Psoralen photochemotherapy (PUVA) is a widely used and frequently convenient and effective method of dermatological therapy, using a combination of psoralen, ingested or topically applied, and cutaneous ultraviolet A irradiation. Both acute and chronic adverse reactions are not infrequent, and in order to minimize these, clear guidelines for PUVA operators are needed. However, apart from brief recommendations for psoriasis,1 such guidelines are lacking, and the British Photodermatology Group (BPG) has therefore recently produced consensus recommendations. Where possible, the guidelines have been formulated on the basis of appropriate controlled studies; where these are lacking, they represent instead a synthesis of the current practices of BPG members. It was agreed that, whenever possible, a senior dermatologist with relevant experience in a department should have overall responsibility for the PUVA service, including training of support clinical staff.

UVA dosimetry and calibration

The UVA exposure dose used in PUVA therapy is normally prescribed in radiometric units, namely Joules per square centimetre (J/cm²) of skin exposed. Constancy of lamp output should be checked weekly using an independent hand-held dose meter. Intercomparisons of dose meters used to measure UVA irradiance (in milliwatts per square centimetre, mW/cm²), carried out in the U.K.,2 Belgium,3 and France,4 have shown wide variations in accuracy, with up to threefold differences in sensitivity at the extreme ends of the range. Much of this variation can be attributed to different methods of calibration.

Most dermatology departments in the U.K. have PUVA units incorporating UVA fluorescent lamps,5 with remarkably similar spectral emissions (plots of radiation intensity at each wavelength) for all makes (Fig. 1). UVA sensors produce an electrical signal which is passed to the meter display, and this depends not only on UVA irradiance from the lamps, but also on their spectral content.6 UVA dose meters should thus be calibrated against UVA fluorescent lamps of the same spectral emission as those in PUVA units. The proper technique is to use a spectroradiometer—an expensive, complex device used mainly in national standards laboratories, and medical physics departments with a special interest in photobiology. This type of measurement, known as spectroradiometry, enables the radiation intensity to be recorded wavelength by wavelength over the whole lamp spectrum. By summing the spectral irradiance across the UVA waveband (315–400 nm) the absolute UVA irradiance can be derived. Calibration of a UVA dosemeter simply involves placing the sensor at the same point as the spectroradiometer, and adjusting the meter display to read the UVA irradiance determined spectroradiometrically.

The Group recommends that medical physics departments with the expertise, instrumentation and suitably calibrated lamps, according to national standards, be invited to provide a calibration service for UVA dose meters used in PUVA therapy, on an annual basis. A department with which one of the authors (B.L.D.) is associated currently provides such a service.

Pretreatment consultation and assessment

Suitability for PUVA

All patients should be assessed for their suitability for PUVA (Table 1) prior to commencement of treatment.

![Figure 1. The spectral emission of UVA fluorescent lamps used in PUVA irradiation equipment.](image-url)
Table 1. PUVA contraindications

**Absolute**
- Xeroderma pigmentosum
- Gorlin’s syndrome
- Hereditary dysplastic naevus syndrome
- Systemic lupus erythematosus
- Dermatomyositis
- Trichothiodystrophy
- Bloom’s syndrome
- Cockayne’s syndrome
- Previous malignant melanoma

**Relative**

**Major**
- Age less than 10 years
- Previous or current non-melanoma skin cancer
- Previous exposure to arsenic or ionizing radiation
- Current premalignant skin lesions
- Concomitant immunosuppressive therapy
- Pregnancy
- Porphyria

**Minor**
- Age less than 16 years
- Cataracts
- Bullous pemphigoid
- Pemphigus
- Previous or concomitant treatment with methotrexate
- Significant hepatic dysfunction
- Previous internal malignancy

*A lower age limit may be acceptable where topical psoralen is used. † Oral psoralen only.

The suggested lower age limits are only guidelines, but PUVA should be given only to children with severe dermatoses unresponsive to safer treatment, because of their presumed greater susceptibility to ocular and cutaneous damage. Previous non-melanoma skin cancer is not an absolute contraindication, especially in older patients, if other treatment options produce unacceptable side-effects, or fail; however, if previous skin cancer has occurred on currently uninvolved sites, these should be shielded during PUVA where practicable. Previous treatment with arsenic or ionizing radiation, but probably not methotrexate, increases the risk of development of squamous cell carcinoma with PUVA, and increased caution is therefore required in deciding whether to use PUVA in such patients. Although no studies have indicated an increased risk of malignant melanoma following PUVA, this possibility cannot be excluded, and patients with important risk factors for melanoma should be given alternative therapies if possible. Concomitant treatment with immunosuppressive agents should also be avoided where possible. On the basis of the limited data so far available, PUVA appears to be safe in HIV-antibody positive patients, at least if they are HIV-antigen p24 negative.

There is no evidence that PUVA is a significant teratogen, but female patients, and male patients’ partners should still preferably avoid conception during treatment. However, for patients in whom treatment cannot be stopped during pregnancy because of severe disease, PUVA may be considered the safest second-line therapeutic option if UVB is ineffective.

Adverse hepatic effects of PUVA are uncommon, except perhaps in conjunction with pre-existing liver dysfunction, and assessment of liver function before PUVA is therefore advised, particularly in patients with risk factors for hepatic disease. If baseline measurements are normal, routine monitoring of liver function during PUVA is not required.

### Risk assessment

**Skin cancer.** As part of the consideration of patients’ suitability for PUVA, an assessment of their skin cancer risk prior to treatment should be undertaken, including documentation of skin type, previous sun exposure, any severe sunburning episodes, sunbed usage, and previous treatment with UVB and PUVA. Patients should also be examined for large or clinically atypical naevi; these should then be excised or covered during irradiation.

**Photosensitivity.** Measurement of antinuclear factor titre before starting PUVA may avoid exacerbating clinically occult lupus erythematosus, although the risk of this is unknown. Anti-Ro (SSA) antibodies should also be measured before PUVA in patients with clinical photosensitivity. Current medication should also be recorded; if a patient is receiving a drug with known phototoxic potential, the cutaneous minimal phototoxic dose (MPD) should preferably be determined prior to PUVA, or a low UVA starting dose of 0.5 J/cm² used.

**Consent.** Although written consent does not waive legal responsibilities or replace careful explanation to the patient, it is recommended as a useful formal means of pointing out PUVA risks to patients. Copies of a consent form currently in use are available on request from P.G.N.

### PUVA for specific dermatoses

**Psoriasis**

Indications for PUVA treatment in chronic plaque psoriasis include (i) severe extensive psoriasis unresponsive to topical therapies, (ii) relapse within 3–6 months
of successful in-patient or day-centre topical treatment, and (iii) patient refusal of topical treatment if UVB phototherapy has failed. If prolonged remission (> 9–12 months or more) is not achieved by PUVA in the last mentioned group, adequate in-patient or day-centre topical treatment should be advised prior to further PUVA.

As PUVA erythema peaks at 48–72 h, twice weekly treatment frequently is recommended to permit greater UVA dosage increments, as well as efficient use of PUVA services. The initial UVA exposure dose should, where possible, be decided on the basis of prior measurement of the minimal phototoxic dose (MPD), rather than skin type (the latter correlates poorly with PUVA erythemal sensitivity), and subsequent increments calculated as a percentage of previous doses. A suggested regimen is shown in Appendix 1. MPD testing identifies unusually sensitive subjects and those who fail to respond completely, in which circumstance a higher psoralen dose may be required. It has been clearly demonstrated that treatment regimens based on MPD measurement achieve more rapid clearance of psoriasis, with lower cumulative UVA dosages, than fixed-dose regimens based on skin type. If MPD testing is impractical, an alternative, more conservative, less efficient regimen based on skin type (Appendix 2) may be used.

Maintenance PUVA, following routine clearance of psoriasis, should be avoided, in order to minimize cumulative UVA exposure; treatment should be discontinued as soon as clearance is achieved. Maintenance PUVA should only be considered if there is rapid relapse of psoriasis following clearance (where it is important to indicate the planned duration of maintenance treatment and the subsequent treatment plan) or as part of a rotational regimen of second-line treatments.

The combination of oral etretinate or acitretin with PUVA therapy has several potential advantages, including reduction in the number of PUVA sessions and/or cumulative UVA dosage, and clearance of psoriasis in patients unresponsive to PUVA alone. Thus, for patients who have reached 50 treatment sessions, or relapsed within 6 months of PUVA, such a combination should be considered, to take advantage of a possible reduced long-term skin cancer risk. An etretinate dosage of 0.5–1.0 mg/kg, or acitretin dosage of 0.3–0.7 mg/kg, is recommended. If the associated prolonged risk of teratogenicity is unacceptable, isotretinoin and PUVA may be combined instead. Commencement of retinoid therapy 7 or more days before PUVA has been suggested, but has not been clearly proved to be advantageous.

Other PUVA combination therapies for psoriasis are less useful. Pretreatment with UVA-transmitting emollients is not apparently helpful, and concomitant use of topical steroids, although producing more rapid clearance, is associated with earlier relapse. PUVA and methotrexate are effective for very severe psoriasis, but should be reserved for such cases, as an increased skin cancer risk, although not proven, is possible. PUVA and cyclosporin appear to confer little therapeutic advantage, and skin cancer may well be more likely.

Mycosis fungoides

PUVA is an effective symptomatic treatment for early mycosis fungoides, and prolonged disease-free intervals are possible, although the long-term course of the disease may be unaltered. PUVA is also a useful adjunctive therapy for late-stage disease.

Optimal PUVA regimens for mycosis fungoides have not been established. However, UVA dosages calculated as for psoriasis from MPD testing, or derived from a set regimen of 0.5 J/cm² initially, followed by twice weekly increments of 0.5–1.0 J/cm² have proved satisfactory. Additional local UVA irradiation of sites relatively shielded during whole body exposure is often useful. Patients may be treated more frequently, but there is no evidence to indicate that this is more effective. Some patients experience a flare of their disease early in treatment, sometimes with the appearance of presumed previously subclinical lesions; however, if the normal skin is not erythematosus, dosage increments should not be abandoned. After clearance, a short maintenance period may be used, but prolonged maintenance should be reserved for patients who repeatedly relapse rapidly when PUVA is stopped. Combined PUVA and retinoid therapy, which results in fewer treatment sessions and lower cumulative UVA doses, should also be considered in such patients, as well as in young patients, and in those with resistant disease.

Atopic dermatitis

PUVA is effective for atopic dermatitis of adults and children. However, clearance is less certain than for psoriasis, and may require about twice the number of treatments; in addition, relapse is more frequent. Thus, only patients with severe disease unresponsive to conventional therapy should be treated, particularly children with growth retardation, and those who require repeated hospital admission.

Optimal PUVA regimens for atopic dermatitis have not
been established. One current effective regimen is to use an initial UVA dose of 70% of the MPD, with 20% increments at each twice weekly treatment. If MPD testing is impossible, 1 J/cm² initially, followed by 0.5–2.0 J/cm² increments twice weekly is usually effective. Early disease exacerbation is common, and should be treated with potent topical or oral corticosteroids, with antibiotics when appropriate, as UVA dosages are increased. After clearance, tapering maintenance treatment is often used; a typical regimen is to continue the final UVA dosage once weekly for 4 weeks and then fortnightly for 8 weeks, although slower reductions have also been recommended. Intermittent or continuous oral acyclovir may be required for cutaneous herpes virus infection. High temperatures in the treatment cubicle may cause patients discomfort and exacerbate their dermatitis, and air conditioning or divided treatment sessions are desirable in such cases.

Vitiligo

Oral PUVA is often used in the treatment of vitiligo, particularly if this is of recent onset, limited extent, and central location. 36, 37 8-Methoxypsoralen (8-MOP) is generally used in the United Kingdom, although 5-MOP may be preferable, as burning is less likely. Recommended absolute contraindications are an age below 10 years and skin type I, and suggested relative contraindications are an age below 16 years and skin type II. It should also be remembered that acral sites rarely respond. A twice weekly regimen incorporating 0.6 mg/kg 8-MOP or 1.2 mg/kg 5-MOP and an initial UVA dose of 0.5 J/cm², with around 0.5 J/cm² increments at each visit, to achieve and maintain mild erythema in vitiliginous areas, has proved effective in many patients.

For patients with limited, large, well-demarcated vitiligo patches, topical PUVA may be preferable, in order to avoid systemic psoralen effects. A 0.15% 8-MOP emulsion, a starting dose of 0.25 J/cm², and 0.25–0.5 J/cm² increments have proved effective.

Progress should be monitored by clinical photographs at about 4-monthly intervals, and treatment should be discontinued once improvement has ceased. Overall treatment duration should be limited to 12–18 months, or very rarely 24 months, and should not necessarily be continuous.

Polymorphic light eruption

PUVA is effective prophylactic treatment for up to 90% of patients with polymorphic light eruption (PLE). 38–41 It is indicated for those who are frequently or severely affected despite regular use of high-protection, broad-spectrum sunscreens; treatment for patients who suffer only because of sunbathing is not recommended. Therapy is probably best commenced about a month before the patient first anticipates that the eruption will occur. Several arbitrary regimens are in use, and there is wide variation in UVA dosage and number of treatments (Appendix 3). Cautious regimens may be most appropriate in markedly photosensitive patients, and PUVA-induced exacerbations of PLE may require UVA dosage reduction, temporary cessation of therapy, or brief use of potent topical or oral steroids. Maintenance treatment is not given routinely, although some clinicians advise regular sensible sun exposure following courses of PUVA.

Other disorders

PUVA is also sometimes used for other dermatoses, with variable results. There are insufficient data to establish precise guidelines, but disorders which have been treated, and recommendations concerning the probable efficacy of treatment are indicated in Table 2. In most of the disorders relapse occurs in the absence of maintenance therapy, and PUVA should usually be tried only as a last resort.

Bath PUVA

Bath PUVA is an effective and increasingly popular alternative to oral PUVA. 42–45 8-MOP or trimethylpsoralen are used, and suggested regimens are shown in Appendix 4. Advantages include shorter irradiation times, and lack of gastrointestinal, hepatic or other systemic adverse effects. 46 Data concerning the ocular risks associated with bath PUVA are lacking, and some centres still recommend eye protection for at least 12 h following treatment. However, closer patient supervision is required due to greater photosensitization.

Hand and foot PUVA

Local PUVA using oral or topical psoralen delivery is a useful and effective second-line treatment for hand and foot psoriasis or dermatitis. Good disease control, and sometimes prolonged remission, can be obtained in 70–80% of patients. 37–39 Effective regimens are described in Appendix 5. Poor response occasionally results from inadequate systemic psoralen delivery, and topical treatment should then be tried. PUVA combined with etretinate may be more effective than PUVA alone for
Table 2. Dermatoses infrequently treated with PUVA

<table>
<thead>
<tr>
<th>Dermatosis</th>
<th>Comment</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urticaria pigmentosa</td>
<td>Helpful in symptomatic patients. Relapse likely without maintenance</td>
<td>69, 70</td>
</tr>
<tr>
<td>Graft-versus-host disease</td>
<td>Cautious trial worthwhile in extensive unresponsive lichenoid disease</td>
<td>71–73</td>
</tr>
<tr>
<td>Pityriasis lichenoides</td>
<td>Indicated in widespread disease if UVB fails</td>
<td>74</td>
</tr>
<tr>
<td>Lichen planus</td>
<td>Response variable; worthwhile in resistant cases</td>
<td>75–78</td>
</tr>
<tr>
<td>Granuloma annulare</td>
<td>Trial probably worthwhile in extensive disease</td>
<td>79, 80</td>
</tr>
<tr>
<td>Alopecia areata</td>
<td>Not recommended; possibly a single 3-month trial, but relapse likely</td>
<td>81–86</td>
</tr>
<tr>
<td>Symptomatic dermographism</td>
<td>Not recommended</td>
<td>87</td>
</tr>
<tr>
<td>Nodular prurigo</td>
<td>Trial worthwhile if unresponsive to conventional treatment</td>
<td>88</td>
</tr>
<tr>
<td>Aquagenic pruritus</td>
<td>Trial worthwhile if any partial response to conventional treatment</td>
<td>89</td>
</tr>
<tr>
<td>Chronic actinic dermatitis</td>
<td></td>
<td>90</td>
</tr>
<tr>
<td>Actinic prurigo</td>
<td>Sometimes effective but close supervision required. Consider referral to centre with special interest</td>
<td>91</td>
</tr>
<tr>
<td>Solar urticaria</td>
<td></td>
<td>92</td>
</tr>
</tbody>
</table>

palmoplantar pustulosis.\textsuperscript{51} Long-term adverse effects have not been noted, and may be less likely than with whole-body PUVA because of the thicker skin of the palms and soles.

**Reduction of adverse reactions to PUVA**

**Acute**

8-MOP is widely accepted as the standard orally administered psoralen for PUVA; 0.6 mg/kg body weight is given 2 h before UVA exposure. However, therapy with this preparation is not infrequently disrupted by acute adverse reactions such as nausea, vomiting, pruritus and erythema. In contrast, 5-MOP, given in a dose of 1–2 mg/kg 2 h before UVA exposure, and now widely available, appears to be almost as effective as 8-MOP, and is virtually free of these effects. However, 8\% of subjects are reported to develop an apparently harmless, asymptomatic, maculopapular, intertriginous eruption early in treatment, which subsides spontaneously despite continued treatment.\textsuperscript{54} Furthermore, to date, *in vitro* photomutagenicity and photocarcinogenicity studies in mice suggest that 5-MOP is not associated with a greater degree of risk than 8-MOP.\textsuperscript{55,56} Thus, 5-MOP may eventually replace 8-MOP. Until then, as its clinical efficacy has been clearly demonstrated, 8-MOP should remain the psoralen of choice for most clinical situations, although 5-MOP should be substituted if acute adverse reactions occur.

**Long-term**

**Cataracts.** Following ingestion of a single therapeutic dose, 8-MOP can be detected in the ocular lens in humans for at least 12 h,\textsuperscript{57} and in rats for 12–24 h.\textsuperscript{58} Eye protection for at least 12 h following oral psoralen ingestion is therefore recommended. However, for children, patients with pre-existing cataracts, and those at increased risk of cataract development, such as atopic dermatitis sufferers,\textsuperscript{69} protection for 24 h is suggested. In the absence of an action spectrum for PUVA cataract induction, precise desirable transmission characteristics for protective glasses cannot be defined, but it has been widespread practice for PUVA staff to check patients’ spectacles for suitability by using radiation from a PUVA cabinet and a hand-held UVA meter.\textsuperscript{60} This seems a reasonable practice, as most PUVA effects are induced by short-wavelength UVA. However, if any UV transmission is detected, then spectrophotometric assessment should be performed to ensure that only long-wavelength UVA is transmitted, or preferably one of several sunglass lenses or coatings for prescription lenses, both tinted and clear, which provide virtually complete protection against UVA (Appendix 6), should be used. Although sideshields are considered preferable, these may reasonably be omitted without a great increase in risk, in the interest of patient compliance. There is currently no evidence from PUVA follow-up studies of an increased incidence of cataracts, and therefore prior
examination by an ophthalmologist need only be considered for patients at increased risk of cataracts, such as those with atopic dermatitis. There is currently insufficient data to provide informed advice on the necessity for, or optimal frequency of, ophthalmological follow-up for such patients. The retina can definitely be damaged irreversibly by PUVA, but adult ocular, and most artificial, lenses are extremely efficient UVA filters. However, patients with aphakia, either congenital or acquired, and young patients, must be given extra supervision to ensure adequate retinal protection. Although specially manufactured UVA-blocking goggles should normally be worn during therapeutic UVA exposure, these may safely be omitted for the treatment of diseased eyelid skin because negligible ocular UVB or UVA exposure occurs when the eyes are shut.

Skin cancer. A statistically significant increase in non-melanoma skin cancer risk, particularly for squamous cell carcinoma, is related to the number of treatments and UVA dose during PUVA therapy. Beyond 200 treatments there appears to be an approximately 10-fold increase in squamous cell cancer risk, and thus a maximum of 150–200 treatment sessions is recommended, except when there is no suitable alternative therapy. It also seems appropriate to advise that ideally no more than around 30 treatments should be given annually, in order to minimize skin cancer risk, and that maintenance PUVA should be avoided whenever possible. In view of the reported higher risk of genital than other skin tumours in men after high doses of PUVA, genital protection with opaque close-weave material is now considered mandatory during treatment. Because of its constant exposure to ambient irradiation, the skin of the face should also be covered during treatment when possible, if it is unaffected by disease. Ultraviolet-opaque but visibly transparent face shields or, alternatively, appropriate fabrics, can be used for this purpose.

Cumulative UVA dosage appears to be less reliable as a guide to skin cancer risk than the number of PUVA treatments. However, for a patient of skin type II/III receiving PUVA with oral 8-MOP, a relatively safe maximum dose is probably of the order of 1000–1500 J/cm². A careful and permanent record of the total number of treatment sessions and cumulative UVA dosage is essential, and long-term PUVA exposure of patients should be kept to a minimum by supervision of treatment by a dermatologist with special PUVA experience. In addition, annual review of patients, when possible, for up to 10 years, is advisable in order to check for malignant and premalignant skin lesions, in those patients who have received more than 150 treatments.

Conclusions

PUVA is an effective and economical dermatological therapy which is usually highly acceptable to patients. With careful supervision its short- and long-term adverse effects can be minimized, so that it remains a valuable treatment option over many years.


References


56 Young AR, Magnus IA, Davies AC, Smith NP. A comparison of the phototumorigenic potential of 8-MOP and 5-MOP in hairless


Appendix 1. A protocol for twice weekly PUVA treatment of psoriasis (ref. 15)

<table>
<thead>
<tr>
<th>MPD</th>
<th>1·0</th>
<th>2·0</th>
<th>4·0</th>
<th>&gt;4·0</th>
<th>Treatment number</th>
</tr>
</thead>
<tbody>
<tr>
<td>0·5</td>
<td>1·0</td>
<td>2·0</td>
<td>4·0</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>0·5</td>
<td>1·0</td>
<td>2·0</td>
<td>4·0</td>
<td></td>
<td>2</td>
</tr>
</tbody>
</table>

Starting dose (J/cm²)

0·7 | 1·4 | 2·8 | 5·6 | 40% increment
0·7 | 1·4 | 2·8 | 5·6 | 40% increment
0·9 | 1·8 | 3·6 | 7·3 | 30% increment
0·9 | 1·8 | 3·6 | 7·3 | 30% increment
1·1 | 2·3 | 4·6 | 9·7 | 25% increment
1·1 | 2·3 | 4·6 | 9·7 | 25% increment
1·4 | 2·7 | 5·5 | 10·9| 20% increment
1·4 | 2·7 | 5·5 | 10·9| 20% increment
1·6 | 3·1 | 6·3 | 12·6| 15% increment
1·6 | 3·1 | 6·3 | 12·6| 15% increment
1·7 | 3·5 | 6·9 | 13·9| 10% increment
1·7 | 3·5 | 6·9 | 13·9| 10% increment
1·8 | 3·6 | 7·3 | 14·5| 5% increment
1·8 | 3·6 | 7·3 | 14·5| 5% increment
1·8 | 3·6 | 7·3 | 14·5| 0% increment
1·8 | 3·6 | 7·3 | 14·5| 0% increment

The starting dose of ultraviolet A is 50% of the previously determined minimal phototoxic dose. Successive treatment doses are given in the vertical columns, with the dose increasing by the percentage shown after alternate treatments. After the 16th treatment, the dose is maintained. If erythema develops, the dose may be decreased by moving one column to the left.

Appendix 2. Dosage regimen for twice weekly PUVA treatment of psoriasis without MPD testing

<table>
<thead>
<tr>
<th>Skin type</th>
<th>J/cm²</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>0·5</td>
</tr>
<tr>
<td>II</td>
<td>1·0</td>
</tr>
<tr>
<td>III</td>
<td>1·5</td>
</tr>
<tr>
<td>IV</td>
<td>2·0</td>
</tr>
</tbody>
</table>

Subsequent increments

40% per week until erythema, then:
- maximum of 20% per week,
- if slight erythema—no increment,
- if moderate erythema—miss a treatment.

Dosage increases may be arbitrarily stopped once 15 J/cm² is reached.

Appendix 3. PUVA regimens for polymorphic light eruption in current use

1 First dose 70% MPD; increase by 20% three times weekly for 5 weeks.
2 First dose 0·5 J/cm²; increase by 40% twice weekly for 4 weeks.
3 First dose 1 J/cm²; increase by 1 J/cm² twice weekly for 2 weeks.

Appendix 4. Administration protocol for whole-body bath PUVA

1 Bath PUVA with 8-MOP
Dissolve 30 ml 1% 8-MOP solution in 80 l water (final concentration 3·75 mg/l)
Bathe for 15 min, followed by UVA exposure immediately
Initial UVA dose 0·2–0·5 J/cm²
Increase by ¼ of initial dose at each treatment
Frequency 2–3 times each week

2 Bath PUVA with trimethylpsoralen (TMP)
Dissolve 50 mg TMP in 150 ml ethanol
Mix in 150 l water (final concentration 0·33 mg/l)
Bathe for 10 min, followed by UVA exposure immediately
Initial UVA dose 0·1–0·4 J/cm²
Increase by ¼ of initial dose at each treatment
Frequency 2–3 times each week
Appendix 5. Regimens for hand/foot PUVA

1 Oral psoralen
8-MOP 0·6 mg/kg or 5-MOP 1·2 mg/kg
First dose 0·5–1·0 J/cm²
Increments 0·5–2·0 J/cm²

2 Topical 8-MOP
8-MOP 0·1% (approx.) in alcoholic or emulsion base
(may be diluted 1:10 if erythema occurs at lowest UVA dose)
Apply 30 min before UVA exposure
First dose 0·5–1·0 J/cm²
Increments 0·5–2·0 J/cm²

3 Bath 8-MOP
Mix 0·75 ml of 1% 8-MOP lotion in 2 l of water (final concentration 3·75 mg/l) at body temperature.
Soak for 15 min. followed by UVA exposure immediately
Initial UVA dose 0·1–0·5 J/cm²
Increments 0·1–0·5 J/cm²
Frequency 2–3 times each week

4 Bath TMP
Dissolve 50 mg TMP in 150 ml ethanol
Mix 2·1 ml in 2 l water
Bathe for 30 min. then give UVA immediately
Initial UVA dose 1–4 J/cm²
Increments 0·5–2 J/cm²
Frequency 3 times each week
Maximum dose 10–20 J/cm²
NB. If dorsa of hands or feet are affected give 50% of dose for palms and soles.

Appendix 6. Recommended eye protection

1 Spectacle lenses
Recommended spectacle lenses are listed below, with current price-range guides. Manufacturers are shown in brackets.

Lower price-range
Standard CR39 with UVX coating [Essilor] (£27)
Standard CR39 lens with UV400 coating [Norville]. Lenses are slightly yellow in colour (£31).
Perfalit Lambda 660 [Rodenstock]. Lenses are brown in colour (£40).

Higher price range
Lens UV PLS 530 [Norville]. These lenses provide good protection and are orange/amber in colour (£72).
Standard CR39 with Claret UV ET filter [Zeiss] (£75).

2 Spectacle frames
Safety spectacles with tinted side-shields provide the greatest, although not essential, protection, e.g. Stigmat 3007 [Norville] (£24).
Patient’s own spectacle frame can usually be fitted with made-to-measure tinted side-shields (£24).

3 Full protective device (1 + 2)
Total cost = spectacle lens + frame
Select lens from section 1 and frame from section 2.
e.g. Lens with UV400 coating £31
     Stigmat 3007 frame £24
     Glazing £4
     Total cost £59

3 Protective shields
These are protective goggles which can be placed over the patient’s own spectacles [NoIR]. Shield is dark amber-coloured, and provides excellent protection (£50).

For advice and supplies please contact:
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