

# Shady sides – manifestations of light dermatoses

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Besides the well-known sun burns, sunlight also can trigger very annoying light dermatoses. Food, pharmaceuticals and cosmetic ingredients are potential influencing factors. Sometimes even preventive measures and medicine are of little help, all the more important is an adequate skin care.

**L**ight induced sensitivities occur more frequently than generally expected. It is not only a matter of sun allergies that can be ascribed to substance-based influences. There is a wide range of symptoms starting from minor hyperpigmentations up to anaphylactic shock situations although this symptom is not very common. The collective term is light dermatoses and their co-factors can be classified into three different groups:

- photosensitizing and phototoxic substances
- metabolic and autoimmune diseases or genetic predisposition
- unknown factors

While photosensitizing and phototoxic substances can affect everybody at some time or another, the other triggers imply a particular prevalence in the form of a personal factor acquired through disease or genetic predisposition.

## Substances as triggers

Photosensitizing and phototoxic substances are ubiquitous and even occur as ingredients of skin care preparations. Photosensitizations caused by UV filters can be ascribed to a UV light-induced stimulation of the molecules. In some filters the conversion of the supplied energy into heat is not fast enough so that aggressive radicals can develop. If these radicals are not rendered harmless by means of appropriate antioxidants they will produce allergens in the surrounding milieu. Such reactions are well-known in connection with benzophenones and p-aminobenzoic acid derivatives (PABA).

The interaction of radiation and atmospheric oxygen in a variety of essential oils results in allergenic reaction products such as for instance ascaridol and 1,2,4-trihydroxymenthane in tea tree oil or bergapten from bergamot oil.

## Rather aggressive

The ethoxilated alcohols and polyethylene glycols (PEG) contained in various cosmetic preparations will form peroxides if exposed to the same conditions that are responsible for triggering Majorca acne. A variety of other compounds will develop from the different fragrance components and halocarbons (preservatives, antiseptics). The abietic acid of the colophony resin, sometimes an ingredient of mascara and eye shadows, will form highly allergenic oxidation products if exposed to UV/atmospheric oxygen; by the way: these highly allergenic oxidation products also occur in recycled newsprint and may cause hand eczema.

Basically the best protection from photosensitizing cosmetic ingredients is just attentively reading the INCI of the product before use and avoiding the relevant compositions. This however implies a certain expertise that cannot be expected from consumers. Even the majority of dermatologists may have a hard time interpreting it.

Further sunlight-induced reactions can be generated by food. Phototoxic furocoumarins from lemon or orange peels cause perioral dermatitis. Hypericin which occurs in St. John's wort oil is responsible for spot-like hyperpigmentations. The consumption of unpeeled buckwheat may cause itching skin rashes (urticaria) after sun exposure due to photosensitizing naphthodianthrone compounds that are similar to hypericin. In case of any suspicion that food components might have triggered photodermatoses, it is recommended to keep a diary particularly since the symptoms on the skin may appear with a time delay.

## Reactions to particular plants

Meadow grass dermatitis which is triggered by the transmission of furocoumarins (psoralenes) after contact with certain plants such as giant hogweed (cartwheel flower), common hogweed (cow parsnip, eltro) or celery also has

phototoxic origin. In this case blisters will form which will subsequently heal but leave hyperpigmentations. A characteristic sign is the lamellar array of the skin rashes caused by a brief contact with the plant and the leak of plant saps.

### Pharmaceuticals as triggers

Light dermatoses caused by the oral intake of photosensitizing pharmaceuticals occur as extensive spots on the skin areas that are not protected by clothing. Sometimes only symptoms such as an intense sun burn will appear. Information on pharmaceuticals that may imply phototoxic reactions can be found in the Red List, a German encyclopaedia on pharmaceuticals in which all the potential side effects are compiled (Ed.: Verlag Rote Liste Service GmbH, Frankfurt am Main). The following pharmaceuticals are involved among others:

- diuretics: thiazide derivatives as e.g. hydrochlorothiazide
- neuroleptics: phenothiazines such as chlorpromazine
- cytostatics: pyrimidine antagonists such as fluorouracil, alkylating drugs such as dacarbazine
- antimalaria drugs such as quinine, quinidine and chloroquine
- antibiotics: as e.g. tetracycline such as doxycycline und minocycline, that are prescribed to treat acne, rosacea and perioral dermatitis, among others, will cause "hypersensitizations"
- plant extracts as e.g. angelica (masterwort, wild parsnip): in the form of a pharmaceutical drug or tea, angelica is used to treat indigestion; the photosensitization is triggered by furocoumarins.
- 8-methoxypsoralene is specifically used for the phototherapy in the case of psoriasis
- vitamin A acid and isotretinoin (isomer)
- hormones
- NSAID (Non-Steroidal Anti-Inflammatory Drugs) as e.g. diclofenac or naproxen

The list could be continued indefinitely and proves how important it is to gather information on potential adverse effects before the intake of pharmaceuticals, above all if a particular sensitivity already is known. Besides the Red List, the DRUGDEX®-full text data base system by Thomson Reuters (USA) is an excellent source for detailed information.

As applicable for a number of other phototoxic reactions, absolute priority should be given to wearing appropriate clothing or a head covering in order to protect the skin against sunlight. Sun screens only offer partial protection since

the different photoreactions are triggered by varying wave lengths. In some cases the responsible wave lengths are in the visible range.

It is recommended to be cautious with the use of halogenated antiseptics. Triclosan, 5-chloro-2-(2,4-dichlorophenoxy)-phenol forms halogenated dibenzodioxins und dibenzofurans on the skin after sun exposure.

### Appropriate skin care

In case of an already existing light dermatosis, there are only limited options for an adequate skin care. Depending on the damage, any kind of every-day skin care may even be inappropriate. Frequently, it is recommended to just wait till the symptoms finally recede. If there are no objections from the dermatological point of view, the following skin care treatment can be suggested:

- Never use creams on weeping skin rashes! Restart skin care only after the skin areas have dried out. Exceptions to the rule might be aqueous sera containing astringent (gallic acids), anti-inflammatory (omega-3, omega-6, boswellia acids, and chamomile) or anti-itching agents (urea, allantoin, fatty acid amides).
- If using oils whose triglycerides contain essential fatty acids (linseed oil, kiwi seed oil, and evening primrose oil), non-fattening and emulsifier free nanodispersions have proved successful particularly in the case of sun-burn like dermatoses.
- After remission of the acute inflammatory phase, skin care creams can be used. In this case it is recommended to avoid occlusive conditions, perfumes, non-degradable emulsifiers and preservatives.
- Also aloe vera and D-panthenol can be applied. Unless there is an allergy to compositae, besides vitamin A also echinacea extracts can stimulate the skin recovery.
- Hyperpigmentations can be treated with a topical application of liposomal vitamin C-phosphate which can be supported by a light peeling in order to remove the pigmented skin layers in the medium run. Vitamin A-derivatives facilitate the cell formation. During the treatment, sun exposure should be avoided respectively adequate sun protection preparations should be applied.

### Internal causes

Origins of so-called secondary light dermatoses are metabolic or autoimmune diseases but also a genetic disposition. Some examples:

- Xeroderma pigmentosum is caused by a genetic defect. It is a life-threatening disease. UV radiation triggers inflammations, keratosis and carcinoma of the skin.
- Autoimmune diseases such as lupus erythematoses result in a hypersensitivity to sun radiation.
- Erythropoietic protoporphyria is a metabolic disorder in which the ability to form the blood pigment heme is impeded by an enzyme defect. The accumulating protoporphyrine absorbs the visible light in the range of 400-410 nm. In this process radicals will form which cause itching and pain after exposure to light. In most cases, over the counter sun protection preparations are useless since their UV filters are not effective in this particular wave range.

If the persons affected stay in areas that protect against the respective wave lengths, they can apply normal skin care preparations. In acute cases only medical therapy will help.

Light dermatoses with endogenic and hence to a large extent unknown triggers are called idiopathic light dermatoses. Similar to the substance-based photodermatoses they belong to the group of primary light dermatoses. Among them are:

- polymorphic light dermatosis with varying degree of severity
- light urticaria: heavily itching swellings up to anaphylactic shock
- actinic prurigo: itching dermatoses
- chronic actinic dermatitis: inflammatory and itching skin areas
- hydroa vacciniformia: redness and blisters leaving scars

The polymorphic light dermatosis is the most prevalent skin condition. Besides sun protective measures, the skin care in idiopathic light dermatoses and substance-based photodermatoses is similar. It is important to coordinate the skin care with the dermatologist in charge.

Dr. Hans Lautenschläger