BEST PRACTICE

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SEPTEMBER 2009

Memory Loss

Skin:

- Topical corticosteroids
- Eczema
- Psoriasis

Cervical Screening

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better medicine

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Waste Not, Want Not

Medication wastage

www.bpac.org.nz keyword: medicinewaste

Medication wastage is estimated to cost billions of dollars per annum worldwide.[1] Factors such as poor compliance, discontinuation of medication, adverse effects and dose changes have led to an ongoing issue of unused or expired medicines being hoarded in some households.[2]

Clearly we need to consider solutions to this problem, but care needs to be taken that any intervention reaps more benefit than the cost of the wastage itself. Throwing out perfectly good medicine seems wrong, but is the alternative more costly? One argument for this is stat dispensing, where the likelihood of the medicine being wasted may be offset by the saved dispensing fee, which can then be used to create health gain elsewhere.

In a recent survey of 452 individuals across New Zealand, 56% reported that they collected all of their prescribed medications from a pharmacy, even if they did not intend to take them. Just over 25% said they collect all of their medication prescription repeats, even if the medications are no longer needed. Over 60% of respondents indicated that there were leftover, or unwanted prescription medications present in their house, at the time of completing the questionnaire.[3]

Investigations into returns of unused medication to community pharmacies in Otago have highlighted the potential significance and volume of these unused medications.[4, 5] One individual return had over 70 different medications, which included cardiovascular, nervous system, musculoskeletal, diabetic and infection medications totalling over $14,500.[4] Another individual returned items worth only $350 but this included 1557 paracetamol/codeine tablets, 1198 paracetamol tablets, 468 doxepin capsules, 362 warfarin tablets and seven 100 g hydrocortisone-17-butyrate creams.[5]

Larger studies have been conducted in Taranaki and Hutt Valley where it was found that inhalers accounted for 20% of the total cost of returned medications, a large proportion of which (69%) were preventer inhalers (unpublished data).

International studies have shown:

- 65% of returned items to pharmacy contained greater than 65% of the original content.[6]
- 66% of returned items to a pharmacy were medications that had been dispensed for greater than a one month period.[7]

Treatment change and bereavement are the most commonly reported reasons for returning medications.[4,7–9] Other reasons include; medicines no longer needed or expired, adverse drug reactions and oversupply.[4, 7–9] Approximately 50% of patients will discontinue using their medications within a few months for reasons which include; forgetting to follow the dosing instructions, adverse effects, inefficacy or condition resolving.[10] Resentment about the need for treatment and secondary gain from persistent symptoms (i.e. sympathy, benefits) may be factors in non-adherence to treatment in some cases.[11]

How can medicine wastage be addressed?

A collaborative approach to reducing medicine wastage is needed. Patient education should focus on addressing the reasons why medicines are wasted in the first place. Amnesties for returning medicines and established collection processes in pharmacies are good ideas, but they only address correct disposal of medicines, rather than reducing the amount that is unused.

At an individual level, medicine wastage should be addressed before it begins. Ask patients regularly if they are using the medicines you are prescribing. Communication skills are important as many patients may be reluctant to confess that they have a stock pile. Ask open questions rather than make assumptions.

Prescribing tips to reduce medicine wastage:

- Treatment change is one of the most common reasons for unused medications. Changes often occur in the early phase of treatment,[8] therefore it may be prudent to prescribe a smaller initial amount of medication or “close control” for the first month of a three month prescription, if it is anticipated that the dose may need to be changed.
- The large number of “as required” medications being returned[4, 9] may indicate oversupply. Specifying an appropriate quantity may reduce wastage and allow better monitoring of the condition.
- There is often a temptation to “give the patient a good deal” and prescribe a bulk amount of medication, but this must be weighed up against the cost to the tax payer and healthcare system. Having a large amount of medications stockpiled is a safety concern and may also create confusion about what is supposed to be taken.

Approximately 50% of patients will discontinue using their medications within a few months

How to correctly dispose of unwanted medicines

There is currently no mechanism for re-using returned medication that is unexpired and in original packaging. The main reason for this is that it cannot be guaranteed that optimal storage of the medicine occurred. The following advice can be given to patients:

- Do return unwanted medicines to a community pharmacy
- Do keep medicines in original containers and packaging (so they are not mistaken for anything else)

- Don't flush
- Don't pour down the sink
- Don't throw in the rubbish
- Don't give to other people

For example; what tablets are you taking, when and how often? Do you know what they are for? Are you experiencing any adverse effects? Do you want to carry on with your current medications?

Check at appropriate times that the medication regimen is clinically appropriate. Ideally this should be a consultation without any other agenda but this often is not realistic. One way to address this is to allow only a certain number of phone repeats for a medication. For example, after every third telephone medication repeat, encourage the patient to attend a consultation for a medicine review. This may also provide a chance to discuss preventative health care.

Acknowledgements:
Thank you to Dr Rhiannon Braund, Lecturer, School of Pharmacy, University of Otago, for contribution to this article.

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CVD Screening

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(National Heart Foundation of New Zealand)

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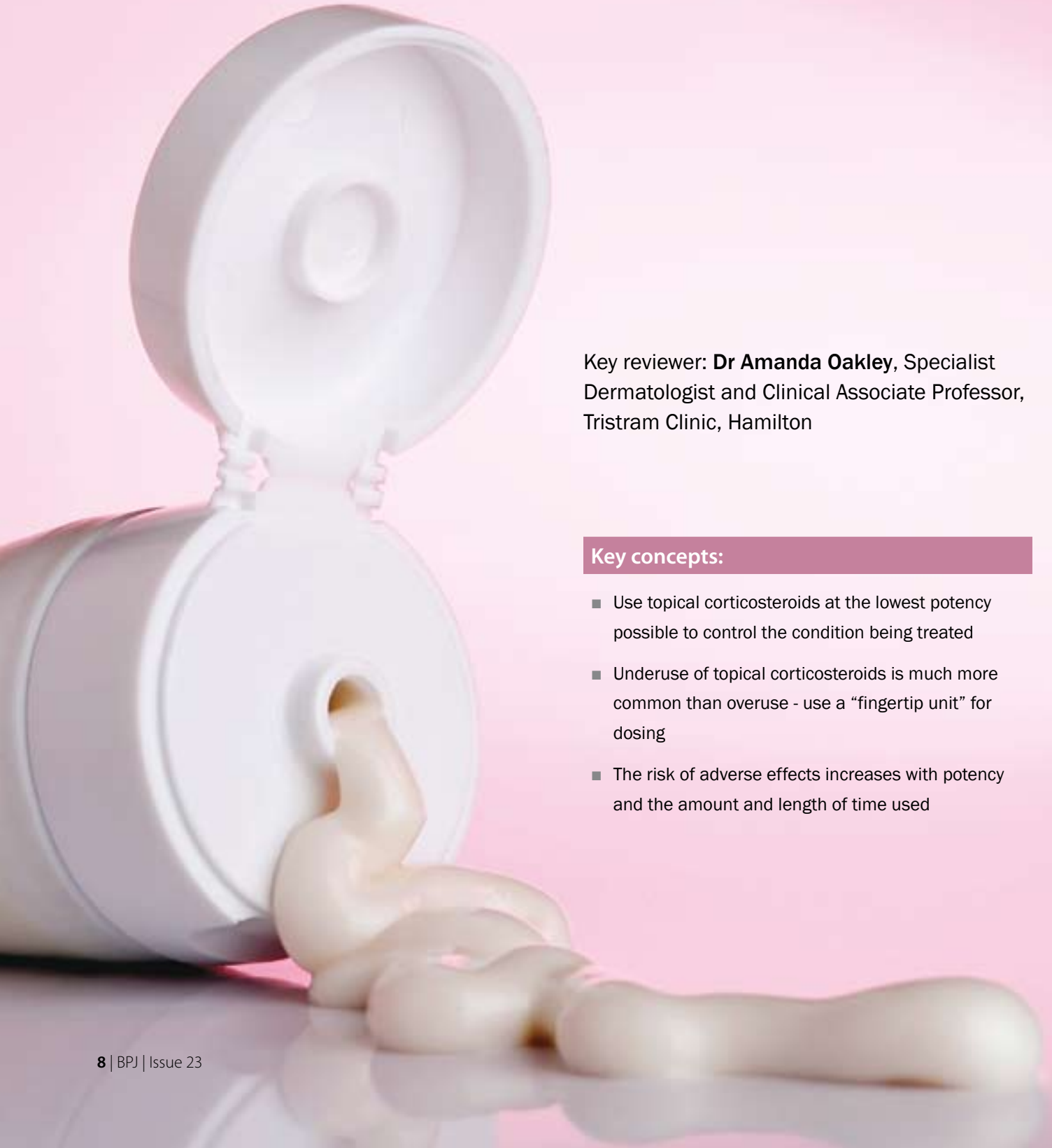
Topical corticosteroid treatment for skin conditions



Key reviewer: **Dr Amanda Oakley**, Specialist Dermatologist and Clinical Associate Professor, Tristram Clinic, Hamilton

Key concepts:

- Use topical corticosteroids at the lowest potency possible to control the condition being treated
- Underuse of topical corticosteroids is much more common than overuse - use a “fingertip unit” for dosing
- The risk of adverse effects increases with potency and the amount and length of time used

Topical corticosteroids are used for many skin conditions (Table 1). They suppress the inflammatory reaction and relieve symptoms however they are not curative and when they are discontinued symptoms can recur.[1]

Topical corticosteroids should not be used for rosacea or acne vulgaris. They may worsen ulcerated or secondarily infected lesions.[1]

Which potency do I choose?

In general use topical corticosteroids at the lowest potency possible to control the condition.

Low potency corticosteroids are typically used when treating large areas or for longer term application. They are also more suitable for use on children or areas of thinner skin such as the face, groin or axilla.[2]

More potent corticosteroids are suitable for severe conditions and for use on areas of the body that have thicker skin such as the palms of the hands and soles of the feet. They should generally not be used under occlusion or on areas of thinner skin.[2]

Occlusion increases the absorption of topical corticosteroids by increasing the hydration of the skin and therefore enhancing penetration. This needs to be considered when selecting corticosteroid potency. Occlusive materials include polyethylene gloves, plastic film (e.g. Gladwrap) or occlusive dressings.[3] Irritation, folliculitis and infection are more likely to occur under occlusion.[2] Corticosteroid related adverse effects are also more likely, especially if occlusion is prolonged, because of increased absorption.

Which formulation do I choose?

Choice of formulation depends on a number of factors, including the type of skin lesion to be treated and its location. Patient preference is important to consider because it can affect compliance.

Table 1: Conditions that may respond to topical corticosteroids[2]

Moderate - high potency corticosteroids	Low - moderate potency corticosteroids
Alopecia areata	Perianal inflammation (severe)
Atopic dermatitis	Asteatotic eczema
Contact dermatitis (severe)	Atopic dermatitis
Discoid lupus	Dry nummular (discoid) eczema
Hyperkeratotic eczema	Intertrigo (short-term)
Lichen planus	Scabies (after scabicide)
Lichen sclerosus	Seborrhoeic dermatitis
Lichen simplex chronicus	**Low potency corticosteroids**
Exudative nummular (discoid) eczema	Dermatitis (face, eyelids, napkin area)
Psoriasis	Intertrigo
Severe hand eczema	Perianal inflammation
Stasis dermatitis	

Ointments are greasy and remain on the skin after they are applied. They are particularly suitable for use on dry, thick or lichenified skin. The potency of a corticosteroid can be affected by its formulation e.g. for any given strength of corticosteroid, an ointment formulation will be more potent than a cream. This is because the occlusive nature of an ointment enhances absorption of the corticosteroid.[2]

Creams are often preferred by patients especially for use on exposed areas such as the face because they vanish when rubbed into the skin.[1, 4] They are suitable for moist or weeping lesions. Creams contain more preservatives and excipients than ointments and so are more likely to cause hypersensitivity or irritation.[4]

Lotions have a thin consistency, making them easier to apply to hairy areas such as the scalp.[1] They contain alcohol and have a drying effect on exudative lesions, but may sting on application.

How much do I prescribe?

Underuse of topical corticosteroids is much more common than overuse. An acute or severe condition that is likely to respond to topical corticosteroids should be treated generously, aiming to get control promptly.

Regularly review patients with chronic skin conditions e.g. atopic dermatitis, to monitor use of topical corticosteroids – they may be using excessive potency or frequency or quantities where a milder preparation or an emollient would be more suitable. Conversely, the patient may be underusing topical corticosteroids because of perceived lack of efficacy, or fear of adverse effects – generous intermittent applications of potent products should be encouraged e.g., "weekend pulse therapy".

Dose using fingertip units

A fingertip unit is a guide to how much corticosteroid to apply to a particular area and describes the amount of product squeezed onto the top third of the finger (see Figure 1).[5]

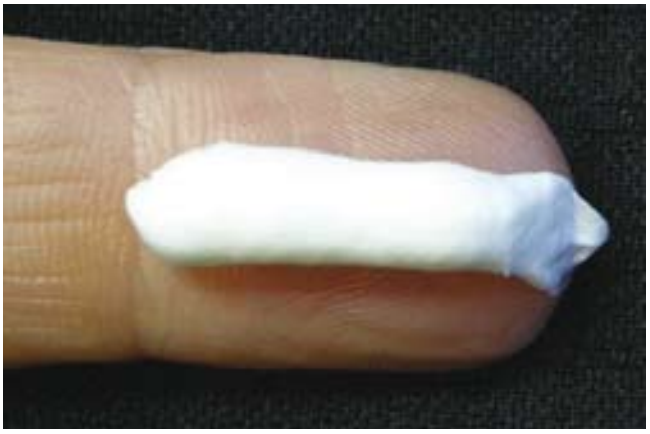


Figure 1: Fingertip unit. Picture supplied by DermNet NZ.

Table 2: Number of fingertip units per body part [5]

Body part	Fingertip units	Approximate quantity to prescribe*
One hand	1	15 g
One arm	3	30 g
One foot	2	15 g
One leg	6	50 g
Face and neck	2.5	30 g
Trunk, front and back	14	100 g
Entire body	~ 40	300 g

* for single daily application for an adult for two weeks

One fingertip unit is equivalent to approximately 0.5 g for a male and 0.4 g for a female. Infants and children should use one quarter to one third of the adult amount.

The number of fingertip units needed varies with the area of skin requiring treatment (Table 2 and Figure 2).

The amount of cream required daily can be used to calculate the correct amount needed on a prescription.

For example: If an adult female applies cream to both arms and hands once daily, she will need 3.2 g per day (i.e. eight fingertip units × 0.4 g = 3.2 g per day) and 22.4 g per week. A 50 g tube should last approximately two weeks, but if she applies it twice daily it will be finished in approximately one week.

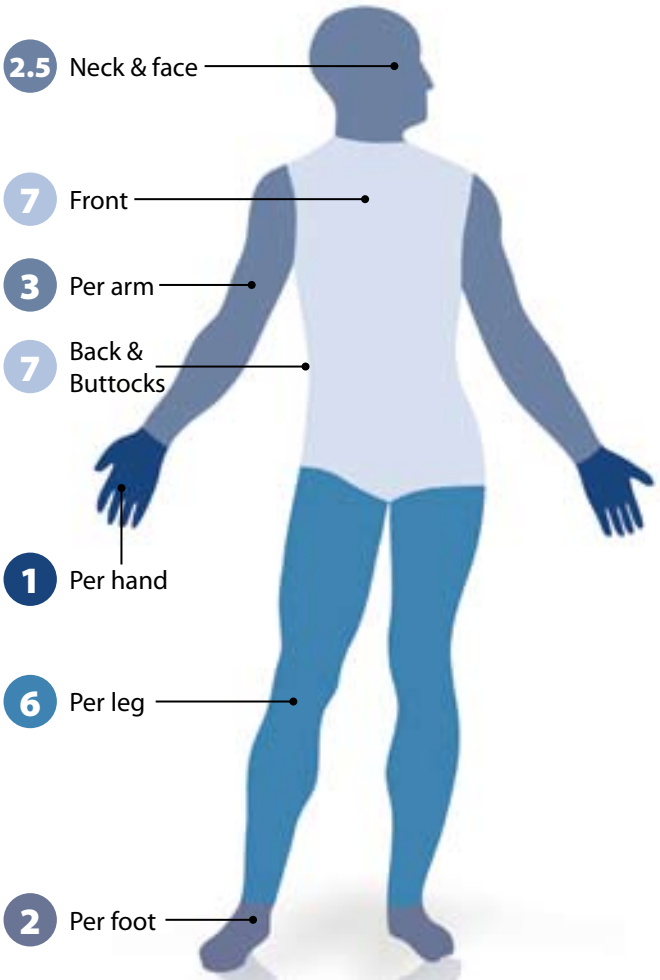


Figure 2: Fingertip units for different areas of the body

How often should topical corticosteroids be applied?

Application of topical corticosteroids is usually recommended once or twice daily depending on the condititon being treated.[6] For the treatment of atopic eczema, applying topical corticosteroids more often than once daily has not been shown to produce significantly better results and may adversely affect patient compliance.[7]

Best practice tip: Prescribe moderate, potent or very potent topical corticosteroids for once daily use only.

How long can topical corticosteroids be used?

Long term use of topical corticosteroids can induce tachyphylaxis (tolerance to the vasoconstrictive action of topical corticosteroids). Adverse effects are uncommon when using mild to potent corticosteroids for less than three months, except when used on the face and neck, in intertriginous areas (skin folds), or under occlusion. However, very potent corticosteroids should not be used continuously for longer than three weeks.[2] If longer use of very potent corticosteroids is required, they should be gradually tapered to avoid rebound symptoms and then stopped for a period of at least one week after which treatment can be resumed.[2]

Should topical corticosteroids be applied before or after emollients?

There are a lack of controlled studies investigating the best order of application of topical corticosteroids and emollients.

The NICE guideline[6] for managing eczema in children advises that if both are applied, an interval of several minutes should be left between the application of a topical corticosteroid and an emollient. Which to use first is debatable – however one guideline suggests that emollients should be applied first because:[8]

- Topical drugs may be more effective when used after emollients
- Corticosteroids may be diluted or transferred to areas that do not require treatment if emollients are applied immediately on top of them

What adverse effects are likely to occur?

The risk of adverse effects increases with potency, the amount of topical corticosteroids used and occlusion. While systemic adverse effects are rare, local adverse effects are more common and include skin atrophy, telangiectasia (especially on the cheeks), acne and corticosteroid or preservative-induced contact dermatitis.[9]

Systemic adverse effects, including adrenal suppression, growth retardation, Cushing's syndrome and hypertension may occur with the use of topical corticosteroids but are

Topical corticosteroids available in New Zealand (prescription only) [10]

Potency class	Products	Plain formulations	Combination formulations
Low	Hydrocortisone (1%)	Hydrocortisone BP cream (PSM) S Lemnis Fatty Cream HC S DP Lotion-HC 1% (+ wool fat, mineral oil) S	Micreme H (+ miconazole) S Pimafucort cream/ ointment (+ neomycin, natamycin) S
Moderate (2–25 times as potent as hydrocortisone)	Clobetasone butyrate (0.05%)	Eumovate cream PS	
	Triamcinolone acetonide (0.02%)	Aristocort cream/ointment S	
Potent (50–100 times as potent as hydrocortisone)	Betamethasone valerate (0.1%)	Beta cream/ointment/ scalp application S Betnovate lotion S	Betnovate C ointment/ cream (+ clioquinol) PS Fucicort (+ fusidic acid 2%) PS
	Betamethasone dipropionate (0.05%)	Diprosone cream/ointment PS Diprosone OV cream/ ointment PS	
	Diflucortolone valerate (0.1%)	Nerisone cream/ointment/ fatty ointment PS	Nerisone C cream (+ chlorquinaldol 1%) NS
	Hydrocortisone 17-butyrate (0.1%)	Locoid cream/lipocream/ ointment/scalp lotion/ crelo topical emulsion S	Locoid C (+chlorquinaldol 3%) S
	Mometasone furoate (0.1%)	Elocon cream/lotion/ ointment S	
	Methylprednisolone aceponate (0.1%)	Advantan cream/ointment S	
	Triamcinolone acetonide (0.1%)		Viaderm KC cream/ ointment (+ neomycin, nystatin, gramicidin) PS
Very potent (up to 600 times as potent as hydrocortisone)	Clobetasol propionate (0.05%)	Dermol cream/ointment S	

Key: S fully subsidised; PS partially subsidised; NS not subsidised

N.B. 0.5%–1% (restricted to ≤ 30 g) hydrocortisone products are available over-the-counter.

very rare. Cataracts and glaucoma have been reported in some cases when topical corticosteroids, including hydrocortisone, have been used for long periods in the periorbital area.[3]

Adverse effects are more likely when topical corticosteroids are used:

- In infants and children
- For prolonged periods
- At high-potency
- Over large areas and under occlusion

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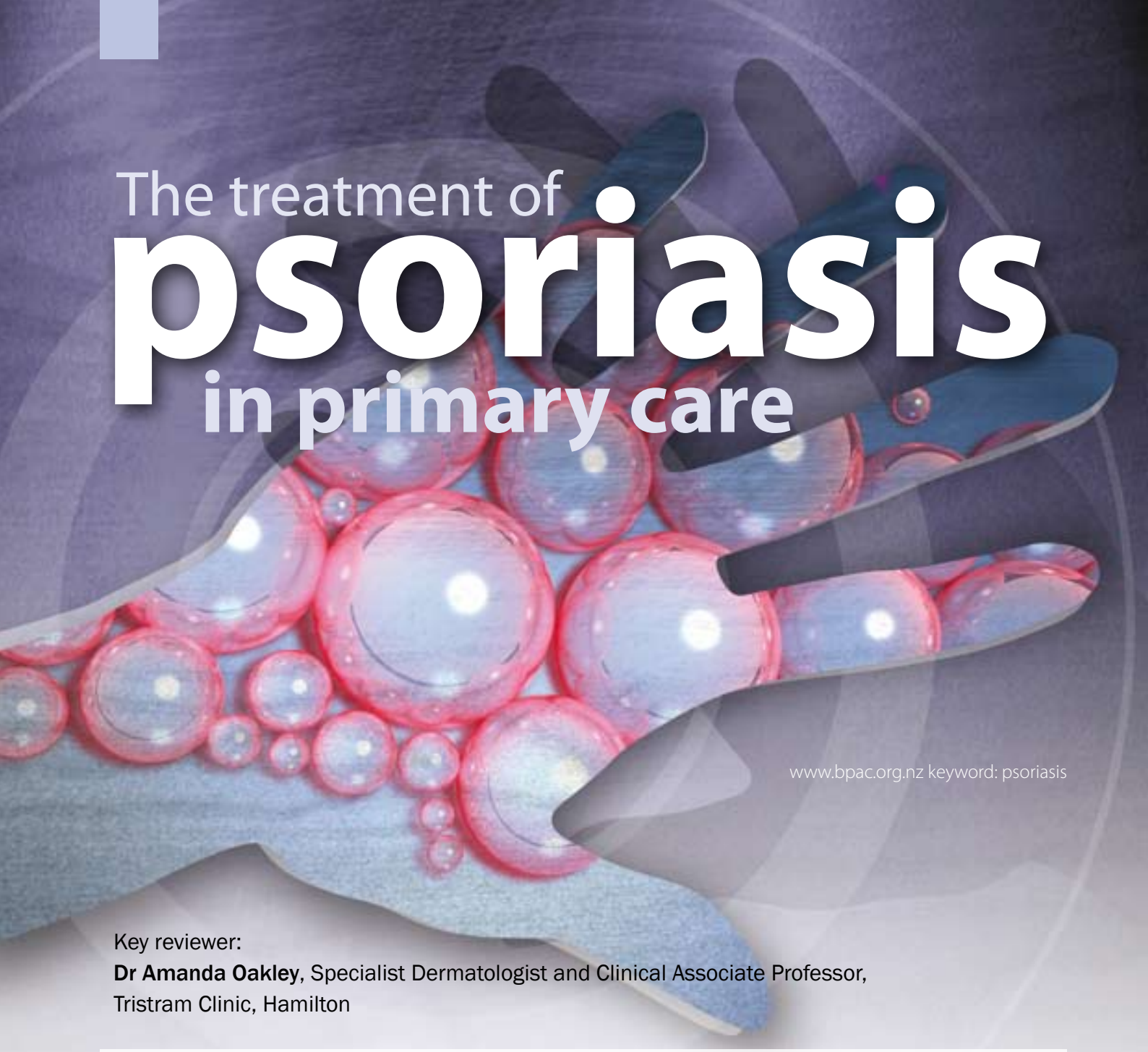
The treatment of psoriasis in primary care



www.bpac.org.nz keyword: psoriasis

Key reviewer:
Dr Amanda Oakley, Specialist Dermatologist and Clinical Associate Professor, Tristram Clinic, Hamilton

Key concepts

- Psoriasis is an emotionally and physically debilitating disease which can significantly affect quality of life
- Psoriasis is aggravated by factors including smoking, high alcohol intake, high glycaemic diet, certain medications (particularly lithium) and emotional stress
- Management includes support with the psychosocial aspects of the disease and advice on lifestyle interventions
- Topical treatments are the first choice for mild psoriasis, and are also used adjunctively for resistant lesions in patients with more extensive disease
- Calcipotriol and intermittent potent topical corticosteroids are prescribed for the majority of patients
- Systemic treatments are used for severe disease and are usually initiated by specialists

Psoriasis is an immune-mediated chronic scaly skin disorder in which there is upregulation of protein expression resulting in excessive keratinocytic proliferation, abnormal keratinocyte differentiation and inflammation. The pathogenesis involves Tumour Necrosis Factor (TNF)-α, dendritic cells and T cells. Normally keratinocytes require 28 – 44 days to migrate from the basal cell layer of the epidermis to the stratum corneum, but in people with psoriasis this migration takes only four days. Excessive cornification hardens the surface of the thickened plaques, which are red because of prominent vascularity.

Psoriasis is one of the most prevalent autoimmune diseases affecting both men and women equally. Approximately 2–3% of the New Zealand population are affected. Psoriasis most often appears in the late teens or in the 50s, but can develop at any age. There is a genetic predisposition and approximately one third of people with psoriasis report having an affected relative.[1,2]

It is an emotionally and physically debilitating disease which can have a significant impact on quality of life and can lead to depressive illness. Up to 25% of patients with psoriasis may also have seronegative arthritis.[2] It is associated with diabetes mellitus and people with moderate and severe psoriasis have a three-fold increased risk of cardiovascular disease.[3] Aggravating factors include smoking, high alcohol intake, high glycaemic diet, certain medications (e.g. lithium, beta-blockers) and emotional stress.[4]

DIAGNOSIS AND MONITORING

Psoriasis is diagnosed clinically, occasionally with the support of histology. There are no specific blood tests to diagnose or monitor psoriasis. Cardiovascular risk should be monitored due to the association between cardiovascular disease and psoriasis.[3]

There are several different types of psoriasis

There are several different types of psoriasis (Box 1, see over page). It may be widespread or localised to one part of the body, such as the scalp or fingernails. Psoriasis affecting the face, hands and feet may be particularly debilitating.

One way to assess severity depends on how much of the body surface is affected (see box below). Clinical trials may use the more complex Psoriasis Area and Severity Index (PASI) score to assess effectiveness of treatment.[4]

Assessing Severity

Severity can be defined by how much of the body surface area is affected:[5]

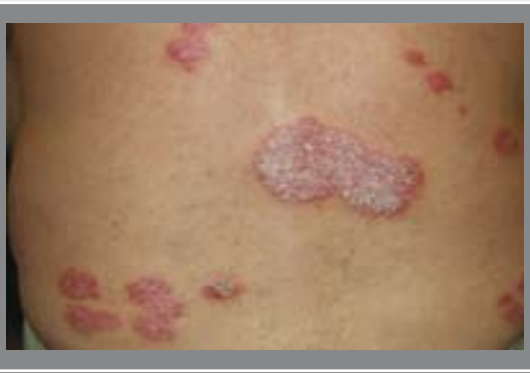
- **Mild psoriasis:** < 5% of the body surface area
- **Moderate psoriasis:** 5–10% of the body surface area
- **Severe psoriasis:** >10% of the body surface area

Quality of life is also taken into consideration when assessing severity.

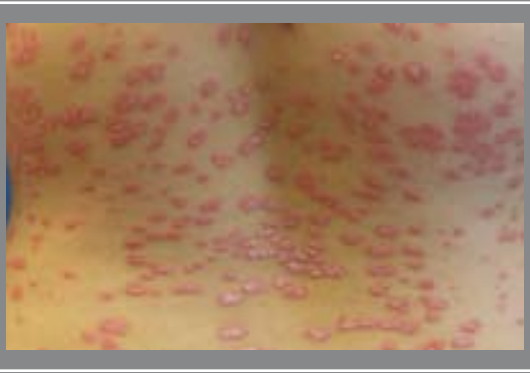
Best practice tip: To estimate the percentage of body surface area affected by psoriasis, consider that the palm of your hand (excluding fingers) is approximately equal to 1% of your body surface area.

Box 1: The main clinical types of psoriasis[4]

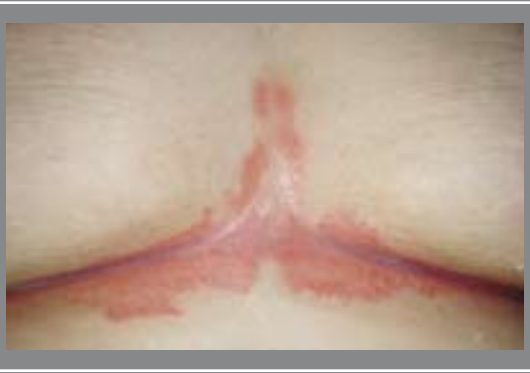
Plaque psoriasis (psoriasis vulgaris) – most prevalent form (~ 80%), thin to thick red plaques covered with a silvery scale, typically found on the elbows, knees, scalp and lower back but may occur anywhere. Most (~ 80%) people affected with plaque psoriasis have mild to moderate disease.



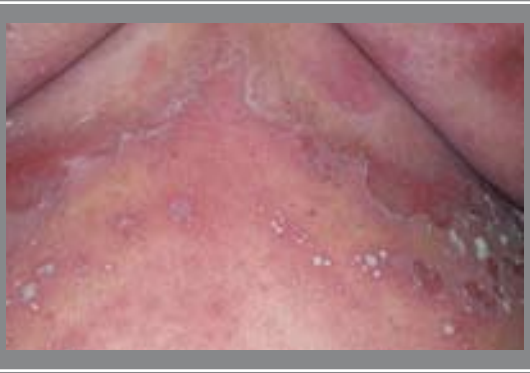
Guttate psoriasis – 5 to 10 mm red plaques on the trunk and limbs, often appearing suddenly after a Streptococcal or viral upper respiratory tract infection.



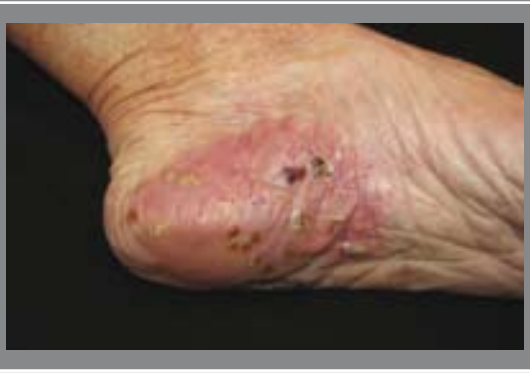
Flexural psoriasis – bright red smooth shiny skin lesions found in skin folds: axillae, groin, under breasts and around genitals and buttocks.



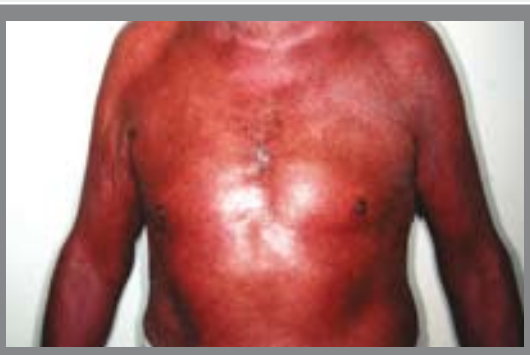
Generalised pustular psoriasis – a severe acute illness characterised by fever associated with crops of sterile pustules arising in or around painful red skin. It can be triggered by systemic or potent topical steroids and other medications, over-exposure to UV light, infections and stress.



Palmoplantar pustulosis – sterile pustules arising on palms and soles strongly associated with smoking (95%). Chronic plaque psoriasis is associated in 25% of cases.



Erythrodermic psoriasis – a particularly inflammatory form of psoriasis that affects most of or the entire body surface and can lead to hypothermia, hypoalbuminaemia and cardiac failure. Severe cases require hospitalisation. Known triggers include the abrupt withdrawal of a systemic psoriasis treatment, allergic reactions, over-exposure to UV light, infection and some medications (e.g lithium, antimalarials).



Nail psoriasis – pitting, onycholysis, subungual hyperkeratosis and other forms of nail dystrophy affect about 50% of patients with other forms of psoriasis.

Pictures supplied by DermNet NZ

MANAGEMENT

Educating the patient about the chronic nature of psoriasis and possible co-morbidities is important. Reassure them that it is not contagious and that treatment can help. The patient may require support to cope with the psychosocial aspects of the disease. Some patients may choose not to treat the skin disease itself as treatments can be burdensome and associated with adverse effects.

Provide general advice regarding the benefits of not smoking, maintaining optimal weight and avoiding excessive alcohol. Although sun exposure is often helpful, fair skinned people should be cautioned regarding the risk of sunburn and long term overexposure, leading to aging of the skin and skin cancer.

Treatment has to be individualised. It will vary depending on the characteristics of the psoriasis being treated: its body location, thickness of lesions, degree of erythema and scale, as well as patient preference or commitment to therapy.

Topical treatments are the first choice for mild psoriasis, and are also used adjunctively for resistant lesions in patients with more extensive disease (who are being treated with phototherapy and/or systemic medications).

Treatments most commonly initiated in general practice include:

- Topical skin therapies:
  - Corticosteroids
  - Calcipotriol
  - Dithranol
  - Coal tar/pine tar (see page 22 for a list of products)
- Scalp treatment
- Nail treatment

TOPICAL SKIN THERAPIES

Regular use of a thick emollient such as fatty cream is helpful to prevent cracking and irritation. Emollients may be supplemented by keratolytic or "descaling" agents, such as 10% urea cream or 2% salicylic acid in white soft paraffin.

Coal tar or pine tar emollient solutions in bathwater may be soothing, reduce itch and allow gentle removal of scale.

Corticosteroids

Topical corticosteroids are used by the majority of people with psoriasis, particularly those with limited disease. Corticosteroids have anti-inflammatory, immunosuppressive and antiproliferative properties.

Lower potency corticosteroids should generally be used for limited periods of time on the face and other areas of thin skin and in infants. Pimecrolimus is an alternative anti-inflammatory cream for psoriasis affecting the face or genitals, but is not effective on other sites where plaques are thick.

In other areas and in adults moderate or high-potency corticosteroids are generally recommended as initial therapy. Thick chronic plaques and plaques on hands and feet may require treatment with the highest potency agents (for a maximum of three weeks). Ointments are the best choice for dry, scaly plaques.

One of the drawbacks of corticosteroid therapy is associated tachyphylaxis, leading to decreased efficacy with continued use, and sometimes resulting in an acute flare-up when therapy is stopped. Once the lesions have improved, these effects can be minimised by switching patients to less potent formulations, or advising them to apply the medication less frequently, i.e. for two to three consecutive days each week. Another strategy is the use of corticosteroid-free times.

Calcipotriol ointment/cream/scalp solution

Calcipotriol (Daivonex, fully subsidised) is a vitamin D analogue that acts mainly by reducing the proliferation of keratinocytes. Treated areas become less scaly but may remain red.

It is not recommended for severe extensive psoriasis unless calcium status is carefully monitored, because of the risk of hypercalcaemia secondary to excessive absorption of calcipotriol.[6]

Calcipotriol cream or ointment (50 mcg/g) can be applied topically to chronic plaques twice daily. It may be reduced to once daily when the condition improves, however it is most effective if applied consistently twice daily.

Calcipotriol is not as well absorbed from the cream formulation as the ointment, so the cream is only used if there is a strong patient preference.

Safety of calcipotriol.[6] The most common adverse effect is localised skin irritation, which may lessen with continued use.

Calcipotriol is not usually recommended for use on the face because it may cause itching and erythema of the facial skin. However some patients are able to use the cream formulation successfully on the face. Irritation may be reduced with hydrocortisone cream. When patients are applying calcipotriol to other parts of their body, they should be advised to wash their hands after application, to avoid inadvertent transfer to the face.

Hypercalcaemia has been reported rarely at the recommended dose, however serum calcium and renal function should be monitored at three monthly intervals, if patients are applying larger quantities. To avoid the risk of hypercalcaemia, calcipotriol should not be used concurrently with calcium or vitamin D supplements.

Calcipotriol total dose should not exceed 5 mg/week, for example:

- 100 g of ointment or cream
- 60 mL of scalp solution plus one 30 g tube of cream or ointment
- 30 mL of scalp solution plus two 30 g tubes of cream or ointment

Direct sunlight and UV radiation may inactivate calcipotriol, so it is best applied after exposure.

Note that calcipotriol is poisonous to dogs.

Calcipotriol use in children. Calcipotriol (50 mcg/g) can be used in children aged six years and over but the datasheet indicates it should not be used for more than eight weeks.[6]

For children aged 6–12 years the maximum weekly dose should not exceed 50 g, while for children over 12 years the maximum weekly dose is 75 g.

Combined use of calcipotriol and other psoriasis treatments. When using multiple topical treatments, instruct patients to apply them at separate times. For example a topical corticosteroid may be used in the morning and calcipotriol used in the evening or calcipotriol may be used twice daily on weekdays, and the topical corticosteroid twice daily at weekends.[4, 5]

Calcipotriol should not be used at the same time as topical salicylic acid because it is inactivated by it.[4]

Dithranol[7]

Dithranol (Micanol cream, fully subsidised) belongs to the family of hydroxyanthrones which have been used in the treatment of psoriasis for more than a century. In the archaic Ingram regimen, a thick paste is applied to large plaques twice daily, under carefully applied dressings. It is then removed in a tar bath and the patient exposed to UV radiation. This regimen is too difficult for home use as dithranol is very irritating to normal skin and causes permanent stains on clothing and bathtubs.

A short-contact regimen may be suitable for well motivated patients with small numbers of large plaques of psoriasis.[4] Dithranol 1% cream can be applied once daily to the plaques, rubbed in gently until it no longer smears, and rinsed off (water only) after 10 minutes. The application time can be increased gradually over seven days to a maximum of 30 minutes.

It should not be applied to areas of thin skin (such as face, genitalia, intertriginous areas) and should be avoided in pregnancy, lactation and in children. Patients should be reminded that it can cause staining of skin, hair, fingernails (gloves are recommended when applying it), clothing and bed linen. If redness or burning occurs the treatment should be reduced or stopped.

Coal tar/pine tar

It is not well understood how coal or pine tar works for psoriasis, but it appears to have an anti-pruritic nature and is keratoplastic – i.e. it normalises keratin growth in the skin to reduce scale build up.[4]

Coal and pine tar are used mainly as a bath additive or scalp application. Coal or pine tar preparations or Egopsoryl TA gel (unsubsidised) can be directly applied to plaques. Coal or pine tar is often combined with salicylic acid 2 – 4% as a keratolytic. See page 22 for a list of products.

Best practice tip: LPC (coal tar solution) and/or salicylic acid may be prescribed in an emulsifying ointment base for chronic plaques on the trunk or limbs or in aqueous cream for application to the scalp. For example, a prescription may be written as: "LPC 10%, salicylic acid 2% in aqueous cream ad 100%. 200 g".

Scalp treatments

Ketoconazole (2% shampoo) may help some patients with diffuse mild scalp psoriasis, although it is more effective in seborrhoeic dermatitis.[4] It is used twice weekly and is best left on the scalp for several minutes before rinsing off.

Coal tar/pine tar shampoos (e.g. Neutrogena T/Gel, Polytar Plus, Fongitar, Ionil-T, Sebitar) are unsubsidised but generally more effective than ketoconazole shampoos. They are used one to two times per week (or as necessary) and are best left on the scalp for several minutes before rinsing off.

Coco-scalp (fully subsidised) is an ointment made from coal tar, sulphur, salicylic acid and coconut oil that can be applied to scaly plaques and left for a minimum of one hour before washing off. It can safely be occluded (e.g. wrapping the hair in plastic cling film) for several hours to achieve better de-scaling results.

Alternatively, in patients with coal tar intolerance or aversion, try salicylic acid 2–4% in aqueous cream, washed out after several hours.

Dithranol 0.1% can be applied to the scalp, after the hair has been washed to remove any grease and while the hair is still damp, and rinsed off (water only) after 30 minutes. It is unsuitable for blonde or grey hair as it may stain.

Corticosteroid scalp applications include (in increasing potency):

- Hydrocortisone-17-butyrate 0.1% (Locoid Scalp Lotion or Crelo)
- Betamethasone valerate 0.1% (Beta Scalp application)
- Mometasone furoate 0.1% (Elocon Lotion)
- Clobetasol propionate 0.05% (Dermol Scalp application)

Topical corticosteroids are applied to the scalp once or twice daily for short courses up to one month in duration, and then two to three days each week for maintenance if required. Overuse may cause psoriasis to worsen. Topical corticosteroids are particularly useful to reduce pruritus although alcohol-based lotions may sting on application and they are ineffective through thick scale.

Calcipotriol scalp solution (50 mcg/mL) can be applied topically to the scalp twice daily, reducing the frequency when improvement occurs. Dose of the solution should not exceed 60 mL per week. If cosmetically acceptable to the patient, calcipotriol in a cream base may be more effective than the solution.

Nail treatments

Treatment of nail psoriasis is often ineffective.[4] Topical scalp preparations (corticosteroids and calcipotriol) can be dripped or rubbed under affected nails and rubbed into the proximal nail fold. The patient should be advised that it may take months or longer for results as nails grow slowly (e.g. 1 mm per month).

SPECIALIST REFERRAL

Referral for phototherapy or systemic therapy should be considered for those with:

- More than 10% to 20% body surface involvement
- Generalised pustular psoriasis (mild, localised or palmoplantar pustulosis may not require referral)
- Erythrodermic psoriasis
- Psoriatic arthritis
- Localised recalcitrant psoriasis
- Psoriasis that significantly interferes with function e.g. on the palms or on the soles of feet

Specialist initiated treatments include phototherapy (narrowband UVB), methotrexate, acitretin,* ciclosporin and biological response mediators (e.g infliximab, adalumimab). Photochemotherapy (PUVA) has been discontinued in most centres except for use in localised hand and foot treatment.

Systemic therapies are not always effective and they may have potential serious adverse effects and risks.

*From 1st April 2009, acitretin has been available for prescription, fully funded on special authority application, by vocationally registered GPs.

Phototherapy

Narrowband UVB phototherapy is available in larger hospitals and some private dermatological practices. The patient stands in a cabinet containing 24 to 56 fluorescent bulbs, primarily emitting a wavelength of 311–312 nm, and is exposed to increasing doses two or three times weekly for 20 to 40 treatments. Thin plaque psoriasis responds best with about 85% of patients achieving 90% clearance. Prolonged remissions are common. Risks of this therapy include burns, presumed increase in skin cancer and premature aging of the skin.[5]

Methotrexate

Methotrexate is a folate antagonist and T-cell suppressive. It is effective for at least 60% of patients with psoriasis, taken as a weekly dose of 10 to 30 mg. Supplementary folic acid (5 mg once weekly) may reduce the risk of adverse effects such as gastrointestinal disturbance and mouth ulceration. Methotrexate is absolutely contraindicated in pregnancy and lactation. It also affects sperm and men should not father children while taking it. Significant liver disease, especially when resulting from alcohol misuse, precludes treatment. Other folate antagonists such as trimethoprim and sulfonamides must not be prescribed with methotrexate, because of an increased risk of marrow suppression.

Monitoring blood count, liver function and serum creatinine every one to three months is essential. Long term liver fibrosis and cirrhosis is a risk and additional specialist monitoring may be required.

Acitretin

Acitretin is a synthetic aromatic analogue of retinoic acid. It acts by normalising epidermal cell proliferation, differentiation and cornification. It is particularly useful for erythrodermic and pustular forms of psoriasis. The thickness and scaling of chronic plaque psoriasis usually improves on treatment. Like isotretinoin, it has recently become fully funded when special authority criteria are fulfilled. However there are significant adverse effects and risks from treatment.

The initial dose is 10–30 mg daily and the maintenance dose depends on clinical efficacy and tolerability.

Safety of acitretin.[8] Acitretin is highly teratogenic and must not be used by women who are pregnant, or of child bearing potential, unless strict contraception is used for four weeks before, during and for three years after treatment. Blood donation is prohibited during, and for one year after completion of therapy.

Alcohol must be avoided during treatment and for two months after treatment with acitretin, particularly by women of child bearing potential. This is because it can lead to the formation of etretinate, a retinoid that is stored in fat cells, and takes several years for the body to clear.

Acitretin is contraindicated in patients who are breastfeeding, or those with severely impaired renal or hepatic function, or chronically abnormal elevated blood lipid values.

Tetracyclines, methotrexate and vitamin A or other retinoids must be avoided.

Hepatic function and serum lipids should be regularly monitored throughout treatment. Acitretin may result in a reversible hyperlipidaemia requiring active management. People with diabetes may experience a change in their glucose tolerance (both improvement or worsening can occur) and so blood glucose levels may require more intensive monitoring in the early stages of treatment.

The most common adverse effects observed are dryness of the lips, epistaxis, peeling palms and soles, dry skin, asteatotic dermatitis and diffuse hair loss (which may be severe). These are symptoms of hypervitaminosis A. Many patients are unable to tolerate acitretin because of fatigue, myalgia, arthralgia or ophthalmic effects (e.g. blurred vision, impaired night vision).

Coal/pine tar preparations used for psoriasis

Coal tar can irritate the skin so patients need to experiment with different products. A higher concentration of coal tar is more effective but has a greater risk of irritation.

Preparations	Quantities and other ingredients
For application to scalp	
Coco-scalp ointment (S)	Coal tar solution 12%, sulfur 4%, salicylic acid 2%, coconut oil
Fongitar shampoo (NS)	Pyrithione zinc 1%, Polytar 1% (tar 0.3%, coal tar solution 0.1%, cade oil 0.3%, arachis oil extract of crude coal tar 0.3%)
Ionil-T shampoo (NS)	Salicylic acid 2%, coal tar solution 5%
Neutrogena T/Gel shampoo (NS)	Coal tar 0.5% (as solubilised coal tar extract 2%)
Polytar Plus shampoo (NS)	Coal tar 4%
Sebitar shampoo (NS)	Pine tar 1%, coal tar solution 1%, salicylic acid 2%
For bathing	
Polytar Emollient liquid (PS)	Polytar 25% (tar 7.5%, coal tar 2.5%, cade oil 7.5%, arachis oil extract of crude coal tar 7.5%), liquid paraffin in a water dispersible base
Polytar Liquid (NS)	Polytar 1% (tar 0.3%, coal tar solution 0.1%, cade oil 0.3%, arachis oil extract of coal tar 0.3%)
For application directly to plaques	
Egopsoryl TA gel (PS)	Coal tar solution 5%, sulfur 0.5%, phenol 0.5%, menthol 0.75%, allantoin 2.5%

Key: (S): fully subsidised; (PS): partially subsidised; (NS): not subsidised

Ciclosporin

Ciclosporin is rapidly effective for psoriasis in doses ranging from 2.5 to 5 mg/kg/day, taken for three to six month courses. However, as psoriasis tends to relapse quickly on discontinuing the drug, many people take it for prolonged periods risking hypertension, renal impairment, increased risk of skin cancer and other adverse effects.

Monitor blood pressure and renal function regularly, along with other routine tests (see BPJ 17, October 2008 "Monitoring DMARDs").

Biological response mediators

Several proteins and monoclonal antibodies that target T cells and TNF-α have come on to the market in the last decade and many new drugs are under investigation. Treatment is well tolerated and often very effective for psoriasis. Long-term studies are ongoing to determine safety, as these agents may increase granulomatous infections, such as tuberculosis and have been reported to exacerbate cardiac failure and demyelinating conditions (e.g. multiple sclerosis).[9]

Infliximab is a TNF-α inhibitor given as an intravenous infusion at weeks zero, two and six, then every eight weeks. It is reserved for very severe psoriasis and can be administered in both hospital or outpatient settings.

Adalimumab is also a TNF-α inhibitor, self-administered as a subcutaneous injection every two weeks. Adalimumab is now funded on Special Authority application (initially by a specialist) for small numbers of patients with severe psoriasis in whom current treatments are ineffective or contraindicated.

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Managing ECZEMA

www.bpac.org.nz keyword: eczema

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Key concepts

- Identify and minimise exposure to factors that exacerbate eczema
- Maintain the barrier function of skin with emollients
- Use anti-inflammatory treatment to control exacerbations

Eczema, also referred to as atopic eczema or atopic dermatitis, is an itchy inflammatory skin condition that is often chronic or relapsing.[1]

There are two main theories about the pathogenesis of eczema. The more traditional view is that eczema is primarily an immune mediated response to environmental factors.[2] However recent research suggests that skin barrier defects play a key role. These defects arise from gene mutations and result in loss of function of structural proteins e.g. filaggrin.[3] It is most likely that eczema arises from a combination of both genetic and environmental factors.

Most cases of eczema first develop in children aged under five years and about one in six school children have some degree of eczema. However, in about two thirds of cases, by the mid teenage years, the flare-ups of eczema have either gone completely, or are much less of a problem. It is unusual to first develop eczema after the age of 20, although eczema may go into remission in childhood and reappear in adult life. There is no way of predicting which children will be affected as adults.

The impact of eczema on families is an important consideration. Studies have found that the care of a child with severe eczema can have a significantly greater impact on family functioning, than the care of children with other chronic conditions, such as diabetes.[4]

Eczema can impact on career choice. A teenager with eczema should be aware that hair dressing, nursing, cooking and cleaning jobs, for example, are associated with a high risk of chronic hand dermatitis. People with eczema who work in the food, hospitality and health industries may find that their occupation adversely influences their eczema, and that they may not be able to work if they have visible eczema, particularly if it is infected.

Diagnosis of eczema

The diagnosis of eczema is usually based on the presence of an itchy rash in addition to a history of atopy and dry skin (see Box 1).

Box 1: Diagnostic criteria for eczema in children and adults[5]

An itchy skin condition (or parental report of scratching) in the last 12 months, plus three or more of the following:

- A history of involvement of the skin creases (antecubital fossae, popliteal fossae, ankles, neck or periorbital skin)
- A personal history of asthma or allergic rhinitis (or history of atopic disease in a first degree relative if a child is less than four years of age)
- A history of a generally dry skin in the last year
- Visible flexural eczema (including eczema affecting cheeks or forehead and outer aspects of limbs in children less than four years of age)
- Onset under the age of two years (but this criteria does not apply until the child is more than four years of age)

A detailed history includes; age of onset, pattern and severity, response to treatments, possible trigger factors, diet, personal and family history of atopic disease and the impact of the condition on children and their parents or carers.[5]

Examination of the rash can reveal the extent, location and severity of eczema and determine whether it is clinically infected.[6]

The affected areas of skin may vary depending on age:[1,7]

- In infants eczema commonly affects the face, trunk and limbs
- In children and adults eczema commonly affects the flexures, however it may be most troublesome on the face and hands

Management of eczema

The main aims of eczema management are to:

- Identify and minimise exposure to factors that exacerbate eczema
- Maintain the barrier function of skin with emollients
- Use anti-inflammatory treatment to control exacerbations

Referral to a specialist may be required for severe eczema or eczema that fails to respond to appropriate treatment.

Urgent referral is required if eczema herpeticum is suspected. This is a severe form of herpes simplex virus infection in a patient with atopic eczema which presents as rapidly worsening, painful plaques, clustered vesicles or punched out erosions.[7] It is often associated with fever and malaise.

Minimise irritants where possible

Factors that exacerbate eczema include infections, heat and sweating, dry skin, low humidity, emotional stress and irritants such as soaps, detergents and some fabrics (e.g. wool).[6] Avoiding these triggers where possible is beneficial during acute flares of eczema as well as for long-term management.[8]

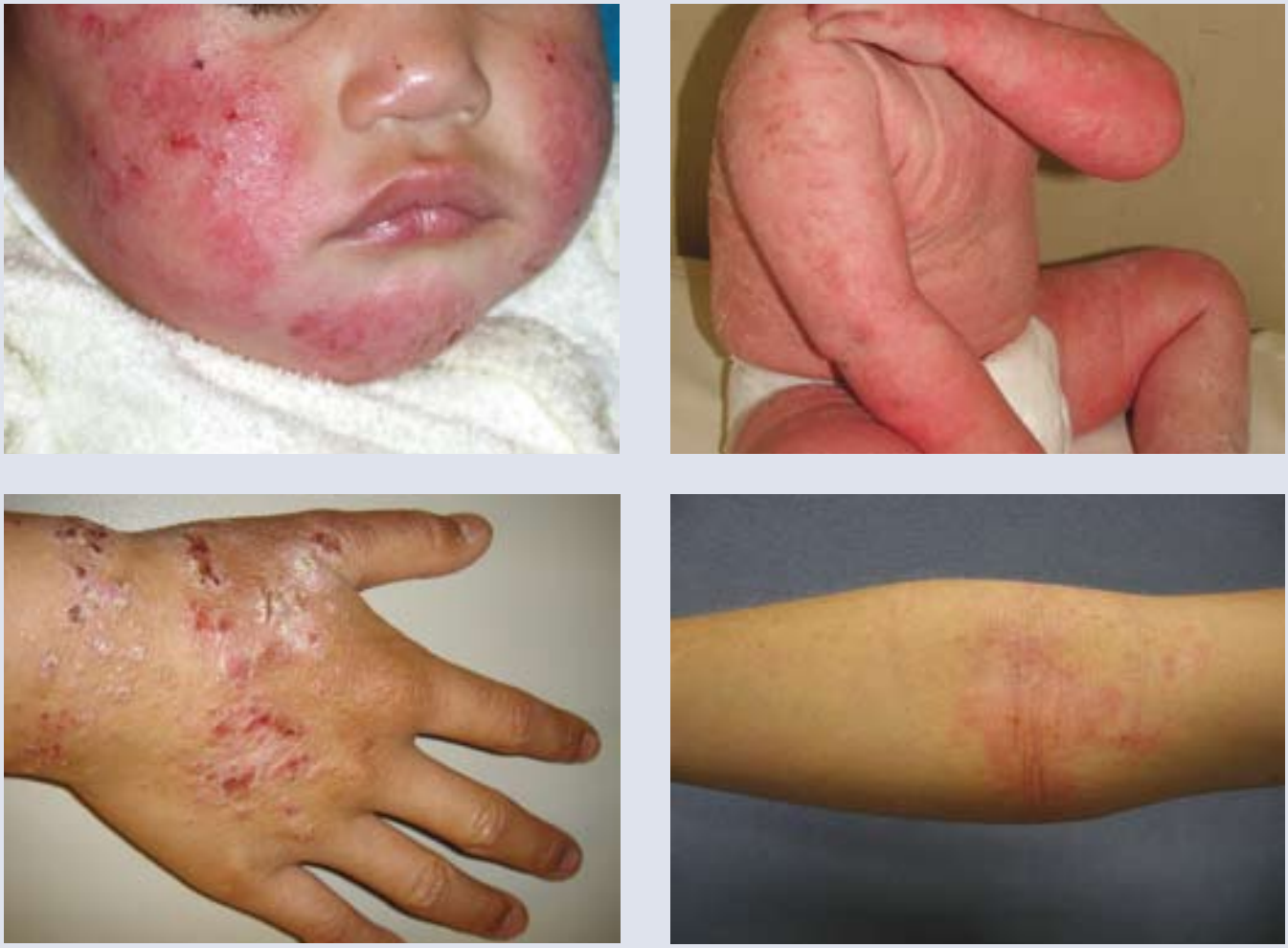

Figure 1: Examples of eczema (pictures suppled by DermNet NZ)

Eczema management algorithm adapted from NICE[5]

Clear:	Mild:	Moderate:	Severe:
■ Normal skin ■ No evidence of active eczema	■ Areas of dry skin ■ Infrequent itching (with or without small areas of redness)	■ Areas of dry skin ■ Frequent itching ■ Redness (with or without excoriation and localised skin thickening)	■ Widespread areas of dry skin ■ Incessant itching ■ Redness (with or without excoriation, extensive skin thickening, bleeding, oozing, cracking and alteration of skin pigment)
↓	↓	↓	↓
Emollients	Emollients	Emollients	Emollients
	Mild topical corticosteroids	**Moderate** potency topical corticosteroids*	**Potent** topical corticosteroids**
			Consider **wet wraps** (see page 30) and referral for systemic therapies

* Avoid use on face, neck, genitals or axillae for longer than 7–14 days

** Avoid use on face, neck, genitals or axillae

The role of airborne allergens such as house dust mites and animal dander in causing eczema is unclear and total elimination of these triggers can be difficult, time consuming and costly and may have limited benefit.[6] However, using mattress covers, low-pile carpet and minimising exposure to pets may be trialled, especially for children who also have asthma and/or rhinitis.[9] Cigarette smoke should be avoided.

Food allergies occasionally play a role in exacerbating eczema, however parents should be cautioned against adopting very restrictive diets because they may be of limited benefit, and may cause serious nutritional deficiencies.[9] In most cases, advise the patient or parent to continue a normal diet. Approximately 10% of children with eczema have a food allergy that aggravates their eczema, but this is much less common in adults. Common allergens include; milk, eggs, nuts, soya and wheat. Food allergy may be suspected if other symptoms such as gastrointestinal upset, vomiting or diarrhoea are present

Box 2: Emollients available in New Zealand

In New Zealand, funded options include aqueous cream, fatty cream,* emulsifying ointment and cetomacrogol cream.

Partially funded options include oily cream, glycerol with paraffin and cetyl alcohol (QV lotion) and wool fat with mineral oil (Alpha-Keri, Hydroderm BK and DP lotions). Urea cream (Nutraplus) is very effective at moisturising dry skin, but may sting if there is active eczema.

* Lemnis Fatty Cream has been discontinued. healthE Fatty Cream has the same formulation as Lemnis Fatty Cream and is now fully funded. N.B. Lemnis Fatty Cream HC (hydrocortisone) is still available.

concurrently.[10] Food allergy is less likely if eczema has developed after age two years, as sensitisation to dietary allergens decreases with age.[5]

Apply emollients liberally, frequently and continuously

The aim of using emollients is to maintain the skin's barrier function to keep moisture in and irritants, allergens and pathogens out.[6]

Most people with atopic eczema have dry skin. Dry skin causes pruritus and also contributes to eczema morbidity by creating microfissures and cracks in the skin, through which irritants and pathogens can enter.[11]

While emollients are universally recommended as the core treatment for eczema, there is limited evidence about their efficacy. One study in infants with moderate to severe eczema found that using emollients significantly decreased the requirement for topical steroids.[12]

Apply emollients three to four times daily

Application of emollients three to four times daily (or more) is ideal, however this may be difficult. For most people regular once a day application is achievable and can still lead to improved outcomes. Emollients are best applied after bathing or showering while the skin is hydrated.

People with eczema should avoid using soaps, detergents or bubble bath. Instead, a soap substitute such as emulsifying ointment or aqueous cream, can be used.[7]

To use emulsifying ointment as a soap substitute, mix it with warm water in the palm of the hand or in a cup (which could be stored for one or two days) to form a lather. Apply this to skin in the same way as soap. While bath oils may be used, there is limited evidence of their effectiveness and patients should be advised that their regular emollient regimen still needs to continue.[13] N.B. Emollients will make the bath/shower greasy and slippery. A bath mat should be used for safety purposes

Prescribe sufficient quantities of a patients preferred emollient

Emollients are available as both creams and ointments. Creams are suitable for red, inflamed skin while ointments are suitable for very dry skin. Ointments are often not well tolerated because some people find them too greasy. It may be more convenient for patients to apply a cream or lotion during the day and an ointment at night.[7]

The best emollient is the one preferred by the patient because it is more likely to be used regularly. It is important to prescribe sufficient quantities of emollient. Approximately 250 – 500 g of emollient per week is required for someone with extensive areas of dry skin.[7]

Best practice tip: Initially prescribe a selection of emollients to allow the patient to choose the one that suits them best.

Topical corticosteroids are used to treat eczema flares

Despite efforts to avoid irritants and the best use of emollients, flares are characteristic of eczema. Topical corticosteroids are the main agents used to control flares. As the intention is to use them short-term, they should be applied in appropriate amounts (see page 10) to all affected areas to gain rapid control.

See page 8 for information on the use of topical corticosteroids including their indications, potencies, adverse effects and precautions.

In general the potency of steroid should be matched to the severity of the flare.[5] For mild flares use a mild steroid, for a moderate flare use a moderately potent steroid and for a severe flare use a potent steroid. Treatment should continue until the flare has resolved.

Avoid using a potent corticosteroid on flares affecting the face, neck, genitals or axillae because the risk of local adverse effects is greater in these areas.[7]

In most cases, once control of a flare is achieved, corticosteroids can be stopped. However, for those who experience frequent flares (i.e. two or three per month) it may be useful to continue topical corticosteroids between flares. There are two ways to do this - either step-down to the lowest potency that controls the eczema or use the same potency but apply less frequently (e.g. two consecutive days per week).[5]

Emollients need to be continued during flares and for maintenance.

Other pharmacological treatments for eczema

Pimecrolimus (Elidel) - is there a place for this?

Yes: For use on sensitive areas e.g. eyelids, groin if hydrocortisone is being used continuously, or is not effective, on these areas.

No: if cheaper low potency topical corticosteroids are proving effective or where it is safe to use more potent topical corticosteroids.

Pimecrolimus is classified as a calcineurin inhibitor. It works by inhibiting T cell cytokine production and prevents the release of inflammatory mediators from mast cells.[10] Pimecrolimus is less effective than 0.1% betamethasone valerate (potent topical corticosteroid).[14] However, unlike topical corticosteroids, pimecrolimus does not cause skin atrophy which may be an advantage on sensitive areas such as the face, eyelids and groin.[8]

Pimecrolimus may cause local irritation (a short-lasting burning sensation) which can be particularly problematic in children who have low tolerance for stinging preparations.[15] The long term safety profile of pimecrolimus is unknown and while the link is uncertain, there is concern that there may be an increased risk of skin cancer and lymphoma.[16]

For these reasons, topical corticosteroids are still considered the first line treatment for eczema.

Pimecrolimus is not currently subsidised – a 15 g tube costs approximately $50.

Antihistamines may be useful to aid sleep for those with severe pruritus

Evidence supporting the use of antihistamines for eczema is weak.[8] However pruritus associated with eczema can cause scratching, leading to excoriation, bleeding and infection. During a flare the itch can result in significant sleep loss for which a short course of sedating antihistamine such as promethazine hydrochloride (Phenergan) may be useful.[9]

A trial of non-sedating antihistamines (e.g. cetirizine) may be of benefit for patients with allergic triggers as they may reduce atopic disease with use over several months.[9]

Secondary infection may require topical or oral antibiotics

Eczema lesions are commonly colonised with *Staphylococcus aureus*.[9] Signs that eczema is clinically infected include; crusting, weeping, pustules or failure to improve with treatment.[7]

If there are extensive areas of infected eczema an oral antibiotic such as flucloxacillin is recommended. For localised areas of infection a topical antibiotic may be used either in conjunction with a corticosteroid or as a combined product. Limit the use of topical antibiotics to one to two weeks as resistance or sensitisation may occur.[7]

Antiseptic use

Evidence of effectiveness of topical antiseptics (e.g. chlorhexidine) for eczema is limited. However they can be used to reduce bacterial load in infection-prone areas.[5]

Routine use of emollients containing antiseptics is not recommended because they may cause sensitisation.[7]

The "swimming pool water" method – sodium hypochlorite, as half a cup of household bleach in bathwater, may reduce the severity of eczema.[17] Patients should be advised to soak for five to ten minutes, and then thoroughly rinse the skin with lukewarm, fresh water to prevent dryness and irritation. Pat dry and apply any prescribed medications and/or emollients. Bleach baths can be used two to three times a week. Do not use if there are extensive areas of broken skin.[9]

Potassium permanganate is an antiseptic that is sometimes used to treat eczema that is weeping or has become infected. Potassium permanganate crystals can be added to bath water at a concentration of 1:10000 (dissolve a few crystals in a container of water until a light purple solution is formed and then add to bath water, which should turn light pink). However the solution may cause brown staining to the skin and nails not to mention the bath so this method has fallen out of favour.

Wet wraps may be useful for severe or extensive eczema

Wet wraps are used to hydrate the skin and prevent scratching. They also enhance penetration of topical steroids into the skin. They are effective for severe or extensive eczema.[9, 18]

Adverse effects that may occur, especially with incorrect or excessive use, include maceration of the skin or secondary infection.

Wet wraps are typically used overnight and removed in the morning. Emollients should continue to be applied frequently throughout the day to the affected areas. Wet wraps may be used for a few nights (maximum five to seven consecutive nights)[9] until the redness, swelling and weeping has settled down (see opposite page for instructions).[19]

The role of oral corticosteroids for treating eczema is limited

Oral corticosteroids may be used to quickly control an eczema flare however there is an associated risk of rebound flares.[18] Frequent or prolonged use can also increase the risk of adverse effects such as growth retardation in children, osteoporosis or elevated blood pressure.[1, 7, 8]

Do skin prick tests, RAST tests or patch tests have a role in diagnosing allergies?

People with mild eczema rarely require investigation for allergies.[5]

Referral to a dermatologist for allergy tests may be appropriate for eczema that has a poor response or failed to respond to conventional topical treatment, or where dietary precipitants are suspected.[20] Commonly used tests for allergies include patch test, skin prick test and radioallergosorbent test (RAST).

Patch testing

Patch testing is used in the investigation of suspected allergic contact dermatitis. It is mainly indicated where eczema is confined to a specific site e.g. only the hands or face. Patches containing standardised allergens are applied to the upper back and tape is used to keep them in place for 48 hours. It is then "read" at various time intervals looking for evidence of an eczema-like rash, that might indicate sensitivity to a particular allergen.

Skin prick testing

Skin prick testing is used to detect the presence of allergen specific IgE to food, aeroallergens, some venoms, antibiotics and latex.[20] Drops of commercially produced

allergen are placed onto a marked area of skin on the forearm or upper back. Using a sterile lancet, a small prick to the skin through the drop is made. If the patient is allergic a small lump will appear at the site of testing over 15 – 20 minutes. This is not an eczematous response, so its relevance to eczema is unclear.

Atopic individuals may get false positive results with skin prick testing because of the sensitivity of eczematous skin to any trauma. In people with eczema or dermographism, scratching the skin may cause a raised mark and be read as a positive test result, even without any allergen. For this reason, skin prick testing is unreliable for diagnosing allergies in people with eczema.[21]

RAST testing

RAST testing is a blood test that measures the level of specific IgE to different allergens and is used to investigate increased sensitivity to a variety of food groups (e.g. eggs, cow's milk and nuts), house dust mite and animal dander.

In atopic eczema the results of RAST testing may be misleading. It is often used in the investigation of patients with atopic eczema however, a patient can have a degree of sensitivity to many allergens but not all will have a clinically significant effect on their eczema.

Specialist care

Children and adults with severe or persistent eczema should be referred to a dermatologist and may also require paediatric and/or immunology assessment.

Other second line treatments used in specialist practice include narrowband ultraviolet-B (UVB) phototherapy and immunosuppressive agents, especially methotrexate, azathioprine and ciclosporin. These agents necessitate regular review and careful monitoring.

Keeping eczema under wraps– recommendations for applying wet wraps:[9, 18]

- Prepare lengths of tubular bandage. Cut two lengths for each arm and leg and two lengths for the torso
- One length of each is soaked in warm water and then wrung out until it is slightly damp
- Bath and wash as usual
- Apply steroid to affected areas (if required) and then emollient to the rest of the skin
- Cover with the damp dressing and then cover this with the dry dressing. Plastic is not a suitable alternative to the dry dressing as it is too occlusive and may be a choking hazard.
- The wrap may be left on overnight but make sure the patient remains in a warm environment

N.B: Tubular bandages (e.g. Tubigrip, Tubifast) are available in a range of sizes from pharmacies. Tubifast bandages and garments are available from the following websites:

Allergy Pharmacy :
www.allergypharmacy.co.nz

Kumfy Kids:
www.kumfykids.co.nz

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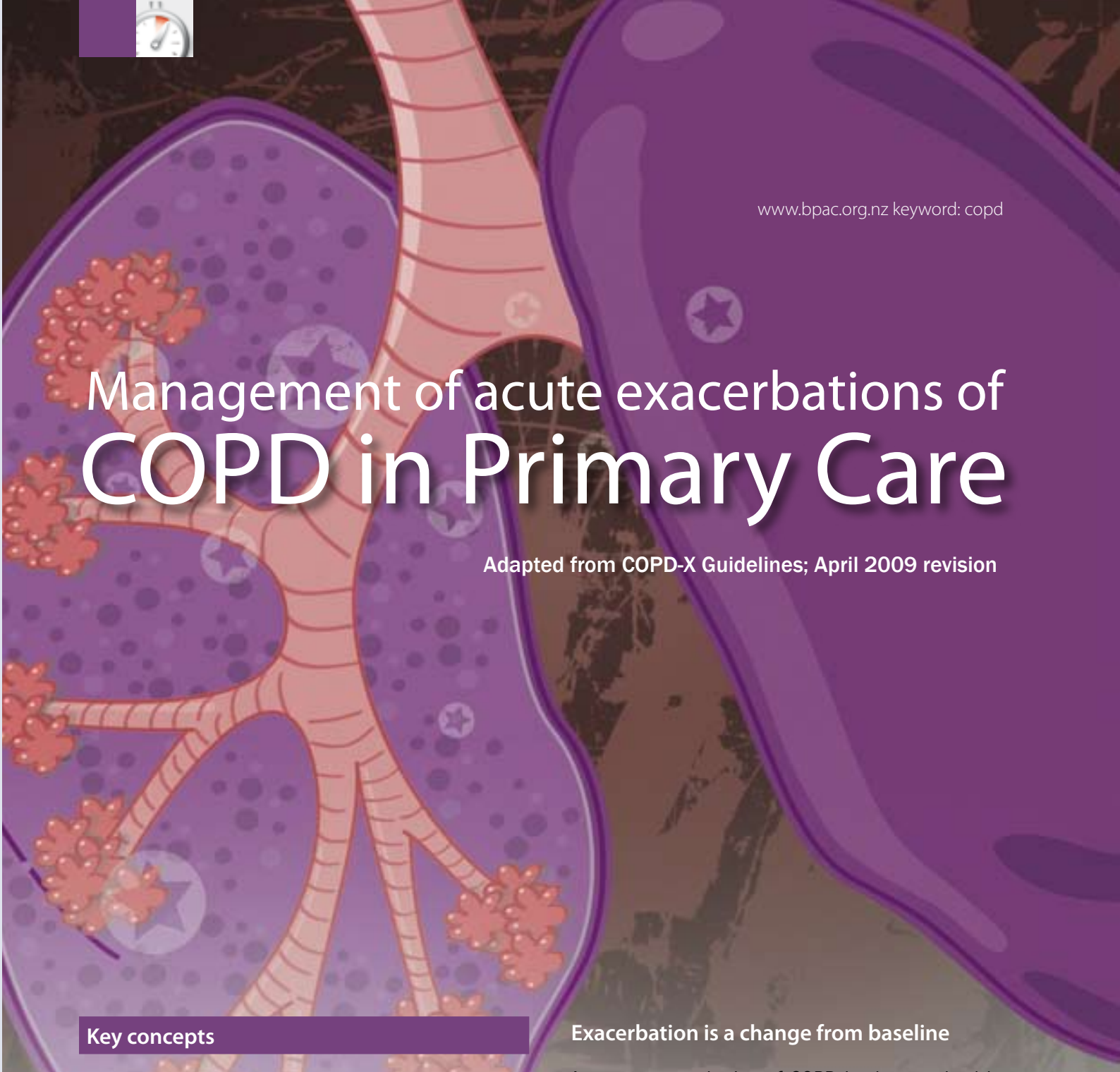
Management of acute exacerbations of COPD in Primary Care

Adapted from COPD-X Guidelines; April 2009 revision



Key concepts

- Early diagnosis and treatment may prevent hospital admission
- Inhaled bronchodilators are effective for the treatment of acute exacerbations
- Oral prednisone reduces the severity and shortens recovery time from acute exacerbations
- Antibiotics may be beneficial if there are clinical signs of infection

Exacerbation is a change from baseline

An acute exacerbation of COPD is characterised by a change in a person's baseline dyspnoea, cough and/or sputum production that is greater than day to day variation (definition from the Global Initiative for Obstructive Lung Disease – GOLD).

Lung inflammation and infection appear to play an important role in the pathogenesis of worsening symptoms. The most common triggers are viral or bacterial infections. Non-infectious causes include left ventricular failure, pulmonary embolus, environmental irritants, chest trauma and inappropriate sedative use.

Early diagnosis and prompt management may prevent progression and admission

Assessment of the severity of the exacerbation includes measurement of blood pressure, respiratory rate and oxygen saturation (if pulse oximetry is available). The need for hospital admission is based on clinical findings and social circumstances.

Educating the patient and their carers about the signs of worsening COPD may be helpful in early detection of an exacerbation. A self-management plan which describes how to step up treatment is also beneficial. The plan should include advice on bronchodilator use, when to start oral prednisone, and the indications for antibiotic use.

Optimise the dose of bronchodilator

During exacerbations of COPD the immediate effect of a bronchodilator is small, but for those with severe obstruction, there may be a significant improvement in clinical symptoms. Bronchodilators may reduce air trapping.

A short acting beta-2 agonist (salbutamol 400 – 800 mcg) or ipratropium 80 mcg can be given by pressurised metered dose inhaler (MDI) and spacer. The dose interval is titrated to response and can range from hourly to six-hourly.

If the patient is using a long acting beta-2 agonist (LABA) or tiotropium, they should be continued during the treatment of the exacerbation.

Glucocorticoids are beneficial

Oral prednisone can speed up the resolution of exacerbations and also reduce the risk of relapse. There is little evidence that IV steroids are better than oral. The optimal oral dose has not been established. Prednisone 40 mg taken as a single daily dose in the morning, for up to two weeks, is sufficient in most cases. It is traditional to do a tapering dose, but this is not necessary after such a short course. Longer courses add no further benefit and have a greater risk of adverse effects.

If the patient is already using an inhaled corticosteroid (ICS), this can be continued while taking a short course of prednisone, but it is useful to check the inhaler technique as the main benefit of ICS is reducing the frequency of exacerbations in those with severe COPD. Patients who are taking long-term, low-dose prednisone should not be using ICS at the same time.

Antibiotics have specific indications

Viral infections are a significant cause of exacerbations. Bacterial infections (predominantly *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Moraxella catarrhalis*) have a primary or secondary role in about 50% of acute exacerbations of COPD. Sputum culture is not routinely required, but may be helpful if there is no improvement with treatment.

The benefits of antibiotic use are unclear. They are only recommended if there are at least two new findings of: increased purulent sputum, increased sputum production or increased dyspnoea.

Antibiotic choice

First-line: Amoxicillin 500 mg three times a day for five days

Alternative: Doxycycline 100 mg twice daily for five days (if penicillin allergic or recent course of amoxicillin). Amoxicillin clavulanate is only indicated if there has been clinical failure with first-line antibiotics.

Ciprofloxacin does not have adequate coverage against *S. pneumoniae* and should not be used for the management of acute exacerbations of COPD.

When to refer

Mortality rates from exacerbations of COPD increase with acute carbon dioxide retention (respiratory acidosis), the presence of co-morbidities (e.g. heart failure and IHD) and complications such as pneumonia. Depending on the circumstances and severity of the exacerbation urgent

hospital admission may be required for ventilatory support and other intensive treatment.

Indications for referral to secondary care;

- Inability to walk short distances when previously mobile
- Inability to eat or sleep because of dyspnoea
- Inability to manage at home even with help
- High risk co-morbid condition
- Altered mental state suggestive of hypercapnia
- Worsening cor pulmonale or hypoxaemia
- New appearance of arrhythmia
- Inadequate response to management in primary care
- Uncertainty of diagnosis

Strategies to reduce the frequency of exacerbations

Exacerbations of COPD, especially if severe, are associated with increased mortality. Strategies to reduce the frequency of exacerbations should be considered and be part of an individual management plan. Strategies include:

- Influenza vaccination (yearly) and pneumococcal vaccination (five yearly)
- Minimising infection risk, such as avoiding contact with people with an active URTI
- Avoiding exposure to smoke and irritants
- Optimising control of co-morbidities
- Use of medication

Inhaled corticosteroids (including when combined with LABA) reduce the rate of exacerbations however they do not improve mortality and their effect on the decline in lung function remains unclear. They should be considered for patients with severe COPD and frequent exacerbations (e.g. two or more exacerbations in a year requiring treatment with an antibiotic or oral corticosteroid). Systemic absorption may occur, especially when high doses are used, therefore the benefit of ICS must be weighed against the risk of adverse effects, such as bruising, cataracts and osteoporosis.

Tiotropium decreases exacerbations as well as improving lung function, symptoms and quality of life. The number needed to treat (NNT) for one year to prevent one exacerbation is 14 and the NNT is 30 to prevent one hospitalization. Adverse effects of tiotropium include dry mouth and infrequently, urinary retention.

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Having a senior moment?

Differentiating normal age-related memory loss from early onset dementia

Key reviewer: **Dr Ian Hosford**, Psychogeriatrician, Hawke's Bay DHB, Hastings

Key concepts

- Normal age-related memory loss affects everybody
- Assessing memory loss can be complex
- A simple place to start is to rule out other causes, especially depression and look for red flags
- Differentiate normal age-related cognitive decline from early stage dementia
- Consider performing a memory test
- Make a plan for follow-up, investigations and referral

www.bpac.org.nz keyword: memory

The aging brain

As we age, brain volume shrinks, neurons are lost at a greater rate than they are regenerated, synapses deteriorate and neurotransmitters become less efficient at carrying information.[1, 2] This process begins when we are in our 20s and starts to accelerate at around age 50, when changes in cognitive function may become noticeable.[1]

A person in their 50s may take longer to recall names and words, learn new tasks or multi-task and attention to detail declines. In their 60s, these changes become more perceptible. It becomes harder to concentrate and to tune out distractions, new memories are more difficult to form and it takes longer to recall information.[1]

The rate at which the brain ages is dependent on many factors such as genetics, hormones, neurotransmitters (e.g. dopamine, serotonin), co-morbidities, experiences and environmental factors.

Types of memory

Different parts of the memory are affected in different ways during the aging process. The terms, short-term and long-term memory, are still used but it is helpful to consider memory functioning in more detail:[2, 3]

Episodic memory is information that is stored with mental tags about when, where and how it was picked up e.g. your first day of school, what you ate for dinner last night. Memories may be stored from minutes to years. The ability to learn new information and recall recently learned memory declines from middle age. Remote memories are more resistant to loss.

Semantic memory is the memory of meanings (factual and conceptual knowledge) e.g. knowing that Wellington is the capital of New Zealand or why a fork is different from a knife. The volume of this memory increases gradually from middle age to the young elderly but declines in very elderly people.

Procedural memory is the "how to" knowledge of skills and procedures, and can be explicit (purposeful, conscious) e.g. learning to ride a bike, or implicit (automatic, unconscious) e.g. knowing the sequence of keys on a computer keyboard. This memory is usually retained into late life.

Working memory is information remembered over a brief period of time (seconds to minutes) before it is dismissed or transferred to a longer-term memory. It can be phonological e.g. name of a person you just met, keeping a phone number in your head as you dial it, or spatial e.g. mentally following a route or rotating an object in your head. With aging, working memory span often becomes shorter, making it more difficult to retain a memory for long enough to use it.

Is this memory loss normal?

A 65 year old male presents complaining of increased forgetfulness and problems with his memory. He is worried that he is developing dementia. What would you do?

History, observation and examination will generally guide the clinician as to when a formal cognitive assessment is required (if at all). Assessing memory loss may in some circumstances be straight forward and require simple reassurance but in other circumstances may be complex and take several consultations.

The following framework may be helpful to structure your approach:

1. Rule out other causes; consider depression, red flags (see page 38)
2. Differentiate normal age-related cognitive decline from early stage dementia
3. Consider performing a memory test
4. Make a plan; follow-up, investigations, referral?

1. Rule out other causes for memory loss

Are there any potentially reversible factors in this case which may explain the memory loss:

- Is the patient taking any medications that cause cognitive impairment?
- Does the patient have a history of drug or alcohol misuse?
- Are there any signs or symptoms of infection?
- Is there evidence of recent head trauma?

Consider depression

Depression is a common cause of memory impairment and often co-exists with dementia in elderly people.[4] Memory may appear selective or patchy rather than generally impaired and symptoms usually have a duration of weeks or months rather than a gradual decline over years.[4] A screening tool such as the Geriatric Depression Scale may be used.

See BPJ 11 (February 2008) "Depression in elderly people".

Are there any red flags?

Referral for more extensive investigation is required for memory loss in the presence of the following factors:[4]

Patient:

- Age less than 60 years

History:

- Rapid (i.e. over one to two months) decline in cognition or function
- Unexplained neurological symptoms (e.g. new onset severe headache, seizures)
- Use of anticoagulants or history of bleeding disorder
- History of cancer
- Family history of neurodegenerative disease

Examination:

- Any new localising sign (e.g. positive glabellar tap, grasp reflex)
- Unusual or atypical cognitive symptoms or presentation
- Gait disturbance

Causes of memory loss[4]

Medical conditions associated with memory loss include:

- Mental illness e.g. depression
- Cerebrovascular disease
- Neurodegenerative disease e.g. Alzheimer's disease, Parkinson's disease, Creutzfeldt-Jakob disease
- Medications (e.g. tricyclic antidepressants, cytotoxics)
- Substance misuse/dependence (e.g. alcohol, benzodiazepines, opiates)
- Brain tumour and infections
- Head injury
- Epilepsy
- Chronic pain, anxiety, stress
- Sleep disorders
- Thyroid disease
- Malnutrition, vitamin deficiencies

2. Differentiate normal age-related cognitive decline from early stage dementia

After ruling out any other explanations for the memory loss, now consider whether this is normal age-related memory decline or early symptoms of dementia.

Normal age-related memory decline is characterised by:

- Subjective memory concern
- Mild episodic memory impairment
- Preserved procedural and semantic memory
- Possible mild non-memory cognitive dysfunction (e.g. attention)
- No functional impairment or behavioural abnormalities[6]

The main distinction between memory loss due to aging and memory loss due to dementia is that problems in age-related memory loss do not affect daily functioning or the ability to live independently.

Mild cognitive impairment is a "grey area" between normal age-related memory loss and dementia, and is defined as objectively impaired neuropsychological test performance but intact activities of daily living.[6]

- Most people are able to maintain their cognitive ability at a functioning level throughout their life.
- Approximately 20% of people aged over 65 years have mild cognitive impairment.[7]
- For some people, mild cognitive impairment is a precursor to dementia. A recent meta-analysis reported that the annual conversion rate from mild cognitive impairment to dementia is approximately 5–10%. Many people with mild cognitive impairment however did not progress to dementia even with ten years follow up.[8]
- Between 3–11% of people aged over 65 years and around 33% of people aged over 85 years have dementia.[7]

Table 1 shows the general distinctions between types of memory impairment.

Table 1: Characteristics of memory impairment (adapted from Neurological Foundation of New Zealand).[9]

Normal age-related "forgetfulness"	Mild cognitive impairment	Dementia
Sometimes misplaces keys, spectacles, or other items	Frequently misplaces items	Forgets what an item is used for or puts it in an inappropriate place
Momentarily forgets an acquaintance's name	Frequently forgets people's names and is slow to recall them	May not remember knowing a person
Occasionally has to "search" for a word	Finding words becomes more difficult	Begins to lose language skills. May withdraw from social interaction
Occasionally forgets to run an errand	Begins to forget events or newly learned information	Loses sense of time. Doesn't know what day it is
May forget an event from the distant past	May forget more recent events or newly learned information	Working memory is seriously impaired. Has difficulty learning or remembering new information
When driving may momentarily forget where to turn. Quickly orients self	May temporarily become lost more often. May have trouble understanding and following a map	Becomes easily disoriented or lost in familiar places, sometimes for hours
Jokes about memory loss	Worries about memory loss. Family and friends notice the lapses	May have little or no awareness of cognitive problems

Is this just memory loss or are there other signs of cognitive impairment?

If the memory loss is accompanied by other signs of cognitive impairment, this may be suggestive of dementia. Signs and symptoms include:

- Aphasia (impairment in producing and understanding speech)
- Apraxia (difficulty in performing motor tasks)
- Agnosia (inability to recognise familiar people, places and objects)
- Disturbance in executive function (difficulty sequencing, organising, abstracting, planning)
- Change in behaviour/mood (i.e. agitation, apathy, anxiety, disinhibition)
- Physical signs including gait disturbance, extra pyramidal symptoms, focal or lateralising neurological signs

Ask the patient if they have any trouble with managing money, using the telephone, using transportation or remembering to take medications. In dementia the earliest changes are seen in the ability to do these tasks.[5] The patient may not be aware of some of these changes, so asking permission to speak to someone who knows them well, may be necessary.

3. Consider performing a memory test

A diagnosis of mild cognitive impairment or dementia is predominantly made from the clinical history provided by the patient and an informant. Memory tests can be used to help confirm and quantify cognitive impairments.

A diagnosis of cognitive impairment can have a significant impact on self-esteem, independence, relationships, employment and plans for the future. Assessing cognitive decline with a memory test is only appropriate if the benefit of early detection of dementia is greater than the harm it may cause.

Early diagnosis allows people to make arrangements such as appointing Enduring Powers of Attorney, updating wills, moving homes and visiting family overseas. It also can provide an explanation for changes that have been occurring in the person for a long time (sometimes years) before they come to see the doctor. There is no cure for dementia but there are treatments that can alter the course of the illness.

Conversation with the patient during the consultation so far may have given clues as to whether a memory test is warranted – how did they answer questions? Did they hesitate to find words or recall facts or sequence of events? Were there any anomalies in their use of language? Is their reported cognitive impairment beyond what could be classified as normal age-related "forgetfulness"?

Which memory test?

A full battery of cognitive tests is usually not appropriate in a primary care setting. A brief, standardised cognitive screening tool may be used.[5]

The Mini Mental Status Examination (MMSE) is the most commonly used memory screening tool. Like many memory tests it is associated with age, educational, language and cultural bias. Some practitioners may be reluctant to use it as it takes ten minutes or more to administer.[10]

The Mini Assessment of Cognition (Mini-COG) and General Practitioner Assessment of Cognition (GPCOG) have an administration time of five minutes or less, a misclassification rate less than or equal to the MMSE (15%), high sensitivity and specificity (≥ 80%) and have been validated in studies relevant to general practice (large sample size, clinical diagnosis used as reference standard).[10] The Six Item Cognitive Impairment Test (6CIT) is also recommended.[7]

The Mini-COG is a good brief initial test that gives a result of "probably demented" or "probably not demented". GPCOG and 6CIT have the advantage of a scoring system so that severity can be monitored over time. However the GPCOG can involve an informant interview which may not be practical at the time of the appointment. See page 43 for tests.

4. Make a plan

Consider findings from the patient history and examination, observations and results of the memory test (if performed). Remember that it is possible for a person to score quite well on a memory test and still have significant cognitive impairment. Conversely a person who functions well can score badly on a cognitive test, e.g., if they are anxious or have mild dysphasia.

At this stage the patient may be classified into one of three alternatives:

a) Suspicion of dementia, for which further assessment and referral is appropriate

b) Suspicion of mild cognitive impairment, for which lifestyle advice and follow-up is appropriate

c) No signs of cognitive impairment, for which lifestyle advice and reassurance is appropriate

Follow-up mild cognitive impairment

In memory loss with no other domains of cognition involved and preservation of function:[4]

- Follow up carefully every three to six months to watch for deterioration
- Perform serial observations with mental state testing (e.g. GPCOG, 6CIT, MMSE) to confirm progression

Ask the patient to recruit a close family member to help them objectively observe any worsening cognitive impairment or impact on activities of daily living.

Consider laboratory investigation

Investigations help to rule out potentially reversible factors such as medical conditions.

If mild cognitive impairment has been identified, general investigation includes CBC, CRP, TSH, vitamin B12, folate, serum electrolytes, calcium and glucose. In some cases referral for a CT/MRI brain scan may be considered. White matter changes are associated with worsening cognitive function.[4, 7]

How to keep your brain healthy

Lifestyle interventions to reduce cardiovascular risk such as regular exercise, eating a balanced diet, low to moderate alcohol intake and being a non-smoker also seem to protect against age-related cognitive decline. A healthy lifestyle, both mental and physical, is the best preventative defence.[2]

Hypertension, stroke and small vessel disease, diabetes, hyperlipidaemia, obesity and hyperhomocysteinaemia have all been associated with an increased risk of age-related cognitive decline.[2]

Exercise

An increased level of fitness is associated with improved memory and learning and a reduction in age-related cognitive decline.[2, 13]

Alcohol

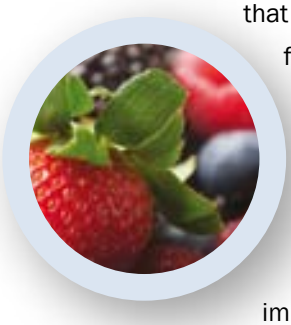
Consumption of small quantities of alcohol (one standard unit of alcohol a day) on a regular basis is thought to stimulate the hippocampus, therefore counteracting cognitive decline. This follows a U or J shaped curve where teetotal or heavy drinkers are disadvantaged.[2]

Diet

A healthy, balanced diet rich in antioxidants (e.g. blueberries, strawberries, cocoa, tea) and omega-3 (e.g. oily fish) may help to slow age-related cognitive decline.[6, 14] It is preferable (and safer) to use naturally occurring sources of antioxidants and omega-3 oils than supplement forms.

Supplements

Gingko biloba is a commonly used supplement for memory loss. In an analysis of 36 trials, it was concluded that gingko biloba appears to be safe with no excess side effects, however there is no consistent or reliable evidence



that it has any clinically significant benefit for people with dementia or cognitive impairment.[15]

Antioxidant supplements such as vitamin A, vitamin E and beta-carotene show no significant improvement in longevity, in fact they may actually increase mortality.[16]

Also see BPJ 14 "Antioxidants and aging".

Pharmacological treatments

There is no pharmacological treatment for delaying age-related cognitive decline or improving mild cognitive impairment.

Cholinesterase inhibitors such as donepezil, galantamine and rivastigmine are often used to temporarily stop or slow cognitive and functional decline in people with Alzheimer-type dementia and dementia associated with Parkinson's disease. Clinical trials show mixed evidence of their effectiveness. These medications are not subsidised.

There is no quality clinical evidence to support the use of oestrogen or hormone replacement therapy to prevent or treat cognitive decline. Some studies report evidence of increased mortality with these treatments.

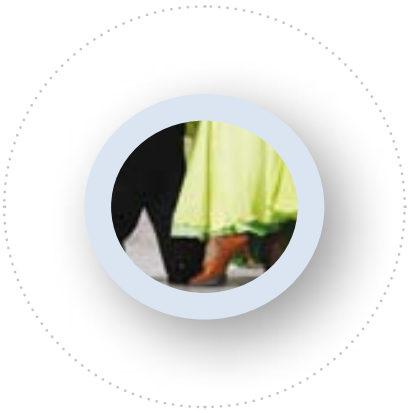
Brain exercises

A higher level of education or occupational attainment is considered to be a protective factor against age-related cognitive decline.[2] However it is never too late to start exercising the brain. There is growing evidence that participating in activities such as reading, puzzles, computer activities and crafts reduces the risk of age-related cognitive decline.[17] Social interaction is beneficial too. In a study involving almost 1000 elderly people, it was discovered that those who participated less frequently in social activity, had a more rapid rate of decline in cognitive function.[18]

In addition to exercising the brain, there are several strategies that can be adopted to help memory recall:[1]

- Place commonly lost items in the same spot every time
- Write things down e.g. make a "to do" list
- Say words out loud e.g. "I have turned off the iron", repeat a persons name after being introduced
- Use memory aids e.g. notepad, diary, wristwatch alarm, voice recorder
- Group items using mnemonics e.g. alphabetise a list, create an acronym or acrostic (using the first letter of each item to form a sentence), use rhymes or create a story to connect the information
- Concentrate and relax when trying to remember
- Sleep on it – research has shown that the brain continues to solve a problem while we sleep

Best practice tip: Encourage elderly patients to take up dancing! This combines physical activity, brain exercise (counting rhythms, learning steps etc) and social interaction. Some patients may prefer to join a walking or exercise group.



Driving safety

If a patient has cognitive impairment that is worsening over time, this is likely to have implications on driving safety. Relatives may sometimes raise concerns. Relinquishing a driver's licence is often a very sensitive issue. A patient with worsening cognitive impairment could be gradually prepared for this (unless they are clearly unsafe) by suggesting that they might require periodic on-road driving assessment or need to restrict their driving to familiar routes and only during daylight.[19]

Note that a medical practitioner has a legal obligation to advise the Land Transport Safety Authority if a patient poses a danger to public safety by continuing to drive when advised not to.

Cognitive screening tools

Copies of the cognitive screening tools mentioned in this article can be accessed on the following websites:

GPCOG www.patient.co.uk/doctor/General-Practitioner-Assessment-of-Cognition-(GPCOG)-Score.htm
Mini-Cog www.hospitalmedicine.org/geriresource/toolbox/pdfs/clock_drawing_test.pdf
6CIT www.patient.co.uk/showdoc/40026041/
MMSE www.nzgg.org.nz/guidelines/0103/Appendix___MMSE___Mini_Mental_State_Examination.pdf

Mini-Cog[11]

1. Ask the patient to repeat three unrelated words e.g. hat, apple, table

   If necessary the clinician may repeat the words up to six times for the patient to learn them

2. Ask the patient to draw a clock, put in all the numbers and set the hands at ten past eleven.

   A normal clock includes: clock circle, numbers in correct order, numbers in correct spaces on clock, two hands of clock, correct time.

3. Ask the patient to recall the three words from question 1.

Score: 3 words recalled + normal clock	probably not demented
Score: 1–2 words recalled + normal clock	probably not demented
Score: 0–2 words recalled + abnormal clock	probably demented

GPCOG Patient examination (Brodaty et al)[12]

Unless specified, each question should be only asked once

Recall

1. Give the patient a name and address and ask them to repeat it and remember it as you will ask them to recall it again *e.g. John Brown, 42 West Street, Kensington. (Allow a maximum of four attempts to repeat the address).*

Time orientation

2. What is the date? *1 point. Exact only*

Clock drawing (visuospatial functioning) Use a page with printed circle

3. Please mark in all the numbers to indicate the hours of a clock. *1 point. Correct spacing required.*
4. Please mark in hands to show 10 minutes past 11 o'clock. *1 point*

Information

5. Ask the patient to tell you something that happened in the news recently (in the past week) *1 point*

Recall

6. Ask the patient to recall the name and address from Question 1. *1 point for each of: John, Brown, 42, West Street, Kensington*

Score = 9	no cognitive impairment, interview not necessary
Score = 5–8	proceed to informant interview
Score = 0–4	cognitive impairment, interview not necessary

GPCOG Informant interview

Ask the informant: Compared to a few years ago:

1. Does the patient have more trouble remembering things that have happened recently?
2. Does he or she have more trouble recalling conversations a few days later?
3. When speaking, does the patient have more difficulty in finding the right word or tend to use the wrong words more often?
4. Is the patient less able to manage money and financial affairs (e.g. paying bills, budgeting)?
5. Is the patient less able to manage his or her medication independently?
6. Does the patient need more assistance with transport (either private or public)?

Score one point for each "no" answer

Score = 4–6	no cognitive impairment
Score = 0–3	cognitive impairment detected

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Cervical smears – achieving equity

Key reviewer: **Dr Hazel Lewis**, Clinical Leader, National Cervical Screening Programme, Wellington

Key concepts

- Target Māori, Pacific and Asian women and women from areas of high deprivation for cervical screening
- Target women over 30 years who have never had a smear or have not had a smear for over five years
- Target interventions carefully to avoid increasing disparities

Following the introduction of the National Cervical Screening Programme (NCSP) in 1990, the overall incidence of cervical cancer in New Zealand has dropped dramatically. Cervical cancer is a disease with a long latency period, taking on average 10 to 20 years to develop. This means that screening for the detection and treatment of precursor lesions can be very effective for women who participate regularly in a screening programme.

An inadequate screening history is associated with increased rates of cervical cancer. A 2004 audit of women with cervical cancer in New Zealand demonstrated that 80% had a suboptimal screening history.[1] Although it is anticipated that the HPV vaccination programme will bring about a further reduction in cervical cancer rates in the future, improving screening rates will continue to be the most effective way to reduce morbidity and mortality from cervical cancer.[2]

Who should have cervical smears

All women who have ever been sexually active are eligible for cervical screening from the time they turn 20 until they turn 70. This includes:

- Lesbians
- Women who have not been sexually active for many years
- Women who still have a cervix following a hysterectomy

Women aged 70 and over who have never had a cervical smear test are advised to have a smear test followed by another a year later. If both tests are normal no further tests are needed.

Many women are not participating in cervical screening

The NCSP has a target of 75% coverage for cervical smears for all eligible women.[3] At this stage European/Other women are the only ethnic group who meet this target (Figure 1), although screening rates are increasing for all ethnicities.

The women who consistently have cervical screening rates less than the NCSP target are:

- Māori
- Pacific
- Asian
- Women from the most deprived areas

Māori women

As rates of screening have improved, the rate of cervical cancer in Māori women has fallen. However Māori women still have higher rates of cervical cancer than non-Māori women, and are four times more likely to die from cervical cancer than European women.[4] This is likely to be due to continued suboptimal rates of screening.

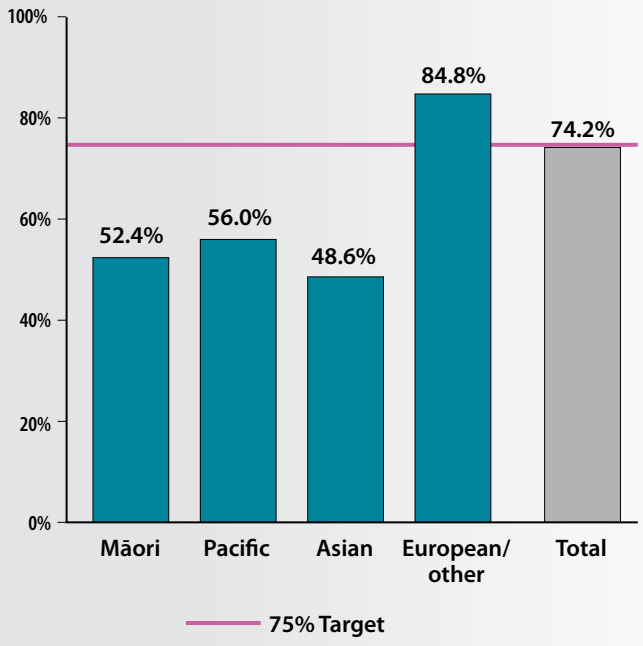


Figure 1: National Cervical Screening Programme three year coverage by ethnicity, March 2009 (hysterectomy-adjusted)[5]

Recommendations for screening following hysterectomy[6]

- Women who have had a total hysterectomy (the cervix has been removed) for a benign condition and have no history of abnormal smears do not need to continue to be screened.
- Women who have had a total hysterectomy and have had abnormal smear results or abnormal cervical histology need to continue being screened.
- Women who have had subtotal hysterectomies (the cervix has not been removed) need to continue being screened.

The screening interval will vary and depends on previous history. For further details, refer to the 2008 Guidelines for Cervical Screening in New Zealand.

Pacific women

Pacific women have a higher rate of cervical cancer than the national average, and are almost twice as likely to die from cervical cancer as European women. Uptake of cervical screening is lower for Pacific women than for European women, with just over half of eligible Pacific women having regular cervical screening.[7]

Asian women

Asian women are significantly less likely to have had cervical screening than European women, with less than half having regular cervical screening.[8] This is the lowest rate for any ethnic group in New Zealand.There are no published statistics on cervical cancer in Asian or Chinese immigrant women in New Zealand.

Women living in areas of high deprivation

Eligible women living in the most deprived areas of New Zealand are less likely than women living in the least deprived areas to have had cervical screening in the last three years.[9]

Mana Wahine

The concept of mana wahine describes the status, power and authority of Māori women. Mana wahine reflects Māori women's connection to the land, as descendants of Papatuanuku, the Earth Mother. Mana wahine has embedded within it a philosophy concerning the sphere of influence, a code of knowledge and behaviour built up over generations. It is the intellectual property that belongs to all Māori women.

Māori women have a long history of trying to bring about changes in the provision of health services to Māori people (e.g. Māori Women's Welfare League). Services run by Māori for Māori may be more acceptable and appropriate for many Māori women. It is therefore important to raise the awareness of specific cervical screening services, that may be available in their own communities, and to ensure Māori women are fully informed of all available services.

Māori women are at the centre of their whānau, hapū and iwi and fulfill an important role in sustaining them. They are te wharetangata, the House of the People. The spiritual link between land and the health and wellbeing of Māori women is reflected in the language used to describe the functional anatomy of te wharetangata. The cervix is the doorway to te wharetangata. The relationship between women and land acknowledges that they carry the same role in terms of providing nourishment: without them humanity is lost.[10]

Me aro koe ki te hā o Hine-ahu-one
Pay heed to the dignity of women

PHO performance management programme and cervical screening

Cervical cancer screening coverage is included as one of the clinical indicators for the PHO performance management programme. This is important because regular cervical screening has been shown to result in a reduction in deaths from cervical cancer, but currently in New Zealand some women are receiving less than adequate levels of cervical screening.

The PHO indicator includes all women aged 20–69 years who have received a cervical smear in the last three years. A higher weighting is given to cervical smears performed in high needs women. High needs women is defined as an enrolee who is Māori, Pacific, or New Zealand deprivation deciles 9 or 10. The PHO enrolment database reports the New Zealand deprivation deciles 9 or 10 classification as Quintile 5.

The overall goal is to achieve a cervical screening rate of greater than 75% for all women. PHOs which initially have low coverage rates are expected to make more of an increase, than PHOs which start with coverage rates nearer to the target.

Make it count

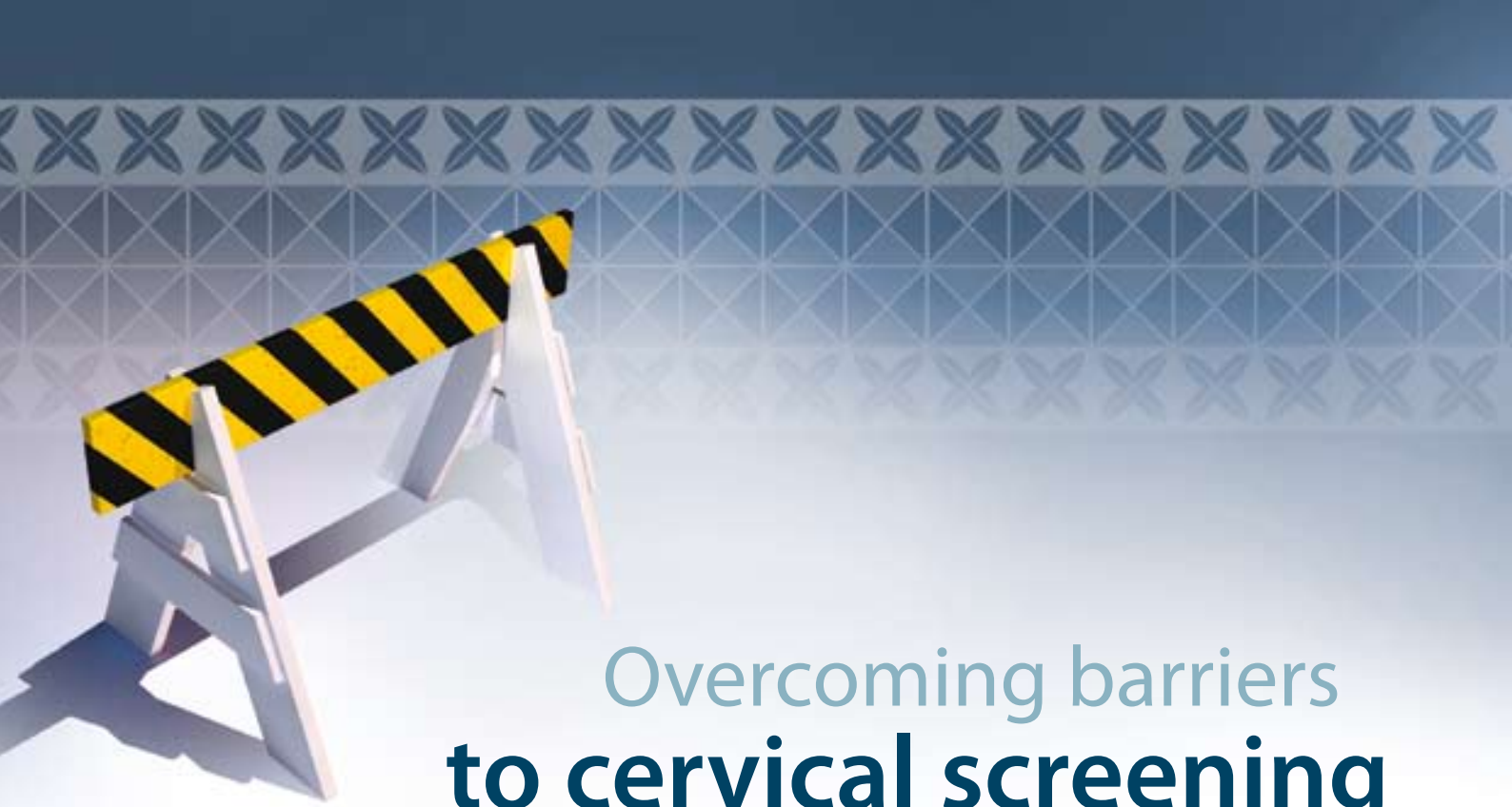
NHI number: Ensure the NHI number is included. A valid NHI means the data can be matched with the National Screening Unit (NSU) and PHO enrolment databases, to ensure it is counted.

Gender: If the gender is recorded as "unknown" the record is accepted, however it is converted to "male" and will not be counted.

Declines/withdrawals: Women who withdraw from the programme and opt off the national register are not counted. This should not affect achievement of targets, as they are based on improvement rather than absolute numbers.

Hysterectomy adjustment: Targets have been developed taking into account the estimated number of women who have had a hysterectomy. Again, this should not affect overall achievement of targets, as they are based on improvement, rather than absolute numbers.

The full PHO indicator definitions are available from: **www.dhbnz.org.nz/Site/SIG/pho/Programme_Documents.aspx**



Overcoming barriers to cervical screening

Barriers

There are many barriers that may explain the reluctance of some woman to attend for cervical screening. Some are common to all women, such as shyness or cost, while other reasons may be specific to some groups, such as cultural and language barriers.

Embarrassment/whakamā/shyness

For many women having a cervical smear is often associated with thoughts of nervousness, vulnerability and embarrassment.

Whakamā is thought to be one of the main barriers to screening for Māori women. This may be due to the strong Māori belief in the sacredness of te wharetangata, and a perceived insensitivity of smear takers.[11]

The association between sexual activity and cervical screening may make many women uncomfortable about presenting for a smear. Some women are also concerned that personal information of a sexual nature may not remain confidential. Traditionally Asian women are typically less open about their sexuality and are generally discouraged from expressing their sexuality until they are married.[12]

Many women are embarrassed by having a smear performed by a male GP. Most Māori women feel more comfortable with a Māori smear taker, although Pacific women generally prefer a non-Pacific smear taker, who they would be less likely to know through their social networks.

Cost

Cost is frequently a barrier to having a cervical smear.[11] There is considerable variation within New Zealand in the cost of having a smear taken. Other cost issues include transport, childcare and wages lost due to taking time off work. In many cases when money is an issue, a test such as a cervical smear, becomes a low priority.

For women who are not enrolled in a PHO, the cost of a consultation is not subsidised, and their consultation will be charged at the “casual” rate.

Cancer fear

Although the fear of cancer is a motivating factor for many women, it is also a significant deterrent for others,[11] as many would rather not know. Different cultures have their own belief systems about cancer and illness.[13] These may act as a barrier for cervical screening, e.g. the belief that cancer means certain death, and there is little that can be done to treat it.

Pain or discomfort

Many women identify the pain and discomfort of having a smear taken as a barrier.[11] This may include a number of factors, such as: pain and discomfort during the procedure, male doctors being less gentle than female doctors, and the invasiveness of the procedure.

Not knowing what to expect

Some women are discouraged by a previous negative experience of having a smear taken, especially if they feel the procedure was not explained properly, or they did not feel fully informed.[11] Furthermore, if these uncertainties are shared with friends or family/whānau, this barrier can become widespread.

New immigrants

Some new immigrants to New Zealand will not be familiar with cervical screening if they have not previously been exposed to a cervical screening programme. Although routine cytological screening is common in wealthier countries, most developing countries lack the infrastructure and trained personnel needed to provide a programme.

Approximately 25% of new immigrants report needing assistance with the health system, which may be further complicated by one in five rating their English language ability as moderate to poor.[14] One study found that in women immigrants to New Zealand not previously exposed to a cervical screening programme, not knowing where to go and not realising it is necessary, were the most frequently cited reasons for never having had a smear.[15]

While the values of European New Zealand women are generally aligned with New Zealand health practice, this is less likely for women with other cultural influences. Acculturation is the process of one cultural group adopting the beliefs and behaviour of another group, usually a minority group adopting habits and language patterns of the dominant group. When women have been in a country for some time and have become more acculturated, they are more likely to participate in cervical screening.

Cultural viewpoint of health

As a way of understanding more complex scientific and medical concepts, people may relate the information back to the things in their life they do understand.[16] Māori, along with many other indigenous people, often hold an overall view of health that is quite different to Western views. For example, common beliefs about cancer may include that it is contagious, it implies certain death and it may be a punishment, curse, payback or is predetermined. People may not seek treatment because they think they have the power to fight cancer through their beliefs, feelings and perceptions.

For some cultures, visits to health providers do not occur until there are symptoms. It is also commonly believed that primary prevention is achieved by the individual, through maintaining a good diet, achieving good spiritual balance and consuming herbs that promote health, rather than by attending a doctor when there are no symptoms of disease.

Overcoming barriers

Being aware of the barriers to cervical screening is not sufficient to overcome them. In addition to national initiatives (see sidebar), carefully targeted planning at a practice level is necessary to address disparities.

National initiatives to promote cervical screening

The importance of increasing cervical screening rates is recognised by the PHO Performance Management Programme. Most PHOs have initiatives to reduce the barriers to having a smear. Many of these initiatives include free smears for Māori and Pacific women and women from areas of high deprivation, as well as women overdue for smears, free women's clinics and mobile cervical screening services.

Recently the NCSP has initiated a significant social marketing campaign/health promotion programme, to educate and encourage Māori, Pacific and Asian women in particular, to have regular cervical smears.

Although the NCSP campaign can be expected to help reduce the disparities in cervical screening rates, there will continue to be women who remain unscreened or under-screened. General practice is in a unique position to encourage all women to participate in the NCSP.

The first step to increasing cervical screening rates is to invest time in becoming familiar with the needs of the local community and establishing the trust of the women being targeted.

Target the disparities. Currently European women are accessing cervical screening services at acceptable levels, and as a result have the lowest levels of cervical cancer and associated mortality. Resources should be targeted to the women who are not receiving cervical smears – Māori, Pacific and Asian women and women from areas of high deprivation.

Although we often refer to the "hard to reach" – it is more helpful to consider a broader range of reasons why women do not have regular cervical smears, e.g. women who are:

- Hard-to-find
- Unconvinced
- Uninformed
- Under-screened
- Undecided

Consider – are these people "hard to reach" or is your service "hard to use"?

Start with your practice population

Starting with your own practice population here are some practical steps:

- Perform a computer search to identify the women in your practice who have never had a smear or who are overdue for cervical smears
- Contact the National Cervical Screening Programme (0800 729 729) to check if smear has been performed by another smear taker, and to check screening histories and recall, if necessary
- Place an alert on a medical record, so the issue can be discussed when the patient next attends.
- Invite all women who are overdue by letter or telephone to participate
- Think of approaches relevant to your practice population

Invest time in building relationships and trust

"It comes down to the basics. Taking time to talk to women, listening to their concerns, respecting their wishes and building trust. It can take some women a long time to be ready, but it is important to keep building that relationship."
– Māori/Pacific Health Nurse

It may take several consultations for some women to feel comfortable about having a smear. Depending on their own personal experiences, some women may never be ready. It is important to acknowledge concerns and fears and provide clear information about the procedure.

Mā te rongo, ka mohio; Mā te mohio, ka mārama; Mā te mārama, ka mātau; Mā te Mātau, ka ora

Through listening comes awareness,
Through awareness comes understanding,
Through understanding comes knowledge,
Through knowledge comes life and well-being.

Make it a positive experience

Emphasise how cervical screening benefits both the patient and their family/whānau. Validate their decision to participate and encourage them to encourage others. It is also important that women are made to feel as comfortable as possible about the process. Women who have had a positive experience are more likely to return.

Ensure women feel prepared

It is important to take the time to ensure the woman is well prepared, relaxed and comfortable prior to the collection of the cervical smear. Important things to consider are:

- Don't rush the woman, if she's not ready this time, talk to her in preparation for the next time
- Encourage her to ask questions to gauge her understanding of the information you are sharing
- Reduce her discomfort by giving her time to absorb the information
- Ask her if she would like to see the speculum. Allow her to hold it if she chooses, and explain how it will be used
- Let her know she can have a support person
- Tell her to let you know if she is uncomfortable during the procedure

Make it less embarrassing

Give women a choice of smear taker and ensure they know the process is confidential. This may be difficult if they know someone in the practice, especially if they are worried that aspects of their sexuality may be revealed.

It is important to take practical steps to reduce embarrassment or vulnerability while the smear is being collected. This may include:

- Being covered while lying on the bed
- Pulling curtains around the bed
- Ensuring the environment is relaxed (e.g. pictures, warm room, music)
- Offering different positions to lie in
- Offering disposable plastic speculums
- Warming the speculum

Provide culturally appropriate smear takers

Some Māori and Pacific health providers have smear takers and may provide a free or low cost service. Cervical Screening health promoters, specialising in the promotion of screening to Māori, Pacific and Asian women, are also available in most areas (contact your DHB) and will be able to assist practices in ensuring their services are culturally appropriate. It may be worth exploring what options for smear taker training are available in your area. Depending on availability, nurse smear taker training is free.

Language difficulties are a major barrier for many Asian and some Pacific and Māori women, therefore it is important to provide access to a smear taker with appropriate language skills where possible, otherwise the use of an interpreter is encouraged. Many PHOs are

clients of Language Line, a telephone interpreting service managed by the Office of Ethnic Affairs. If the service is available through the PHO, an interpreter can be available via the telephone almost immediately. There are 40 different languages available with a choice of gender of the interpreter.

Communicating results

Reassure patients that an abnormal cervical smear result rarely indicates cancer but rather screening provides the opportunity to detect early changes and if necessary, treatment can be initiated.

Ask patients about their preferred method of receiving results. They may wish that even normal results are communicated to them. Make sure the patient knows when their next smear is due and place a recall on your PMS.

Tell the patient that they may be asked to return for a repeat test if the smear is reported as unsatisfactory. The rate of unsatisfactory smears is likely to be less with the advent of liquid based cytology.

Cost solutions

In recognition of cost being a major barrier for many women, most PHOs have initiatives in place to provide free or low cost cervical screening, for targeted populations.

Māori/Pacific providers, family planning clinics and practice nurses may offer a low cost or free alternative. Become familiar with the services available in your area and ensure the patients who would benefit most have access to these services.

Competencies for Smear Taker Training is a new document available.[17] This has been developed by the NCSP in consultation with stakeholders and replaces the NCSP Training Standards for Smear Takers 2002. It is available from **www.nsu.govt.nz**

September is Cervical Screening Awareness Month

The NCSP is promoting September as "Cervical Screening Awareness Month".[18] This is a national initiative, supported by the New Zealand Cancer Society and the Family Planning Association. There will be an increased awareness of the cervical screening programme, providing the opportunity for local promotional activities, supported by national advertising and promotions.

The aim is to get women talking about the benefits of cervical screening, and to encourage the women around them to have a cervical smear.

All clinics and smear-taking practices throughout New Zealand have been provided with promotional resources for the month, including:

- Awareness Month posters for display in practices during September
- Free 30 mL hand and body lotion samples for distribution to women who have their smear test during September (while stocks last)
- Miniature promotional stands for display on reception counters

Resources

For information about cervical screening in New Zealand visit: **www.cervicalscreening.govt.nz**

Resources are available to help inform women about cervical screening and encourage participation.

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Quiz feedback: How to treat acne

In BPJ 20 (April 2009) we published an article on the treatment of acne. GPs were invited to complete a quiz on this topic and the responses were discussed with our GP panel. **Dr Amanda Oakley**, Specialist Dermatologist and Clinical Associate Professor, Tristram Clinic, Hamilton, provided expert commentary on several key issues that were highlighted.

A full version of the quiz feedback can be found online at **www.bpac.org.nz** (search by Publication/CME quiz feedbacks)

The psychological impact of acne

People are affected psychologically to varying degrees by their acne – some have severe acne and seem not to be bothered by it at all and do not even raise it as a concern (especially young males) and some have only a few lesions on their face but are significantly depressed and anxious because of this.

Should doctors be actively asking patients if they want to treat their acne?

Regardless of the number or severity of lesions, acne that causes significant psychological distress is classified as "severe acne".

It is always useful to ask how much the acne is bothering the patient, to determine its perceived severity and the likely adherence to potentially tedious and long term treatment regimens. Patients are usually relieved to be asked how the acne affects them and will readily admit to low self-esteem. Questions may reveal significant embarrassment, withdrawal from social encounters, family friction and clinical depression.

The Cardiff Acne Disability Index (CADI) is a five item questionnaire aimed at adolescents and young adults.[1] It is simple to use, however results do not always correlate with clinical acne severity.

Question 1 asks whether acne has induced negative feelings such as aggression, frustration or anger.

Question 2 asks whether the acne has interfered with social encounters.

Question 3 asks whether the acne has prevented swimming. This question might be altered to include other sports with communal changing rooms.

Question 4 asks about the effect of the acne on the patient's feelings i.e., the degree of concern or depression caused by it.

Question 5 asks about the patient's assessment of severity.

The CADI is available online from several sources including: **www.dermatology.org.uk/quality/quality-cadi.html**

Does junk food cause acne?

Myths about junk food causing acne are still prevalent among young people and their parents. Is there any evidence of a link between certain foods and cause or exacerbation of acne? Also, are there any dietary sources that are beneficial?

Food may influence acne. Acne is absent or much less common in some rural populations than in Westernised urban environments. Some studies have suggested this may be related to dairy products, perhaps because of hormones in milk. Others have evaluated the role of high-glycaemic foods, fat intake or fatty acid composition. Acne is associated with polycystic ovaries and insulin resistance may also play a role.[2]

It is difficult to know how to advise patients. We should probably at least encourage a low-glycaemic, low-fat diet. The Stone Age "hunter-gatherer" diet has been reported to be beneficial. But these diets are difficult for New Zealand teenagers to follow.

Which OTC topical acne products are best?

What advice can a GP give to a patient for selecting an OTC medicated product?

Over-the-counter acne medications may be effective, well tolerated and cosmetically elegant for some patients. But good information about these ingredients is hard to find.

I advise basing your recommendations on products containing benzoyl peroxide. This has comedolytic, keratolytic and anti-inflammatory action. It is available as wash-off or leave-on lotions, gels and creams in various concentrations and is priced from $20 to $30 for 40 g (about one months supply). The low concentrations (2.4 – 4%) are just as effective as higher strength products, and are less irritating.

Salicylic acid (beta hydroxy acid) remains popular and can be found in cleansers and leave-on treatments. It has mild comedolytic and anti-inflammatory effects, but may cause irritant dermatitis (like benzoyl peroxide and topical retinoids). Other useful components include glycolic acid (alpha hydroxy acid), azelaic acid, resorcinol, sulphur and sodium sulfacetamide. Antiseptics such as triclosan are popular as cleansers. Zinc, retinoic acid, niacinamide, tea tree oil, green tea and ayurvedic therapies also are frequently used.

The cost of topical acne treatments

Although topical treatments such as benzoyl peroxide or adapalene are most appropriate for treating mild acne, often GPs consider prescribing doxycycline or other oral antibiotics due to cost issues. The topical treatments are not subsidised and can be unaffordable to many people. What is the advice on this?

Although topical acne therapy remains unsubsidised, we must tell our patients that it is recommended and likely to be of benefit. They are often already spending a great deal of money on remedies of dubious benefit. Ask them!

Antibiotics do not take care of comedones, which should be managed initially with topical benzoyl peroxide and/or topical retinoids. New combination topical products will enhance compliance and results (e.g., benzoyl peroxide / clindamycin and benzoyl peroxide / adapalene). Topical antibiotics as sole treatment are not recommended due to lack of efficacy and bacterial resistance.

Systemic antibiotics are warranted if there are many or deep inflamed lesions, but they do not work any faster than topical treatment. Combined oral contraceptive agents, especially those with antiandrogens such as cyproterone or drospirenone, are effective for women with seborrhoea and mild to moderate acne.

Doxycycline: when to expect to see results

How long does a patient have to take doxycycline until improvement is seen? A "significant improvement" from the patient's perspective is often different from that of the doctor. Once improvement has been achieved, should doxycycline be tapered or stopped?

Whatever drug is being studied, improvement occurs steadily but slowly, plateauing at about six months. There is probably little benefit reviewing before three months treatment has been completed, except to encourage compliance and to manage adverse effects. But many patients achieving 60% reduction in the number of spots

report "no benefit" from the treatment as their expectation is for complete clearance.

Dose tapering has not been well studied in acne. It is useful in rosacea, but rosacea responds much more quickly and completely to doxycycline in most cases.

I favour using doxycycline 100 mg daily until acne clears and then stopping, rather than small doses for six months. We need to balance efficacy in an individual with increasing bacterial resistance in the community. But topical therapy must be continued as maintenance therapy. If significant acne recurs, it is probably time to consider oral isotretinoin.

The role of minocycline

What is the role of minocycline in the treatment of acne?

The most common oral antibiotics for treating acne vulgaris are the tetracycline derivatives, although erythromycin, trimethoprim, co-trimoxazole and clindamycin have also been used extensively. The rationale is the effect on *Propionibacterium acnes* as well as the intrinsic anti-inflammatory properties of these antibiotics. Sensitivity of *Propionibacterium acnes* to erythromycin is lower than to doxycycline or minocycline.

Doxycycline (100 mg) is fully subsidised and effective if taken regularly in doses ranging from 50 mg to 200 mg daily. Adverse effects are common but rarely serious (nausea, oesophagitis, photosensitivity).

Minocycline is considered second-line. It is partially subsidised. It may be more effective than doxycycline in patients that forget to take their pills, as it is thought to stay in the sebaceous glands for several days. It has some serious potential adverse effects (dizziness, hypersensitivity reactions, hepatitis, lupus erythematosus, long-lasting bluish pigmentation), but these are very rare.

Confidence in prescribing isotretinoin

Prescribing isotretinoin for acne with the aid of a decision support tool such as bestpractice Decision Support allows prescribers to feel confident that they are covering all required aspects and are prescribing safely. However it is unclear at this stage how to fulfil the medico legal requirements of prescribing isotretinoin. Does a training course need to be completed?

Bpac comment: On the Special Authority application form for isotretinoin, the prescriber has to indicate that they: "have an up to date knowledge of the treatment options for acne and are aware of the safety issues around isotretinoin and are competent to prescribe isotretinoin". No specific training course currently exists. In order to fulfil this Special Authority requirement, the GP must be competent to undertake the treatment in the same way as for any other clinical situation. It is the responsibility of the individual to familiarise themselves with isotretinoin and the treatment of acne. It is strongly recommended that a decision support prescribing tool is used.

It takes a long time to become expert and comfortable prescribing isotretinoin. Dermatologists in training are closely supervised for four years and may treat hundreds of patients with this drug. The route to acne-clearance is rarely straightforward, requiring dose adjustment and interruption, and careful management of adverse effects. The patient should be prepared for this. Prolonged consultations and careful follow-up are necessary. It is best to have a working relationship with a local dermatologist; but many dermatologists are struggling to accept the changed prescribing environment and prefer to see the patient themselves.

Myths and legends abound. Many patients with acne and /or their parents are misinformed and may demand or refuse appropriate treatment. Proceed with care!

The teratogenicity of isotretinoin

In the special authority criteria for isotretinoin, it states that the patient must agree not to become pregnant during the course of treatment. Can a patient be trusted if they say they are not sexually active or is it simply not good practice to prescribe isotretinoin if a patient refuses oral contraceptives?

I am nervous every time I prescribe isotretinoin to a female. It is essential to ensure she understands the implication of pregnancy. I obtain signed consent. I talk about sexual activity, improved self esteem leading to new relationships, pregnancy testing, contraception, emergency contraception, rape and termination of pregnancy. I make sure she knows who to call or email for further advice. I do not prescribe if I am not convinced she can be relied on. But mistakes happen and you have to be prepared for that.

The overall risk of birth defects is estimated to be up to 30% after exposure during embryogenesis. The burdensome iPLEDGE system in the USA may not have reduced the numbers of pregnancies. Pregnancy testing does not prevent pregnancy. Studies have shown that some exposed pregnant women did not receive counselling. Some women did not use contraceptives due to motivational, cultural and religious barriers.

"Yet because acne is so horrific and so common, even the most conservative risk/benefit analysis finds that, overall, isotretinoin provides far more benefit than risk."[3]

Final word

As GPs become more familiar with treating acne in primary care, what insights can we gain from our dermatologist colleagues?

As for any other medical condition, it is important to take a thorough history and examine the patient carefully. Determine the severity and impact of the acne, and the result of treatment to date. Explain a stepwise and multipronged approach to treatment. Encourage adherence to your recommendations and provide a listening ear.

Isotretinoin is very effective but it should be reserved for severe, treatment-resistant or very persistent acne in well-motivated patients. It is best not to prescribe it yourself unless you are thoroughly informed and are managing numerous patients with acne.

Normally, in mild acne, on the first visit you should be discussing cleansers, and prescribing topical benzoyl peroxide. On review, if necessary add retinoid +/- topical antibiotic. Never prescribe an antibiotic alone – it will not work and you will encourage bacterial resistance. Continue follow-up.

In more extensive or inflammatory acne, use topical benzoyl peroxide (less expensive) or retinoid or both (combined preparation now available) together with oral doxycycline. Review to ensure compliance.

In female patients, you may decide to use "the pill". Low-dose combined oral contraceptives with minimal androgen effect contain ethinylestrodiol and desorgestrel, gestodene or norgestimate. If there is clearly an indication for an antiandrogenic progesterone (e.g., polycystic ovarian disease, hirsutism), the choice is between cyproterone and drospirenone. The latter is not subsidised but may be better tolerated. Evaluate the effect after six months.

For women in whom oestrogens are contraindicated, spironolactone may be a better choice of anti-androgen and may be combined with progesterone-only contraceptive.

Acknowledgement

Thank you to Dr Amanda Oakley and our GP Discussion panel – Dr Neil Whittaker, Dr Susie Lawless and Dr Janine Bailey.

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All about Proton Pump Inhibitors (PPIs)

- It is clear that patients who take PPIs unnecessarily or for unclear indications should be advised to stop. However some patients may experience "rebound hyperacidity" when PPIs are ceased after two months use or more.[1, 2] This can be managed using antacids as rebound hyperacidity may also occur after stopping histamine (H2)-blocker therapy.
- Reassure your female patients - a recent meta-analysis of seven studies involving over 1500 women concluded that PPIs can be used safely during pregnancy. PPI use was not associated with elevated risk for major congenital malformations, spontaneous abortions, or preterm delivery. Omeprazole accounted for 87% of the PPI exposures in this study.[3]
- Several recent studies have suggested that there is a potential interaction between some PPIs and clopidogrel. It has been reported that omeprazole, lansoprazole and rabeprazole (not available in New Zealand) adversely affect the anti-platelet activity of clopidogrel but pantoprazole does not show this effect.[4] H2-receptor blockers (e.g. ranitidine) and antacids are not associated with this interaction. Medsafe in New Zealand is currently reviewing this possible medicine interaction. In the meantime Medsafe has advised that clopidogrel datasheets will be updated to include this precaution. Prescribers should review patients using clopidogrel and ensure that PPIs are not used concomitantly unless absolutely necessary.
- As of 1st September, patients can purchase Losec (omeprazole) 10 mg tablets over-the-counter at pharmacies.

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Fire hazard with paraffin based skin products

Bandages, dressings and clothing in contact with paraffin based skin products e.g. liquid paraffin, white soft paraffin, emulsifying ointment are easily ignited with a naked flame or cigarette. Ensure that patients and caregivers are aware of this potential fire risk when prescribing these products.

For more information visit: www.npsa.nhs.uk/nrls/alerts-and-directives/rapidrr/paraffin-skin-products

Do you have a brilliant idea that you would like to share with your colleagues? Can you tell us about a mistake that you have learnt from so others don't fall into the same trap? What's new in primary care that people would want to know? Share your practice tips with us. Email: **editor@bpac.org.nz**

No Clear Benefit for Aspirin in Primary Prevention of Cardiovascular Disease

Journal Watch, Vol. 29, No.14, July 15, 2009

Risks might outweigh benefits in patients who receive other modern preventive therapies.

In patients with histories of symptomatic occlusive vascular disease, aspirin's ability to prevent future adverse cardiovascular events significantly outweighs its association with elevated risks for major bleeding. However, use of aspirin for primary prevention of occlusive vascular disease remains controversial; prospective trials and meta-analyses have shown no overall net benefit for aspirin and have lacked power to identify subgroups of patients in which aspirin's benefits might significantly outweigh its risks.

To overcome these limitations, investigators pooled individual patient data from six large prospective primary prevention trials of aspirin, in which 95,000 patients without diabetes and without histories of occlusive vascular disease were randomised to receive aspirin or no aspirin for two or more years.

Compared with patients who received no aspirin, aspirin recipients had a statistically significant but a very small absolute reduction (0.51% vs. 0.57% annually) in serious vascular events — primarily major adverse coronary events and strokes — with no significant differences among patient subgroups and no differences in mortality. Patients who received aspirin suffered significantly fewer ischemic strokes but significantly more hemorrhagic strokes, with no significant net effect on stroke incidence. Patients who received aspirin experienced significantly more major extra-cranial bleeds (0.10% vs. 0.07% annually).

Comment

Although these data suggest a possible small net benefit for aspirin in patients without histories of symptomatic occlusive vascular disease, the authors point out that few of these patients had the benefits of statins and other modern, low-risk, primary preventive interventions, and they suggest that the benefits of adding aspirin to these newer therapies no longer outweigh the risks.

— Bruce Soloway, MD

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How much evidence do we need to change practices in which we firmly believe?

Journal Watch General Medicine July 30, 2009

http://general-medicine.jwatch.org

Enough already! Randomised trials show that tight glucose control in patients with long-standing type 2 diabetes isn't beneficial.

Should the glycosylated haemoglobin (Hb_{A1c}) level goal in patients with long-standing type 2 diabetes be 7%? 6.5%? Lower? Although many clinicians believe in tight control for patients with type 2 diabetes, recent studies suggest that this practice is not beneficial. Several recently published commentaries cite evidence that challenges current beliefs and practices.

In the first major trial (done in the 1960s) of tight glucose control in patients with type 2 diabetes, oral glucose-lowering agents were associated with higher cardiovascular

mortality and no differences in microvascular complications compared with placebo.[1] Insulin also was not associated with clinical benefit.

In three recent large randomised trials (ACCORD,[2] ADVANCE[3] and VADT[4]), tight control in patients with long-standing type 2 diabetes did not lower overall mortality, cardiovascular-related mortality, stroke, amputations or even clinical (as opposed to surrogate) microvascular endpoints. Differences in specific outcomes in these trials might be related to different treatments or to duration of diabetes in participants. In some studies, fewer intensively treated patients reached composite outcomes (such as "any diabetes complications"), but the bulk of improvement was in nonclinical outcomes (e.g., incident albuminuria). Tight control was associated with severe hypoglycemia and weight gain. In the UKPDS study,[5] published a decade ago, non-obese intensively treated participants with newly diagnosed type 2 diabetes were less likely to reach microvascular endpoints (including "need for photocoagulation", but not visual loss) but showed no difference in mortality (cardiovascular, diabetes-related, or all-cause) compared with non-obese control patients. Among obese participants, metformin alone lowered long-term mortality and myocardial infarction rate, but sulfonylureas and insulin did not; tight control did not lessen risk for microvascular complications. Metformin and sulfonylureas in combination were associated with excess diabetes-related deaths and all-cause mortality.

Because trials do not support tight control and because of the cost, burden, and harms associated with tight control, we should be emphasising cardiovascular risk reduction (particularly control of blood pressure and cholesterol levels) and healthy lifestyles for patients with type 2 diabetes.[6] Several groups of editorialists suggest aiming for Hb_{A1c} levels of 7.0% or 7.5% in patients in whom this goal is achievable with one medication and adjusting this target for others based on symptoms, side effects, treatment burden, and patient values and preferences.[6,7,8] Commentary authors suggest that the Hb_{A1c} goals for practice guidelines should not be <7% and that, to encourage individualised treatment, performance measures should set an upper limit (e.g., 9%) rather than a lower limit (e.g., <7%).[7]

Comment:

Randomised trial results often are not available to answer important clinical questions. In this case, they are. We shouldn't ignore them. Many clinical trials are completed that show benefits, and much time passes, before new treatments are adopted; similarly, many trials that show lack of benefit, or even harm, might be required before clinicians abandon ineffective practices that have become routine. Haynes and Haynes ask, "What does it take to put an ugly fact through the heart of a beautiful hypothesis?" and they quote poetry: "The chains of habit are too weak to be felt until they are too strong to be broken."[9] Social psychology literature suggests that people cling to belief even in the face of mountains of evidence to the contrary. But, as physicians and scientists, we should embrace change when new evidence consistently contradicts our prior beliefs and clinical practice.

— Richard Saitz, MD, MPH, FACP, FASAM

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ACE Inhibitors and ARBs for Preventing Nephropathy and Retinopathy in Type 1 Diabetes

Journal Watch, Vol. 29, No.15, August 1, 2009

Treatment did not prevent nephropathy but did prevent development of retinopathy.

Many clinicians believe that inhibition of the renin–angiotensin system prevents diabetic nephropathy. Similarly, researchers have hypothesised that such inhibition could prevent diabetic retinopathy. In this international randomised trial, investigators enrolled 285 normotensive patients with type 1 diabetes (mean glycosylated haemoglobin level, 8.5%; mean diabetes duration, about 11 years) and normal urine albumin levels. Patients received daily enalapril (an ACE inhibitor 20 mg), losartan (an angiotensin-receptor blocker [ARB]; 100 mg), or placebo; follow-up was five years. The primary endpoints were nephropathy (change in mesangial fractional volume, as measured by renal biopsy) and retinopathy (progression of two steps or more on a retinopathy scale, as measured by retinal photos).

At five years, the investigators found no significant differences among the groups in the primary renal endpoint or in glomerular filtration rate changes. However, cumulative incidence of microalbuminuria was significantly higher in the losartan group (17%) than in the enalapril group (4%) or the placebo group (6%). Placebo recipients were significantly more likely to reach the retinopathy endpoint (38%) than were enalapril recipients (25%) or losartan recipients (21%); the difference remained significant after controlling for blood pressure.

Comment

In this randomised trial, neither enalapril nor losartan prevented histologic progression of nephropathy among normotensive patients with type 1 diabetes and normoalbuminuria. This study is notable for longer follow-up than prior studies and for the use of biopsies to monitor nephropathy. According to the editorialists, these results trump prior results and should lead to elimination of ACE-I or ARB use for preserving kidney function in normotensive type 1 diabetic patients with normoalbuminuria and to re-evaluation of current protocols for managing microalbuminuria in patients with type 1 or 2 diabetes. However, among patients like those in this study (most of whom had minimal or no retinopathy), treatment with an ACE-I or ARB might prevent progression of retinopathy; studies have not shown a benefit of such therapy for type 1 patients with established retinopathy or in type 2 patients with or without retinopathy.

— Jamaluddin Moloo, MD, MPH

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Tanning Beds Are Human Carcinogens: Report from the WHO International Agency for Research on Cancer

Journal Watch Dermatology July 30, 2009

http://dermatology.jwatch.org

Tanning beds cause skin and eye cancer.

The International Agency for Research on Cancer (IARC) is a section of the World Health Organisation whose mission is to develop strategies for cancer prevention and control. In June 2009, the agency convened a working group of 20 scientists to reassess the carcinogenicity of various sources of radiation. In the past, the IARC has found sufficient proof that solar radiation is a human carcinogen involved

in the development of basal cell carcinomas, squamous cell carcinomas, and melanoma. The current working group now finds unquestionable evidence that UV-emitting tanning devices cause melanoma of the skin and of the choroid and ciliary body of the eye. This determination is based on a comprehensive meta-analysis finding that risk for cutaneous melanoma increases by 75% when tanning device use begins before age 30, compared with non-use (Int J Cancer 2006;120:1116). The group also cites case-controlled studies showing increased incidence of ocular melanoma among users of UV-emitting tanning devices (Int J Cancer 2004;112:896). Their conclusions are supported by mechanistic studies in animal models that show a cytidine-to-thymidine transition in DNA caused by UVA radiation. In humans, this mutation is found in TP53 in premalignant solar keratosis and in malignant skin tumours.

Comment: This report, by a highly respected independent group, validates what dermatologists have known for a long time - that tanning beds cause melanoma. Tanning beds now justly take their place along with x-rays and gamma rays at the forefront of radiation carcinogens. Physicians can cite the IARC conclusions when counselling patients, especially younger individuals, about the hazards of tanning bed use.

— Craig A. Elmets, MD

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Video Decision-Support Tool Is Effective for Advance Care Planning in Dementia

Journal Watch, Vol. 29, No.13, July 1, 2009

A video depiction of advanced dementia persuaded older people to choose comfort care.

Visual images can enhance healthcare communication and decision making. In this randomised trial, Boston investigators determined the effects of a video decision-support tool on older people's (age, ≥65) preferences for future care if they develop advanced dementia.

The 106 people in the control group heard a narrator describe advanced dementia; the 94 people in the video group listened to the narrative and also watched a two minute video depiction of an 80-year old woman who had advanced dementia and could not communicate, ambulate, or feed herself. Of the control patients, 64% chose comfort care, 19% chose limited care and 14% chose life-prolonging care (3% were uncertain). Of the video group, 86% chose comfort care, 9% chose limited care and 4% chose life-prolonging care (1% were uncertain); these differences were statistically significant. Knowledge of dementia was significantly greater in the video-intervention group than in the control group. Furthermore, after six weeks, significantly more participants in the control group than in the video group changed their preferences (29% vs. 6%).

Comment

Unsurprisingly, older people who hear and watch depictions of advanced dementia are more likely to prefer comfort care for dementia and have more stable preferences than patients who only hear a description of dementia. These results should encourage development of video decision-support tools for other scenarios (e.g., life-sustaining technologies such as haemodialysis, left ventricular assist devices).

— Paul S. Mueller, MD, MPH, FACP

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The video described here can be found free of charge at: www.acpdecisions.com/acpdecisions/videos.html

Flucloxacillin and impetigo

Dear bpac,

Are there any alternatives for impetigo when flucloxacillin is rejected because of taste?

GP, Northland

Oral antibiotics are recommended for the treatment of impetigo if there is extensive or severe infection (including systemic symptoms), areas on which it would be impractical to use topical drugs, or if there is bullous impetigo.

Despite its taste, flucloxacillin is strongly recommended as first line antibiotic treatment.[1] It is a relatively safe medication to use in children, and as it is a narrow spectrum antibiotic, it does not contribute to increasing bacterial resistance.

Given the potential for bacterial resistance and adverse effects with other options, parents should be encouraged to persevere with giving flucoxacillin (unless allergic). PHARMAC has produced a leaflet with tips for administering an unpalatable medicine – "Practical tips for giving medicine to kids". Available from: www.pharmaconline.co.nz/physicalproductdetails.aspx?id=3143

There are two main options if attempts to use flucloxacillin have failed or if the child is pencillin allergic - erythromycin or cefaclor, although both have disadvantages. Erythromycin increases reistance in the target bacterium and cefaclor is associated with adverse skin and joint reactions in children.[3] It may be tempting to use amoxicillin clavulanate but it is not recommended as empiric treatment as use of broad spectrum antibiotics increases bacterial resistance in the community.

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