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Ultraviolet A1 phototherapy: a British Photodermatology Group workshop report

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Summary

Whole-body ultraviolet (UV)A1 (340–400 nm) phototherapy was first introduced 30 years ago, but is currently available in the UK in only three dermatology departments. A workshop to discuss UVA1 was held by the British Photodermatology Group in May 2009, the aim of which was to provide an overview of UVA1 phototherapy and its role in practice, and to identify areas in which further studies are required. The conclusions were that UVA1 phototherapy is an effective treatment in several inflammatory skin diseases, including localized scleroderma and atopic eczema (AE); however, deficiencies and limitations exist in the published evidence base. For most diseases, such as AE, other treatments also exist, which are generally more effective than UVA1. However, for some diseases, particularly morphoea, the evidence of efficacy is stronger for UVA1 than for other treatments. Acute adverse effects of UVA1 are minimal. The risk of long-term adverse effects, particularly skin cancer, is unknown. Medium to high doses of UVA1 are needed for efficacy in most situations, but the equipment to deliver such doses is large, expensive and difficult to install. UVA1 is currently underprovided, and the recommendation of the workshop is that more tertiary centres should have access to UVA1 phototherapy in the UK.

Introduction

Long-wavelength ultraviolet A (UVA1) (Fig. 1) was first reported as a skin disease treatment in 1981.[1] Thirty years later, the published evidence on how best to use it remains limited and of variable quality.

A workshop to discuss UVA1 was held by the British Photodermatology Group in May 2009, which aimed to answer several questions asked by clinicians: 'Should I be using UVA1 phototherapy?'; 'Which patients benefit from UVA1 phototherapy?; and 'Is UVA1 phototherapy safe, and is it practical?'.

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Workshop methods

Dermatologists, physicists and scientists with experience of or an interest in UVA1 were invited. All but one (BE, from Munich) were based in the three UVA1 centres in the UK. Each participant at the workshop reviewed an allocated area of therapy, and presented this to the group. The literature review methods were not standardized, but levels of evidence were based on the Scottish Intercollegiate Guidelines (SIGN) system (see Appendix 1).

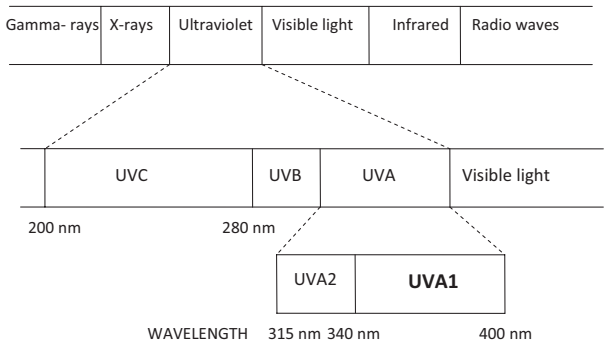

Figure 1 The electromagnetic spectrum, illustrating the relative position of UVA1 (340–400 nm) radiation.

Discussion at the workshop highlighted relevant publications. Because the published studies on UVA1 remain limited in number, much of the workshop outcome was based on consensus expert opinion.

Possible mechanisms of action of UVA1

UVA1 is different from other UV phototherapies because it penetrates deeper and has some different biological effects. The cellular effects of UVA1 have more in common with visible light than with UVA2 (320–340 nm) which behaves more like UVB.[2]

The key molecules that absorb UVA1 radiation, leading to the biological effects, have not been conclusively identified. UVA1 mainly exerts its effects via oxygen-dependent indirect mechanisms, through absorption by endogenous photodynamic photosensitizers including lipids and proteins.[3,4] There is also recent evidence that DNA can directly absorb UVA1 to form cyclobutane pyrimidine dimers.[5] The effects of UVA1-generated singlet oxygen ($^1O_2$) on T lymphocytes are likely to contribute to its efficacy in inflammatory disease. The greater susceptibility of malignant T cells to free-radical damage may explain the efficacy of UVA1 in mycosis fungoides.[6] Other immune cells such as Langerhans cells and mast cells may also be affected by UVA1, with effects distinct from those induced by UVA2 or UVB. UVA1-induced $^1O_2$ and hydrogen peroxide modulate the activity of matrix metalloproteinases produced by fibroblasts. Collagenase mRNA is up-regulated in morphoea fibroblasts after UVA irradiation,[7,8] a mechanism that is thought to underlie the efficacy of UVA1 in sclerotic and fibrotic dermatoses.[9]

What is known about the efficacy of UVA1 in dermatological disease?

The points taken into consideration in the workshop regarding the efficacy of UVA1 for treating various skin diseases are summarized in Table 1. There have been reviews of UVA1 disease indications,[10] and specifically for fibrosing diseases such as morphoea.[9,11] Most publications describe UVA1 in terms of low, medium or high dose; these doses describe individual treatments rather than cumulative dose over a course. Low doses can be delivered using low-output, fluorescent lamp equipment, whereas medium and high doses require larger and more expensive metal-halide lamp equipment. There are no internationally agreed definitions of different treatment doses but < 10 J/cm^2 is generally considered 'very low dose', 10–29 J/cm^2 'low dose', 30–59 J/cm^2 'medium dose' and > 60 J/cm^2 'high dose'.

Atopic eczema

UVA1, at least at medium to high doses, is effective, but has not been shown to be more effective than the standard phototherapies (narrowband UVB[12,13] and psoralen UVA[14]) for atopic eczema (AE). In many of the studies, UVA1 was used as monotherapy, rather than an adjunct to other standard therapies, which may limit extrapolation to clinical practice. UVA1 has been found to be more effective than comparators in 'acute eczema'[15–20] and not more effective than comparators in 'chronic eczema' (Table 1).[12,13,21] However, there has been no direct comparison of efficacy of UVA1 in 'acute' vs. 'chronic' eczema, so it is unclear whether UVA1 has a specific role in the treatment of acute eczema. The workshop view was that UVA1 is valuable for some patients with AE unresponsive to other phototherapies; however, these are few, and for most centres, the expense of high-output UVA1 apparatus solely for this indication would be difficult to justify.

Fibrosing skin disease

UVA1 is definitely effective and valuable in the treatment of fibrotic skin conditions, for which it can induce lengthy periods of remission, and for which other therapeutic options are limited (Table 1).[10,11,22] For localized morphoea, UVA1 is the only treatment with published controlled-study evidence of efficacy.[23] The workshop concluded that it was clear that UVA1 in fibrosing conditions can be beneficial for patients if disease restricts joint or chest movement, and also in stopping or slowing widespread disease. A common side-effect is local skin darkening, which may limit its use, especially where the primary problem is cosmetic.

Table 1 Diseases that may respond to UVA1 phototherapy.

Disease	Does UVA1 work?	Level of evidence	Suggested line of therapy if UVA1 available (workshop opinion)	Comment
Localised scleroderma (morphoea)	Yes	1–	Second (after topical steroids)	Medium and high doses seem more effective than low doses. Can worsen appearance but improve function, e.g. joint movement. PUVA is considered a standard therapy and has not yet been directly compared with UVA1
Sclerodermic GVHD	Possibly	3	First	Case series and case reports
Nephrogenic systemic fibrosis	Possibly	3	Second (after renal transplantation if this is appropriate)	Case reports only
HS	Possibly	3	Position not clear	Case series
Lichen sclerosus	Possibly	3	Second (after topical steroids for extragenital disease)	Open, uncontrolled evidence and case reviews only
Systemic sclerosis	Possibly	3	Position not clear	May be useful for sclerotic skin changes of the hands
Scleroedema of Bushke	Possibly	3	Second (after improvement of diabetic control if relevant)	Case series and case reports
SLE	Probably	1+	Position not clear	Appears to have beneficial effect on systemic and possibly also dermatological features of disease
CTCL	Possibly	3	Third (In disease beyond patch stage after topical steroids and PUVA)	Possibly as effective as PUVA. Effect may be due to local and not systemic effect on lymphocytes
Urticaria pigmentosa	Possibly	−1	Position not clear	Mast-cell numbers in histology specimens seem to decrease. Not all reports have shown benefit. May be more useful for headaches and bone pain than for appearance of rash
Chronic urticaria	Possibly	3	Position not clear	Case reviews. Often other treatment more appropriate than UVA1 (e.g. H_1-and H_2 antihistamines, leucotriene receptor antagonists, UVB and PUVA)
Granuloma annulare	Possibly	3	Third (after topical steroids and PUVA)	May be beneficial in patients who have not responded to PUVA, but effect may be temporary
Cutaneous sarcoidosis	Possibly	3	Position not clear	Case report
Atopic eczema	Yes	1+	Third (after topical steroids and UVB or PUVA)	Studies have involved UVA1 as monotherapy. May be inferior to PUVA
Dishydrotic hand eczema	Possibly	1–	Third (after topical steroids and PUVA)	May be as effective as PUVA
Subacute prurigo	Probably	1–	Third (after topical steroids and UVB or PUVA)	Possibly more effective than narrow-band UVB
Psoriasis	Possibly	3	Fourth (after topical therapy, UVB or PUVA and systemic agents)	Probably inferior to broadband UVB
PRP	Possibly	3	Position not clear	Case report in combination with acitretin
Pityriasis lichenoides (PLEVA and PLC)	Possibly	3	Third (after topical steroids and UVB for PLC). Position not clear for PLEVA	Open study and case review

CTCL, cutaneous T-cell lymphoma; GVHD, graft-versus-host disease; HS, hypereosinophillic syndrome; PLC, pityriasis lichenoides chronica; PLEVA, pityriasis lichenoides et varioliformis acuta; PRP, pityriasis rubra pilaris; PUVA, psoralen ultraviolet A; SLE, systemic lupus erythematosus; UV, ultraviolet.

Acute adverse effects of UVA1

Skin pigmentation (tanning), which can persist for months, is the commonest problem. Patients should be warned that in morphoea, the lesions may be more noticeable after treatment, because of hyperpigmentation. Erythema is rarely a problem, particularly if prior testing to ascertain the minimal erythema dose (MED) has been performed. With high-output therapy, an immediate asymptomatic erythema generally occurs, lasting for about 2 h after treatment. As UVA1 can provoke polymorphic light eruption, initial exposure of a small test area can indicate whether whole-body exposure is likely to induce an eruption. Uncommon acute adverse effects include recrudescence of herpes simplex, cholinergic urticaria, and transient and reversible changes in the appearance of moles.

The workshop recommended the need for caution with UVA1 and concomitant photoactive medications, given that most drug-induced photosensitivity is UVA-dependent. Potential photosensitizers include fluoroquinolone antibiotics and St John's wort (hypericin).[24] Dietary psoralens have no significant effect.[25] If a patient starts a photoactive drug during UVA1 treatment, the UVA1 MED should be rechecked. The suggested MED assessment dose ranges are 7–56 J/cm^2 for skin phototype I, 7–80 J/cm^2 for phototype II, and 10–112 J/cm^2 for phototypes III and IV.

Chronic adverse effects of UVA1

Studies of chronic UVA1 effects in humans are limited. Three retrospective studies involving 423 patients, who received between 4 and 116 treatments in total,[26–28] reported no chronic effects. A case report of melanoma after UVA1 and PUVA treatment has been published.[29] There is some limited information on UVA1 from animal models, showing that it can induce squamous cell carcinomas and melanomas.

Managing potential long-term risks

As with other phototherapies, there is no clearcut evidence on which to base a recommendation for a maximum lifetime number of treatments.[30] Erring on the side of caution, the workshop's opinion was that patients receiving over 200 UVA1 whole body treatments should be considered at possibly being at increased risk of skin cancer and managed accordingly. As with other phototherapies, UVA1 should be provided in the context of a clinical governance system, to ensure appropriate and safe use and follow-up.

UVA1 equipment

Available sources

UVA1 can be generated using fluorescent tubes or filtered metal halide lamps (for a list of suppliers, see Appendix 2). The Waldmann UV 7001K® (Herbert Waldmann GmbH & Co., Vilingen-Schwenningen, Germany) whole body treatment unit equipped with 40 Philips® TL 100 W/10R UVA1 fluorescent lamps (Philips Electronics UK Ltd, Guilford, U.K.) delivers a mean patient irradiance of 20 mW/cm^2, and is suitable for low-dose UVA1 therapy. Practical delivery of medium and high doses requires the higher (approximately 60 mW/cm^2) irradiance of metal-halide units such as the Sellamed 3000® unit for localized exposures and the Sellamed 24000® bed (Sellas Medical Devices GmbH, Gevelsburg, Germany) (Fig. 2) for whole-body

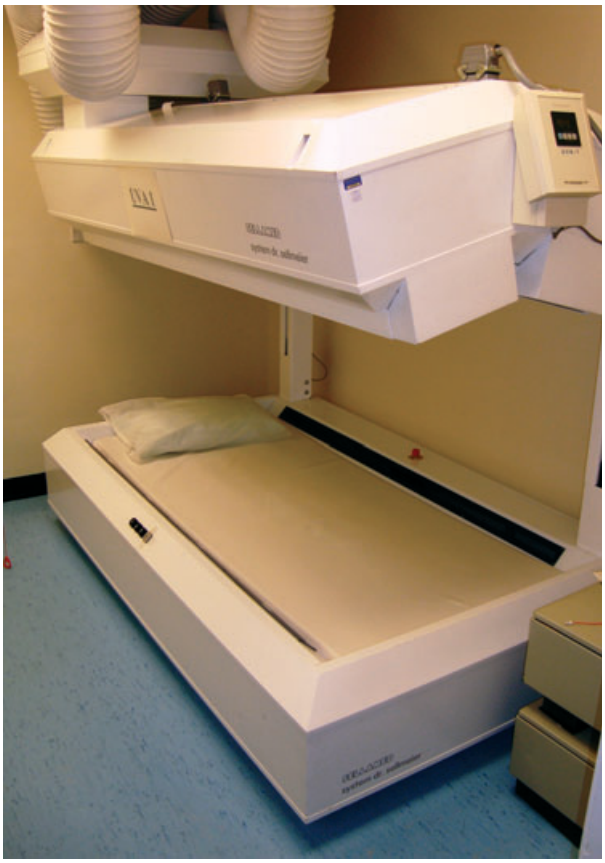

Figure 2 The Sellamed 24000® bed for delivering ultraviolet A1 phototherapy.

treatment. Because of the heat-removal systems and electrical cabling required, these high-dose sources are expensive to install.

Calibration/dosimetry/maintenance

The workshop advised performing UVA1 dosimetry regularly, and one method was outlined. Firstly, the sensitivity factor of the meter should be established, and the spectrum lamp measured using a calibrated spectroradiometer. The integrated irradiance within the UVA1 range is then determined by spectroradiometry, and a correction factor calculated. Secondly, the irradiance is measured at different locations within the treatment area; the average value over these locations then defines the treatment irradiance. For a small source such as a 300 × 300 mm field (e.g. Sellamed 3000® unit), 10 locations may suffice, but a high-output Sellamed 24000® unit may need 30–40 locations measured. A representative spectrum produced from the Sellamed 24000® unit is shown in Fig. 3.

Practical and financial issues around the use of ultraviolet A1 equipment in dermatology units

The main limitation on the availability of UVA1 in the UK and elsewhere is the cost and practical issues associated with the machines. A high-output UVA1 device currently costs around £30 000, plus around £10 000 for installation. Following installation, high temperatures in the treatment room (requiring heat-removal equipment) and long treatment times (limiting the numbers of patients that can be treated) are issues that need consideration. Low-dose UVA1 using fluorescent sources is simpler to deliver and is cheaper to install, but more studies are required to discover which patients can be effectively treated with low-dose UVA1.

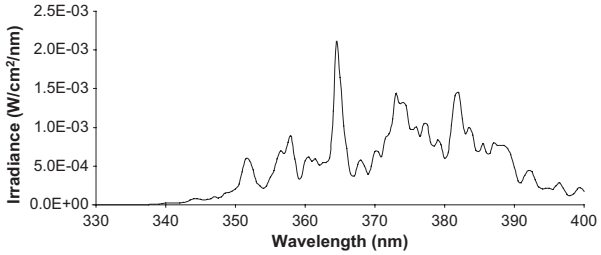

Figure 3 Example of output spectrum of Sellamed 24000® unit. Representative spectra at 650 mm from bottom of bed to top of canopy. Detector at 250 mm from bottom of bed.

Conclusions

Over the past 15 years, it has become clear that UVA1 phototherapy is a valuable treatment for some patients with some uncommon and treatment-resistant skin conditions, and for a few patients with common conditions inadequately responsive to 'standard' phototherapies. Although its mechanism of action is not yet fully understood, the formation of reactive oxygen species seems to be more important than the direct interaction of UV with DNA. UVA1 is the first-line treatment for progressive morphoea or morphoea that is restricting movement. Although effective in AE, UVA1 is best used for cases in which other phototherapies have been inadequate. UVA1 therapy is generally well tolerated with few acute adverse effects, but the lack of long-term studies mean caution should be exercised because of the possible increased risk of skin cancer and risk of photoageing. A number of different UVA1 devices are commercially available. As with other phototherapies, accurate maintenance and dosimetry is essential. Although some data on UVA1 treatment regimens have been published, the evidence for these is often lacking.

Crucial areas for future study are the establishment of the efficacy of UVA1 in different diseases, and identification of the minimum effective regimen for individual doses and treatment courses. If lower individual treatment doses, even with the requirement for longer treatment courses for a cumulative effect, are effective, this would make UVA1 a practical and affordable option for more dermatology units, as treatment with medium and high individual doses requires expensive and bulky equipment, thus restricting the availability of UVA1.

Key research questions for future study

- How does UVA1 phototherapy compare with more readily available phototherapies, particularly PUVA, in the treatment of widespread morphoea?
- Is narrowband UVB or UVA1 most effective as an adjunctive (to topical corticosteroid) therapy for severe AE?
- Is the combination of UVA1 plus narrowband UVB better for AE than either therapy alone?
- Is UVA1 appropriate as an adjunctive therapy for cutaneous lupus erythematosus, or should it be reserved for systemic lupus erythematosus (SLE)?
- What is the minimum therapeutically effective dose, and is irradiance (determining practicable individual treatment doses) important for each disease in which UVA1 is shown to be effective?

Learning points

- UVA1 is not a new treatment, but its adoption in phototherapy centres worldwide has been slow.
- UVA1 is effective for various diseases, and is the first-line phototherapy for some types of morphoea.
- Some individual patients with AE benefit from UVA1, but in practice, its role is as second- or third-line phototherapy in AE.
- UVA1 is effective at very low doses to treat SLE.
- More pragmatic studies are needed, using UVA1 as an adjunct to other standard treatments, but for most indications, medium to high treatment doses are more effective than low doses.
- Delivery of medium and high doses of UVA1 requires expensive, bulky, heat-producing, high-output equipment, which is likely to be the main reason for the slow adoption of UVA1.

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Appendix 1

The strength of published evidence was graded according to a modified version of the ranking system adopted by the Scottish Intercollegiate Guidelines Network (SIGN).

Level	Description
1++	High-quality meta-analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias
1+	Well-conducted meta-analyses, systematic reviews, or RCTs with a low risk of bias
1–	Meta-analyses, systematic reviews, or RCTs with a high risk of bias
2++	High-quality systematic reviews of case control or cohort or studies, high-quality case–control or cohort studies with a very low risk of confounding or bias, and a high probability that the relationship is causal
2+	Well-conducted case–control or cohort studies with a low risk of confounding or bias, and a moderate probability that the relationship is causal
2–	Case–control or cohort studies with a high risk of confounding or bias, and a significant risk that the relationship is not causal
3	Non-analytical studies, e.g. case reports, case series
4	Expert opinion

RCT, randomized controlled trial.

Appendix 2

UK-based suppliers of equipment that can be used to deliver ultraviolet A1 phototherapy.

UK distributor/ manufacturer	Manufacturers represented
Arthrodax Healthcare International Ltd (http://www.athrodax.co.uk)	Waldmann Medizintechnik GmbH (Germany) (http://www.waldmann.com); Sellas Medizinische Gerate GmbH (Germany) (http://www.sellas.de)
Hospital Lamp Supplies (http://www.hybec.com)	Division of Hybec Ltd
MGC Lamps Ltd (http://www.mgc-lamps.com)	

CPD questions

Learning objective

To show an understanding of UVA1 phototherapy, including mode of action, indications and adverse effects.

Question 1

A 30-year-old woman has been diagnosed with morphoea restricting movement at her shoulders and right elbow. She has heard of ultraviolet (UV)A1 phototherapy and wants to know more about how it might work in her condition. Which of the following is likely to be the most important mechanism of action of UVA1 in treating morphoea?
a) Apoptosis of malignant T lymphocytes
b) Effects on Langerhans cell function
c) Formation of thymine dimers
d) Increased fibroblast matrix metalloproteinase activity
e) UVA1-generated production of singlet oxygen

Question 2

A 24-year-old man of skin phototype III with a lifelong history of atopic eczema (AE) attends with repeated flares of his eczema. His eczema has been severe over the past 3 years, with poor control despite use of 200–300 g betamethasone dipropionate 0.05% ointment weekly, along with emollients. After 1 week of clobetasol propionate 0.05% ointment, his eczema has improved, but it continues to affect him severely. Which of the following treatments would be most appropriate?

a) Broadband UVA phototherapy
b) Broadband UVB phototherapy
c) High-dose UVA1 phototherapy
d) Narrowband UVB phototherapy
e) Psoralen ultraviolet A photochemotherapy (PUVA)

Question 3

A 36-year-old woman is very distressed by the progressive worsening, with new lesions developing, of a symmetrical eruption of pale brown patches predominantly over the trunk. Based on the clinical presentation and histopathological findings of a skin biopsy, her condition has been diagnosed as widespread morphoea. Which of the following treatments would be most appropriate?
a) Calcipotriol ointment
b) Narrowband UVB
c) Oil–water emollient cream
d) Tacrolimus ointment
e) UVA1 phototherapy

Question 4

A 43-year-old woman with extensive plaque psoriasis, who has failed to respond to a variety of topical treatments, attends the clinic seeking phototherapy. Which of the following phototherapies would be the treatment of first choice?
a) Bath PUVA
b) Broadband UVB
c) Narrowband UVB
d) Oral PUVA
e) UVA1

Question 5

A 35-year-old man with systemic lupus erythematosus has heard of UVA1 and seeks more information about it. Which of the following wavebands corresponds to UVA1 emission?

a) 280–320 nm
b) 311–312 nm
c) 320–400 nm
d) 340–400 nm
e) 360–430 nm

Instructions for answering questions

This learning activity is freely available online at https://www.wileyhealthlearning.com/ced.aspx.

Users are encouraged to
- Read the article in print or online, paying particular attention to the learning points and any author conflict of interest disclosures
- Reflect on the article
- Register or login online at www.wileyhealthlearning.com/ced.aspx and answer the CPD questions
- Complete the required evaluation component of the activity

Once the test is passed, you will receive a certificate and the learning activity can be added to your RCP CPD diary as a self-certified entry.