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**Review**

**Update on treatment of photodermatitis**

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**Abstract**

Photodermatoses are a group of skin conditions associated with an abnormal reaction to ultraviolet (UV) radiation. There are several of the photosensitive rashes which mainly affect the UV exposed areas of the skin. It can be classified into four groups: immunology mediated photodermatoses, chemical and drug induced photosensitivity, photoaggravated dermatoses, and genetic disorders. A systematic approach including history, physical examination, phototesting, photopatch testing, and laboratory tests are important in diagnosis of a photodermatitis patient. In order to optimally treat a disease of photodermatoses, we need to consider which treatment offers the most appropriate result in each disease, such as sunscreens, systemic medication, topical medication, phototherapy, and others. For all groups of photodermatoses, photoprotection is one of the essential parts of management. Photoprotection, which includes suncreening and wearing photoprotective clothing, a wide brimmed hat, and sunglasses, is important. There are also promising emerging photoprotective agents.

**Keywords: photodermatoses; ultraviolet; sunscreen; phototherapy; photoprotective**

List of Abbreviations

AP	Actinic Prurigo
BCME	Buddleja cordata methanolic extract
CAD	Chronic Actinic Dermatitis
Ig	Immunoglobulin
IMP	Immunologically Mediated Photodermatoses
IVIG	IV Immunoglobulin

MED	Minimal Erythema Dose
MMF	Mycophenolate mofetil
MPA	Mycophenolic Acid
NB-UVB	Narrowband UVB
PL	<i>Polypodium leucotomos</i>
PLE	Polymorphic Light Eruption
PUVA	Photochemotherapy
ROS	Reactive Oxygen Species
SPF	Sun Protection Factor
SSUV	Solar Simulated UV
TPMT	Thiopurine s-methyltransferase
UV	Ultraviolet
UVR	Ultraviolet Radiation

## Introduction

Photodermatoses are a group of diseases that involve abnormal cutaneous reactions provoked by exposure to ultraviolet (UV) radiation [1, 2]. This may refer to the development of a new rash, the onset of subjective symptoms, or aggravation of an existing skin disease [3]. Photodermatoses can be classified into four groups: immunology mediated photodermatoses, chemical and drug induced photosensitivity, photoaggravated dermatoses, and genetic disorders with defective DNA repair or with chromosomal instability [1]. Age distribution is rather homogenous. Several disorders can present at any age, but the elderly population is more susceptible in drug induced photosensitivity [4] although over 25% of all photosensitivity cases begin at or before 15 years of age [5].

The immunologically mediated photodermatoses (IMP or idiopathic photodermatoses) describe several groups of skin disorders caused by optical radiation, predominantly in the UVA wavelength region. The exact pathomechanism in IMP remains to be elucidated, although an auto-immunologic mechanism is most likely. Polymorphic light eruption (PLE), chronic actinic dermatitis (CAD), actinic prurigo (AP), solar urticaria, and hydroa vacciniforme are the most important conditions in IMP [6]. Chemical- and drug-induced photosensitivity can be caused by topical or systemic exogenous agents, as in phototoxicity and photoallergy; these reactions can also be caused by endogenous agents like in cutaneous porphyrias and pellagra [4].

Skin diseases that occur without UV radiation (UVR) exposure but are occasionally exacerbated by UVR are called photoaggravated dermatoses. In conditions, such as lupus erythematosus, photoaggravation occurs, and the mainstay of prevention management is photoprotection. In psoriasis and atopic dermatitis, exposure to UVR results in improvement in a majority of

patients but may exacerbate a subset [7]. Genophotodermatoses with different kinds of genetic defects in the UV repair processes (Xeroderma pigmentosum, Cockayne syndrome, trichothiodystrophy) or with other biochemical abnormalities (Hartnup disease) are very rare genetic disorders. Besides the characteristic skin symptoms, these disorders are usually associated with an early development of cutaneous and ocular malignancies with specific extracutaneous features [8]. Classifications of photodermatoses are listed in Table 1.

**Table 1.** Classification of Photodermatoses

Immunology mediated photodermatoses	<ul style="list-style-type: none"> <li>• Polymorphous light eruption</li> <li>• Chronic actinic dermatitis</li> <li>• Actinic prurigo</li> <li>• Solar urticaria</li> <li>• Hydroa vacciniforme</li> </ul>
Chemical and drug induced photosensitivity	<ul style="list-style-type: none"> <li>– Caused by exogenous agents : Photoallergy Phototoxicity</li> <li>– Caused by endogenous agents : Cutaneous porphyrias Pellagra</li> </ul>
Photoaggravated dermatoses	<ul style="list-style-type: none"> <li>• Acne vulgaris</li> <li>• Seborrheic dermatitis</li> <li>• Lupus erythematosus</li> <li>• Atopic dermatitis</li> <li>• Rosacea</li> <li>• Darier’s disease</li> <li>• Dermatomyositis</li> <li>• Erythema multiforme</li> <li>• Grover’s disease</li> <li>• Lichen planus</li> <li>• Psoriasis</li> </ul>
Genetic disorders with defective DNA repair or with	<ul style="list-style-type: none"> <li>– Xeroderma pigmentosum</li> </ul>

chromosomal instability

- Bloom syndrome
- Cockayne syndrome
- Hartnup disease
- Rothmund–Thomson syndrome
- Kindler syndrome
- Trichothiodystrophy

Although these diseases have different pathophysiologic mechanisms, not all have been clearly defined [2]. A systematic approach including history, physical examination, phototesting, photopatch testing, and laboratory tests are essential steps in evaluating a photodermatosis patient [9]. There are many available options for treatment such as sunscreen, systemic medication, topical medication, phototherapy, and other photoprotective agents that will be discussed in this review.

## Sunscreens

Sunscreens can prevent sunburn, photosensitivity reactions, premature aging of skin, and skin cancer. Therefore, not only using sunscreen is essential, but also restricting or avoiding UVR and wearing protective clothing should be combined for skin protection [10]. Sunscreens are a fundamental part of photoprotection in the management of photodermatoses.

Solar UV radiation (290–400 nm) reaching the earth's surface contains no more than 5–10% UVB (290–320 nm) along with 90–95% of UVA (320–400 nm) [11]. The most effective sunscreen for every patient depends on identification of the photon wavelength responsible for inducing the sensitivity reaction. This can be done through the determination of a minimal erythema dose (MED) to UVA and UVB radiation [4], but occasionally visible light is the culprit.

UVB and UVA filters are categorized into organic and inorganic filters. The principle of photoprotection in organic sunscreens is the absorption of UV irradiation. In general, their chemical structure consists of aromatic compounds and two functional groups acting as 'electron releasers' and 'electron acceptors.' Upon UV irradiation, the molecule tends to delocalize the electrons to reach a higher state of energy. The excitation caused by absorption of UV light is the favored state of the molecule. There are many efficient UVB filters such as p-aminobenzoic acid, octocrylene, salicylates, and cinnamates [11], but only a limited number of organic UVA filters are available, such as the benzophenones (oxybenzone, dioxybenzone, and sulisobenzone), butyl methoxydibenzoylmethane (avobenzone), and methyl anthranilate. All except avobenzone are primarily protective only against UVA-2 (320-340 nm), whereas the absorption of avobenzone extends into UVA-1 (340-400 nm). Because avobenzone is photolabile, degradation occurs rapidly on exposure to sunlight. By combining it with photostable UV filters, such as octocrylene, salicylates, or oxybenzone, it can be photostabilized [4]. One important point is the systemic absorption of sunscreens after topical application. The particles of solid inorganic sunscreens such as zinc oxide and titanium dioxide are micronized in many formulations, thus reducing the reflection of visible light giving them a more transparent appearance and allowing smoother application [11]. Inorganic sunscreens are very photostable and may be safer [12]. The main advantage of inorganic sunscreens versus organic sunscreens is their low allergenic potential and the disadvantages are their visual appearance because of the solid consistency and white pigmentation residue on the skin [11].

Sunscreen efficacy is measured first by its sun protection factor (SPF), which is a globally accepted index of protection from erythema after a single exposure to solar-simulated radiation, primarily the effect from UVB exposure, and to a lesser extent from UVA [4]. The SPF is defined as the ratio of UVR required to produce a MED in sunscreen-protected skin to the amount required to produce the same degree of erythema in unprotected skin [12]. Restriction of UVR exposure and the use of high SPF sunscreens with efficacy against both UVB and UVA are important therapies for most groups of photodermatoses.

## Phototherapy and photochemotherapy (PUVA)

At first glance it appears strange to use light treatment to prevent a condition that is caused by light and the mechanisms by which UVB and PUVA induce tolerance to sunlight are not completely understood [13]. The principle of phototherapy and

photochemotherapy is the prevention of exacerbations of the disease by increasing the tolerance against solar radiation (light hardening). Epidermal thickening and pigment induction have been discussed as possible protection factors [6].

UVB is the simplest form of phototherapy. UVB radiation primarily penetrates into structures of the epidermis and superficial dermis. The radiation is absorbed by the major endogenous chromophores with immunological importance, such as nuclear DNA, trans-urocanic acid, and cell membranes. In addition to its effect on the cell cycle, UV light induces the release of prostaglandins and cytokines. Following UVB exposure, keratinocytes and lymphocytes secrete a number of pro-inflammatory cytokines such as IL-1, IL-10, and TNF- $\alpha$ , which suppress Langerhans cells and thereby induce immunosuppression [14].

Narrowband UVB (NB-UVB) phototherapy is more efficient than conventional broadband UVB therapy, emphasizing therapeutic onset and remission duration. Keratinocytes, circulating and cutaneous T-cells, neutrophils, monocytes, Langerhans cells, mast-cells, and fibroblasts are susceptible to low doses of NB-UVB and are thus more or less a selective target of the photons. NB-UVB induces local and systemic immunosuppressive effects, which may particularly contribute to the beneficial effect of this light source [14].

UVA light penetrates deeper into dermal structures, when compared to UVB light. UVA radiation carries less energy than UVB radiation, but owing to its long wavelength, penetrates the skin the deepest with as much as 50% reaching the dermis. Because of its longer mean wavelength, UV-1 radiation penetrates more deeply into the skin than UVA-2 and thus affects not only epidermal structures, but also mid and deep dermal components, especially blood vessels [14].

PUVA utilizes a combination of psoralens and UVA radiation. PUVA can reverse the pathologically altered patterns of keratinocyte differentiation markers and reduce the number of proliferating epidermal cells. PUVA strongly suppresses infiltrating lymphocytes, which varies depending on different T-cell subsets. PUVA is far more potent in induction of apoptosis in T-lymphocytes and antigen presenting cells than in keratinocytes, which may explain its efficacy in inflammatory skin diseases [14]. Numerous studies are available indicating that phototherapy and photochemotherapy are effective for treatment of photodermatoses and can improve the quality of life.

## Systemic therapy

### 1. Glucocorticoids

Systemic corticosteroids in short courses may be necessary in PLE, especially in the spring. In most sensitive patients, systemic steroids may be needed at the inception of the phototherapy [15]. It also can be used in severe reactions of phototoxicity, photoallergy [4], and AP [16]. Systemic steroids are effective in some cases of CAD but chronic toxicity of systemic steroids limits chronic usage [15].

### 2. Antimalarial

The antimalarial drugs - chloroquine and hydroxychloroquine, have shown efficacy in the treatment of PLE. Chloroquine, however, has shown an unacceptable risk of ocular toxicity that is believed to be a result of its affinity for melanin. Both the drugs are effective in reducing the severity of the signs and symptoms of PLE. Hence, hydroxychloroquine was well-tolerated with a superior quality of response as compared to chloroquine. Thus, hydroxychloroquine turns out to be a better option for treating PLE. Comparative studies have reported that hydroxychloroquine has a significantly lower risk of causing ocular toxicity than chloroquine [17]. In porphyria cutanea tarda, if phlebotomy is ineffective or if the patient has contraindications to phlebotomy, low dose hydroxychloroquine (200 mg twice weekly) can be effective. Antimalarials most likely chelate porphyrins and make them more soluble, thereby promoting renal excretion [1].

### 3. Azathioprine

Azathioprine is a synthetic purine analog derived from 6-mercaptopurine. It is a purine antagonist and its active metabolites act by disrupting the function of endogenous purines. It has a cytotoxic and immunosuppressive mechanism of action. It is used in dermatology for treatment of immunobullous diseases, generalized eczematous disorders, and photodermatoses. There is an enzyme in the metabolism of azathioprine called thiopurine s-methyltransferase (TPMT). It is very important to measure the TPMT activity before initiating therapy so that proper dosing of azathioprine can be used [18]. Some consider it the first choice for long-term immunosuppression of CAD. Azathioprine has also been reported to be helpful in severe PLE [19]. However, side effects, especially gastrointestinal disturbance, may limit the use of azathioprine in a small portion of patients [20].

#### 4. Cyclosporine

Cyclosporine, also known as cyclosporine A, was isolated from the soil fungus *Tolypocladium inflatum*. It is a calcineurin inhibitor that acts selectively on T cells. A case of CAD that was unresponsive to  $\beta$ -carotene and photoprotection rapidly improved on cyclosporine 4.5 mg/kg/day. Two cases of CAD that were unresponsive to high-dose steroids responded rapidly to cyclosporine 4 mg/kg/day for 3 months, with improvement of the pruritus and skin lesions [21].

Cyclosporine can be used as a prophylactic treatment for moderate to severe PLE. A case report of a patient with psoriasis and PLE showed that cyclosporine 3.3 mg/kg/day used to treat the patient's psoriasis also prevented exacerbations of her PLE. Three additional case reports revealed that cyclosporine 3 to 4 mg/kg/day 1 week before travel to a sunny climate with discontinuation upon return prevented flares of PLE without complications [21].

A case of treatment-resistant solar urticaria improved with cyclosporine 4.5 mg/kg/day. The patient was able to tolerate the sun for at least an hour with minimal urticaria as opposed to a few minutes without cyclosporine therapy. The solar urticaria returned once cyclosporine was discontinued. Cyclosporine may be useful in short courses in cases where treatment is only necessary during the summertime [21]. Side effects are dose dependent, related to the duration of therapy, and reversible on discontinuation once treatment guidelines are followed and careful monitoring is practiced [22].

#### 5. Antihistamines

Antihistamines (hydroxyzine, diphenhydramine, or doxepin) may be used for pruritus in PLE and AP. The nonsedating H1 agents such as loratadine, cetirizine HCl, and fexofenadine may increase the minimal urticaria dose 10-fold or more in solar urticaria. Higher doses, twice or more the standard recommendation may be required [15]. In phototoxicity and photoallergy, antihistamines may also be helpful [4].

#### 6. Thalidomide

Thalidomide has proven to be the most effective drug in the treatment of AP. In fact, the response to thalidomide may be a marker in the diagnosis of AP. The initial dose is 100–200 mg/day and it can be reduced when improvement is observed [23]. Apart from teratogenicity, the most important adverse effect was the induction of often irreversible peripheral neuropathy, occurring in 21–50% of people. Drowsiness, dizziness, mood changes, constipation, and xerostomia are more commonly reported side effects [24].

#### 7. Mycophenolate mofetil (MMF)

MMF is a prodrug of an older drug, mycophenolic acid (MPA). MPA was isolated as a fermentation product of *Penicillium stoloniferum*. It was studied as a potential antibiotic until the 1970s, when its antiviral, antitumor, and immunosuppressive properties were elucidated. MMF has also been used in actinic dermatitis. MMF as monotherapy has shown to induce significant improvement within only 6 weeks and eventual complete clearance on a maintenance dose of MMF. MMF may also allow a reduced dosage of prednisone required during PUVA desensitization. MMF proved to have higher bioavailability, efficacy, and fewer GI side effects [25].

#### 8. IV immunoglobulin (IVIG)

IVIG is composed of human plasma derived from pools of 1000 to 15,000 donors. The purified immunoglobulin is stabilized with glucose, maltose, sucrose, mannitol, sorbitol, glycine, or albumin. IVIG is made up of more than 90% immunoglobulin (Ig) G and small amounts of IgM and IgA. The total amount of immunoglobulins that are infused with a 2-g/kg dose is enormous; serum IgG will increase approximately 5-fold. It has been reported as effective modalities of treatment in solar urticaria. IVIG is relatively safe, particularly in comparison with alternative immunosuppressive treatments, but we should be aware of potential adverse reactions. Infusion-related side effects occur in less than 10% of patients and are generally mild and self-limiting. These side effects include headache, myalgias, flushing, fever, chills, fatigue, nausea or vomiting, low back pain, chest discomfort, hypotension and hypertension, tachycardia, and skin eruptions [26].

#### 9. $\beta$ -carotene

$\beta$ -carotene supplements are widely used as an oral sun protectant. However, studies demonstrating the protection of oral treatment with  $\beta$ -carotene against skin responses to sun exposure are inadequate. The protective effects are thought to be related to the antioxidant properties of the carotenoid. Upon UV-irradiation, the skin is exposed to photooxidative damage, which is induced by

the formation of reactive oxygen species (ROS). Photooxidative damage affects cellular lipids, proteins, and DNA and is considered to be involved in the development of photodermatoses [27].  $\beta$ -carotene has been reported to be helpful in PLE but large trials have not been performed to determine their efficacy. However, it has been reported with varying success as a treatment of erythropoietic protoporphyria [1].

## Topical therapy

### 1. Glucocorticoids

At times topical steroid, frequently of super or high potency in several daily to weekly pulses, are necessary to control the pruritus and clear the eruption in PLE, but in CAD is only effective in some cases [15]. It is also effective for mild disease in AP [16]. For symptomatic treatment of acute reactions in phototoxicity and photoallergy, topical corticosteroids are the drug of choice [4].

### 2. Antioxidant

Particularly with respect to UVB-induced skin damage such as erythema formation, the photoprotective effects of antioxidants are significant when applied in distinct mixtures in appropriate vehicles. Topical application of such combinations may result in a sustained antioxidant capacity of the skin, possibly owing to antioxidant synergisms. Because UVA-induced skin alterations are largely determined by oxidative processes, topical administration of antioxidants might be particularly promising. In fact, topical application of antioxidants or antioxidant mixtures resulted in a remarkable increase in the minimal dose to induce immediate pigment darkening after UVA exposure and diminished the severity of UVA-induced photodermatoses [28]. Topical antioxidants such as tocopherol seem to have a favorable influence on the course of PLE [6].

### 3. Calcineurin inhibitors

Tacrolimus shows a beneficial effect on CAD; this could be attributed to the fact that CAD is characterized by a lymphohistiocytic infiltrate producing a chronic eczematous dermatitis. Tacrolimus blocks the activation of lymphocytes and other immune system cells and also inhibits the release of mediators from cutaneous mast cells and basophils [29]. Topically applied tacrolimus and pimecrolimus significantly reduced the production of thymine dimers as markers for DNA photodamage after UVB irradiation [30]. It is also effective for mild disease in AP [16].

## Other photoprotective agents

### 1. Polypodium leucotomos (PL) extract

PL is a natural extract from tropical fern leaves with potent antioxidant and anti-inflammatory properties, likely because of its high concentration of polyphenolics and other antioxidant moieties [31]. PL supplementation acts at a molecular and cellular level to enhance endogenous antioxidant systems and inhibit generation of ROS, thus decreasing UV mediated oxidative DNA mutations. By reducing UV-induced inflammatory responses and inhibiting extracellular matrix remodeling, PL demonstrates some protective effects against PUVA induced phototoxicity [32].

PL extract 240mg taken twice daily for 60 days was a safe and effective means for reducing the damaging effects of UV radiation [33]. Oral administration of only two doses of PL was able to lead to a significant decrease in erythema, sunburn cells, DNA damage, UV-induced epidermal hyperproliferation, and mast cell infiltration in human skin [34]. The photoprotective activity of PL was significant. PL extract administration has shown to be an effective and safe method, leading to a significant protection of skin in IMP [35].

### 2. Baicalin

Baicalin is the predominant flavonoid isolated from the roots of *Scutellaria lateriflora* Georgi (Huang Qin). Baicalin displayed an antioxidant effect in the treatment of several diseases. Furthermore, recent studies have confirmed the photoprotective effect of baicalin against acute and chronic UVB-induced photodamage. This effect is thought to be associated with reduction of oxidative stress [36, 37]. Baicalin could reduce the levels of oxidative products, protect the mitochondrial membrane potential, and enhance cell viability, thus attenuating UVA-induced oxidative stress damage and apoptosis in human skin fibroblasts [36].

We have previously suggested that baicalin protects the cells from UVA-induced oxidative stress damage and apoptosis [36]. Topically applied baicalin inhibited chronic UVB irradiation of mouse skin induced epidermal thickening [37].



### 3. Ginsenoside Rg1

Ginsenoside, extracted from the root, stem, and leaves of ginseng, consists of three major moieties, Rg, Rb, and Rh. Ginsenoside Rg1, one of the protopanaxatriol groups, has multiple pharmacological effects. Recent studies have shown that the anti-aging abilities of ginsenoside Rg1 are related to its antioxidant and absorbance of ROS. Ginsenoside Rg1 can play a role in anti-PUVA-induced premature senescence as a result of its antioxidant activity by protecting telomeres. Thus, ginsenoside Rg1 may be a new promising agent for UV-protection in the future [38].

### 4. Buddleja cordata methanolic extract (BCME)

In recent years, there has been considerable interest in using botanical agents to prevent skin damage resulting from solar UV-irradiation. Buddleja cordata is a plant that is known as “tepozan.” Some people in Mexico use the leaves of this plant to treat tumors, abscesses, sores, and burns. BCME exhibits absorbance in the UVB spectrum, contains significant scavenging ability for the hydroxyl radical, and is not a genotoxic agent in the micronucleus test. The present findings demonstrate that BCME is endowed with good in vivo skin photoprotective properties and that this is likely related to the polyphenol content of BCME (verbascoside and linarin). BCME possess sunscreen properties that it may exhibit some potential to prevent photodamage [39].

### 5. Goji berry

The goji berry, *Lycium barbarum*, has long been recognized in traditional Chinese medicine for various therapeutic properties based on its antioxidant and immune-modulating effects. Reeve et al [40] describe the potential for orally consumed goji berry juice to alter the photodamage induced in the skin of mice by acute solar simulated UV (SSUV) irradiation. Dilutions of goji berry juice between 1% and 10% dose-dependently protected against SSUV-induced immunosuppression and against suppression induced by the mediator, cis-urocanic acid, measured by the contact hypersensitivity reaction. Antioxidant activity in the skin was demonstrated by the significant protection by 5% goji juice against lipid peroxidation induced by UVA radiation [40].

### 6. Green tea and white tea

Tea polyphenols have been found to exert beneficial effects on the skin via their antioxidant properties. Green tea has received much interest because of the beneficial role polyphenols play in skin cancer prevention. White tea is the least processed of the teas and may retain higher levels of polyphenols. Topical application of green and white tea offered protection against harmful effects of UVR. Hence, both green tea and white tea are potential photoprotective agents that may be used as a sun protection [41].

### 7. Propolis

Propolis is a honeybee product that has been used in traditional medicine for antioxidant, immune-stimulating, anti-inflammatory, and anti-cancer effects. The potential of the topical application of a crude ethanolic extract of Sydney propolis to protect against UVR-induced impairments associated with an increased risk of photocarcinogenesis has been tested in the hairless mouse. Sydney propolis was able to effectively reduce cutaneous inflammation, immunosuppression, and lipid peroxidation induced by UV exposure. It is concluded that Sydney propolis might have strong beneficial photoprotective effects [42].

## Conclusion

Photodermatoses are a challenging area for patients and dermatologists. A systematic approach is essential to arrive at the correct diagnosis. Avoidance of UVR exposure and the use of high SPF sunscreens with efficacy against both UVB and UVA can minimize the risk of photosensitivity effects. Phototherapy, systemic medication, topical medication, and other photoprotective agents are valuable in the management of photodermatoses (Table 2). There are also several promising photoprotective agents that may become effectively used in the future.

Table 2. Summary of treatment on photodermatoses

	Sunscreens	Phototherapy	Systemic medication	Topical medication	Other

Polymorphous light eruption	+	NB-UVB	Corticosteroid*, Antioxidant, Antimalarial, Cyclosporine*, Azathioprine*, Antihistamine, β-carotene	Corticosteroid	<i>Polypodium leucotomos</i> extract
Chronic actinic dermatitis	+	Low dose PUVA, NB-UVB	Cyclosporine, Azathioprine, Mycophenolate mofetil, Corticosteroid	Corticosteroid, Calcineurin inhibitors	Avoidance contact allergen, <i>Polypodium leucotomos</i> extract
Actinic prurigo	+	NB-UVB, PUVA	Corticosteroid*, Antihistamines, Thalidomide	Corticosteroid, Calcineurin inhibitors	<i>Polypodium leucotomos</i> extract
Solar urticaria	+	NB-UVB, UVA, PUVA	Antihistamines, Cyclosporine, IV immunoglobulin	-	<i>Polypodium leucotomos</i> extract
Hydroa vacciniforme	+	NB-UVB, PUVA	-	-	<i>Polypodium leucotomos</i> extract
Photoallergy	+	UVA, UVB, PUVA	Corticosteroid*, Antihistamines	Corticosteroid	Avoid causative agent
Phototoxicity	+	UVA,	Corticosteroid*,	Corticosteroid	Avoid

		UVB, PUVA	Antihistamines		causative agent, <i>Polypodium leucotomos</i> extract
Porphyria cutanea tarda	+	-	Antimalarial	-	Avoidance of precipitating factors, Phlebotomy
Erythropoietic protoporphyria	+	UVB, NB-UVB, PUVA	β-carotene	-	Therapy for hepatic disease
Photoaggravated dermatoses	+	-	-	-	Disease specific treatments

\*severe cases

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