

Induction of tolerance in solar urticaria by ultraviolet A 'rush hardening': is this true desensitization?

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Idiopathic solar urticaria (ISU) is uncommon, being estimated to affect 3.9 per 100 000 population in Scotland.¹ Spontaneous resolution may occur,² but it typically persists for years.³ It can severely impair quality of life.⁴

Various phototherapies may be useful. ISU is diverse, with provoking wavelengths ranging from ultraviolet B (UVB) to ultraviolet A (UVA) to visible, and, even infrared rays. The wavebands used for phototherapy need not match the provoking wavebands. Narrowband (NB)-UVB is sometimes selected to treat ISU in patients whose urticaria is triggered by longer UVA and visible wavelengths but not by UVB.^{5,6} Standard NB-UVB treatment for ISU probably works through a variety of mechanisms inducing photoprotection (hardening), as well as in other ways also relevant in chronic spontaneous urticaria.^{7,8} The term 'desensitization', often used to describe prophylactic phototherapy for ISU, is not strictly accurate when describing standard NB-UVB or broadband UVA monotherapy with either three-times weekly or twice-daily treatments.^{9,10} However, using UVA in the way first described by Beissert *et al.*¹¹ as 'rush hardening' is more akin to true 'desensitization' as in Hymenoptera venom immunotherapy.

In this issue, Masuoka *et al.*¹² report two people with UVA and visible waveband ISU treated with frequently repeated exposures, initially to small body areas, to a broadband UVA source until urticarial responses no longer occurred, followed by UVA exposure every 1–2 weeks for 4–6 months. Before treatment both patients had urticaria induced by injecting autologous serum irradiated *in vitro* with visible light; afterwards, irradiated autologous serum tests (using both serum stored from before treatment and from after treatment) were negative. Wealing on histamine prick tests and intradermal injection of compound 48/80 (a mast-cell degranulating agent) persisted after treatment.

Could a factor in serum that induced urticaria after irradiation have lost its effectiveness in inducing urticaria after storage? Possibly, but negative tests with serum collected after UVA treatment make this explanation seem unlikely. How can we explain that after UVA rush hardening irradiated autologous serum no longer produced urticaria, yet a direct chemical effect mast-cell degranulating agent and histamine still produced urticaria? In the past ISU has been passively transferred, with serum from an affected patient capable of producing localized solar urticaria when injected into an unaffected volunteer. That this serum factor was heat labile¹³ supported the hypothesis that it could be IgE. Masuoka *et al.*¹² note in

their introduction that an anti-IgE monoclonal antibody can be effective in ISU, which provides some support for this idea,¹⁴ although omalizumab may reduce mast-cell degranulation through mechanisms not involving specific IgE.¹⁵ The authors suggest that UVA 'rush therapy' led to continued binding of IgE receptors on mast cells or that it inhibited internalization of photoantigen (chemically altered after UV or visible light absorption)/IgE/mast-cell IgE receptor complexes. This is possible. Their findings suggest that this 'rush hardening' approach does not work through the nonspecific immunological and photoprotective effects considered most important in standard ultraviolet 'hardening' for the photodermatoses, but that it more specifically blocks a serum factor.

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Conflicts of interest

None declared.

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