does obesity predispose to atopic dermatitis, or vice versa? *Exp Dermatol* 2023;32:975-85. [PUBMED](#) | [CROSSREF](#)
35. Silva FMCE, Oliveira EE, Ambrósio MGE, Ayupe MC, Souza VP, Gameiro J, et al. High-fat diet-induced obesity worsens TH2 immune response and immunopathologic characteristics in murine model of eosinophilic oesophagitis. *Clin Exp Allergy* 2020;50:244-55. [PUBMED](#) | [CROSSREF](#)
36. Zhang LJ, Guerrero-Juarez CF, Chen SX, Zhang X, Yin M, Li F, et al. Diet-induced obesity promotes infection by impairment of the innate antimicrobial defense function of dermal adipocyte progenitors. *Sci Transl Med* 2021;13:eabb5280. [PUBMED](#) | [CROSSREF](#)
37. Stufano A, D'Amore S, Schino V, Danza P, Iavicoli I, Lovreglio P. Metabolic syndrome and cardiovascular risk factors in a fishing community in Southern Italy. *Saf Health Work* 2024;15:464-71. [PUBMED](#) | [CROSSREF](#)
38. Wanke I, Skabytska Y, Kraft B, Peschel A, Biedermann T, Schitteck B. *Staphylococcus aureus* skin colonization is promoted by barrier disruption and leads to local inflammation. *Exp Dermatol* 2013;22:153-5. [PUBMED](#) | [CROSSREF](#)
39. Lal C, Weaver TE, Bae CJ, Strohl KP; Mechanisms and Clinical Management. Excessive daytime sleepiness in obstructive sleep apnea. *Ann Am Thorac Soc* 2021;18:757-68. [PUBMED](#) | [CROSSREF](#)
40. Wong JL, Martinez F, Aguila AP, Pal A, Aysola RS, Henderson LA, et al. Stress in obstructive sleep apnea. *Sci Rep* 2021;11:12631. [PUBMED](#) | [CROSSREF](#)
41. Jeong S, Kim J, Oh SS, Kang HT, Ahn YS, Jeong KS. The risk of insomnia by work schedule instability in Korean firefighters. *Ann Occup Environ Med* 2024;36:e24. [PUBMED](#) | [CROSSREF](#)
42. Yeager MP, Guyre CA, Sites BD, Collins JE, Pioli PA, Guyre PM. The stress hormone cortisol enhances interferon- γ -mediated proinflammatory responses of human immune cells. *Anesth Analg* 2018;127:556-63. [PUBMED](#) | [CROSSREF](#)
43. Gutierrez-Coronado O, Gutierrez PTV, Villasenor-Garcia MM, Viveros-Paredes JM, Puebla-Perez AM. Activation of the hypothalamic-pituitary-adrenal axis induces the differential release of pro-inflammatory cytokines in BALB/C mice. *Eur Sci J* 2015;11:367-80.
44. Estefan J, Ferreira DC, Cavalcante FS, Dos Santos KRN, Ribeiro M. Investigation of possible relationship between atopic dermatitis and salivary biomarkers, stress, and sleep disorders. *World J Clin Cases* 2023;11:3958-66. [PUBMED](#) | [CROSSREF](#)
45. Arndt J, Smith N, Tausk F. Stress and atopic dermatitis. *Curr Allergy Asthma Rep* 2008;8:312-7. [PUBMED](#) | [CROSSREF](#)
46. Schut C, Weik U, Tews N, Gieler U, Deinzer R, Kupfer J. Psychophysiological effects of stress management in patients with atopic dermatitis: a randomized controlled trial. *Acta Derm Venereol* 2013;93:57-61. [PUBMED](#) | [CROSSREF](#)
47. Hu JM, Lin CS, Chen SJ, Chen CY, Lin CL, Kao CH. Association between obstructive sleep apnea and atopic dermatitis in children: a nationwide, population-based cohort study. *Pediatr Allergy Immunol* 2018;29:260-6. [PUBMED](#) | [CROSSREF](#)
48. Gupta MA, Simpson FC, Vujcic B, Gupta AK. Obstructive sleep apnea and dermatologic disorders. *Clin Dermatol* 2017;35:319-27. [PUBMED](#) | [CROSSREF](#)
49. Yang H, Park H. Usefulness of the Berlin, STOP, and STOP-BANG questionnaires in the diagnosis of obstructive sleep apnea. *J Sleep Med* 2019;16:11-20. [CROSSREF](#)
50. Pivetta B, Chen L, Nagappa M, Saripella A, Waseem R, Englesakis M, et al. Use and performance of the STOP-BANG questionnaire for obstructive sleep apnea screening across geographic regions: a systematic review and meta-analysis. *JAMA Netw Open* 2021;4:e211009. [PUBMED](#) | [CROSSREF](#)