Continuum of Light Sensitivity in Atopic Dermatitis

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Continuum of light sensitivity in atopic dermatitis: A retrospective analysis of 139 cases in Scotland

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Background: Previous reports have characterized photosensitivity in atopic dermatitis (AD), but with differences in terminology and criteria.

Objective: This study aims to assess outcomes in 139 patients with AD referred for photodiagnostic testing and to establish diagnostic criteria for photosensitivity in AD.

Methods: Clinical and photodiagnostic data were reviewed, categorizing photosensitivity into photoexacerbated AD, photosensitive AD, and chronic actinic dermatitis.

Results: Of the patient cohort, the mean age was 42.6 ± 16.7 years, and 61.9% were men. In total, 51.1% of the patients with photoexacerbated AD had normal monochromator phototesting, and 7.9% of the patients with photosensitive AD displayed slight-to-moderate ultraviolet (UV)-A sensitivity (≥30% of normal minimal erythema dose [MED]) and mostly normal or slightly reduced UV-B MEDs (≥80% of normal MED). Conversely, 41% of the patients had chronic actinic dermatitis, and 93% of this group demonstrated significant UV-B sensitivity, with very low UV-B MEDs (<80% of normal MED) and/or very low UV-A MEDs (<30% of normal MED). No significant differences in sex, age, or skin phototype were observed between the groups. Serial phototesting revealed changes in photosensitivity status over time in 8 patients.

Limitations: A small sample size and retrospective design.

Conclusions: This study highlights the heterogeneity of photosensitivity patterns in patients with AD and the importance of follow-up assessments due to potential shifts in disease spectrum over time. (J Am Acad Dermatol 2024;91:1086-93.)

Key words: atopic dermatitis; chronic actinic dermatitis; photoaggravated; photoexacerbated; photosensitivity; phototesting.

INTRODUCTION

Photoaggravated dermatoses refer to skin conditions that may be present in the absence of light but are exacerbated on exposure to optical radiation.[1] Many skin diseases can be photoaggravated, including atopic dermatitis (AD). Although it is well established that AD is improved by sunlight exposure or phototherapy in most patients, in a subset of patients with AD, their condition is exacerbated by sunlight.[1]

Photosensitivity in AD has been investigated but has not been characterized as a unifying entity, with reports of discrete states of photoaggravated AD, photosensitive AD, photo-exacerbated AD, photosensitive eczema, and more definitively, chronic actinic dermatitis (CAD).[2] There have been

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differences in the terminology and criteria applied for the diagnosis of light sensitivity status in AD between phototesting centers.[2-5] The objective of this study was firstly to retrospectively review the photodiagnostic data of patients with AD who were referred to the Scottish photobiology service (SPS) and to use these data to establish diagnostic criteria for photosensitivity status in AD.

CAPSULE SUMMARY

- This study characterizes the photosensitivity patterns seen in a subset of patients with atopic dermatitis.
- In patients with AD with suspected light exacerbation, it is important to be aware of the continuum of photosensitivity, categorized as: photoexacerbated AD, photosensitive AD, and chronic actinic dermatitis, which can change within patients over time.

METHODS

The photobiology unit at Ninewells Hospital and Medical School, Dundee, is the photodiagnostic tertiary referral center in Scotland (the SPS). We reviewed patients with AD who were referred to the SPS for investigation from January 2017 to January 2023, based on clinical suspicion of light sensitivity, such as rash predominately on the photodistributed sites, history of rash worsening after sunlight exposure or phototherapy, or a change in pattern or seasonality of AD. The demographic data, clinical features, photodiagnostic investigation findings, and other related outcomes were collected. Lupus was ruled out through clinical assessment and lupus serology where indicated. Patients with possible drug-induced photosensitivity were excluded. The National Health Service Tayside Caldicott Guardian (IGTCAL12180) approval was obtained.

The photobiological assessment included monochromator phototesting.[6] Geometric dose-series of irradiations were undertaken on the back at the following narrow wavebands (±half-maximum bandwidth): 305 ± 5 nm, 335 ± 27 nm, 365 ± 27 nm, 400 ± 27 nm, 430 ± 27 nm. The minimal erythema dose (MED), defined as the lowest dose to produce just perceptible erythema at 24 hours after irradiation, was determined at each waveband. An abnormal MED in each waveband was defined when compared with the MEDs derived in our healthy volunteer population (skin phototype I-III).[7] Narrowband ultraviolet (UV)B (TL-01) MED testing was also undertaken, with a cut-off at 0.025 J/cm^2, with MEDs below this being abnormal based again on data derived from a skin phototype I to III population.

Photoprovocation was performed in selected patients by using broadband UV-A (320-400 nm), with cumulative doses varying from 5 to 40 Joules/cm^2 undertaken over 1 to 3 consecutive days. Where indicated, photopatch testing was undertaken using the standard methodology and European battery,[8,9] consisting of 19 photoallergens. The UVA irradiation at 5 J/cm^2 (or lower dose or unirradiated if patients had low or severely low UVA MEDs) of one set of photoallergens was undertaken 24 hours later. Readings were performed at 24 and 48 hours after irradiation. Assessment and interpretation of readings used the international contact dermatitis research group guideline.

Final diagnosis after photodiagnostic assessment was categorized into 1 of the 3 groups: photoexacerbated AD (PEAD), photosensitive AD (PSAD), and CAD based on the diagnostic criteria, as shown in Fig 1.

RESULTS

A total of 139 patients with AD who were referred for photodiagnostic assessment over the period of the study were included. There were more men (61.9%) than women, and the mean age ± standard deviation (SD) at diagnosis was 42.6 ± 16.7 years. Most patients were of skin phototype (SPT) I to III (88.1%). Comorbidities of allergic rhinitis and asthma were present in 81.4% and 79.1%, respectively. When categorized according to final diagnoses, PEAD was diagnosed in 71 patients (51.1%), followed by CAD (57 patients, 41%), and PSAD (11 patients, 7.9%). Table I highlights the demographic data and clinical characteristics of these 3 groups. No difference in sex, age, or SPT was seen between the diagnostic categories ($P > .05$).

The light exposure time required to trigger symptoms and rash was usually <1 hour in the patients diagnosed with CAD (84%). Although around half of the patients diagnosed with PSAD and PEAD reported that ≥1 hour of exposure was required to trigger symptoms and rash. Time to onset of rash after the triggering exposure was <1 day in patients with CAD (100%), whereas nearly half of patients with PEAD reported that it would take >1 day to develop rash after triggering levels of sun exposure. The symptoms and rash of PEAD (87%) and PSAD (100%) were mostly perennial, but surprisingly, the perennial history of rash was lower in CAD (58.3% vs 87%-100%; $P = .041$). The proportion of patients with a history of rash triggered with light through clothing (58.1% vs 29.2%; $P = .023$) was higher in patients with CAD, with only a nonsignificant trend seen with rash

Abbreviations used:

AD:	atopic dermatitis
CAD:	chronic actinic dermatitis
DLQI:	Dermatological Life Quality Index
Ig:	immunoglobulin
MED:	minimal erythema dose
PEAD:	photoexacerbated atopic dermatitis
PLE:	polymorphic light eruption
PSAD:	photosensitive atopic dermatitis
SPT:	skin phototype
SPS:	Scottish photobiology service
UV:	ultraviolet

triggered through window glass (54.3% vs 33.3-34.6%; $P = .365$). Induction of symptoms and rash was limited to photo-exposed sites only in about 25% of the patients with AD cohort, and this proportion did not vary between the 3 diagnostic groups. Although the hardening data were not complete ($n = 17$), hardening was observed in 33.3% and 50% of patients with PEAD and CAD, respectively.

Monochromator phototesting was normal in patients with PEAD. Patients diagnosed with PSAD showed slight-to-moderate UV-A sensitivity defined as reduction in 24-hour MED to 335 ± 27 nm, 365 ± 27 nm, or 400 ± 27 nm (UV-A region, within 30% of normal MED range). Normal UVB MEDs (305 ± 5 nm) were observed in patients diagnosed with PSAD, except for 2 patients who had slightly low UV-B MEDs (within 80% of the normal range). In contrast, in patients diagnosed with CAD, 53 of 57; 93% had marked sensitivity to UVB (305 ± 5 nm), with MEDs <80% of the normal MED range and/or very low UV-A MEDs at <30% of the normal MED range (Table II).

Notably, 3 patients from the CAD group who demonstrated normal UV-B MEDs at 305 ± 5 nm but showed markedly abnormal UV-A MED (<50% MED UV-A) were of SPT IV or V. These patients were diagnosed with CAD, as the normal range is based on skin phototypes I to III. Another patient with CAD had a normal 305 ± 5 nm UV-B MED on first phototesting but was diagnosed with CAD based on subsequent lower 305 ± 5 nm MEDs on repeat phototesting.

TL-01 MEDs were evaluated in 113 patients. All patients in the PEAD group had normal TL-01 MEDs. One of the 11 patients in the PSAD group had an abnormally low TL-01 MED, although with only minimal erythema evident below a dose of 0.097 J/cm^2. In the CAD group, 37.5% (15 from 40) had abnormally low TL-01MEDs, typically with moderate to palpable erythemal reactions. Thus, an abnormally low TL-01 MED may indicate underlying abnormal photosensitivity conditions (CAD or PSAD) in AD (Fig 2) but cannot be relied on as a screen as a normal TL-01 MED does not definitively rule out CAD or PSAD, and other factors, such as SPT and degree of erythemal response, must be taken into account.

Broadband UV-A photoprovocation testing was performed in 25 patients, showing positive papules or eczema in 5 patients. The proportion of patients with positive photopatch (60.9%)/patch testing (76.4%) was high but not significantly different across the 3 groups. Vitamin D insufficiency (25-50 nmol/L) and deficiency (<25 nmol/L) were highly prevalent in our patient cohort. Only 36.4% of patients with AD had adequate vitamin D levels. However, there were no significant differences in vitamin D status between the 3 groups. Similarly, there was no significant difference between the groups in total immunoglobulin (Ig)E levels, or Dermatological Life Quality Index (DLQI) scores

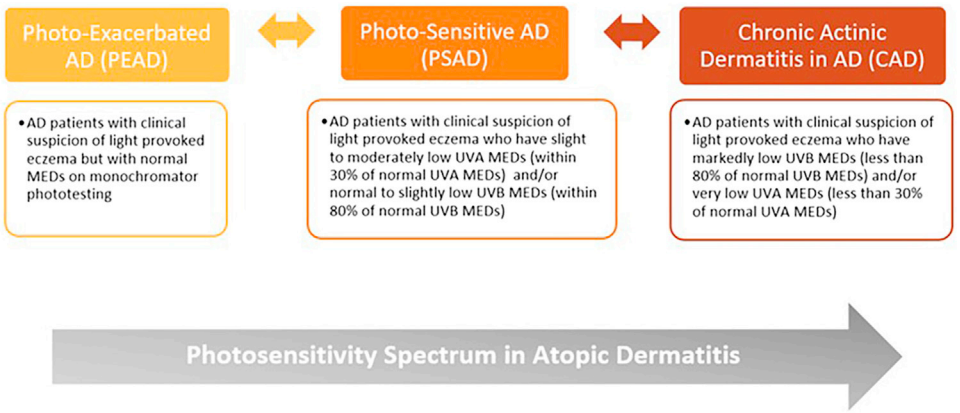

Fig 1. The spectrum of photosensitivity in patients with atopic dermatitis and a clinical suspicion of photosensitivity. *AD*, Atopic dermatitis; *CAD*, chronic actinic dermatitis; *MED*, minimal erythema dose; *UV*, ultraviolet.

Table I. Demographic data and clinical characteristics in patients with AD and a clinical suspicion of light sensitivity

	All patients with AD	Photoexacerbated AD (PEAD)	Photosensitive AD (PSAD)	Chronic actinic dermatitis (CAD)	
	N = 139	*n* = 71	*n* = 11	*n* = 57	
	n (%)	*n* (%)	*n* (%)	*n* (%)	*P**
Demographics					
Sex					
Male	86 (61.9)	39 (54.9)	8 (72.7)	39 (68.4)	.233
Age (y), mean ± SD	42.6 ± 16.7	43.9 ± 16.9	38.0 ± 16.7	41.9 ± 16.5	.504
Skin phototype (*n* = 101)					
I-III	89 (88.1)	44 (89.8)	7 (87.5)	38 (86.4)	.898
IV-VI	12 (11.9)	5 (10.2)	1 (12.5)	6 (13.6)	
Comorbidity					
Allergic rhinitis (*n* = 86)	70 (81.4)	39 (83)	4 (50)	27 (87.1)	.059
Asthma (*n* = 86)	68 (79.1)	39 (83)	4 (50)	25 (80.6)	.108
Clinical characteristics					
Median duration of symptoms, months (IQR), (*n* = 112)	36 (18.8-96)	30 (24-60)	36 (12.8-48)	48 (18-120)	.530
Rash distribution (*n* = 114)					
Photo-exposed site	29 (25.4)	13 (21)	2 (18.2)	14 (34.1)	.314
Timing of rash related to sunlight exposure					
Time exposure (*n* = 47)					
Minutes (0-59)	33 (70.2)	11 (55)	1 (50)	21 (84)	.131
Hours (1-5)	14 (29.8)	9 (45)	1 (50)	4 (16)	
Time to onset (*n* = 39)					
Minutes (0-59)	11 (28.2)	5 (26.3)	-	6 (31.6)	.002
Hours (1-23)	19 (48.7)	5 (26.3)	1 (100)	13 (68.4)	
Days (1-2)	9 (23.1)	9 (47.4)	-	-	
Duration of rash (*n* = 52)					
Days (1-6)	30 (57.7)	16 (64)	1 50	13 (52)	.709
Weeks (1-3)	21 (40.4)	8 (32)	1 50	12 (48)	
Months (≥1)	1 (1.9)	1 (4)		-	
Perennial (*n* = 51)	38 (74.5)	20 (87)	4 (100)	14 (58.3)	.041
Hardening (*n* = 17)	7 (41.2)	2 (33.3)	0 (0)	5 (50)	.784
Trigger through clothing (*n* = 58)	25 (43.1)	7 (29.2)	0 (0)	18 (58.1)	.023
Trigger through window glass (*n* = 64)	29 (45.3)	9 (34.6)	1 (33.3)	19 (54.3)	.365
Trigger through indoor light (*n* = 55)	5 (9.1)	2 (7.7)	2 (100)	1 (3.7)	.004
DLQI score, mean ± SD, (*n* = 117)	17.1 ± 6.8	17.8 ± 6.7	17.4 ± 7.3	16.0 ± 6.9	.398

AD, Atopic dermatitis; *ACD*, allergic contact dermatitis; *CAD*, chronic actinic dermatitis.
**P* value compared between PEAD, PSAD, and CAD.

($P > .05$). Notably, median IgE levels were very high (>1000 ku/L), indicating that our patient cohort was a markedly atopic group with severe AD.

Two patients were first diagnosed as PSAD, and subsequent serial phototesting indicated that they had evolved into CAD, with marked UV-B sensitivity. In contrast, on follow-up, 4 patients with CAD had normalization of phototesting and reverted to PEAD, and 2 patients showed resolution of 305 ± 5 nm (UV-B) sensitivity and had only residual 335 ± 27 nm or 365 ± 27 nm (UV-A) sensitivity, indicating reversion to PSAD. All these changes in light sensitivity status occurred over a 1 to 2 year follow-up period (Fig 3). Five patients in the PEAD group were also diagnosed with coexisting polymorphic light eruption (PLE). These patients generally had a history consistent with PLE, notably a delayed onset of papular or vesicular rash after sun exposure, which was quite distinct from their history of AD. Monochromator phototesting was normal in all 5 of these patients, and photoprovocation testing was undertaken in 3 of them, showing typical papular PLE changes in 2.

Table II. Photobiological assessment and other investigations in patients with AD and a clinical suspicion of light sensitivity

	All patients with AD	Photoexacerbated AD (PEAD)	Photosensitive AD (PSAD)	Chronic actinic dermatitis (CAD)	
	N = 139	*n* = 71	*n* = 11	*n* = 57	
	N (%)	*n* (%)	*n* (%)	*n* (%)	*P**
Photobiological assessment					
Abnormal delayed erythematous response in monochromator phototesting					
305 ± 5 nm (*n* = 139)	55 (39.6)	0	2 (18.2)	53 (93.0)	-
335 ± 27 nm (*n* = 137)	56 (42.3)	0	7 (63.6)	49 (87.5)	-
365 ± 27 nm (*n* = 139)	49 (35.3)	0	5 (45.5)	44 (77.2)	-
400 ± 27 nm (*n* = 137)	15 (10.9)	0	1 (9.1)	14 (25)	-
430 ± 27 nm (*n* = 129)	1 (0.8)	0	0 (0)	1 (1.9)	-
Abnormal TL-01 MED (*n* = 113)	16 (14.2)	0	1 (9.1)	15 (37.5)	-
Photoprovocation test (*n* = 25)	5 (20)	4 (30.8)	-	1 (11.1)	-
Photopatch test (*n* = 46)					
Positive reaction	28 (60.9)	10 (55.6)	-	18 (64.3)	.554
PACD	5 (10.9)	3 (16.7)	-	2 (7.1)	
ACD	18 (39.1)	7 (38.9)	-	11 (39.3)	
PACD + ACD	5 (10.9)	-	-	5 (17.9)	
Other investigations					
Patch test (*n* = 90)					
ACD	68 (76.4)	28 (68.3)	7 (87.5)	33 (82.5)	.280
Median total IgE (ku/L), IQR, (*n* = 97)	1535 (205-5000)	1778.5 (235.5-5000)	2598 (37.5-5000)	1012.5 (138.5-3451)	.592
Vitamin D (*n* = 66)					
Deficient (<25 nmol/L)	17 25.8	5 20.8	3 50	9 25	.326
Insufficient (25-50 nmol/L)	25 37.9	8 33.3	3 50	14 38.9	
Adequate (>50 nmol/L)	24 36.4	11 45.8	-	13 36.1	

AD, Atopic dermatitis; *ACD,* Allergic contact dermatitis; *CAD* chronic actinic dermatitis; *DLQI,* dermatology life quality index; *IgE,* immunoglobulin E; *MED,* minimal erythematous dose; *PACD,* photoallergic contact dermatitis.
**P* value compared between PEAD, PSAD, and CAD.

DISCUSSION

This study sought to further clarify the characteristics and diagnostic categories of photosensitivity in patients with AD who had been referred for investigation of possible light sensitivity. Based on our data and the existing literature,[2-5] while we consider that there are 3 diagnostic categories, PEAD, PSAD, and CAD, this should be considered as a continuum of disease.

The variability in light sensitivity observed among this subset of patients with AD, where light has an exacerbating role, underscores the dynamic nature of the condition, likely influenced by factors such as disease progression, environmental exposure patterns, and treatment efficacy. For instance, individuals initially diagnosed with PSAD may exhibit worsening symptoms and evolve into CAD, characterized by marked UV-B sensitivity. On the contrary, some patients with CAD may experience normalization of phototesting results and revert to a less severe form of light sensitivity, such as PEAD or PSAD. These fluctuations highlight the importance of longitudinal monitoring and repeat phototesting in the management of patients with AD and the importance of both the patient and clinical team being aware of the potential for change.

Furthermore, our data emphasize that it can be impossible to distinguish between the 3 diagnoses based on clinical characteristics alone. Although narrowband UV-B TL-01 MED testing is an essential investigation to undertake in patients with AD, if considering phototherapy, the TL-01 MED on its own is not sufficient as a screening tool, and more detailed monochromator phototesting is advised if there is diagnostic doubt.

On review of existing literature, there is variation in the terminology used and the criteria applied for photosensitivity in AD. The excellent study of Rutter et al,[4] thoroughly reviewed 120 patients with photoaggravated AD. Abnormal monochromator phototesting was found in 23%, and broadband UV provocation testing was positive in 93% of their patients. However, 39% of the patients of SPT V-VI had monochromator sensitivity to at least 1 waveband,

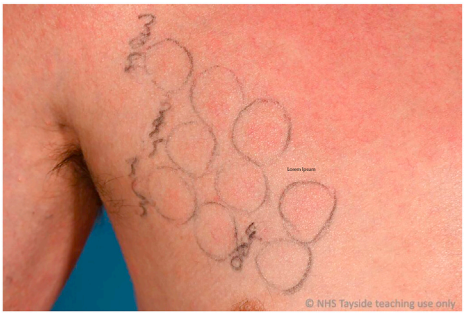

Fig 2. A young man of SPT II, with a history of severe atopic dermatitis develops worsening involvement of head and neck, initially mistaken for photoaggravation; Phototherapy was considered but TL-01 MED was abnormally low. Further monochromator phototesting at chest wall (as shown) revealed abnormal broadband UV sensitivity MED at 305 ± 5 nm <10 (lowest normal [LN] 33) mJ/cm^2; at 335 ± 27 nm <1200 (LN 3900) mJ/cm^2; at 365 ± 27 nm <6800 (LN 18,000) mJ/cm^2; 400 ± 27 nm <56 000 (LN 56,000) mJ/cm,^{2} and narrow band UVB TL-01 MED 23 (LN 25) mJ/cm^2) and a diagnosis of chronic actinic dermatitis was made. *MED*, Minimal erythema dose.

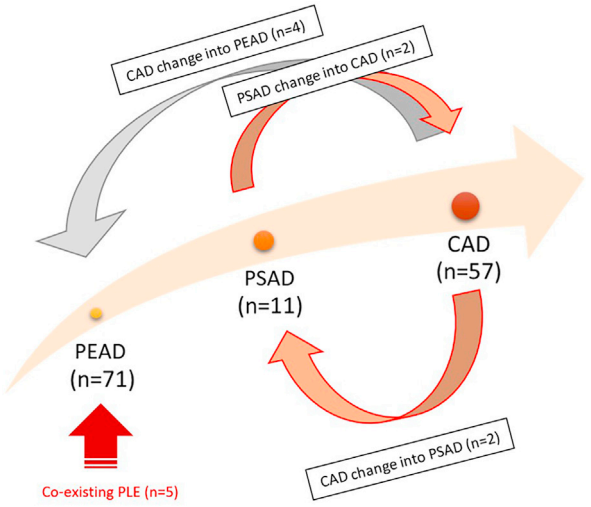

Fig 3. Longitudinal changes in light sensitivity status. Two patients with PSAD progressed to CAD with marked UV-B sensitivity, whereas 4 patients with CAD reverted to PEAD. Two patients transitioned from CAD to PSAD, exhibiting resolution of UV-B sensitivity. Coexisting PLE was diagnosed in 5 patients with PEAD. *CAD*, Chronic actinic dermatitis; *PEAD*, photoexacerbated atopic dermatitis; *PSAD*, photosensitive atopic dermatitis; *PLE*, polymorphic light eruption.

indicating that these patients may fit better within the diagnostic criteria for CAD, particularly as again, the normal range for MEDs employed in the United Kingdom at present was derived from healthy volunteers of SPT I to III. Furthermore, studies have also indicated that patients of higher skin phototypes are overrepresented in those diagnosed with early-onset CAD.[10] Recent studies from China identified photosensitive AD in patients with AD who demonstrated any abnormalities in phototesting outcomes, which included abnormal MED or minimal phototoxic dose values or having abnormal skin responses at the phototesting sites (pruritus, pain, papules, and diffuse erythema).[5] Studies from Germany included 17 photosensitive patients with AD, all of whom showed positive photoprovocation tests but had normal MEDs from broadband phototesting. Photoprovocation reactions in their patients were described as of 2 types: papular and eczematous reactions, which shared a similar histologic pattern.[3] In contrast, studies from the Netherlands identified photosensitive AD only in patients who had positive photoprovocation tests of an eczematous type.[2]

Our proposed diagnostic criteria for photosensitivity in AD are not dependent on larger area photoprovocation testing, as we have shown that a negative photoprovocation test cannot rule out photosensitivity in AD, and conversely, a positive photoprovocation may represent aggravation of subclinical disease rather than true light sensitivity. As larger area photoprovocation testing is less well standardized and normal range data are not available, we consider narrow waveband small area phototesting using a monochromator is more reliable as a means of diagnosing photosensitivity status in this patient cohort, although it may be complemented by larger area UV provocation.

Polymorphic light eruption often coexists with AD, given the prevalence of PLE in 18% of Northern Europeans.[11] Awareness of a coincidental history of PLE based on careful history taking to elicit the key features of a delayed onset papular or vesicular rash following sun exposure, resolving over a few days, is important as there is potential for photoaggravation of AD through the route of PLE-triggering and subsequent koebnerization of AD after either natural sunlight exposure or phototherapy.[2]

Positive photopatch and patch testing was reported in 60.9% and 76.4% of our cohort, emphasizing the importance of patch or photopatch testing as an investigation in AD with photosensitivity. Interestingly, a recent meta-analysis did not show a higher prevalence of allergic contact dermatitis in a general AD group.[12] Of importance, in this study positive patch or photopatch testing did not significantly differ between PEAD, PSAD, and CAD.

Although only 36.4% of our AD patient cohort had adequate vitamin D levels, there was no significant difference in vitamin D status between the 3 groups. The high proportion of patients with low vitamin D levels likely reflects sun avoidance behavior in this patient cohort, compounded by living in Scotland.[13]

Vitamin D assessment or supplementation is thus an essential investigation in patients with suspected or confirmed photosensitivity, particularly for bone health.

Photosensitivity diseases can significantly adversely affect the quality of life, such as limiting social and occupational activities and influencing clothing choices.[14] Consequently, our patients demonstrated a mean DLQI of around 17, indicating a severe adverse effect on quality of life and surpassing the previously reported DLQI range of 4.2 to 10.2 seen in general AD cases.[15-18]

The strengths of this study are that it is a well-characterized patient cohort of reasonable size and with well-defined clinical and photodiagnostic phenotypic data derived from a Scottish patient cohort who were assessed through the tertiary SPS. We recognize that most patients with AD will find their disease is improved by sun exposure, and thus we have analyzed data on the minority of patients with AD overall, those with possible light-induced worsening of their disease. Thus, any conclusions from our study must bear this in mind and must not be extrapolated to the general population with AD. A further unavoidable limitation of our study was that our diagnostic criteria were based on monochromator phototesting outcomes using a reference range for people of SPT I to III, in keeping with other published studies. This recognizes that there are currently no available normal population reference ranges available for people of higher SPT IV to VI, and this is a priority to assess moving forward.[6]

In conclusion, for patients with AD referred for photodiagnostic investigations, we propose the criteria and identify the diagnostic categories of PEAD, PSAD, and CAD. These entities represent a spectrum of disease, which lies on a continuum, with real-life change between the diagnostic groups occurring in some patients over time. Our findings additionally highlight the importance of photodiagnostic investigations, as clinical assessment alone does not suffice. Furthermore, repeated phototesting over time can be informative in detecting a change in diagnostic grouping of these patients, particularly in the event of a change in clinical features, and awareness of this is important for patients and their clinicians.

Conflicts of interest

None disclosed.

REFERENCES

1. O'Gorman SM, Murphy GM. Photoaggravated disorders. *Dermatol Clin*. 2014;32(3):385-398, ix. https://doi.org/10.1016/j.det.2014.03.008
2. ten Berge O, van Weelden H, Bruijnzeel-Koomen CAFM, de Bruin-Weller MS, Sigurdsson V. Throwing a light on photosensitivity in atopic dermatitis: a retrospective study. *Am J Clin Dermatol*. 2009;10(2):119-123. https://doi.org/10.2165/00128071-200910020-00004
3. Ellenbogen E, Wesselmann U, Hofmann SC, Lehmann P. Photosensitive atopic dermatitis—a neglected subset: clinical, laboratory, histological and photobiological workup. *J Eur Acad Dermatol Venereol*. 2016;30(2):270-275. https://doi.org/10.1111/jdv.13451
4. Rutter KJ, Farrar MD, Marjanovic EJ, Rhodes LE. Clinicophotobiological characterization of photoaggravated atopic dermatitis. *JAMA Dermatol*. 2022;158(9):1022-1030. https://doi.org/10.1001/jamadermatol.2022.2823
5. Gao H, Zhang Z, Ma L, Xu Y, Hu Y, Luo X. Photosensitivity is an important cause of refractory facial erythema in atopic dermatitis: a retrospective study of 82 Chinese patients. *Photodermatol Photoimmunol Photomed*. 2023;39(4):318-324. https://doi.org/10.1111/phpp.12836
6. Ibbotson SH, Allan D, Dawe RS, et al. Photodiagnostic services in the UK and Republic of Ireland: a British Photodermatology Group Workshop Report. *J Eur Acad Dermatol Venereol*. 2021;35(12):2448-2455. https://doi.org/10.1111/jdv.17632
7. Moseley H, Naasan H, Dawe RS, Woods J, Ferguson J. Population reference intervals for minimal erythemal doses in monochromator phototesting. *Photodermatol Photoimmunol Photomed*. 2009;25(1):8-11. https://doi.org/10.1111/j.1600-0781.2009.00391.x
8. Bruynzeel DP, Ferguson J, Andersen K, et al. Photopatch testing: a consensus methodology for Europe. *J Eur Acad Dermatol Venereol*. 2004;18(6):679-682. https://doi.org/10.1111/j.1468-3083.2004.01053.x
9. European Multicentre Photopatch Test Study (EMCPPTS) Taskforce. A European multicentre photopatch test study. *Br J Dermatol*. 2012;166(5):1002-1009. https://doi.org/10.1111/j.1365-2133.2012.10857.x
10. Tan K-W, Haylett AK, Ling TC, Rhodes LE. Comparison of demographic and photobiological features of chronic actinic dermatitis in patients with *Lighter vs Darker* skin types. *JAMA Dermatol*. 2017;153(5):427-435. https://doi.org/10.1001/jamadermatol.2016.5861
11. Rhodes LE, Bock M, Janssens AS, et al. Polymorphic light eruption occurs in 18% of Europeans and does not show higher prevalence with increasing latitude: multicenter survey of 6,895 individuals residing from the Mediterranean to Scandinavia. *J Invest Dermatol*. 2010;130(2):626-628. https://doi.org/10.1038/jid.2009.250
12. Hamann CR, Hamann D, Egeberg A, Johansen JD, Silverberg J, Thyssen JP. Association between atopic dermatitis and contact sensitization: a systematic review and meta-analysis. *J Am Acad Dermatol*. 2017;77(1):70-78. https://doi.org/10.1016/j.jaad.2017.02.001
13. Rhodes LE, Webb AR, Berry JL, et al. Sunlight exposure behaviour and vitamin D status in photosensitive patients: longitudinal comparative study with healthy individuals at U.K. latitude. *Br J Dermatol*. 2014;171(6):1478-1486. https://doi.org/10.1111/bjd.13325
14. Rutter KJ, Ashraf I, Cordingley L, Rhodes LE. Quality of life and psychological impact in the photodermatoses: a systematic review. *Br J Dermatol*. 2020;182(5):1092-1102. https://doi.org/10.1111/bjd.18326
15. Beikert FC, Langenbruch AK, Radtke MA, Kornek T, Purwins S, Augustin M. Willingness to pay and quality of life in patients with atopic dermatitis. *Arch Dermatol Res*. 2014;306(3):279-286. https://doi.org/10.1007/s00403-013-1402-1

16. Paul C, Griffiths CEM, Costanzo A, et al. Factors predicting quality of life impairment in adult patients with atopic dermatitis: results from a patient survey and machine learning analysis. *Dermatol Ther (Heidelb)*. 2023;13(4):981-995. https://doi.org/10.1007/s13555-023-00897-0
17. Silverberg JI, Gelfand JM, Margolis DJ, et al. Patient burden and quality of life in atopic dermatitis in US adults: a population-based cross-sectional study. *Ann Allergy Asthma Immunol*. 2018;121(3):340-347. https://doi.org/10.1016/j.anai.2018.07.006
18. Silverberg JI, Gelfand JM, Margolis DJ, et al. Severity strata for POEM, PO-SCORAD, and DLQI in US adults with atopic dermatitis. *Ann Allergy Asthma Immunol*. 2018;121(4):464-468.e3. https://doi.org/10.1016/j.anai.2018.07.004

JAAD Game changer

JAAD Game Changers: Characteristics and outcomes of nonmelanoma skin cancer (NMSC) in children and young adults

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How did this article change the practice of dermatology?

- Twenty-eight patients with a total of 182 cases of nonmelanoma skin cancer (NMSC) over a 21-year period were identified in patients <22 years of age, suggesting that NMSC is a rare yet recurrent problem in affected children and young adults. Seventy-eight percent of the patients with squamous cell carcinoma developed additional squamous cell carcinomas, keratoacanthomas, actinic keratoses, or a combination of these while 43% of the patients with basal cell carcinoma had additional basal cell carcinomas.
- Forty-six percent of the children and young adults in this study with NMSC had iatrogenic risk factors, including prolonged immunosuppression, chemotherapy, voriconazole use, radiation exposure, or a combination of these. Of these patients, 62% had subsequent cancerous or precancerous lesions.
- Children and young adults with these exposures, including hematopoietic stem-cell transplantation, as well as liver, lung, or kidney transplantation, are at increased risk of NMSC.

Conflicts of interest: None disclosed.

Note: A Game Changer is a short narrative stating how an article that originally appeared in *JAAD* changed the game of dermatology. The Game Changer author is not the author of the original article.

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