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Recommendations and Basic Principles of Phototherapy

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Phototherapy has long been a cornerstone in dermatology, offered in most dermatology clinics globally. Despite the advent of several new biologic and systemic therapeutics, phototherapy remains a favoured treatment option due to its low side-effect profile and efficacy for treating mild-to-moderate inflammatory dermatoses. It can be used for a variety of skin conditions, including psoriasis, eczema, vitiligo, lichen planus, mycosis fungoides, pityriasis lichenoides, nodular prurigo, pruritus, and morphea.

In this article, we will provide an overview of the basic principles of phototherapy, as well as offering recommendations for managing a phototherapy service. Our focus will be on whole-body phototherapy, without the use of psoralens.

Introduction

Phototherapy, in the form of sunlight, has been used since ancient times, with historical evidence indicating its use in Egyptian and Indian cultures to treat skin diseases. The modern practice of phototherapy began with the pioneering work of Danish physician Niels Finsen, who used ultraviolet (UV) radiation to treat lupus vulgaris. For his contributions, Finsen was awarded the Nobel Prize in 1903.^{1,2}

By the 19th century, phototherapy began to become established as a medical treatment. In 1925, Goeckerman combined UV radiation with crude coal tar to treat psoriasis. However, it was not until the 1970s that broadband UVB (BB-UVB) became a widely accepted treatment for various inflammatory skin diseases. A paradigm shift occurred in the 1980s, when Parrish and Jaenicke demonstrated that the action spectrum and most effective therapeutic wavelength for treating psoriasis was 313 nm. Following this, Philips introduced fluorescent narrow-band (NB-UVB) phototherapy lamps that emit light between 310 and 311 nm, which were later adopted by other manufacturers.^{1,2}

Phototherapy and Mechanism of Action

Ultraviolet radiation (UVR) exerts its effects on the skin through multiple biological mechanisms, involving both the innate and adaptive immune systems. It has an immunosuppressive effect on T-cell function and induces antigen-specific tolerance. In addition, UVB reduces DNA synthesis, which is helpful for skin conditions such as psoriasis, where there is accelerated DNA synthesis. It induces the production of cytokines such as interleukin (IL)-6, IL-1, and activates pathways including the expression of tumour suppression gene p53, which leads to cell cycle arrest and allows for DNA repair. Over time, phototherapy leads to epidermal hyperplasia and tanning.³

Wavelengths associated with each type of phototherapy are shown in **Table 1**. In simple terms, the longer the wavelength, the deeper it penetrates into the skin. UVB mainly penetrates into the epidermis and upper dermis. In contrast,

Phototherapy Type	Wavelength Range (nm)	Penetration Depth
NB-UVB	311–313	Epidermis
BB-UVB	280–320	Epidermis, upper dermis
Combined UVAB	280–400	Deep dermis
UVA1	340–400	Deep dermis

Table 1. Wavelengths associated with different types of phototherapy; courtesy of Tashmeeta Ahad, BM BCh(Oxon), MA(Cantab), MRCP(UK)(Derm).

Abbreviations: NB-UVB: narrow-band ultraviolet B, BB-UVB: broadband ultraviolet B, UVAB: ultraviolet A and B, UVA1: ultraviolet A1

UVA is able to penetrate to the deep layers of the dermis and potentially beyond. When choosing phototherapy wavelengths, it is important to consider the following factors, such as photon energy (inversely proportional to wavelength) and action spectrum (measure of the importance of each wavelength in producing a particular photobiologic response). Nucleic acids, DNA, and chromophores in skin mainly absorb UV photons at approximately 300 nm (UVB). Apart from DNA and nucleic acids, UVR also acts on other chromophores such as urocanic acid and tryptophan found in skin. UVR may also induce apoptosis of keratinocytes forming 'sunburn cells'. The mechanism of DNA damage underpinning photocarcinogenesis includes the creation of dimeric DNA photoproducts, namely **i)** Cyclobutane pyrimidine dimers, and **ii)** 6–4 photoproducts. In contrast to UVB, UVA causes DNA damage via indirect photon absorption, such as type 1 and type 2 photosensitized reactions, and the creation of reactive oxygen species and free radical damage.^{1,2,4}

Phototherapy Modalities

Phototherapy modalities used for whole-body phototherapy are shown in **Table 1**. Narrow-band UVB (NB-UVB) is the most commonly used type of phototherapy, although centres such

as ours in Vancouver, British Columbia offer BB-UVB combined UVA and UVB as well as UVA1 whole-body phototherapy.

Phototherapy is typically administered over several weeks, with patients undergoing treatment 2–3 times a week. Typically, to treat skin conditions such as psoriasis or eczema, patients may require at least 25 treatments to achieve adequate improvement. The UV dose administered is increased over time as photoadaptation occurs, allowing patients to tolerate higher doses without burning. This is due to epidermal hyperplasia and tanning. The objective is to aim for a suberythemogenic dose each time, which is just below the threshold required to cause erythema (redness/sunburn) of the skin, to achieve a photobiologic response.

Phototherapy Equipment

There are various types of phototherapy devices used to treat patients, ranging from full-body units to targeted handheld devices. Most hospital clinics and dermatology offices will incorporate full-cabinet devices, although 3D panels and single panels may be used for home phototherapy. Handheld devices are also available. Phototherapy machines use specialized UV lamps, most commonly fluorescent bulbs. Traditionally, mercury arc lamps were used.^{1,2} More recently, LED-based systems are being explored, due to their ability to provide precise wavelengths with energy efficiency.

Dosimetry and Calibration

Accurate dosimetry is critical in phototherapy to ensure patients receive the correct dose. UVB and UVA irradiance must be regularly monitored using calibrated metres. Any changes in lamp performance, such as bulb replacements, can alter the output and must be adjusted to maintain consistent dosages. Dosimeters integrated into the cabinet may be unreliable, although for an office-based practice they may have to be relied upon if access to external calibration checks, such as those provided by medical physics, are unavailable.^{1,5} These differences mean that UV dose outputs across different machines and

clinics are often not comparable. It is important to periodically verify the irradiance of phototherapy equipment. The following formula can be used for calculating the dose and corresponding exposure

$$\text{Irradiance (mW/cm}^2\text{)} \times \text{Time (seconds)} = \text{Dose (mJ/cm}^2\text{)}$$

times to make calibration adjustments:

Treatment Protocols

Although published references for treatment protocols, such as by the American Academy of Dermatology,⁶ are available, these protocols may need to be modified based on various factors. These include the type of equipment used and its calibration, the skin phototype profile of the patient population being treated, the available clinical supervision in clinics (e.g. nurse/medical office assistant), and ability to manage adverse effects such as erythema. These and other factors may influence the 'aggressiveness' of a treatment protocol.

There are 3 main components to consider when determining a phototherapy treatment protocol.

- **Starting Dose:** Determined based on the patient's Fitzpatrick skin type or Minimal Erythema Dose (MED).
- **Incremental Doses:** Increased gradually over time to maximize efficacy while avoiding erythema. Typical increments tend to be 10% or 20%.
- **Frequency:** Treatment frequency typically starts at 2–3 times per week.

Starting Dose

Different individuals are likely to tolerate varying initial doses of UV. Factors influencing this include skin pigmentation, phenotype, medications which may cause drug induced photosensitivity,⁸ and underlying photosensitivity disorders.⁹ The starting dose for phototherapy can be chosen based on an individual's MED, or empirically chosen based on skin pigmentation properties, such as the Fitzpatrick skin phototype⁷ (Table 2). The MED is defined as the minimum UV dose

Fitzpatrick Skin Phototype	
I	Always burns, does not tan
II	Burns easily, tans poorly
III	Tans after initial burn
IV	Burns minimally, tans easily
V	Rarely burns, tans darkly easily
VI	Never burns, always tans darkly

Table 2. Fitzpatrick Skin Phototype.⁷

Skin Phototype	NB-UVB Starting Dose (mJ/cm ²)	BB-UVB Starting Dose (mJ/cm ²)
I	130	20
II	220	25
III	260	30
IV	330	40
V	350	50
VI	400	60

Table 3. Example starting dose for UVB (in mJ/cm²); courtesy of Psoriasis and Phototherapy Clinic, Vancouver General Hospital.

Abbreviations: NB-UVB: narrow-band ultraviolet B, BB-UVB: broadband ultraviolet B

that induces erythema within 24 hours. In Europe and the UK, a patient's MED may be used as the primary method of selecting a starting dose for phototherapy. However, due to time and labour constraints, most North American clinics tend to use empirical dosing based on the Fitzpatrick skin phototype.

The starting dose for phototherapy is typically set at 70% of a patient's MED or is based on empirical guidelines tailored to the Fitzpatrick skin phototype (**Table 3**).

Incremental dose

Typically, incremental dosing of 10%–20% may be used, with treatment administered 2–3 times a week. The maximum doses can reach approximately 3000 mJ/cm², as tolerated. Incremental dosing may need to be adjusted depending on side effects, most commonly erythema (**Table 4**) and if treatments are missed (**Table 5**).

Adverse Events and Safety Procedures

Patients should be informed about both acute and chronic side effects and provide consent. It is advisable to incorporate a patient consent form and provide written patient information before starting phototherapy. A nursing education session or similar briefing, prior to starting a treatment course, which outlines what phototherapy entails, safety procedures, expectations, and potential adverse events will help mitigate medicolegal risks. Patients should be advised on the importance of protective equipment such as eye goggles and of wearing the same clothing to avoid non-photoadapted skin from becoming exposed or burned. The doses administered and side effects observed should be documented in detail following each visit.⁵

Acute Side Effects

- Erythema/burning
- Tanning
- Reactivation of herpes virus infection (cold sore)
- Itching
- Activation of photosensitivity disorder

Chronic Side Effects

- Photoageing
- Skin cancer risk is theoretical with UVB phototherapy, because existing literature has shown no evidence of increased skin cancer risk with UVB phototherapy.^{10,11} However, guidelines recommend offering routine skin cancer screening to patients who have had more than 500 UVB exposures or may have other risk factors.⁵

Grade of Erythema	Reaction to Previous Exposure	Dose Increment
0	No erythema or pain	10–20%
1	Mild erythema without pain	5–10%
2	Mild erythema with minimal pain/discomfort lasting <24 hours	0% Keep same fluence
3	Moderate erythema with pain/discomfort lasting >24 hours	-10%
4	Severe erythema with symptoms e.g., blistering/tenderness	Hold treatment for at least 1 week

Table 4. Adjustment to UV incremental dosing based on erythema/adverse events. (Approximate values; variations possible); courtesy of Psoriasis and Phototherapy Clinic, Vancouver General Hospital.

Time Since Last Treatment	Decrease in Fluence
1 week	25%
2 weeks	50%
3 weeks	75%
4 weeks	Baseline fluence

Table 5. Adjustment to UV incremental dosing for missed treatments. (Approximate values; variations possible); courtesy of Tashmeeta Ahad, BM BCh(Oxon), MA(Cantab), MRCP(UK)(Derm).

Conclusion

Phototherapy remains a fundamental treatment in dermatology for inflammatory skin diseases. By understanding the underlying mechanisms, using equipment appropriately, and individualized treatment protocols for each patient, dermatologists can provide effective and safe phototherapy services.

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Financial Disclosures

None declared.

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