



Original Research Article

OBSERVATIONAL MAPPING OF PEDIATRIC ATOPIC DERMATITIS TRIGGERS USING WEARABLES AND ENVIRONMENTAL SENSORS: ITCH-EXPOSURE ASSOCIATIONS

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ABSTRACT

Background: Atopic dermatitis (AD) is a chronic, pruritic, and relapsing inflammatory skin disease frequently triggered by environmental factors. Identifying real-world triggers remains challenging, particularly in children, where daily variations in temperature, humidity, particulate matter, and allergens can influence disease severity. Advances in wearable and environmental sensing technologies enable continuous monitoring of physiologic and environmental parameters, offering new insights into how exposures relate to itch and sleep disturbances in pediatric AD. The aim is to map real-world associations between itch intensity and environmental exposures using wearable biosensors and environmental sensors in pediatric patients with atopic dermatitis attending a tertiary care hospital.

Materials and Methods: An observational study was conducted on 76 pediatric patients with clinician-diagnosed AD. Participants were equipped with wrist or ankle-worn accelerometer-based devices and indoor environmental monitors measuring temperature, humidity, CO₂, volatile organic compounds (VOCs), and particulate matter (PM_{2.5}). Outdoor pollution and pollen data were linked from public datasets. Itch intensity was reported through electronic diaries on a 0–10 numerical rating scale. Linear mixed-effects models assessed associations between environmental exposures and itch intensity, while generalized mixed models analyzed binary outcomes such as sleep disruption. Analyses were performed using IBM SPSS version 26.0, with $p < 0.05$ considered statistically significant.

Results: The mean participant age was 7.42 ± 3.12 years, with 56.58% males and 50.00% moderate disease severity. Data completeness exceeded 89%, confirming strong adherence. Elevated indoor PM_{2.5} ($\beta = +0.37$, $p = 0.001$), outdoor PM_{2.5} ($\beta = +0.28$, $p = 0.013$), higher indoor temperature ($\beta = +0.21$, $p = 0.025$), and pollen count ($\beta = +0.32$, $p = 0.010$) were significantly associated with increased itch intensity, while higher humidity showed a protective effect ($\beta = -0.18$, $p = 0.017$). Children with severe itch exhibited 12.57 ± 4.76 scratch events/hour, lower sleep efficiency (76.49%), and more awakenings (4.41/night) compared to mild cases ($p < 0.001$).

Conclusion: Continuous monitoring with wearable and environmental sensors effectively captured short-term associations between environmental exposures and itch intensity in children with AD. Elevated particulate levels, warmer indoor temperatures, and lower humidity significantly worsened symptoms and sleep quality. These findings highlight the clinical potential of digital monitoring to personalize trigger management and improve outcomes in pediatric atopic dermatitis.

Keywords: Atopic dermatitis, Pediatric, Wearable sensors, Environmental exposure, Itch intensity.

INTRODUCTION

Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease characterized by intense pruritus, xerosis, and eczematous lesions, with onset commonly in infancy and persistence through childhood into adolescence.^[1] The clinical course is heterogeneous—ranging from intermittent, mild disease to persistent, moderate-to-severe activity that imposes substantial physical, psychosocial, and economic burdens on children and their families.^[1,2] In pediatric populations, sleep disruption from nocturnal itch, daytime inattention, and school absenteeism are well-documented sequelae that magnify the overall disease impact.^[2,3] Against this backdrop, there is growing interest in complementing traditional clinic-based assessments with real-world, longitudinal monitoring that better captures symptom variability in the environments where children actually live. Pathobiology in AD centers on a dynamic interplay between epidermal barrier dysfunction and immune dysregulation. Genetically determined defects in structural proteins such as filaggrin compromise stratum corneum integrity, increasing transepidermal water loss and permeability to irritants, allergens, and microbes; downstream type 2 immune polarization further perpetuates itch–scratch cycles and inflammation.^[4] In practical terms, barrier fragility makes pediatric AD uniquely sensitive to external triggers—heat, humidity shifts, airborne pollutants, aeroallergens, and indoor environmental quality—that can provoke flares despite stable pharmacologic regimens.^[1,4] Recognizing and quantifying these triggers at individual and population levels is therefore essential for precision self-management and anticipatory guidance. Conventional tools—periodic clinician scoring (e.g., SCORAD, EASI) and caregiver recall—are indispensable but provide only snapshots. They may miss short-lived spikes in itch or exposure that unfold over hours or days, and they are vulnerable to recall bias. Contemporary guidelines increasingly emphasize patient education, proactive emollient therapy, trigger mitigation, and step-up anti-inflammatory treatment, yet they acknowledge persistent gaps in monitoring day-to-day symptom dynamics and adherence in home settings.^[3] Novel digital health approaches—electronic diaries, wearables, and connected environmental sensors—offer a path to fill these gaps by enabling high-frequency, minimally burdensome measurement of symptoms and exposures between visits.^[5,6] Among digital endpoints, objective quantification of scratching is especially valuable because scratching both indicates and amplifies itch, disrupts sleep, and further injures the skin barrier. Wrist- or hand-mounted accelerometer-based systems can translate movement signatures into scratch events or duration, yielding continuous, ecologically valid readouts at home.⁶ Importantly, recent analytical validation studies demonstrate that such devices can reliably

detect nocturnal scratching in adults with AD under real-world conditions, supporting their suitability for clinical research and, increasingly, for patient self-management.^[6] Parallel advances in mobile health (mHealth) interventions and teledermatology have shown promise for improving self-efficacy, treatment adherence, and quality-of-life outcomes in AD, underscoring the feasibility and potential impact of digitally enabled care pathways for children and families.^[5] Environmental measurement has evolved just as rapidly. Compact indoor stations can continuously capture temperature, relative humidity, carbon dioxide (as a ventilation proxy), volatile organic compound (VOC) indices, and particulate matter (PM_{2.5}), while geospatial feeds provide neighborhood-level outdoor air quality and meteorology. This creates an opportunity to link minute-scale physiologic and behavioral signals (sleep, activity, scratch) and caregiver-reported itch with concurrently measured indoor/outdoor exposures. For example, warmer, drier microclimates can heighten itch via barrier dehydration and sweat-related irritation; conversely, excessively humid conditions may foster microbial overgrowth—implicating a U-shaped comfort zone that varies across individuals and seasons.^[1,7] Airborne particulates and irritant gases can activate epidermal and neuronal pathways that potentiate pruritus, while aeroallergens add an immunologic stimulus that is particularly relevant in sensitized children.^[1,2,4] A data-integrated approach that accounts for both physiologic vulnerability (barrier and immune state) and real-time exposures may therefore be superior to single-timepoint clinic assessments for explaining day-to-day symptom variability. Electronic diaries (ecological momentary assessments) complement sensor streams by capturing itch intensity in the child's or caregiver's voice, anchoring the physiologic and environmental time series to the patient-centered outcome that matters most: how much it itches now. When diaries are prompted several times per day and paired with wearables and environmental sensors, they enable within-person analyses that can detect small, time-lagged effects of exposures on itch. Such designs are especially well suited to pediatric AD, where symptom fluctuations are frequent, families are highly engaged, and interventions often hinge on near-term behavioral adjustments—ventilating a room, altering sleepwear or bedding, changing bath routines, or modifying outdoor activity on high-pollen or high-PM days.^[5,7]

MATERIALS AND METHODS

This was an observational study conducted at a tertiary care hospital to map real-world itch–exposure associations in pediatric atopic dermatitis (AD). Children with clinician-diagnosed AD were monitored using a combination of wearable biosensors and environmental sensors, alongside caregiver or patient-reported itch measures, to

examine temporal linkages between symptoms and environmental factors during routine daily life. The study protocol adhered to the Declaration of Helsinki and institutional research governance standards. Seventy-six pediatric patients with a confirmed diagnosis of AD were consecutively enrolled from dermatology and allergy clinics. Inclusion required a clinician diagnosis of AD, willingness to wear sensors during waking and sleeping hours as appropriate, and caregiver consent with assent when applicable. Exclusion criteria were comorbid dermatologic conditions likely to confound itch assessment (e.g., extensive psoriasis), inability to safely wear devices, and concurrent systemic therapies initiated specifically for AD within the immediate pre-enrollment period that would make symptom trajectories uninterpretable. Demographic and clinical characteristics, including disease severity (EASI and/or SCORAD), treatment regimens, and relevant comorbidities, were abstracted from the medical record and baseline interview.

Methodology: The primary outcome was patient- or caregiver-reported itch intensity assessed repeatedly using an electronic diary on a 0–10 numerical rating scale (NRS), with anchors of 0 (no itch) and 10 (worst imaginable itch). Secondary outcomes included sleep disruption (self-/caregiver-reported and actigraphy-derived sleep parameters), scratch behavior quantified by the wearable accelerometer algorithm, and clinician-rated disease severity (EASI/SCORAD) at in-person visits. Rescue medication use (topical corticosteroids, calcineurin inhibitors, oral antihistamines) was recorded to enable adjustment for time-varying treatment. Participants were provided a wrist- or ankle-worn device containing a triaxial accelerometer and optical photoplethysmography (PPG). Devices captured raw acceleration at ≥ 25 Hz and heart rate/heart rate variability (HR/HRV) at manufacturer-standard sampling. A validated scratch-detection classifier (threshold- and frequency-domain features from accelerometry) generated minute-level scratch probability and nightly scratch counts. Additional features included step counts, sleep/wake intervals, total sleep time, sleep efficiency, and nocturnal awakenings from actigraphy. Device clocks were synchronized daily to server time to ensure alignment with environmental data streams.

Home indoor environmental quality was measured with a multi-parameter station placed in the participant's primary sleeping area capturing temperature, relative humidity, particulate matter (PM_{2.5}), carbon dioxide (CO₂) as a proxy for ventilation, and volatile organic compound (VOC) index. Ambient outdoor conditions at the participant's residential geohash centroid were retrieved from municipal/central pollution and meteorological feeds, including outdoor PM_{2.5}, PM₁₀, ozone (O₃), nitrogen dioxide (NO₂), ambient temperature, humidity, and heat index. Pollen metrics (tree, grass, weed) were obtained from the regional aeroallergen network by daily count category.

Geolocation from the wearable (if enabled by caregiver) was used only to select the nearest environmental stations; precise locations were not stored. All sensors were factory-calibrated; indoor PM_{2.5} monitors underwent zero-checks using particle-free filters before deployment. Data were ingested in near-real time to a secure database with automated quality checks for range, step changes, flat-lining, and time drift.

Caregivers received prompts on a mobile app to enter itch NRS three to five times per day and a nightly summary of sleep disturbance, visible rash worsening, and use of rescue medications. Contextual covariates included exposure events likely to affect symptoms: bathing frequency, emollient use, known food or contact triggers, pet contact, household cleaning, and time spent outdoors. Missed entries triggered gentle reminders but did not mandate completion.

All data streams were time-stamped in Coordinated Universal Time and merged at the minute level, then aggregated to analysis windows (15-minute for acute associations; hourly and daily for lagged effects). Device non-wear was identified using standard acceleration variance thresholds and removed. Implausible environmental values outside device specifications were flagged and replaced with short-gap linear interpolation when gaps were ≤ 60 minutes; longer gaps were treated as missing. Diary entries with out-of-range values were rejected at entry. For modeling, intermittent missingness in covariates was handled using maximum likelihood within mixed-effects models; sensitivity analyses used multiple imputation with chained equations for variables with $>10\%$ missingness.

Statistical analysis: Analyses were performed using IBM SPSS Statistics, version 26.0. Descriptive statistics summarized baseline characteristics and sensor adherence. Primary analyses used linear mixed-effects models for continuous itch NRS and generalized linear mixed models with appropriate link functions for binary outcomes (e.g., sleep disruption yes/no), specifying random intercepts for participants and, when supported, random slopes for exposure terms. Autocorrelation was addressed using first-order autoregressive [AR(1)] structures on residuals. Time-varying confounders included age group, sex, baseline severity, day of week, school/holiday indicator, indoor–outdoor time, and rescue medication use. Exposure effects were expressed as β coefficients per interquartile-range (IQR) increase or odds ratios (ORs) per IQR, with 95% confidence intervals. Multiplicity was controlled using the Benjamini–Hochberg false discovery rate for families of related hypotheses. Model diagnostics assessed linearity, homoscedasticity, overdispersion, and influential observations. Sensitivity analyses examined alternative lag windows, exclusion of days with febrile illness, and models restricted to nights with ≥ 6 hours of valid actigraphy. Two-sided p-values <0.05 were considered statistically significant.

RESULTS

Baseline Demographic and Clinical Characteristics [Table 1]

A total of seventy-six pediatric patients with atopic dermatitis (AD) were included in the study. The mean age of participants was 7.42 ± 3.12 years, with the largest proportion belonging to the 6–10 years age group (40.79%), followed by 1–5 years (36.84%) and 11–15 years (22.37%). There was a slight male predominance, with 56.58% males and 43.42% females, although the difference between sexes was not statistically significant ($p = 0.482$). The mean disease duration was 3.84 ± 2.15 years, indicating a chronic course in most children.

In terms of disease severity, half of the participants (50.00%) had moderate AD (SCORAD 25–50), 27.63% had mild disease, and 22.37% had severe AD. The prevalence of allergic rhinitis (44.74%) and asthma (25.00%) reflected the typical atopic triad, with allergic rhinitis showing a statistically significant association ($p = 0.027$) with disease presence. Topical corticosteroid use was high (80.26%, $p = 0.013$), consistent with moderate to severe disease profiles. Regular emollient use was reported in 89.47% of participants ($p = 0.044$), demonstrating good adherence to baseline skin care regimens.

Sensor Adherence and Data Completeness [Table 2]

Wearable device and environmental sensor adherence were high, ensuring reliable data capture. The mean daily device wear time was 20.16 ± 2.04 hours, with an overall data completeness rate of 89.42%, showing excellent compliance among participants. Valid accelerometer nights averaged $83.79 \pm 9.28\%$, and environmental sensor

completeness reached $87.25 \pm 8.91\%$, confirming consistency across participants. Itch diary completion was similarly robust, with $91.18 \pm 6.24\%$ of expected entries submitted. Indoor sensors maintained $93.41 \pm 3.87\%$ uptime, reflecting minimal technical loss. Only 9 participants (11.84%) had missing data exceeding 10%, indicating minimal data attrition and supporting the validity of subsequent analyses.

Distribution of Environmental Exposures and Physiological Parameters [Table 3]

Environmental and physiological monitoring demonstrated wide variability across participants. The mean indoor temperature was $26.38 \pm 2.41^\circ\text{C}$, significantly higher during daytime compared to nighttime ($p = 0.041$). The indoor relative humidity averaged $52.74 \pm 8.23\%$, with a trend toward lower humidity during the day, though not statistically significant ($p = 0.076$). Indoor air pollution levels, represented by PM_{2.5} concentration ($37.61 \pm 14.92 \mu\text{g}/\text{m}^3$), showed a significant day–night variation ($p = 0.024$), while outdoor PM_{2.5} levels were higher overall ($49.84 \pm 19.43 \mu\text{g}/\text{m}^3$).

Indoor CO₂ levels averaged 945.63 ± 172.54 ppm, indicating suboptimal ventilation in some households and a significant fluctuation across day and night ($p = 0.012$). The VOC index (mean 1.74 ± 0.56) was within acceptable indoor air quality ranges. Pollen concentrations averaged 145.23 ± 64.11 grains/m³, peaking during outdoor activity periods but showing no significant day–night difference ($p = 0.089$).

Physiologically, children's nocturnal heart rate was 86.47 ± 9.35 bpm, significantly higher during periods of reported itching ($p = 0.033$). Sleep efficiency averaged $82.63 \pm 7.92\%$, which was significantly reduced during high itch phases ($p = 0.021$), suggesting that worsening symptoms directly impacted sleep quality.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants (n = 76)

Variable	Category	Frequency (n)	Percentage (%)	p-value
Age group (years)	1–5	28	36.84	—
	6–10	31	40.79	—
	11–15	17	22.37	—
Sex	Male	43	56.58	0.482
	Female	33	43.42	
Mean age (years)	—	7.42 ± 3.12	—	—
Disease duration (years)	—	3.84 ± 2.15	—	—
SCORAD severity	Mild (<25)	21	27.63	—
	Moderate (25–50)	38	50.00	—
	Severe (>50)	17	22.37	—
Atopic comorbidities	Allergic rhinitis	34	44.74	0.027*
	Asthma	19	25.00	
Topical corticosteroid use	Yes	61	80.26	0.013*
Emollient regular use	Yes	68	89.47	0.044*

*Significant at $p < 0.05$

Table 2: Sensor Adherence and Data Completeness

Parameter	Mean \pm SD	Range
Daily device wear time (hours)	20.16 ± 2.04	15–24
Valid accelerometer nights (%)	83.79 ± 9.28	61.3–100.0
Valid environmental sensor days (%)	87.25 ± 8.91	65.5–100.0
Daily itch diary entries completed (%)	91.18 ± 6.24	73.2–100.0
Mean indoor sensor uptime	93.41 ± 3.87	80–100
Missing data (<10%)	n = 9 participants	11.84%

Association Between Environmental Exposures and Itch Intensity [Table 4]

Linear mixed-effects modeling revealed multiple significant associations between environmental parameters and itch intensity. An increase in indoor PM_{2.5} was strongly correlated with higher itch scores ($\beta = +0.37$, 95% CI 0.15–0.59, $p = 0.001$), and outdoor PM_{2.5} also showed a positive association ($\beta = +0.28$, $p = 0.013$). Higher indoor temperature ($\beta = +0.21$, $p = 0.025$) significantly worsened itch intensity, consistent with the known aggravating role of heat and sweat.

Conversely, relative humidity demonstrated a negative association ($\beta = -0.18$, $p = 0.017$), indicating that drier environments exacerbate itching episodes. Indoor CO₂ concentrations showed a positive but non-significant trend ($\beta = +0.10$, $p = 0.168$), suggesting that poor ventilation may contribute but is not an independent determinant. Pollen count exhibited a significant positive correlation ($\beta = +0.32$, $p = 0.010$), highlighting the impact of outdoor allergen exposure on symptom exacerbation. Overall,

environmental factors collectively explained a substantial portion of short-term variability in itch ratings.

Sleep and Scratch Outcomes in Relation to Itch Severity [Table 5]

Significant differences were observed across itch severity groups. Children with severe itch (NRS ≥ 7) exhibited markedly elevated mean scratch counts (12.57 ± 4.76 events/hr) compared with those with moderate (8.31 ± 3.45) and mild itch (4.92 ± 2.03) ($p < 0.001$). Sleep efficiency decreased progressively with worsening itch, from $86.77 \pm 6.42\%$ in the mild group to $76.49 \pm 8.84\%$ in the severe group ($p < 0.001$). Similarly, nightly awakenings increased from 1.82 ± 0.74 to 4.41 ± 1.31 episodes as itch intensity rose ($p < 0.001$).

Self- or caregiver-reported sleep disruption was present in 77.78% of children with severe itch, compared to 51.28% in moderate and 23.81% in mild groups ($p = 0.002$). These findings indicate a strong, dose-dependent relationship between itch severity and sleep quality deterioration.

Table 3: Distribution of Environmental Exposures and Physiological Parameters

Variable	Mean \pm SD	Minimum	Maximum	p-value (Day vs. Night)
Indoor temperature ($^{\circ}\text{C}$)	26.38 \pm 2.41	22.1	30.8	0.041*
Indoor relative humidity (%)	52.74 \pm 8.23	35.5	68.2	0.076
Indoor PM _{2.5} ($\mu\text{g}/\text{m}^3$)	37.61 \pm 14.92	12.4	78.9	0.024*
Outdoor PM _{2.5} ($\mu\text{g}/\text{m}^3$)	49.84 \pm 19.43	17.6	104.3	—
CO ₂ concentration (ppm)	945.63 \pm 172.54	610	1280	0.012*
VOC index	1.74 \pm 0.56	0.8	3.2	—
Pollen (grains/ m^3)	145.23 \pm 64.11	40	298	0.089
Nocturnal heart rate (bpm)	86.47 \pm 9.35	65	112	0.033*
Sleep efficiency (%)	82.63 \pm 7.92	61.5	94.0	0.021*

*Significant at $p < 0.05$

Table 4: Association Between Environmental Exposures and Itch Intensity (Linear Mixed-Effects Model)

Exposure Variable	β Coefficient (per IQR increase)	95% CI	p-value	Interpretation
Indoor PM _{2.5} ($\mu\text{g}/\text{m}^3$)	+0.37	0.15 – 0.59	0.001*	Higher PM _{2.5} increased itch intensity
Outdoor PM _{2.5} ($\mu\text{g}/\text{m}^3$)	+0.28	0.06 – 0.50	0.013*	Significant positive effect
Indoor temperature ($^{\circ}\text{C}$)	+0.21	0.03 – 0.39	0.025*	Warmer indoor temperature worsened itch
Relative humidity (%)	-0.18	-0.33 – (-0.03)	0.017*	Lower humidity associated with increased itch
CO ₂ (ppm)	+0.10	-0.04 – 0.24	0.168	Not significant
Pollen count	+0.32	0.08 – 0.56	0.010*	Pollen peaks increased itch ratings

*Significant at $p < 0.05$

Table 5: Sleep and Scratch Outcomes in Relation to Itch Severity

Itch Severity Group	Mean Nightly Scratch Count (events/hr)	Sleep Efficiency (%)	Awakenings (per night)	Sleep Disruption Reported (%)	p-value
Mild itch (NRS 0–3)	4.92 \pm 2.03	86.77 \pm 6.42	1.82 \pm 0.74	23.81	—
Moderate itch (NRS 4–6)	8.31 \pm 3.45	81.28 \pm 7.53	3.02 \pm 1.02	51.28	0.002*
Severe itch (NRS ≥ 7)	12.57 \pm 4.76	76.49 \pm 8.84	4.41 \pm 1.31	77.78	<0.001*

*Significant at $p < 0.05$

DISCUSSION

Our cohort's baseline profile 50.00% moderate and 22.37% severe SCORAD, with frequent comorbidities (allergic rhinitis 44.74%, asthma 25.00%) and a slight male predominance (56.58%)—

fits within global pediatric patterns reported by Odhiambo et al. (2009) in ISAAC Phase Three, where eczema symptom prevalence in 6–7-year-olds ranged 0.9% (India) to 22.5% (Ecuador) and was often accompanied by sleep disturbance as a marker of severity; ISAAC also noted sex differences

varying by age which contextualize our non-significant sex split ($p = 0.482$).^[8]

High feasibility of continuous home monitoring in our study— 20.16 ± 2.04 h/day wear, itch diary completion 91.18%, accelerometer nights 83.79%—parallels the positive implementation experience summarized by Khan et al. (2024), who reviewed AD wearables and found actigraphy and skin-sensing modules most consistently aligned with disease activity tracking; this supports our strong data completeness (89.42%) and the reliability of our sensor-derived endpoints.^[9]

Regarding indoor particulates, we observed a robust association between indoor $PM_{2.5}$ and itch ($\beta = +0.37$ per IQR; $p = 0.001$) at a mean indoor $PM_{2.5}$ of $37.61 \pm 14.92 \mu\text{g}/\text{m}^3$. Kim et al. (2021) reported that a $10 \mu\text{g}/\text{m}^3$ increase in indoor $PM_{2.5}$ increased AD symptom scores by 16.5% (95% CI 6.5–27.5) in spring and 12.6% (4.3–21.5) in winter, with larger effects in sensitized or more severe children concordant with our direction and implying indoor air quality as a modifiable trigger.^[10]

Our outdoor $PM_{2.5}$ effect ($\beta = +0.28$; $p = 0.013$) aligns with population-level event studies. During the 2018 California Camp Fire, Fadadu et al. (2021) showed wildfire-related $PM_{2.5}$ was associated with increased weekly clinic visits for AD (rate ratio 1.49 pediatric; 1.15 adult) and a 7.7% rise in weekly pediatric itch visits per $10 \mu\text{g}/\text{m}^3$ $PM_{2.5}$ increase supporting our finding that ambient particulates acutely amplify AD symptom burden.^[11]

Thermal-moisture conditions in our data higher indoor temperature worsening itch ($\beta = +0.21$; $p = 0.025$) and higher relative humidity protective ($\beta = -0.18$; $p = 0.017$)—mirror Kim et al. (2017) who, in a 17-month pediatric panel (35,158 person-days), found that $+5 \text{ }^\circ\text{C}$ temperature and $+5\%$ RH were associated with 12.8% and 3.3% decreases in AD symptoms, respectively, while pollutants ($PM_{10}/NO_2/O_3$) increased risk; the agreement in directionality underscores the clinical relevance of optimizing indoor microclimate.^[12]

Our pollen–itch association ($\beta = +0.32$; $p = 0.010$) is consistent with Nishie et al. (2012), a self-scoring diary study during Japanese cedar/cypress season, where itch flares were significantly higher on high-pollen days in half of participants (7/14), demonstrating that aeroallergen peaks can measurably intensify pruritus particularly in sensitized children.^[13]

We observed day–night CO_2 differences ($p = 0.012$) and a non-significant positive trend with itch ($\beta = +0.10$; $p = 0.168$), suggestive of a ventilation signal. In a small intervention series, Beck et al. (AIVC proceedings) showed that moving AD patients to homes with better air exchange and optimized indoor climate tracked with improvements in clinical and subjective measures across monthly assessments, indicating that ventilation is a plausible co-factor alongside particulates, heat, and humidity.^[14]

The sleep–itch dose-response in our cohort—severe-itch nights had more scratches (12.57 vs 4.92

events/h; $p < 0.001$), lower sleep efficiency (76.49% vs 86.77%; $p < 0.001$), and more awakenings (4.41 vs 1.82/night; $p < 0.001$) aligns with Bawany et al. (2020), who synthesized evidence that 47–80% of children with AD experience sleep disruption, reinforcing sleep as a central morbidity domain and validating our sensor-derived sleep metrics.^[15]

Our scratch quantification used a validated actigraphy classifier; Ji et al. (2023) demonstrated that actigraphy-based nocturnal scratch detection showed strong agreement with video-adjudicated benchmarks for scratch duration and intensity, supporting the reliability of our events/hour endpoint used to distinguish mild vs severe-itch nights.^[16]

Finally, the broader climatic context complements our day–night and seasonal signals (warmer, drier indoor periods linked to worse itch and lower sleep efficiency). In a nationwide cohort of 100,304 children, Yokomichi et al. (2021) found AD incidence to peak for births in October–December, implicating early-life sunshine duration/humidity patterns and reinforcing that both acute exposures (temperature, RH, pollen) and climatic milieu shape pediatric AD expression—consistent with our observed temperature–humidity–pollen triad.^[17]

CONCLUSION

This observational study demonstrated that integrating wearable biosensors and environmental sensors provides a reliable and high-resolution approach to mapping real-world itch–exposure dynamics in children with atopic dermatitis. Elevated indoor and outdoor $PM_{2.5}$, increased temperature, and reduced humidity were significantly associated with heightened itch intensity and worsened sleep quality, confirming the critical role of microenvironmental conditions in disease aggravation. Continuous sensor adherence and robust data completeness underscored the feasibility of this technology for pediatric use. The findings highlight that individualized monitoring of environmental and physiologic variables can guide tailored trigger avoidance strategies, promote better symptom control, and inform precision-based management of pediatric atopic dermatitis in real-life settings.

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