

DIGITAL SCREEN-INDUCED PHOTODERMATITIS OF THE PERIORBITAL REGION:A CROSS-SECTIONAL STUDY OF HIGH-EXPOSURE OFFICE WORKERS

Nazarova Shabnam Otajon kizi

Asia International University

<https://doi.org/10.5281/zenodo.20109718>

ABSTRACT: Background: Prolonged exposure to high-energy visible (HEV) light emitted by digital screens has been increasingly implicated in various photodermatological conditions, yet its specific role in periorbital dermatitis among office workers remains poorly characterised.

Objectives: To determine the prevalence of screen-induced periorbital photodermatitis and identify modifiable risk factors in a cohort of full-time IT professionals.

Methods: A cross-sectional study was conducted among 312 IT professionals (mean age 31.4 ± 6.8 years; 57.1% male) at six technology companies in Tashkent, Uzbekistan (2023–2024). Dermatological examination, UV/HEV photometry, a validated Screen Exposure Index (SEI) questionnaire and serum 25-hydroxyvitamin D were assessed.

Results: Periorbital photodermatitis was diagnosed in 87 participants (27.9%). Logistic regression identified daily screen time ≥ 8 hours (OR 3.41, 95% CI 1.89–6.15, $p < 0.001$), Fitzpatrick phototype II–III (OR 2.17, 95% CI 1.12–4.20, $p = 0.022$), and absence of blue-light filtering (OR 2.58, 95% CI 1.44–4.63, $p = 0.001$) as independent risk factors. Vitamin D deficiency was significantly more prevalent among affected individuals ($p = 0.004$).

Conclusion: Digital screen exposure is an independent risk factor for periorbital photodermatitis in office workers. Implementation of workplace blue-light filtering standards and regular dermatological screening are recommended.

Keywords: photodermatitis, high-energy visible light, blue light, periorbital dermatitis, occupational dermatology, digital screen exposure, screen time.

1. INTRODUCTION

The rapid proliferation of digital technology has resulted in unprecedented levels of screen exposure among the working population. In 2023, the global average daily screen time reached 6.8 hours, with information technology (IT) professionals frequently surpassing 10 hours per working day [1]. While the ocular consequences of prolonged screen use have been extensively studied, the dermatological implications—particularly photodermatological effects on periorbital skin—remain comparatively unexplored.

Digital screens, including LED and OLED displays, emit substantial quantities of high-energy visible (HEV) light in the 400–500 nm wavelength range. Unlike ultraviolet (UV) radiation, HEV light penetrates deeply into the dermis and has been associated with reactive oxygen species (ROS) generation, collagen degradation, and melanogenesis [2]. The periorbital

region, due to its anatomical proximity to screens, thin epidermal layer, and reduced melanocytic density, may be particularly susceptible to cumulative HEV-induced photodamage [3].

Despite isolated clinical reports of periorbital hyperpigmentation and inflammatory dermatitis associated with screen use, no systematic epidemiological study has quantified screen-induced periorbital photodermatitis (SIPD) as a distinct occupational dermatosis. The condition poses a diagnostic challenge because its clinical presentation overlaps with allergic contact dermatitis, atopic dermatitis, and conventional photodermatitis from UV exposure. Furthermore, modifiable risk factors—such as blue-light filter usage, screen brightness settings, and occupational ergonomics—have not been systematically evaluated in a dermatological context.

The primary objectives of this study were: (1) to determine the prevalence of SIPD among full-time IT professionals; (2) to characterise its clinical and dermoscopic features; and (3) to identify independent modifiable and non-modifiable risk factors using multivariate logistic regression.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

A cross-sectional observational study was conducted between March 2023 and February 2024 at six technology companies in Tashkent, Uzbekistan. The study was approved by the Institutional Ethics Committee of Tashkent State Medical University (Protocol No. 14/2022, 18 November 2022) and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

2.2 Participants

Eligible participants were full-time employees aged 18–55 years with a minimum of one year of continuous occupational screen exposure of ≥ 6 hours per day. Exclusion criteria included: pre-existing chronic photodermatoses (e.g., lupus erythematosus, polymorphous light eruption), topical or systemic retinoid use within six months prior to enrolment, any concurrent facial dermatitis of known aetiology, pregnancy, and immunosuppressive therapy. Of 374 individuals invited, 312 met inclusion criteria and constituted the final sample.

2.3 Data Collection

A structured self-administered questionnaire captured demographic data, screen usage habits, protective measures (blue-light filter use, screen brightness adjustment, anti-glare coatings), skincare routines, and relevant medical and family history. Daily screen time was expressed as the Screen Exposure Index (SEI), calculated as the product of mean daily hours and screen luminance (cd/m^2), validated against objective photometric workstation measurements.

2.4 Clinical and Dermoscopic Examination

All dermatological examinations were performed by two board-certified dermatologists blinded to questionnaire data. SIPD was defined as periorbital erythema, hyperpigmentation, fine scaling, or pruritus localised to the upper and/or lower eyelid region, without identifiable

contact allergen or UV-specific distribution, in direct temporal relationship with screen use, reproducible upon re-exposure, and alleviated by screen abstinence. Dermoscopy (DermLite DL4; $\times 10$ polarised mode) was employed to characterise vascular patterns and pigmentation. Interobserver agreement was assessed using Cohen's kappa ($\kappa = 0.83$).

2.5 Biochemical Analyses

Venous blood was drawn for serum 25-hydroxyvitamin D [25(OH)D] quantification by electrochemiluminescence immunoassay (Roche Cobas e601). Vitamin D deficiency was defined as 25(OH)D < 20 ng/mL per Endocrine Society guidelines [4]. Serum IgE and a standard patch-test panel (European Baseline Series, TRUE TEST®) were conducted to exclude allergic aetiologies.

2.6 Statistical Analysis

Data were analysed using SPSS v.27.0 (IBM Corp., USA). Continuous variables are presented as mean \pm standard deviation (SD) or median with interquartile range (IQR). Categorical variables are presented as frequencies and percentages. Group comparisons were performed using the independent-samples t-test or Mann–Whitney U test for continuous data and the chi-squared (χ^2) or Fisher exact test for categorical data. Variables with $p < 0.10$ in univariate analysis were entered into a binary logistic regression model. Statistical significance was set at $p < 0.05$.

3. RESULTS

3.1 Participant Characteristics

The study cohort comprised 312 participants (178 male [57.1%], 134 female [42.9%]; mean age 31.4 ± 6.8 years). The majority held software development (42.3%) or systems administration (28.5%) roles. Mean daily screen time was 9.2 ± 1.8 hours and mean occupational tenure was 5.7 ± 3.2 years. Fitzpatrick skin phototype distribution was: type II (18.9%), type III (47.1%), type IV (29.5%), and type V (4.5%).

3.2 Prevalence and Clinical Features

SIPD was diagnosed in 87 of 312 participants (27.9%; 95% CI 22.9–33.3%). Affected individuals demonstrated significantly higher SEI scores (median 83.4 vs. 61.2 arbitrary units [AU]; $p < 0.001$) and longer occupational tenure (6.9 ± 3.4 vs. 5.2 ± 3.0 years; $p = 0.001$) compared with unaffected participants. On dermoscopy, the most consistent findings were diffuse homogeneous brownish pigmentation (74.7%), dotted/glomerular vessels (58.6%), and fine desquamation (41.4%). Table 1 summarises participant characteristics stratified by diagnosis.

Table 1. Demographic and clinical characteristics of study participants.

Variable	All (n=312)	SIPD+ (n=87)	SIPD- (n=225)
Age, years (mean \pm SD)	31.4 ± 6.8	32.1 ± 7.0	31.1 ± 6.7

Male sex, n (%)	178 (57.1)	44 (50.6)	134 (59.6)
Daily screen time ≥ 8 h, n (%)	196 (62.8)	71 (81.6)*	125 (55.6)
SEI score (median, IQR)	68.3 (54.1–79.6)	83.4 (71.2–94.8)*	61.2 (51.0–74.3)
Phototype II–III, n (%)	205 (65.7)	66 (75.9)*	139 (61.8)
Blue-light filter used, n (%)	138 (44.2)	24 (27.6)*	114 (50.7)
Vitamin D deficient, n (%)	149 (47.8)	53 (60.9)*	96 (42.7)
Positive patch test, n (%)	18 (5.8)	5 (5.7)	13 (5.8)

* $p < 0.05$ vs. SIPD–. SEI: Screen Exposure Index; IQR: interquartile range; SD: standard deviation.

3.3 Risk Factor Analysis

Multivariate logistic regression identified three independent risk factors for SIPD (Table 2). Daily screen time ≥ 8 hours conferred the highest risk (OR 3.41, 95% CI 1.89–6.15; $p < 0.001$), followed by absence of blue-light filtering (OR 2.58, 95% CI 1.44–4.63; $p = 0.001$) and Fitzpatrick phototype II–III (OR 2.17, 95% CI 1.12–4.20; $p = 0.022$). Vitamin D deficiency reached borderline significance (OR 1.72, 95% CI 0.98–3.02; $p = 0.059$). Female sex, age, and screen brightness were not independently significant.

Table 2. Multivariate logistic regression: independent predictors of SIPD.

Variable	OR	95% CI	p-value
Screen time ≥ 8 h/day	3.41	1.89–6.15	< 0.001
No blue-light filter	2.58	1.44–4.63	0.001
Fitzpatrick phototype II–III	2.17	1.12–4.20	0.022
Vitamin D deficiency	1.72	0.98–3.02	0.059
Female sex	1.38	0.78–2.44	0.271

Occupational tenure (per year)	1.09	0.99–1.20	0.083
--------------------------------	------	-----------	-------

OR: odds ratio; CI: confidence interval. Model Nagelkerke $R^2 = 0.31$; Hosmer–Lemeshow goodness-of-fit $p = 0.62$.

4. DISCUSSION

This study, to our knowledge, is the first to describe SIPD as a discrete occupational dermatosis among IT professionals and to quantify its independent risk factors in a Central Asian population. The prevalence of 27.9% is notably higher than the 8–12% reported for occupational photodermatoses attributed to UV light in equivalent worker cohorts [5], suggesting that HEV light may constitute a clinically underappreciated photodermatological hazard.

The strong association with daily screen time ≥ 8 hours (OR 3.41) is consistent with dose-response data from in vitro keratinocyte models, in which HEV irradiance above 25 J/cm² produced significant oxidative DNA damage and pro-inflammatory cytokine upregulation [6]. Our median SEI among affected individuals (83.4 AU) corresponded to an estimated HEV irradiance of approximately 22–28 J/cm² per working day under standard office luminance conditions, corroborating the biologically plausible threshold.

The protective effect of blue-light filtering (OR 2.58 for absence of filter) has important clinical and public-health implications. Commercial screen filters and coating lenses attenuating 380–450 nm HEV light by 20–50% are now widely available [7]. Our findings provide the first epidemiological evidence that their absence significantly increases dermatological risk at the occupational level, lending quantitative support to their inclusion in occupational health guidelines.

The association of Fitzpatrick phototype II–III with SIPD—rather than darker phototypes—is initially counter-intuitive, as increased constitutive melanin confers photoprotection. However, periorbital melanocytic density is relatively low across all phototypes, and ROS-mediated photodamage may disproportionately affect individuals with moderate melanin whose melanosomes provide insufficient quenching of HEV-generated singlet oxygen species [2]. Notably, phototype V individuals (4.5% of our cohort) showed no SIPD cases, suggesting that a threshold of melanin-based protection may operate at higher phototype levels.

Vitamin D deficiency was significantly more prevalent in the SIPD group ($p = 0.004$) and approached independent significance in the multivariate model (OR 1.72; $p = 0.059$). This relationship may reflect bidirectional pathophysiology: prolonged indoor screen work limits solar UV-B exposure necessary for cutaneous vitamin D synthesis, while vitamin D insufficiency per se may impair keratinocyte DNA repair capacity and reduce antioxidant defences [4]. Prospective intervention studies evaluating whether vitamin D supplementation modulates SIPD severity are warranted.

This study has several limitations. First, the cross-sectional design precludes causal inference. Second, recruitment was limited to six Tashkent companies, which may limit generalisability to populations with different ambient UV environments or screen usage patterns. Third, objective measurement of HEV irradiance at participants' workstations was available for only a random subsample ($n = 89$), and SEI was used as a surrogate metric for the remainder.

Fourth, the role of other potential contributors—such as electromagnetic field exposure, occupational stress-related skin barrier dysfunction, or dietary factors—was not assessed. Finally, histological confirmation of SIPD was not performed, as it was deemed disproportionate for a cross-sectional epidemiological study.

5. CONCLUSION

SIPD is a prevalent and hitherto under-recognised occupational dermatosis affecting approximately one in four full-time IT professionals in this cohort. Prolonged screen time, absence of blue-light filtering, and lighter Fitzpatrick phototypes constitute independent modifiable and non-modifiable risk factors. These findings support the introduction of regulatory blue-light exposure limits in workplace standards, routine dermatological screening in high-risk occupations, and patient education regarding available photoprotective technologies. Longitudinal studies and randomised trials of blue-light filtration interventions are now required to establish causality and guide evidence-based prevention.

REFERENCES

1. DataReportal. Digital 2024: Global Overview Report. DataReportal – Global Digital Insights. 2024. Available at: <https://datareportal.com/reports/digital-2024-global-overview-report>.
2. Nakashima Y, Ohta S, Wolf AM. Blue light-induced oxidative stress in live skin. *Free Radic Biol Med*. 2017;108:300–310. doi:10.1016/j.freeradbiomed.2017.03.010.
3. Duteil L, Cardot-Leccia N, Queille-Roussel C, et al. Differences in visible light-induced pigmentation according to wavelengths: a clinical, histological and in vitro study in comparison with UVB exposure. *Pigment Cell Melanoma Res*. 2014;27(5):822–826. doi:10.1111/pcmr.12273.
4. Holick MF, Binkley NC, Bischoff-Ferrari HA, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2011;96(7):1911–1930. doi:10.1210/jc.2011-0385.
5. Meding B, Wrangsjö K, Järholm B. Fifteen-year follow-up of skin symptoms among workers in Swedish construction industry. *Contact Dermatitis*. 2005;53(5):271–277. doi:10.1111/j.0105-1873.2005.00692.x.
6. Regazzetti C, Sormani L, Debayle D, et al. Melanocytes sense blue light and regulate pigmentation through opsin-3. *J Invest Dermatol*. 2018;138(1):171–178. doi:10.1016/j.jid.2017.07.833.
7. Leung TW, Li RW, Kee CS. Blue-light filtering spectacle lenses: optical and clinical performances. *PLoS One*. 2017;12(1):e0169114. doi:10.1371/journal.pone.0169114.
8. Schade N, Esser C, Krutmann J. Ultraviolet B radiation-induced immunosuppression: molecular mechanisms and cellular alterations. *Photochem Photobiol Sci*. 2005;4(9):699–708. doi:10.1039/b418865f.