

Diagnostic performance and methodological concordance of the autologous sweat skin test for sweat allergy in a tropical setting: A pilot cross-sectional study

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Abstract

Background: Sweat allergy is frequently observed in patients with cholinergic urticaria (CholU) and atopic dermatitis (AD), yet data from high-sweating, tropical regions are scarce. The autologous sweat skin test (ASwST) is considered the reference diagnostic assay, but protocols differ.

Objectives: To determine the prevalence of sweat allergy—assessed by the ASwST—in patients with CholU and AD in a tropical setting, and to evaluate concordance between the testing methods.

Methods: In this cross-sectional pilot study, 29 CholU patients, 41 AD patients, and 20 healthy controls underwent intradermal injection of filtered autologous sweat at 20 μ L and 50 μ L. Skin responses were assessed via wheal and flare formation. Positivity rate of sweat allergy using ASwST, clinical correlates, and inter-volume agreement were calculated.

Results: ASwST was positive in 8/29 CholU patients (27.6%) and 11/41 AD patients (26.8%); all controls were negative. In AD, positivity occurred across disease-severity strata. Overall inter-volume agreement reached 80%: 51/70 tests (72.9%) were negative for both volumes, and 5/70 (7.1%) were concordantly positive. The remaining 14/70 cases (20.0%) reacted only at 50 μ L, yielding a significantly higher positivity rate than 20 μ L (27.1% vs 7.1%, $P = 0.001$).

Conclusion: In tropical Thailand, ASwST identified sweat allergy in approximately 25% of CholU and AD patients, lower than many temperate-zone reports. Either injection volume is acceptable; however, 50 μ L increases diagnostic yield without compromising specificity. Larger, multicenter studies should clarify whether ambient heat, humidity, or cutaneous microbiota modulate sweat-allergy.

Key words: Atopic dermatitis, Autologous sweat skin test, Cholinergic urticaria, Intradermal testing, MGL_1304, Sweat, Sweat allergy, Tropical dermatology

Citation:

Paringkarn, T., Limphoka, P., Tuchinda, P., Chularojanamontri, L., Kanistanon, D., Phongtanthakun, W., Yenyuwadee, S., Saengthong-aram, P., Ketyungyoenwong, A., Julraksa, M., Srinoulprasert, Y., Kulthanan, K. (2025). Diagnostic performance and methodological concordance of the autologous sweat skin test for sweat allergy in a tropical setting: A pilot cross-sectional study. *Asian Pac J Allergy Immunol*, 43(3), 531-538. <https://doi.org/10.12932/ap-110625-2092>

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Introduction

Sweat is a major trigger of cholinergic urticaria (CholU) and atopic dermatitis (AD).^{1,2} CholU, a subtype of chronic inducible urticaria, presents as multiple pinpoint wheals (1–3 mm) with surrounding erythema after rises in core temperature. The lesions, most often on the trunk, may coalesce and cause intense pruritus; some patients describe stinging or pain without itch. Severe episodes can lead to angioedema, respiratory compromise, dizziness, and, rarely, anaphylaxis.³

AD is a chronic, relapsing eczematous disorder characterized by intense pruritus.¹ Sweating, alongside irritants, foods, psychological stress, environmental factors, and changes in the skin microbiome, is a recognized exacerbating stimulus.¹

Human sweat is a complex fluid composed of water, electrolytes, proteins, and pro-inflammatory cytokines.² It contains antigens capable of triggering histamine release from basophils and immediate hypersensitivity reactions in CholU and other allergic conditions.^{4–7} The *Malassezia globosa*-derived protein MGL_1304 appears to be the dominant allergen driving type I sweat allergy.^{2,8} Experimental models—including intradermal injection of autologous sweat or acetylcholine, basophil activation assays, and histamine-release tests—have repeatedly confirmed the contribution of sweat to rapid allergic responses.^{4,9} Moreover, serum levels of specific immunoglobulin E (IgE) against MGL_1304 are elevated in patients with AD and CholU.^{8,10–12}

The autologous sweat skin test (ASwST) detects immediate hypersensitivity to sweat through intradermal injection of autologous sweat, followed by assessment of the wheal-and-flare response.^{4,13} Although some investigators treat ASwST as the diagnostic gold standard for sweat allergy,^{9,13} protocols vary in injected volume (20 μ L vs 50 μ L), dilution, and interpretation criteria. A review of published methods (**Table 1**) shows positive rates of 37.5%–100% in patients with CholU^{9,13–15} and 38%–95% in those with AD.^{4,16} The wide sensitivity range indicates heterogeneity in sweat-allergy reactivity that may track with clinical phenotype; healthy controls seldom react to ASwST.^{9,13,14}

In tropical countries such as Thailand, persistently hot, humid climate leads to frequent, intense sweating, creating an ideal context for evaluating sweat allergy. We therefore assessed ASwST in Thai patients with CholU and AD, compared concordance between the 20 μ L and 50 μ L injection volumes to gauge test consistency, and explored associations between ASwST results and clinical characteristics.

Methods

Subjects

The study enrolled 70 participants: 29 with CholU and 41 with AD. CholU was diagnosed clinically—pruritic pinpoint wheals precipitated by heat or exercise¹⁷—with confirmation by numerous small wheals during the pulse-controlled ergometry test.^{3,18} AD was diagnosed according to the Hanifin–Rajka criteria.¹⁹

All participants discontinued oral antihistamines ≥ 7 days and systemic immunomodulators (corticosteroids, cyclosporine, biologics) ≥ 14 days before testing. Twenty healthy volunteers served as controls. The protocol was approved by the Siriraj Institutional Review Board, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand (reference: SI 606/2023); written informed consent was obtained from all participants. Demographic and clinical data were recorded, and AD severity was graded with the Scoring Atopic Dermatitis index.²⁰

Sweat collection and preparation

Participants first acclimatized for 20 min, then cycled on a static bicycle in a room kept at 20–22°C. The face, neck, trunk, and arms were rinsed with water only and dried with a sterile towel. Each participant next donned a disposable, head-free rain cover that enclosed the upper torso. Cycling continued for 30 min or until ≥ 1 mL of sweat had accumulated, whichever occurred first. Sweat was collected with sterile, disposable pipette tips.

ASwST

Sweat samples were diluted 1:100 (v/v) in sterile 0.9% saline, and total protein was quantified by absorbance at 280 nm with a NanoDrop 2000c spectrophotometer (Thermo Fisher Scientific, Waltham, MA, USA). The samples were filtered through 0.45 μ m membranes (Corning, Corning, NY, USA), aliquoted, and stored at 4°C. Skin testing was performed 1–2 weeks later.

Autologous sweat (20 μ L and 50 μ L) and equal volumes of saline (negative controls) were injected intradermally into the volar forearm with a 1 mL syringe fitted with a 27-gauge, half-inch needle. A skin-prick test with histamine 100 μ g/mL (0.01%; Bencard Allergie, Munich, Germany) served as the positive control. Wheal-and-flare diameters were measured at 15 min and 30 min.

Positive results of autologous sweat were considered according to previous studies shown in **Table 1**. A test was deemed positive if either of the following criteria was met: (i) for the 20 μ L injection, a wheal diameter ≥ 6 mm at 15 min,^{9,14,21} or (ii) for the 50 μ L injection, a wheal ≥ 1.5 mm larger than the saline control at 30 min.¹⁵

Statistical analysis

We conducted all analyses with PASW Statistics, version 18 (SPSS Inc, Chicago, IL, USA). Descriptive results are presented as frequencies and percentages for categorical variables and as means with standard deviations or medians with ranges for continuous variables. Associations between categorical variables were examined with chi-square or Fisher exact tests, as appropriate. Concordance between the 2 ASwST volumes (20 μ L vs 50 μ L) was assessed with the McNemar test. A 2-sided $P < 0.05$ denoted statistical significance.

Table 1. Summary of published studies using the autologous sweat skin test in cholinergic urticaria and atopic dermatitis.

Author	Country	Climate zone	Study population		Methods						Positive results of ASwST
			Subjects (N)	Healthy control (N)	Dilution of sweat (titer)	Filter (µm)	Amount of sweat (µl)	Negative control	Reading time (min)	Positive result criteria	
Adachi K et al (1989) ¹²	Japan	Temperate	AD (45)	22	Undiluted and twofold serial dilution	Not defined	Not defined	15	Wheal > 9*9 mm or erythema > 20*20 mm	- AD: 43/45 (95.5%) - Healthy control: 4/22 (18.2%)	
Adachi J et al (1994) ¹³	Japan	Temperate	CholU (20) Acute and chronic urticaria (11)	20	Undiluted and twofold serial dilution	0.22	20	15	Wheal > 9*9 mm or erythema > 20*20 mm	- CholU: 20/20 (100%) - Acute or chronic urticaria: 3/11 (27.3%) - Healthy control: 0/20 (0%)	
Hide M et al (2002) ¹⁴	Japan	Temperate	AD (66) Allergic rhinitis (7)	27	Not defined	0.22	20	15	Diameter of wheal ≥ 8 mm or diameter of flare ≥ 20 mm	- AD: 56/66 (84.4%) Mild: 14/17 (82.4%) Moderate: 18/20 (90.0%) Severe: 21/26 (80.8%) Undefined: 3/3 (100%) - Allergic rhinitis: 5/7 (71.4%) - Healthy control: 3/27 (11.1%) - Not associated with severity of AD	
Fukunaga A et al (2005) ⁹	Japan	Temperate	CholU (18)	10	1:100	0.45	20	15	Diameter of wheal ≥ 6 mm and wheal induced by sterile saline < 2 mm	- CholU: 11/17 (64.7%) - Healthy control: 0/10 (0%)	
Kozaru T et al (2011) ⁷	Japan	Temperate	CholU (6)	None	1:100 1:1000	0.45	20	15	Diameter of wheal ≥ 6 mm	- CholU: 6/6 (100%)	
Kim J et al (2015) ¹⁴	Korea	Continental	CholU (18)	10	1:100	Not defined	20	15	Diameter of wheal ≥ 6 mm	- CholU: 6/16 (37.5%) - Healthy control: 0/10 (0%)	
Ilves T et al (2016) ¹⁶	Finland	Continental	AD (50)	24	Not defined	0.22	20	15	Diameter of wheal ≥ 2 times larger than immediate post-injection size and at least half diameter of histamine	- AD: 19/50 (38%) - Healthy control: 1/20 (4.2%) - Associated with severity of AD	
Oda Y et al (2020) ²¹	Japan	Temperate	AD (6) CholU (32) Both AD and CholU (9)	None	1:100 1:1000	Not defined	20	15	Diameter of wheal ≥ 6 mm	- All patients: 35/47 (74.4%)	
Altrichter S et al (2020) ¹⁵	Germany	Temperate	CholU (39)	70	1:100	0.45	50	15 and 30	Diameter of wheal > 1.5 mm compared to negative control	- CholU: 28/38 (73.7%) - Healthy control: 1/69 (1.4%)	

Abbreviations: 0.9% NaCl, 0.9% sodium chloride; AD, atopic dermatitis; ASwST, autologous sweat skin test; CholU, cholinergic urticaria

Results

Baseline participant characteristics

Seventy patients (29 CholU, 41 AD) and 20 healthy volunteers were enrolled. Baseline data are detailed in **Table 2**. Compared with the CholU cohort, patients with AD had an earlier disease onset and a greater prevalence of allergic rhinitis and asthma.

Utilization of ASwST

Table 3 summarizes clinical variables stratified by ASwST reactivity; all 20 healthy volunteers showed negative responses.

ASwST was positive in 8/29 CholU patients (27.6%; **Figure 1A**). These 8 patients tended to have longer disease duration than ASwST-negative peers, although the difference was not significant ($P = 0.103$). No other demographic differences emerged.

In the AD cohort, 11/41 patients (26.8%) were ASwST-positive (**Figure 1B**). Demographic and clinical variables, including disease severity, did not differ significantly between reactive and non-reactive groups. The median values of the Scoring Atopic Dermatitis index were lower in ASwST-positive patients, but the difference was not significant ($P = 0.142$).

Table 2. Baseline demographic and clinical characteristics of participants.

	Cholinergic urticaria N = 29 n (%)	Atopic dermatitis N = 41 n (%)	Healthy volunteers N = 20 n (%)
Age, years (mean ± SD)	35.9 ± 13.3	33.4 ± 11.3	31.8 ± 7.1
Age onset, year (mean ± SD)	30.4 ± 15.0	16.8 ± 12.4	-
Sex			
Female	11 (37.9)	23 (56.1)	10 (50.0)
Male	18 (62.1)	18 (43.9)	10 (50.0)
Personal history of atopy			
Atopic dermatitis	0	41 (100)	0
Allergic rhinitis	13 (44.8)	33 (80.5)	3 (15.0)
Asthma	1 (3.4)	5 (12.2)	0
Family history of atopy	13 (44.8)	29 (70.7)	2 (10.0)
Aggravating factor			
Exercise	21 (72.4)	8 (19.5)	-
Hot Bath	2 (6.9)	4 (9.8)	-
Hot and spicy food	7 (24.1)	3 (7.3)	-
Stress	4 (13.8)	6 (14.6)	-

Abbreviations: SD, standard deviation

Table 3. Comparison of participants with positive versus negative autologous sweat skin test results.

	ASwST positive N (%)	ASwST negative N (%)	P value
Healthy volunteers (N = 20)	0	20 (100)	-
Cholinergic urticaria (N = 29)	8 (27.6)	21 (72.4)	-
Age, year (mean ± SD)	38.5 ± 13.6	35.0 ± 13.5	0.532
Disease duration, year (median, IQR)	6.0 (11.0)	2.0 (4.0)	0.103
Sex			0.433
Male	4 (50)	14 (66.7)	-
Female	4 (50)	7 (33.3)	-
Personal history of atopy			-
Atopic dermatitis	0	0	-
Allergic rhinitis	2 (25)	11 (52.4)	0.238
Asthma	0	1 (4.8)	1.000
Family history of atopy	3 (37.5)	10 (47.6)	0.697

Table 3. (Continued)

	ASwST positive N (%)	ASwST negative N (%)	P value
Atopic dermatitis (N = 41)	11 (26.8)	30 (73.2)	
Age, year (mean \pm SD)	34.6 \pm 8.5	33.0 \pm 12.2	0.686
Disease duration, year (median, IQR)	10 (25.0)	14 (18.0)	0.896
Sex			1.000
Male	5 (45.5)	13 (43.3)	-
Female	6 (54.5)	17 (56.7)	-
Personal history of atopy			-
Allergic rhinitis	9 (81.8)	24 (80)	1.000
Asthma	1 (9.1)	4 (13.3)	1.000
Family history of atopy	8 (72.7)	21 (70.0)	1.000
Disease severity			-
SCORAD (mean \pm SD)	21.2 \pm 6.3	25 \pm 7.5	0.142
Severity group			0.736
Mild	7 (63.6)	17 (56.7)	-
Moderate	4 (36.4)	13 (43.3)	-

Abbreviations: ASwST, autologous sweat skin test; IQR, interquartile range; SCORAD, Scoring Atopic Dermatitis; SD, standard deviation

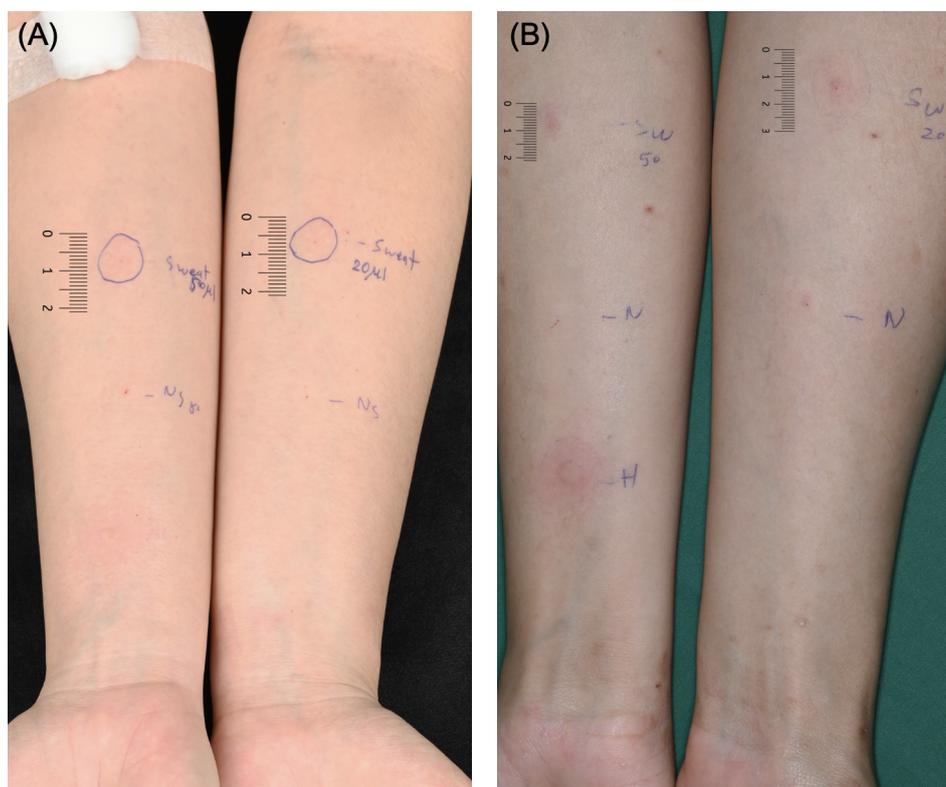


Figure 1. demonstrates positive reaction of the autologous sweat skin test (A) Patient with cholinergic urticaria (B) Patient with atopic dermatitis.

Table 4. Comparison of autologous sweat skin test outcomes using 20- μ L versus 50- μ L injection volumes.

ASwST Result		Injected volume 20 μ L [†] ; n (%)		Total
		Positive	Negative	
Injected volume 50 μ L [‡]	Positive	5 (7.1)	14 (20.0)	19 (27.1)
	Negative	0	51 (72.9)	51 (72.9)
Total		5 (7.1)	65 (92.9)	70 (100.0)

[†]Positive result defined as a wheal diameter \geq 6 mm at 15 min.

[‡]Positive result defined as a wheal diameter induced by diluted sweat \geq 1.5 mm larger than that induced by the negative control at 30 min.

Abbreviations: μ L, microliters; ASwST, autologous sweat skin test

Among ASwST-positive CholU cases, 5 displayed non-follicular wheals—2 purely non-follicular and 3 mixed—and 3 displayed follicular wheals. In AD, ASwST positivity was not linked to disease severity: 7/24 patients (29.2%) with mild disease and 4/17 (23.5%) with moderate disease were positive ($P = 0.736$).

Agreement between ASwST injection volumes

Table 4 compares results obtained with 20 μ L and 50 μ L of autologous sweat. Overall concordance was 80.0%. Negative agreement accounted for 72.9%, and dual-positive agreement for 7.1%. In the remaining 20.0% of cases, only the 50 μ L injection elicited a positive reaction, giving a significantly higher positivity rate for the larger volume than for the smaller (27.1% vs 7.1%; $P = 0.001$).

Discussion

Sweat allergy is frequently reported in patients with CholU and AD.^{6,22} Several studies have shown that it arises from type I immediate hypersensitivity to sweat-specific allergens.^{13,16,21} MGL₁₃₀₄, an antigen derived from *M. globosa*, has been identified as a potential key allergen.⁸

In 1989 and 1994, Adachi et al used radioallergosorbent testing to detect specific IgE antibodies to sweat allergens in patients with AD.^{12,13} They also employed the ASwST to diagnose sweat allergy in CholU.^{12,13} Currently, various diagnostic approaches, including basophil activation and histamine release tests, local provocation with intradermal acetylcholine, and measurement of specific IgE to MGL₁₃₀₄, are available to improve diagnostic sensitivity.^{4-6,9,10,13,21} However, ASwST remains the gold standard in certain studies, with most such investigations conducted in Japan.

To our knowledge, this is the first study to provide data on ASwST utilization in a tropical climate, where higher ambient temperature and humidity may influence the incidence of sweat allergy.

In our study, positive ASwST results were observed in 27.6% of patients with CholU and 26.8% of those with AD. As summarized in our literature review (Table 1), previous investigations have used varied methods and interpretations for ASwST. We employed filtered autologous sweat diluted to 1:100, in keeping with prior protocols. However, 2 injection volumes—20 μ L and 50 μ L—have been studied. Reported positive rates ranged from 37.5% to 100% with 20 μ L,^{7,9,14,21} whereas Altrichter et al, using 50 μ L, documented a 73.7% positive rate.¹⁵ By comparison, we found 7.1% positivity with 20 μ L and 27.1% with 50 μ L. We observed 80.0% agreement between these 2 volumes, suggesting both are reliable. However, 20.0% of cases were positive only with the 50 μ L volume.

Although a standard injection volume for ASwST has neither been formally established nor validated, use of 50 μ L may be considered based on extrapolated evidence from the autologous serum skin test.²³ This approach could enhance the detection of sweat allergy and offer added clinical value by identifying more cases. However, it should be noted that while a larger injected volume may potentially increase the positive rate, differences in positivity criteria between the two volumes may also have contributed to the observed difference. Further studies are warranted to confirm the 50 μ L volume and clarify its clinical interpretation. Our study also involved a larger sample size than prior reports, likely increasing statistical power. Nonetheless, we observed relatively low positivity rates, regardless of injection volume, which could reflect a lower true positivity rate in our tropical population. These findings highlight the need to investigate other possible contributing factors.

In patients with CholU, the ASwST positivity rate in our study was lower than previously reported values of 37% to 100%.^{9,13-15} According to Tanaka et al, symptoms of wheals and pruritus in CholU patients with positive autologous sweat reactions improved after consecutive desensitization using purified sweat antigen.²⁴ After rapid desensitization with autologous sweat, most CholU patients with sweat hypersensitivity also showed symptom improvement and decreased histamine release.⁷ In tropical regions, frequent sweating may lead to natural desensitization, which can yield negative ASwST results. This phenomenon may explain the lower ASwST reactivity observed in our study, possibly from prior sweat-induced desensitization.

Fukunaga et al^{3,17} categorized CholU into 4 subtypes: conventional sweat allergy-type CholU, CholU-peripheral angioedema, follicular-type CholU, and CholU with anhidrosis. The first 2 subtypes typically produce positive ASwST results but negative autologous serum skin test findings. The follicular subtype shows negative ASwST but positive autologous serum skin test results, whereas CholU with anhidrosis displays negative outcomes on sweat allergy testing.

Conventional sweat allergy-type CholU presents with non-follicular wheals and a positive ASwST.^{3,17} Follicular occlusion has been proposed as the underlying mechanism, leading to sweat leakage and subsequent mast cell degranulation provoked by sweat-derived antigens.^{25,26} In our study, among CholU patients who tested positive on ASwST, 5 of them (62.5%) had non-follicular wheals, including 2 with exclusively non-follicular lesions and 3 with mixed non-follicular and follicular wheals. Previous reports noted non-follicular wheals in 63.6% and 50.0% of ASwST-positive patients, respectively.^{9,14} These findings support non-follicular wheals as a characteristic feature of the conventional sweat allergy-type, although other morphologies can occur, suggesting potential phenotypic diversity. Our limited number of ASwST-positive CholU patients calls for additional investigations in tropical settings to clarify the clinical features of sweat allergy in CholU.

In patients with AD, previously reported ASwST positivity rates range from 38% to 95%,^{4,12,16} whereas we found 26.8%. The skin microbiome is believed to drive sweat allergy, primarily through MGL_1304, a major allergen derived from *M. globosa* in human sweat.²⁷ The skin of patients with AD shows greater microbial heterogeneity than that of healthy controls,²⁸ and their serum MGL_1304-specific IgE levels are significantly higher.²⁹ However, the distribution of *Malassezia* spp. differs by region. In Japan,³⁰ *M. globosa* and *M. restricta* predominate in both atopic dermatitis and healthy populations. By contrast, in Thailand—a tropical climate—*M. furfur* predominates among infants with infantile seborrheic dermatitis,³¹ although this finding may not extend to older individuals or those with AD. Another Thai study noted higher *Malassezia* spp. levels in areas with substantial air pollution, suggesting that regional pollution may affect colonization patterns.³² Additionally, specific IgE to MGL_1304 was about 50% lower in a German population than in Japanese patients.^{15,29} These findings imply that variations in climate, pollution, and genetic factors likely contribute to differences in sweat allergy prevalence and positivity rate worldwide.

Although previous reports indicate that *Malassezia* spp. –specific IgE correlates with disease severity in AD,^{16,33} our ASwST findings did not align with AD severity, in agreement with an earlier study.⁴ Advancing knowledge of regional variations in sweat allergy prevalence and positivity rate and clinical phenotypes will be important for improving management of this condition.

Our study has limitations. We did not use other diagnostic methods—beyond ASwST—to evaluate sweat allergy, which might have provided greater sensitivity and stronger correlations. Future research, including basophil activation testing or measuring specific IgE to MGL_1304 in tropical regions, may offer additional insights and enhance diagnostic accuracy.

Conclusion

ASwST proved useful for detecting sweat allergy in CholU and AD under tropical conditions, although the positivity rate was relatively low at approximately 25% to 30%. We observed 80% agreement in ASwST results between the 2 tested volumes, with 20% positivity noted only when using the 50 μ L volume. This finding suggests that both methods can be reliably applied, and that the 50 μ L volume may offer advantages in improving detection. Further research is needed to elucidate the factors contributing to this low positivity rate and to refine diagnostic protocols.

Acknowledgments

The authors gratefully acknowledge Assistant Professor Chulaluk Komoltri for statistical analysis support and Dr Sariya Sittivanaruk for valuable help with coordination.

Competing interests

All authors have neither conflict of interest nor financial support to declare.

Funding

This work was supported by an intramural fund from the Dermatoimmunology Unit, Siriraj Foundation.

References

- Katayama I, Aihara M, Ohya Y, Saeki H, Shimojo N, Shoji S, et al. Japanese guidelines for atopic dermatitis 2017. *Allergol Int.* 2017; 66(2):230-47.
- Hiragun T, Hide M. Sweat Allergy. *Curr Probl Dermatol.* 2016;51:101-8.
- Fukunaga A, Washio K, Hatakeyama M, Oda Y, Ogura K, Horikawa T, et al. Cholinergic urticaria: epidemiology, physiopathology, new categorization, and management. *Clin Auton Res.* 2018;28(1):103-13.
- Hide M, Tanaka T, Yamamura Y, Koro O, Yamamoto S. IgE-mediated hypersensitivity against human sweat antigen in patients with atopic dermatitis. *Acta Derm Venereol.* 2002;82(5):335-40.
- Tanaka A, Tanaka T, Suzuki H, Ishii K, Kameyoshi Y, Hide M. Semi-purification of the immunoglobulin E-sweat antigen acting on mast cells and basophils in atopic dermatitis. *Exp Dermatol.* 2006;15(4): 283-90.
- Takahagi S, Tanaka T, Ishii K, Suzuki H, Kameyoshi Y, Shindo H, et al. Sweat antigen induces histamine release from basophils of patients with cholinergic urticaria associated with atopic diathesis. *Br J Dermatol.* 2009;160(2):426-8.
- Kozaru T, Fukunaga A, Taguchi K, Ogura K, Nagano T, Oka M, et al. Rapid desensitization with autologous sweat in cholinergic urticaria. *Allergol Int.* 2011;60(3):277-81.
- Hiragun T, Ishii K, Hiragun M, Suzuki H, Kan T, Mihara S, et al. Fungal protein MGL_1304 in sweat is an allergen for atopic dermatitis patients. *J Allergy Clin Immunol.* 2013;132(3):608-15 e4.
- Fukunaga A, Bito T, Tsuru K, Oohashi A, Yu X, Ichihashi M, et al. Responsiveness to autologous sweat and serum in cholinergic urticaria classifies its clinical subtypes. *J Allergy Clin Immunol.* 2005;116(2): 397-402.

10. Ishii K, Hiragun M, Hiragun T, Kan T, Kawaguchi T, Yanase Y, et al. A human monoclonal IgE antibody that binds to MGL_1304, a major allergen in human sweat, without activation of mast cells and basophils. *Biochem Biophys Res Commun.* 2015;468(1-2):99-104.
11. Ishibashi Y, Kato H, Asahi Y, Sugita T, Nishikawa A. Identification of the major allergen of *Malassezia globosa* relevant for atopic dermatitis. *J Dermatol Sci.* 2009;55(3):185-92.
12. Adachi K, Aoki T. IgE antibody to sweat in atopic dermatitis. *Acta Derm Venereol Suppl (Stockh).* 1989;144:83-7.
13. Adachi J, Aoki T, Yamatodani A. Demonstration of sweat allergy in cholinergic urticaria. *J Dermatol Sci.* 1994;7(2):142-9.
14. Kim JE, Jung KH, Cho HH, Kang H, Park YM, Park HJ, et al. The significance of hypersensitivity to autologous sweat and serum in cholinergic urticaria: cholinergic urticaria may have different subtypes. *Int J Dermatol.* 2015;54(7):771-7.
15. Altrichter S, Schumacher P, Alraboni O, Wang Y, Hiragun M, Hide M, et al. Sensitization against skin resident fungi is associated with atopy in cholinergic urticaria patients. *Clin Transl Allergy.* 2020;10:18.
16. Ilves T, Virolainen A, Harvima IT. Immediate Wheal Reactivity to Autologous Sweat in Atopic Dermatitis Is Associated with Clinical Severity, Serum Total and Specific IgE and Sweat Tryptase Activity. *Int Arch Allergy Immunol.* 2016;170(2):84-91.
17. Fukunaga A, Oda Y, Imamura S, Mizuno M, Fukumoto T, Washio K. Cholinergic Urticaria: Subtype Classification and Clinical Approach. *Am J Clin Dermatol.* 2023;24(1):41-54.
18. Commens CA, Greaves MW. Tests to establish the diagnosis in cholinergic urticaria. *Br J Dermatol.* 1978;98(1):47-51.
19. Hanifin J, Rajka G. Diagnostic Features of Atopic Dermatitis. *Acta Dermato-Venereologica.* 1980.
20. Severity scoring of atopic dermatitis: the SCORAD index. Consensus Report of the European Task Force on Atopic Dermatitis. *Dermatology.* 1993;186(1):23-31.
21. Oda Y, Washio K, Fukunaga A, Imamura S, Hatakeyama M, Ogura K, et al. Clinical utility of the basophil activation test in the diagnosis of sweat allergy. *Allergol Int.* 2020;69(2):261-7.
22. Takahagi S, Tanaka A, Hide M. Sweat allergy. *Allergol Int.* 2018; 67(4):435-41.
23. Konstantinou GN, Asero R, Maurer M, Sabroe RA, Schmid-Grendelmeier P, Grattan CE. EAACI/GA(2)LEN task force consensus report: the autologous serum skin test in urticaria. *Allergy.* 2009;64(9):1256-68.
24. Tanaka T, Ishii K, Suzuki H, Kameyoshi Y, Hide M. [Cholinergic urticaria successfully treated by immunotherapy with partially purified sweat antigen]. *Arerugi.* 2007;56(1):54-7.
25. Horikawa T, Fukunaga A, Nishigori C. New concepts of hive formation in cholinergic urticaria. *Curr Allergy Asthma Rep.* 2009;9(4):273-9.
26. Murota H, Yamaga K, Ono E, Katayama I. Sweat in the pathogenesis of atopic dermatitis. *Allergol Int.* 2018;67(4):455-9.
27. Hiragun T, Ishii K, Hiragun M, Suzuki H, Kan T, Mihara S, et al. Fungal protein MGL_1304 in sweat is an allergen for atopic dermatitis patients. *J Allergy Clin Immunol.* 2013;132(3):608-15.e4.
28. Sugita T, Suto H, Unno T, Tsuboi R, Ogawa H, Shinoda T, et al. Molecular analysis of *Malassezia* microflora on the skin of atopic dermatitis patients and healthy subjects. *J Clin Microbiol.* 2001;39(10): 3486-90.
29. Hiragun M, Hiragun T, Ishii K, Suzuki H, Tanaka A, Yanase Y, et al. Elevated serum IgE against MGL_1304 in patients with atopic dermatitis and cholinergic urticaria. *Allergol Int.* 2014;63(1):83-93.
30. Harada K, Saito M, Sugita T, Tsuboi R. *Malassezia* species and their associated skin diseases. *J Dermatol.* 2015;42(3):250-7.
31. Wanankul S, Chindamporn A, Yumyourn P, Payungporn S, Samathi C, Poovorawan Y. *Malassezia furfur* in infantile seborrheic dermatitis. *Asian Pac J Allergy Immunol.* 2005;23(2-3):101-5.
32. Chueachavalit C, Meeaphansan J, Payungporn S, Sawaswong V, Chanchaem P, Wongpiyabovorn J, et al. Comparison of *Malassezia* spp. colonization between human skin exposed to high- and low-ambient air pollution. *Exp Dermatol.* 2022;31(9):1454-61.
33. Glatz M, Buchner M, von Bartenwerffer W, Schmid-Grendelmeier P, Worm M, Hedderich J, et al. *Malassezia* spp.-specific immunoglobulin E level is a marker for severity of atopic dermatitis in adults. *Acta Derm Venereol.* 2015;95(2):191-6.