Family Practice Edition

Skin Therapy Letter©

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Clinical Evidence. Practical Advice.

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EDITOR-IN-CHIEF

Dr. Stuart Maddin, Chairman of SkinCareGuide, is one of North America's well-known and respected dermatologists, and is the author of numerous dermatologic journal articles, monographs and textbooks. In addition to providing consultative input to a number of pharmaceutical and biotech companies, he is the director of the clinical trials unit at the Division of Dermatology, UBC. Dr. Maddin has also acted in an advisory capacity to a number of drug regulatory agencies, such as the Health Protection Branch (Ottawa), the AAD-FDA Liaison Committee and WHO (Geneva). As well as being the founder of the Dermatology Update symposia, now in its 21st year, he is past President of the Canadian Dermatology Association and served as Secretary-General of the International Committee of Dermatology—International League of Dermatological Societies.

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A Message from Dr. Stuart Maddin, Editor-in-Chief

In a dermatology career that spans more than 50 years, I have long appreciated the important role of the family practitioner in the diagnosis and treatment of skin conditions and diseases. As I meet physicians from across Canada, I am often asked about the latest developments in diagnosis and treatment. I am therefore pleased to introduce you to a brand new publication written especially for the family physician: Skin Therapy *Letter*© – Family Practice Edition.

The history of Skin Therapy *Letter*© began 10 years ago this month, and is the only publication of its kind to be indexed by the US National Library of Medicine. We send Skin Therapy *Letter*© to every dermatologist in Canada and recently have begun adding the 15,000+ dermatologists in the United States to that list. This makes Skin Therapy *Letter*© one of the world's principal sources of information on skin disease.

In this first issue of Skin Therapy *Letter*© – Family Practice Edition, you will find practical information about treating eczema, including information about the recent US FDA black box warning and how that may or may not affect your practice. We also deal with hyperhidrosis, a condition which for many patients is highly embarrassing and has a huge impact on their quality of life. The summer months bring the sun and a heightened concern about its effects. We explore UVA and UVB light and suggest what patients should be advised to do to protect themselves from harmful rays.

I hope that you will find this helpful. We would enjoy receiving your feedback, topic suggestions and comments. Please email us at physicians@skincareguide.com.

Welcome.

Atopic Eczema

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Diagnostic Features of Eczema or Atopic Dermatitis (AD)

Diagnostic Features of Eczema or Atopic Dermatitis (AD): a chronic relapsing condition in patients with a personal or family history of atopy. Usually starts before the age of 2 years and usually improves or resolves in older children and adults.

- **Itching** must be present to make the diagnosis.
- **Dry Skin** is always present.
- **Typical Rash Location** varies with age of patient. Infants: face usually involved. Diaper area and axilla usually clear. Extensor arms and legs involved due to friction from crawling. Children 4-10 years: flexures, sides of the neck, earlobes.
- **Inflammed Skin** usually seen.
- **Secondary Infection** – *Staphylococcus aureus* very common, molluscum and herpes infections often more extensive.

Treatment: Self-help and Medical Treatments

1. Patient Self-help – Patients may not improve if triggers are not removed

Aggravating/Trigger Factors to be Avoided

- Skin irritants such as soap, bubble bath, detergents, fabric softeners and perfumed products.
- Frequent bathing, especially if not followed by moisturizers. Sweating may exacerbate pruritus.
- Skin infection will tend to promote AD
- Food allergies can play a role in a small percentage of young patients with AD (e.g. eggs, milk, nuts, peanuts, fish, shellfish, wheat, and soy account for over 90% of food allergies)
- Environmental allergens such as house dust mites

Self-Help

1. Use a mild cleanser; 2. Moisturize often; 3. Hydrocortisone; 4. Cool bathing; 5. Use perfume free products; 6. Oral antihistamines; 7. Avoid triggers

Mild Cleansers

- Mild soap or nonsoap cleanser like Spectrojel®, Spectroderm®, Cetaphil®, plain white Dove®
- Emulsifying ointment USP (ask pharmacist)

Moisturizers

Use at least 250mg of cream/ week. Must be thick like butter or greasy like petroleum (e.g., Vaseline® Petroleum Jelly, Aquaphor® ointment, Creamy Vaseline®, 25% water in Hydrophilic Petrolatum, Aqueous cream, Aveeno® cream, unscented cold cream, Eucerin® cream, Cetaphil® cream, Cliniderm® cream)

2. Medical Treatment

Relief and suppression of eczema flares check list:

✓ Itch Relief – Dry Skin Therapy – Inflammation Suppression with anti-inflammatories

✓ Infection Control

Itch Relief

Oral-sedating antihistamines at bedtime. Hydroxyzine, Benadryl®, start at low dose and increase as tolerated. Moisturize and use topical anti-inflammatory. Wet compresses using gauze or face cloth dipped in tepid water, rung out and then laid on oozing skin is soothing.

Long-term Control by Preventing New Flares

Control of a flare may be easier than motivating a patient to continue intermittent use of an anti-inflammatory or to regularly moisturize the skin and avoid triggers. Using anti-inflammatory topicals at the first signs and symptoms can minimize the use of medication and give smoother control of the disease.

Dry Skin

Frequent moisturizing especially within 2-5 minutes after bathing and when skin is wet. Most patients under moisturize. Check the quantity used. Use a minimum of 250mg/week (more if possible).

Anti-inflammatory Drugs

Corticosteroids and Calcineurin inhibitors are useful for short- and long-term use.

Topical Corticosteroids (TCS)

- Gold standard of treatment for AD
- Quick acting anti-inflammatory action. Potency from mild

Treatment: Self-help and Medical Treatments (continued)

to very potent.
- General rule is that one uses the lowest potency possible for control of the disease. Goal is to be off the steroid more often than on the steroid.
- Useful for flare prevention.
- In conjunction with calcineurin inhibitors, they have a role as rescue medication when severe flares develop.
- Low potency corticosteroids are best used in the skin folds, face and neck.
- Moderate potency steroids are needed for thick lichenified eczema in older children and for acute flares on the body.
- Side-effects such as skin atrophy, tachyphlaxis, and adrenal suppression can occur but these are usually seen if the drug is used for too long, too often, or too much especially on the face folds, or inner thigh. Very young and old patients are more at risk. No harm will come from using potent corticosteriods for short periods, i.e., days at a time.
- Two issues of concern are steroid phobia by patients and steroid allergy:
  a) Steroid phobia — patients need to understand that the body naturally produces steroids and that side-effects are unlikely if topical steroids are used appropriately.
  b) Steroid allergy – Uncommon. Patch testing is required to confirm.

If a patient is not responding to a topical steroid, as you would expect, consider compliance, secondary infection, or an allergy to the medication.

Topical Calcineurin Inhibitors

Pimecrolimus (Elidel™ 1% cream)

Nonsteroid approved for short- and long-term intermittent use in mild-to-moderate AD over 2 years of age. Guidelines suggest use when other standard treatments fail or there is concern regarding risk of side-effects.
- Rapid relief (1-2 weeks) due to targeted anti-inflammatory action
- Used in practice to bring AD under control and also intermittently thereafter at first signs and symptoms of disease activity to prevent flare-ups. Topical corticosteroids can also be used but some physicians reserve them for more severe flares.
- Well-controlled studies in infants, children and adults demonstrate significant reduction in incidence of flares with the use of corticosteroids.
- Long-term studies show efficacy and safety in infants from 3-23 months, but it is not approved for this age range.
- Burning or stinging can be a problem but the likelihood is usually relative to disease severity. Patients do much better if warned of this transient effect.
- Long term safety – see Author's Comment on recent FDA advisory.

Tacrolimus (Protopic™0.03% and 0.1%ointment)

Non-steroid approved for short-term intermittent use in moderate-to-severe atopic AD over 2 years of age (0.03% >2yrs, 0.1% >15yrs). Guidelines suggest use when other standard treatments fail or concern regarding risk of side effects.
- Rapid relief (1-2 weeks) due to targeted anti-inflammatory action
- Used in practice to bring AD under control and also intermittently thereafter at first sign of disease activity or flare. TCS could be used as rescue medications for severe flares
- Long-term studies show safety and efficacy in > 2yr old.
- Burning or stinging can be a problem but the likelihood is usually relative to disease severity.
- Long term safety – see Author's Comment on FDA advisory.

Infection Control

Clinical experience shows that AD may respond to anti Staph antibiotics even when there are no signs of a typical impetigo. Localized AD with probable secondary infection (swab if in doubt) use mupirocin cream, fucidic acid cream/ointment or oral cloxacillin or cephalosporin if widespread.

Authors' Comments

Recently asked question: What is the role of calcineurin inhibitors in the control of eczema following the recent FDA Health Advisory?

It is the authors' opinion that the recent Advisory about calcineurin inhibitors increasing the risk of cancer is based more on fear than fact. Granted that long-term use of oral immunosuppressive agents, such as in the transplant population, does raise the incidence of lymphoma, the degree of suppression is dose related. Rates of lymphoma and skin cancers in clinical trials and postmarketing surveillance reveal cancer rates that are much lower than would be expected in a control population. Evidence shows that both pimecrolimus and tacrolimus have very low systemic absorption when used topically as recommended for AD. Animal studies using oral formulations showed a higher rate of lymphoma, but at very high doses.

What is clear is that people with AD have very significant disease that impacts most negatively on their quality of life. For the physician and patient there is no option other than to treat this disabling condition. With any medication there are always potential risks, but based on the available information the benefits of these medications far outweigh the risks. Resistant cases have been treated with UV light, azathioprine, cyclosporine, mycophenolate mofetil and systemic steroids.

It is significant that in a recent survey of leading dermatologists in North America and Europe conducted and published by Skin Therapy *Letter*© the majority of these doctors reported that they will not change their habit of prescribing topical calcineurin inhibitors, but are now likely to spend a little more time counseling and informing their patients who show concern.

Hyperhidrosis

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Hyperhidrosis is characterized by sweating in excess of the physiological needs to maintain thermal homeostasis. No formal definition exists but for practical purposes any degree of sweating that interferes with activities of daily living, can be viewed as hyperhidrosis. The cause is unknown, however treatment options do exist. This disease is much more common than once thought and greatly impacts upon quality of life (QOL). Treating these patients can be exceptionally rewarding both for the patient and the physician.

Topical Treatment	**Controls mild disease**
Botulinum Toxin A (BTX-A)	**Effective** [Naumann M, Hamm H, Lowe NJ. *Br J Dermatol* 147:1-9 (2002a); Naumann M, Lowe NJ, Kumar CR, Hamm H. *Arch Dermatol* 139:731-6 (2003); Lowe NJ, Yamauchi PS, Lask GP, Patnaik R, et al. *Dermatol Surg* 28(9):822-7 (2002).]
Iontophoresis	**Effective** [Reinauer S, Neusser A, Schauf G, Hölzle E. *Br J Dermatol* 129:166-9 (1993).]
Surgery	**Considered only after all other options have failed due to potential complications.** [Zacherl J, Huber ER, Imhof M, et al. *Eur J Surg* 580(suppl):S43-6 (1998).]

Table 1: Treatment options for hyperhidrosis

Epidemiology and Etiology

In a recent survey of 150,000 households in the US, 2.8% of the population reported having unusual or excessive sweating.[Strutton DR, Kowalski JW, Glaser DA, Stang PE. Presented at the American Academy of Dermatology 61st Annual Meeting, San Francisco CA.] The axilla is the most common affected site, followed by the feet, palms and face. No gender differences were noted and onset typically occurred during childhood or adolescence. The exact cause of focal hyperhidrosis is unknown, although sympathetic overstimulation of normal eccrine glands is the most likely etiology. Interestingly, studies have shown an association between the sympathetic hyperactivity seen in hyperhidrosis with other autonomic disorders such as cardiac hyperexcitability. There is likely also a heritable component to this neurogenic overactivity, as 30%-50% of patients have a positive family history.[Haider A, Solish N. *CMAJ* 172(1):69-75 (2005 Jan 4).]

Hyperhidrosis Type	**Clinical Presentation**
Primary (idiopathic)	Focal – localized to the axillae, palms, feet, face
Secondary	Generalized, though it can present in a localized, focal pattern.

Table 2: Clinical Presentation of hyperhidrosis

Impact on Quality of Life

Hyperhidrosis has a profound impact on social interactions and work related activities. Routine social interactions such as holding hands, shaking hands or hugging become awkward. Patients report a sense of humiliation and embarrassment associated with soaked or stained clothing as well as perceived odours.[Naumann M, Hamm H, Lowe NJ. *Br J Dermatol* 147:1-9 (2002a); Haider A, Solish N. *CMAJ* 172(1):69-75 (2005 Jan 4).]

Drugs/Toxins	Alcoholism, substance abuse
Cardiovascular	Heart failure, shock
Respiratory Failure	
Neurologic	Parkinson's disease, spinal cord injury, cerebrovascular accident
Endocrine	Hyperthryroidism, diabetes mellitus, pheochromocytoma, carcinoid syndrome, acromegaly, pregnancy, menopause
Infections	
Malignancies	Hodgkin's disease, myeloproliferative disorders

Table 3: Etiology of secondary hyperhidrosis

Diagnosis and Patient Evaluation

A history focussing on the location of excessive sweating, duration of the presentation, associated symptoms or comorbidities, family history, age of onset and any specific triggers allows one to differentiate primary from secondary hyperhidrosis. The physical exam will be guided by any suggestion of secondary hyperhidrosis, and will also attempt to confirm the distribution of disease.

Measurement of Hyperhidrosis

Each evaluation should attempt to determine the volume of sweat production, the distribution of hyperhidrosis and the affect on QOL. The starch iodine test provides a qualitative assessment of both volume of sweat production and extent of distribution.

- The area to be tested is dried and an iodine solution (1%-5%) is applied.
- After a few seconds, starch is sprinkled over this area.
- The starch and iodine interact in the presence of sweat to develop a purplish sediment.

This test is most helpful when delineating the area for treatment. To assess impact on QOL, various measures have been validated, such as the hyperhidrosis disease severity scale (HDSS). (Table 2)

My sweating is never noticeable and never interferes with my daily activities.	Score 1
My sweating is tolerable but sometimes interferes with my daily activities.	Score 2
My sweating is barely tolerable and frequently interferes with my daily activities.	Score 3
My sweating is intolerable and always interferes with my daily activities.	Score 4

Table 4: Hyperhidrosis Disease Severity Scale (HDSS) is a four-point scale to determine the degree of severity of hyperhidrosis.

Topical Treatments for Hyperhidrosis

Aluminum chloride hexahydrate is considered to be the most effective topical agent for focal, mild axillary hyperhidrosis. It works through mechanical obstruction of the eccrine sweat gland pore. Another topical product is glycopyrrolate, a topical anticholinergic product available as topical pads for mild cases. The main limiting side-effects of all of these products are skin irritation, lack of efficacy in moderate-to-severe axillary hyperhidrosis, and poor response on the palms and soles.

Systemic Treatments

In addition to glycopyrrolate mentioned in Table 5, other agents such as clonazepam, diltiazem, clonidine and nonsteroidal anti-inflammatories have been reported to be useful in isolated cases.

TREATMENT	INDICATION	COMMENTS
Topical treatments	Axillary, facial, less commonly palmar and plantar hyperhidrosis	Short term action. Efficacy in mild cases. Major side effect is local irritation. Palms and soles less responsive
Systemic treatments, e.g., glycopyrrolate	Main role in generalized and compensatory hyperhidrosis.	Limited efficacy due to anticholinergic side-effects, e.g., dry mouth, blurred vision, constipation, urinary retention, palpitations.
Iontophoresis	Palmar and plantar hyperhidrosis	Well tolerated. Dryness and irritation are common. Efficacy can reach 90%. Major limitations are the equipment expense and procedure is time consuming
Surgical sympathectomy	Hyperhidrosis unresponsive to topical or systemic treatment and BTX-A	Major limitation is possible surgical adverse events and an unacceptable high rate of compensatory hyperhidrosis. ETS surgery should be considered as a last resort.
Botulinum toxin injections	Axillary, palmar, plantar and facial hyperhidrosis	Commonly used for moderate-to-severe disease or if mild cases do not respond to topical treatment. Safe, effective and well-tolerated treatment with excellent patient satisfaction. Drug usually covered by third party insurance.

Table 5: Hyperhidrosis treatment options

Treatments Considerations with Botulinum Toxin

The main contraindications to botulinum toxin therapy include neuromuscular disorders such as myasthenia gravis, pregnancy and lactation, organic causes of hyperhidrosis, and medications that may interfere with neuro-muscular transmission. [Haider A, Solish N. *CMAJ* 172(1):69-75 (2005 Jan 4).] Appropriate selection of patients is essential to ensure a satisfactory treatment response and avoid unnecessary frustrations.

Condition	Results	Side-effects	Comments
Axillary hyperhidrosis [Naumann M, Hamm H, Lowe NJ. *Br J Dermatol* 147:1-9 (2002a); Naumann M, Lowe NJ, Kumar CR, Hamm H. *Arch Dermatol* 139:731-6 (2003); Solish N, Benohanian A, Kowalski JW. *Dermatol Surg* 31(4):405-13 (2005 Apr).]	>90% response rates with statistically significant improvements in QOL	Safe, well tolerated	Typical starting doses are 50 units of BTX-A per axilla. The mean duration of effect is 6–7 months.
Palmar hyperhidrosis [Lowe NJ, Yamauchi PS, Lask GP, Patnaik R, et al. *Dermatol Surg* 28(9):822-7 (2002).]	>90% response rates	Pain at the site of injection; transient minor weakness of intrinsic hand muscles lasting 2-5 wks.	Treatment consists of 100 units per palm. Intradermal injection into the palm is painful, so anesthesia is strongly recommended. Regional nerve blockade (median and ulnar) is most commonly used. Topical anesthesia is ineffective.
Plantar hyperhidrosis [Haider A, Solish N. *CMAJ* 172(1):69-75 (2005 Jan 4).]	Excellent response rates have been reported	Pain at the site of injection	Treatment doses and technique are similar to the palms, occasionally requiring a higher dose. Regional nerve block is generally required for anaesthesia and involves the posterior tibial and aural nerves. Duration of effect is 4-6 months.
Facial hyperhidrosis [Haider A, Solish N. *CMAJ* 172(1):69-75 (2005 Jan 4).]	Clinically significant improvements seen		Forehead is most common affected area with the main site of injection being a band along the hairline, extending into the temporal scalp. Can also involve the upper lip, nasolabial folds, malar regions. Duration of effect is 5-6 months.

Table 6: Review of BTX-A treatment to focal areas.

Skin Conditions and the Sun

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The skin has evolved to protect us from the harmful effects of ultraviolet light. Sunscreens were first developed to prevent sunburns by blocking UVB; they allowed us to prolong our time in the sun, but that resulted in increased exposure to UVA. Most modern sunscreens attempt to block the whole spectrum of UV light, however not all so-called broad spectrum sunscreens protect skin from the whole range of UVA.

A Comparison on UVA and UVB

UVA and UVB light have different characteristics.

Comparisons	UVA (320-400nm)	UVB (290-320nm)
Levels	Levels are constant throughout the year	Amounts vary and increase in the summer, at noon, and on the equator
Penetration	Penetrates into the lower dermis	Most only penetrates the epidermis
Levels through glass	Penetrates glass	Does not go through glass
Other	95% of UVL is UVA	SPF of sunscreens only measures UVB blockage

Table 1: Comparison of UVA and UVB light

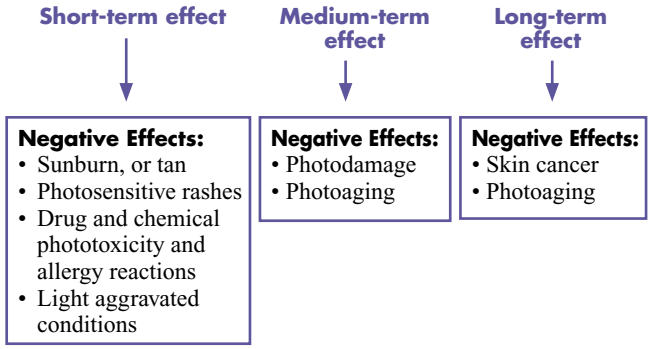
Effects on the Skin	UVA*	UVB
Carcinogenic level	May be important in causing melanoma	More carcinogenic than UVA
Changes to the skin	• Tans the skin • Causes most of the aging effects seen in the skin	Sunburns the skin
Systemic Effects	Immunosuppressive	Needed for Vitamin D production
Other effects	• Phototoxic reactions to drugs and chemicals • Responsible for many photodermatoses	

Table 2: Effects on the skin.

**It should be noted that sun-tanning beds use mainly UVA light. There is no such thing as a "safe" suntan. Recently the US FDA began investigating whether suntan beds should be illegal for anyone under the age of 18 years.*

Long-term Effects on the Skin

Most of us know that sun exposure has immediate positive and negative effects on the skin. The medium and longer term effects are negative. Positive effects include a sense of warmth, pleasure and Vitamin D production.



Photosensitive Rashes

These occur only when activated by UV light. Most of them are brought on by UVA. Photosensitive rashes (detailed below) can be thought of as:

1 – Idiopathic reactions to UV light (Polymorphous light eruption - PLE)

The timing of the onset of the rash in relation to sun exposure and its duration, as well as the type of reaction produced on the skin, is key to making the diagnosis. As always there is some variation.

2 – Phototoxic and photoallergic skin reactions

There are a number of drugs and chemicals that can produce a reaction in the skin. These can be either phototoxic or photoallergic reactions.

3 – UV aggravation of existing conditions

There are many pre-existing conditions that are aggravated by sunshine. Some of the important light aggravated conditions are:

- Rosacea
- Melasma
- Dermatomyositis
- Viral Exanthem
- Darier's disease
- Seborrehic dermatitis
- Discoid lupus
- Herpes simplex
- Pemphigus
- Porphyrias
- Systemic lupus erythematosus (SLE)

Polymorphous Light Eruption

Sun Exposure Causes

- Rash starts within hours of exposure and lasts for days even with no further sunshine. Solar urticaria is seen within minutes of exposure.
- Rash can be seen in the form of papules, papulovesicles or plaques, hence the term polymorphous.
- Plaques are less commonly seen.

Polymorphous light eruption (PLE)

- Mostly caused by UVA
- Occurs in early spring or summer, and often during vacation periods
- Is mostly symmetrical, red papules and plaques.
- Occurs in exposed areas but not necessarily all the exposed areas
- Occurs in 10%-20% of the population
- May be confused for allergic reaction to sunscreen
- Skin tends to be less reactive to sun as the summer progresses.
- The type of rash tends to remain constant for each patient.

Treatment of PLE

- Sun avoidance and protection with broad spectrum sunscreen (UVA and UVB)
- Topical steroids and antihistamines
- UVA and UVB light therapy may help some at the start of the season, which can harden the skin and prevent the reaction.
- Hydroxychloroquine, 400mg daily for 2 weeks in the spring or before a vacation may help

Photoxic vs. Photoallergic Reactions

Phototoxic drugs or chemicals

- Sunburn-like
- Usually seen within hours
- Usually caused by UVA

Includes tetracyclines, sulfa, amioderone, fursomide, naproxen, piroxicam, chlorpromazine, ciprofloxacin, thiazides

Photoallergic reactions

- An eczema like reaction. Can be thought of as a delayed hypersensitivity type reaction.
- Causes: Sunscreens, fragrances/aftershave (like musk ambrette, sandalwood oil), chlorhexidine

Phytophotodermatitis

- A special type of reaction to topical contact with a sensitizer called psoralen contained in a number of plants.
- UVA plus psoralen will produce a blistering reaction often seen in streaks; a brown pigmentation is produced which may last for months.
- Plants containing psoralen are responsible including lime, yarrow, cow parsley, celery, lemon, fig

Photoprotection

Should be encouraged to prevent the immediate, medium and long-term ill effects of excessive sun exposure. Some sun exposure is desirable for vitamin D production.

Two ways to encourage photoprotection:

1. Sun avoidance

- Avoid the sun between 10am and 3pm.
- Try to stay in the shade.
- Wear protective, tightly woven clothes and a broad brimmed hat.

2. Sun protection

- Use a Broad-spectrum sunscreen in a sufficient quantity.
- SPF = the ratio of minimal erythema dose (MED) of protected skin/MED of unprotected skin. This is a crude biological measure.
- The SPF factor is calculated using 2mg/cm^2 of sunscreen. Most people apply only 25-50% of this.
- Reapply sunscreen every 2 hours; UVL causes some chemical sunscreens to become inactive over time.

Sunbed Tan

- Will not give as much protection as a tan produced by the sun.
- The UVB component of a natural tan produces a protective thickening of the epidermis, though the SPF protection is only about 2-6. This protection not found in a UVA tan.

Self Tanners

- Very popular, producing good, even colour.
- Contain dihydroxyacetone (DHA); reacts with amino acids containing keratin. DHA concentration varies from 2%-6%; higher numbers give a darker colour.
- DHA has an SPF of 2%-3%. Some have a low SPF screen added that lasts only a few hours.
- Coloured skin does not provide protection against photodamage.
- Bronzers are dyes that are added to the skin; can be washed off.
- Beta-carotene, tyrosine, tanning accelerators such as psoralen are not recommended.

Sunscreen Use

- Broad spectrum only should be used.
- SPF related to UVB protection only; does not provide a reference to UVA protection.
- All sunscreens have UVB protection; reflected in the SPF.
- If skin sunburns in 10 minutes, properly applied sunscreen at SPF 15 means skin will burn in 150 minutes.
- Physical screens reflect light; chemical screens absorb UV, converting energy into heat
- SPF15 blocks 87.5% of UVB and SPF 50 blocks 98% of UVB.

Sunscreen Choices

Sunscreen with full spectrum UVA protection contains:
Avobenzone (Parsol 1789), Mexoryl Sx, and Zinc oxide working together. The first two have slightly different peaks of protection. Titanium Dioxide, Dioxybenzone Methyl anthranilate and Octocrylene provide UVA protection, but not along the whole spectrum. Some recommended general sunscreens: Ombrelle® 30, 45, 60, cream and lotion. (This broad spectrum sunscreen was pioneered in Canada); Anthelios® 30, 45, 60; Neutrogena® Heathy Defense Sunblocks 30, 45 with parsol.

Special sunscreens:
Lip protection: SCC is more commonly seen in men and women who don't wear lipstick: Ombrelle® Lip Balm SPF30; RoC Minesol® Lipstick SPF 20; Neutrogena Stick 30; Antherpos® SPF 50.
For joggers these can also be used above the eyebrows to prevent the screen from entering the eyes. Can also be used on the nose.

Spray for athletes, or for people with hairy or oily skin: Ombrelle® Sport Spray 15; Coppertone® Sport 15 and 30; Neutrogena® Healthy Defense Spray 30.

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