module 231

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Welcome to the two hundred and thirty first module in the *Pharmacy Magazine* Continuing Professional Development Programme, which looks at actinic keratoses and skin cancer.

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forthismodule

GOAL:
To provide an update on the treatment of actinic keratoses and skin cancer.

OBJECTIVES:
After completing this module you should be able to:

- Describe the clinical features of skin cancers and actinic keratoses
- List risk factors for the development of skin cancers
- Identify high-risk groups.

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CPD

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Actinic keratoses and skin cancer

Contributing author: Dr Christine Clark, clinical pharmacy writer specialising in skin conditions

Introduction

Although it is not part of a community pharmacist's remit to diagnose skin cancer, community pharmacies are often the first port of call when someone finds a suspicious lesion on his/her skin.

It is therefore important to understand the differences in prognosis and management for the various lesions that might be encountered. In addition, understanding of the risk factors for the development of skin cancer has increased considerably in recent years and, in this regard, pharmacists are in a good position to identify and advise the high-risk groups and deliver healthy living messages to the general public. This module covers the management of primary skin cancers and actinic keratoses.

Numbers diagnosed

Traditionally, interest has focused on melanomas, which, although relatively rare, can metastasise and be fatal. In 2010, around 100,000 people were diagnosed with skin cancer in the UK. More than 12,000 of these cancers were malignant melanoma. Each year, more than 2,200 people die from skin cancer[1].

In recent years awareness of non-melanoma skin cancers (NMSCs) has grown considerably and these are now recognised as a growing health-economic burden. This group includes squamous cell carcinomas (SCCs), basal cell carcinomas (BCCs) and the pre-cancerous actinic keratoses (AKs).

This group vastly out-

numbers the melanoma group. In fact BCCs alone are estimated to account for 80 per cent of skin cancers and NMSCs for around a third of all cancers detected in the UK. In England over 69,000 people were registered with NMSCs in 2007 but, due to incomplete registration, the actual number of cases may be over 100,000[2]. NMSCs are commonest in the older age groups, typically those over 70 years of age.

Actinic keratoses are very common. In the UK, 15 per cent of men and 6 per cent of women have AK, with the prevalence increasing to 34 and 18 per cent respectively in those over 70 years of age[3]. Actinic keratoses are managed with a number of topical agents, bringing their own demand for support and advice.

Melanoma

Melanoma (also known as malignant melanoma [MM]) is relatively rare, representing about 10 per cent of skin cancer cases, but it is becoming commoner. In the UK approximately 13,000 new cases are diagnosed each year[4].

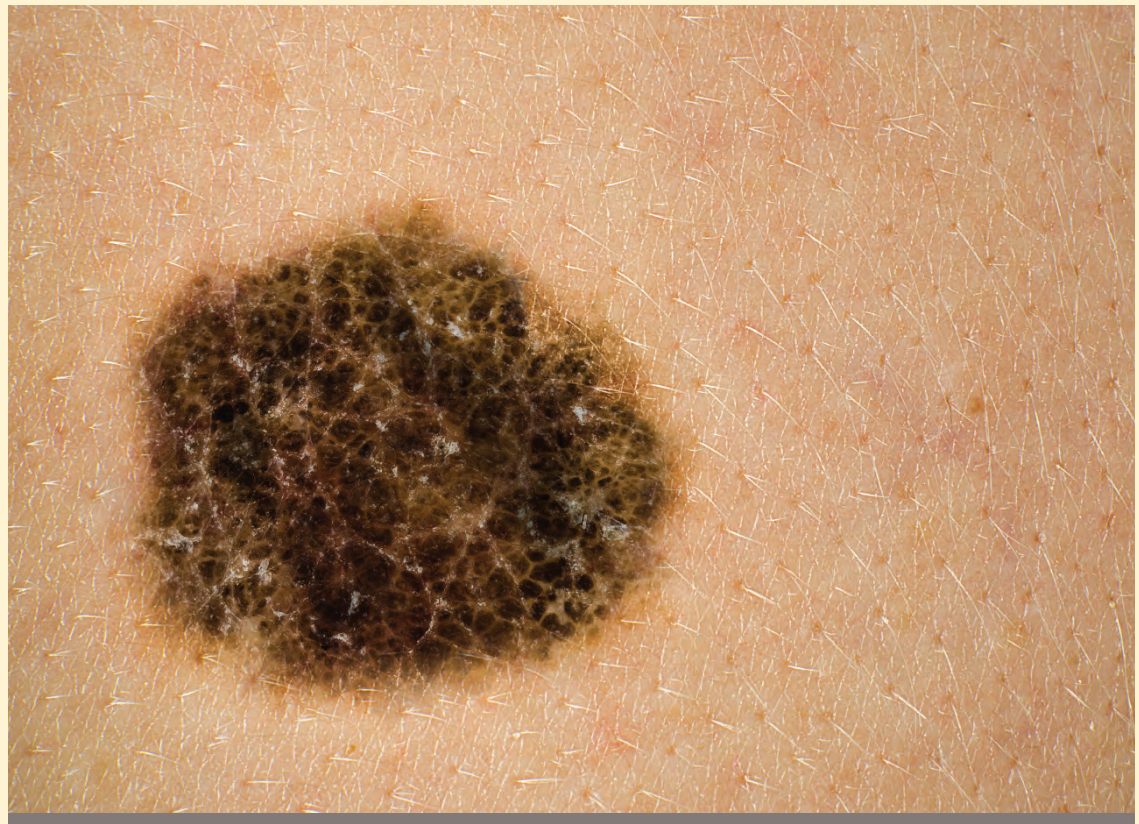
Melanoma is one of the commonest cancers in people aged 15-34 years and more than a third of cases occur in people under 55 years of age[4]. About 20 per cent of MM patients develop metastases that lead to death. In the UK more than 2,000 people die every year from MM. In general, the mortality rate is only slowly increasing because of early detection.

A melanoma is a cancerous growth of melanocytes from the basal layer of the epidermis. Non-cancerous growth of melanocytes causes melanocytic naevi (moles) and ephelides (freckles).

The commonest sign of melanoma is the appearance of a new mole or a change in an existing mole, most commonly on the back, legs, arms or face, although it can be on any part of the body.

Melanomas usually have an irregular shape and uneven colouring. They may also be larger than normal moles and can sometimes be itchy or bleed.

The 'ABCDE' (or ABCD) checklist [see box] has been developed to help differentiate between a normal mole and a melanoma. Normal moles are usually round or oval with a smooth edge and are no more than 6mm (1/4 inch) in diameter.



Malignant melanoma – relatively rare but potentially deadly

ABCDE melanoma checklist

- **A**symmetrical: melanomas have two very different halves and an irregular shape
- **B**order: melanomas have an uneven or ragged border
- **C**olours: melanomas will be a mix of two or more colours
- **D**iameter (or Dynamic): melanomas are larger than 6mm (1/4 inch) in diameter
- **E**nlargement or Elevation: a mole that grows over time or is raised is more likely to be a melanoma

Risk factors

The main risk factors for the development of MM are:

- Genetic predisposition
  - pale skin that does not tan easily
  - red or blonde hair
  - blue eyes
  - a large number of freckles or moles
  - previous diagnosis of skin cancer
  - close relative with MM
- Exposure to UV radiation – including natural sunlight and sunbeds; repeated episodes of sunburn
- Age
- Immunosuppression, including diseases such as HIV, and immunosuppressive drugs such as anti-rejection drugs after organ transplants.

The main treatment for melanoma is surgical removal of the lesion. If treated at an early stage, this is usually successful. However there is a chance that there may be a recurrence later, so patients are taught to examine their skin and lymph nodes and to adopt sun-protection measures to minimise the risk.

Squamous cell carcinoma

Squamous cell carcinoma (SCC) starts in the superficial layers of the epidermis. SCC accounts for about 10 per cent of all cases of skin cancer and about 4 to 5 per cent of cases metastasise if left untreated[5]. Although the exact cause is unknown, SCC is linked with over-exposure to ultraviolet (UV) light. It is important to note that the majority of SCCs (60-80 per cent) start as AKs.

A SCC appears as a firm pink lump and may have a flat, scaly and crusted surface. The lump

is often tender to the touch, bleeds easily and may develop into an ulcer. The main risk factor is cumulative UV exposure[5].

Basal cell carcinoma (rodent ulcer)

Basal cell carcinomas (BCCs) arise from the basal cells of the epidermis and usually appear in sun-exposed areas. They account for about 80 per cent of all cases of skin cancer[6]. Although BCCs rarely metastasise, they can be locally destructive. The main causes are exposure to UV radiation as well as genetic predisposition.

BCCs usually appear as small red or pink lumps, although they can be pearly-white or 'waxy' looking. A BCC can also look like a red, scaly patch. The lump grows slowly and may become crusty, bleed or develop into a painless ulcer. The main risk factors include genetic predisposition (fair skin, red hair, large number of moles), UV exposure and immunosuppression.

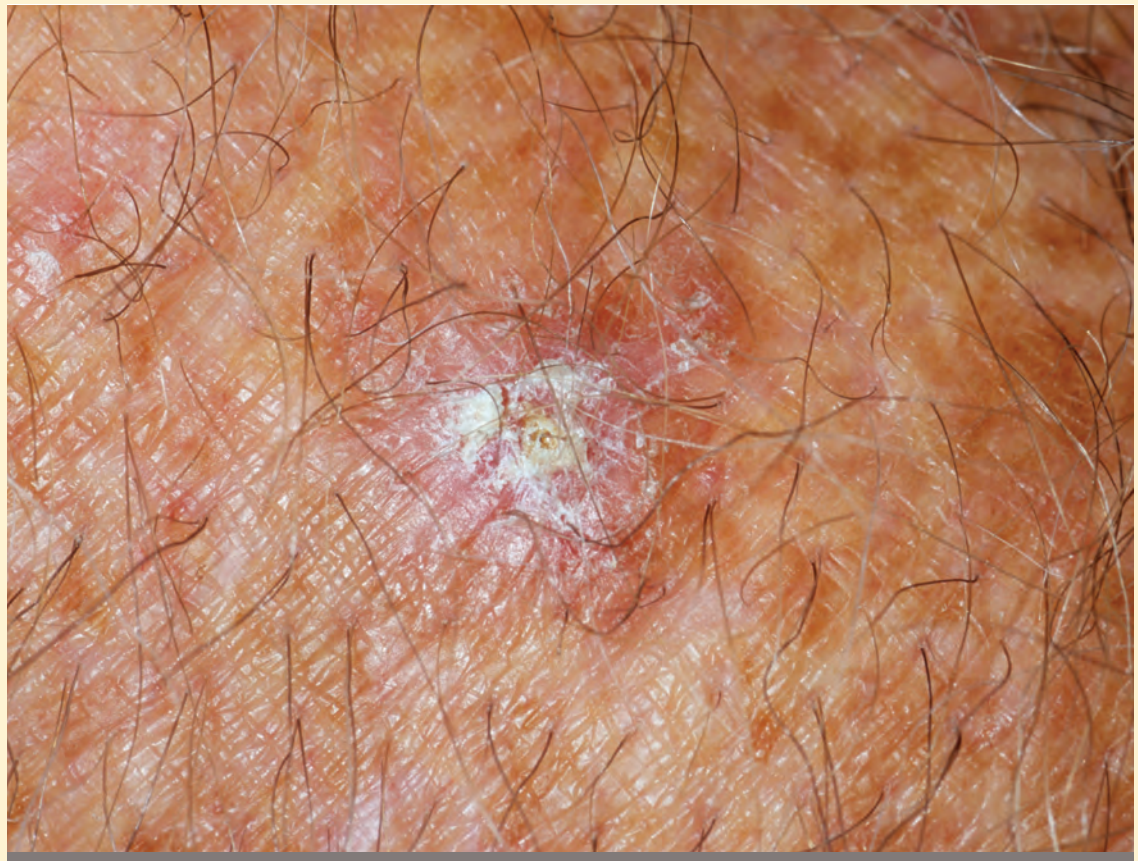
Actinic keratoses

Actinic keratoses (AKs, also known as solar keratoses) are common and usually harmless but they have the potential to become malignant. An AK lesion is also sometimes described as a 'carcinoma in situ', indicating that abnormal cells are present but they have not spread beyond the immediate area. AKs are small, rough spots that develop on the skin. They range from barely perceptible rough spots of skin to raised, hyperkeratotic plaques and can vary from skin-coloured to reddish-brown. The size of AKs varies from one to three millimeters in diameter but they can reach several centimetres.

AKs rarely develop as solitary lesions. They appear most commonly as multiple discrete, flat or raised keratotic lesions and usually gradually enlarge into broader, more elevated lesions[7]. Occasionally they can form small horns or spikes. The presence of multiple lesions is described as 'field cancerisation' or 'field change'. It is estimated that over a one-year period 15-25 per cent of lesions will spontaneously disappear without any intervention[8].

Sometimes it is difficult to distinguish an AK lesion from a small patch of very dry skin or eczema but these will usually disappear with intensive emollient treatment. Patients may also be anxious about seborrhoeic keratoses, which can look alarming but are harmless (see panel).

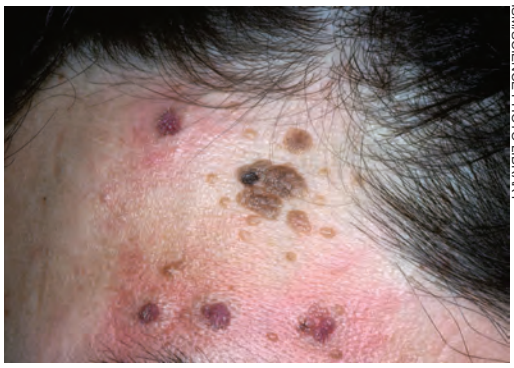
AKs are caused by frequent or intense exposure to UV radiation over many years, and so usually appear on sun-exposed skin areas including the face, ears, bald scalp, forearms and backs of the hands. The highest prevalence rates are found in countries that are both close to the equator and have large fair-skinned populations, such as Australia[7]. The prevalence



It is sometimes difficult to distinguish actinic keratosis (pictured) from dry skin or eczema

Seborrhoeic keratoses

Seborrhoeic keratoses (also known as seborrhoeic warts, basal cell papillomas or senile keratoses) are very common harmless growths on the skin. They are much commoner than skin cancers. Seborrhoeic keratoses are often pigmented and their appearance can sometimes be alarming. A dark seborrhoeic keratosis can look similar to a melanoma.



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Seborrhoeic keratoses have a rough surface and range in colour from golden brown to mid-brown to almost black. Small flat seborrhoeic keratoses can often become more raised and larger with time. Their size varies from less than one centimetre to several centimetres across. They usually have a characteristic 'stuck on' appearance.

Seborrhoeic keratoses occur most often on the trunk, but they are also common on the head and neck. Their numbers vary – one person may have just one seborrhoeic keratosis while others can have hundreds. Once present, they usually stay, and new ones often appear over the years.

Seborrhoeic keratoses are not infectious and do not become malignant. They are usually not treated although they can be removed if they are troublesome; for example, catching on clothing.

'Actinic keratoses are caused by **frequent or intense exposure to UV radiation** over many years'

PHARMACY MAGAZINE CPD MODULE
REFLECT
PLAN
ACT
EVALUATE

Reflection exercise 1

Skin cancer is an important public health issue. Find out what information and resources are available locally including relevant treatment pathways. Reflect on what information you could provide from your pharmacy to align your advice to local services.

in Queensland, for example, exceeds 55 per cent in men and 37 per cent in women aged 30-70 years.

The risk factors for development of AKs are:

- Caucasians (blue eyes and fair skin)
- Proximity to the equator
- Outdoor occupation
- Male sex (due to increased likelihood of having an outdoor occupation)
- Increasing age (due to cumulative exposure)
- Immunosuppression (e.g. following organ transplantation).

The risk of the malignant transformation of an AK into a SCC ranges from less than 0.025 per cent to 16 per cent a year[7]. While it is currently not possible to determine which AKs will progress to SCC, it is known that progress is more likely in immunocompromised individuals (see high-risk groups).

A number of signs and symptoms suggest the development of a SCC and these would prompt a GP to refer the patient for specialist assessment and treatment[9]:

- Recent growth associated with pain and/or bleeding
- A raised lesion (papule or nodule)
- Presence of ulceration, induration, tenderness with surrounding inflammation
- Lesions on the lips (a common site for SCC).

Table 1: Topical treatments for AKs

Topical product	Mechanism of action	Licensed indication	Dosing	Additional comments
Efudix cream (5FU 5%)	5FU inhibits thymidylate synthetase and causes cell death in actively proliferating cells	AK	Every night for four weeks	After four weeks stop the treatment and consider use of a mild or moderate topical steroid for two to four weeks to help settle any inflammation
Actikerall cutaneous solution (5FU 0.5% + salicylic acid 10%)	5FU inhibits thymidylate synthetase and causes cell death in actively proliferating cells	Treatment of slightly palpable and/or moderately thick hyperkeratotic AK	Once a day for 6-12 weeks	After four weeks stop the treatment and consider use of a mild or moderate topical steroid for two to four weeks to help settle any inflammation
Solaraze (diclofenac 3%)	Mechanism of action against actinic keratoses is not known	AK	For large areas of field change use twice a day for 12 weeks	
Picato gel (ingenol mebutate)	Postulated mode of action includes rapid lesion necrosis by mitochondrial swelling and membrane disruption and specific neutrophil-mediated, antibody-dependent cellular cytotoxicity by antibodies produced from B cells that bind to antigens on dysplastic epidermal cells	Non-hyperkeratotic, non-hypertrophic AK	Face/scalp: daily for three days; Trunk/ extremities: daily for two days	Response assessed after 57 days
Aldara (imiquimod 5%)	Up-regulates a variety of cytokines, which, in turn, invoke a non-specific immune response (interferons, natural killer cells) and a specific immune response (T cells)	Non-hyperkeratotic, non-hypertrophic AK	Small areas of field change: use three nights a week (e.g. Monday, Wednesday and Friday) for four weeks. Apply overnight and wash off the following morning	Warn patients to expect marked erythema with crusting of the skin. Rest periods may be needed
Zyclara (imiquimod 3.75%)	As above	Non-hyperkeratotic, non-hypertrophic, visible or palpable AK of the full face or balding scalp	Large areas of field change: apply once daily for two 2-week treatment cycles separated by a 2-week treatment-free period	Side-effects milder than with 5% imiquimod

Management of AKs

The uncertainty about which AKs will transform into SCCs makes prevention of AKs an important public health issue and a key part of their management. As AKs are so prevalent, it is expected that the majority will be managed in primary care[8]. Education of patients regarding UV protection, self-examination and detection of early lesions plays an important part in overall management[7].

Topical treatment

No treatment or treatment with emollient only is considered satisfactory for mild AKs[8]. Single AKs can be treated using targeted treatment such as curettage, cryotherapy, photodynamic therapy (PDT) or topical medicines such as 5-fluorouracil (5FU) either as Efudix (5FU 5%) or Actikerall (5FU 0.5% + salicylic acid 10%).

Curettage involves surgical removal of the lesion; cryotherapy involves freezing the lesion with liquid nitrogen; and PDT involves applying the photosensitiser methyl aminolevulinate (Metvix) and exposing the area to red light. For multiple lesions (field change), topical diclofenac, ingenol mebutate or imiquimod is recommended (see Table 1).

All the topical treatments for AKs are directed at destroying the sun-damaged cells so they all,

to a greater or lesser extent, induce inflammation in the skin. The skin may weep, peel, crack or even blister and then crust or scab over. The area may be itchy or sore, painful and burning (caused by the abnormal cells dying) and is a sign that the treatment is working. After completing the treatment, such reactions will settle over a few weeks – something that patients need to be aware of because it may be important for the timing of treatment.

Sun protection and high-risk groups

AK is regarded as an ongoing disease that requires regular follow-up – half-yearly to yearly – and long-term management[7]. Sun protection is necessary to prevent the further development and recurrence of AK lesions. The recommended preventative measures are shown in Table 2.

Mechanism of UV-induced damage/ AK formation

Chronic sun exposure can cause UV-induced mutations in the DNA of skin cells. Normally, endogenous repair mechanisms mend any damage to the DNA. If the damage cannot be repaired then apoptosis (programmed cell death) is triggered and the damaged cell is removed, thereby preventing further damage and protecting the body. However UV radiation not only induces DNA damage but also influences the repair and apoptotic mechanisms in two important ways:

- UV radiation impairs the local (cutaneous) immune function and the lowered immune status is associated with an increased risk for the development of SCC
- UV radiation is also known to increase the expression of viral genes that have anti-apoptotic activities and delay DNA repair mechanisms.

The human papilloma virus (HPV) is believed to play a role here. As a result, skin cells with damaged DNA persist in the skin and could subsequently develop into skin cancer[7].

Table 2: Preventive measures for AK

- **Avoid over-exposure to UV light – limit the time spent in the sun, especially around midday**
- **Use a high-factor (SPF 50) sunscreen on all exposed skin, including the lips, and cover skin when possible**
- **Avoid tanning beds as these emit ultraviolet A (UVA) rays, which penetrate deeper into the skin and are believed to cause AK lesions**
- **Check skin regularly and report any changes to a healthcare professional**

Fair skin is a risk factor for the development of actinic keratoses

High-risk groups including organ transplant recipients

Several distinct groups have an increased risk of developing skin cancer. Many of these individuals are not aware of the risk so do not change their behaviour accordingly. High-risk groups include:

- Patients with a family history of skin cancer
- Patients with current or previous NMSC
- Those with a weakened immune system (e.g. immunosuppressant medication and/or disease affecting the immune system).

Skin cancer is the commonest malignancy in organ transplant recipients (OTRs). People who have undergone organ transplants and are therefore taking immunosuppressant treatment are 250-times more likely to develop an AK than immunocompetent individuals[7]. They also have

Table 3: Increased incidence of skin cancer in the OTR population[10]

Type of skin cancer	Increase in incidence
Actinic keratosis	250-fold
Squamous cell carcinoma	100-fold
Squamous cell carcinoma of lip	20-fold
Basal cell carcinoma	10-fold
Melanoma	Three-fold

Reflection exercise 2

Find out what advice local organ transplant services give concerning the risks of skin cancer in immunosuppressed patients. Reflect on how you might reinforce this.

a 100-fold higher risk of developing invasive SCCs. OTR patients are also at greater risk of developing metastatic SCCs.

SCCs are the commonest type of skin cancer in OTR patients, whereas in immunocompetent patients BCCs predominate. This means that all patients who have received an organ transplant need ongoing preventive treatment and regular skin monitoring. In general, about 40 per cent of immuno-suppressed patients develop an invasive SCC compared to approximately 10 per cent of immunocompetent patients.

Organ transplant recipients are now routinely educated about the risks of developing skin cancer. Despite these efforts studies show that some patients still fail to protect their skin adequately. Surveys of transplant patients reveal a lack of compliance with UV prevention recommendations. One study[11] showed that:

- Only 54 per cent of transplant recipients remembered receiving advice about the prevention of skin cancer
- Only 40 per cent of kidney transplant recipients reported using a sunscreen
- 90 per cent of those used a sunscreen with a sun protection factor of less than 10.

Reflection exercise 3

Consider the problems of using a high factor sunscreen on a daily basis and find out which products would be suitable. Reflect on how you would explain the options and help a patient to select the most appropriate product.

Sun protection – think SMART (see opposite)

Lack of compliance may also be due to the fact that prescribed medication is difficult or unpleasant to use and good quality sunscreens are expensive. The problem is often compounded by a reluctance to prescribe products that are seen as being mainly cosmetic. Another important factor is the popular myth that some sunscreens are carcinogenic. However, the evidence for the effectiveness of high-factor sunscreens is compelling.

One study involving 120 transplant patients compared patient education about sun protection with education plus provision of a high-factor sunscreen over a two-year period[12]. The results showed that 53 per cent of AKs in the sunscreen group went into spontaneous remission. Eight new invasive SCCs occurred in the control group compared with none in the sunscreen group. Adherence to the sunscreen regimen was high in the study. The product used was Actinica – a broad spectrum sunscreen that provides a SPF greater than 50 and high-level UVA protection.

Vitamin D supplementation

People who are avoiding exposure to sunlight and using sunscreens assiduously are at risk of vitamin D deficiency. In the study described above[11] vitamin D levels were decreased in all patients without adequate substitution, but were consistently lower in the sunscreen group compared to the control group.

Further reading

- Improving outcomes for people with skin tumours including melanoma. The Manual. NICE February 2006
- Improving outcomes for people with skin tumours including melanoma (update): The management of low-risk basal cell carcinomas in the community. NICE May 2010
- ESNM14: Actinic keratosis: ingenol mebutate gel. NICE March 2013
- Skin cancer (non-melanoma) prevention. NHS Choices www.nhs.uk/Conditions/Cancer-of-the-skin/Pages/Prevention.aspx
- Protect your skin and eyes in the sun. NHS Choices www.nhs.uk/Livewell/skin/Pages/Sunsafe.aspx
- Cancer Research UK. Melanoma risks and causes www.cancerresearchuk.org/about-cancer/type/melanoma/about/melanoma-risks-and-causes
- Spencer JM *et al.* Actinic keratosis treatment and management. http://emedicine.medscape.com/article/1099775-treatment

Sunlight (UVB) is required for vitamin D synthesis in the skin and this is essential for bone and muscle function. About 15 minutes of sunlight on unprotected skin – ideally between 11am and 3pm when UVB radiation is most intense – is all that is needed for satisfactory vitamin D synthesis.

There has long been uncertainly about the 'right' level of vitamin D and what, if any, supplements are required – something that new NICE guidelines, published in November 2014, should help to resolve[13]. Experts recommend vitamin D supplementation for adults, if sunscreens are used regularly, at a dose of 10mcg per day[14].

General sun protection advice

The SunSmart message is a useful way to remember sun protection measures:

- **S**pend time in the shade between 11am-3pm
- **M**ake sure you never burn
- **A**im to cover up with a T-shirt, hat and sunglasses
- **R**emember to take extra care with children
- **T**hen use sunscreen with a minimum of SPF15 and good UVA protection.

In addition people should be reminded:

- To report mole changes or unusual skin growths to their GPs
- To take special care of children's skin – ideally by covering them up with a suitable garment and/or keeping them in the shade
- Wearing a wide-brimmed hat can reduce the amount of UV rays that reach the face and eyes
- Sunbeds are not a safe alternative to lying outside in the sun. Skin will still be exposed to harmful UV rays. Health risks linked to sunbeds and other UV tanning equipment include skin cancer, premature skin ageing, eye irritation and cataracts.

For more information, see NHS Choices 'Protect skin and eyes'.

Supporting high-risk patients

For immunosuppressed individuals including OTR patients:

- Check routine use of sunscreen and advise if appropriate
- Help to select a suitable sunscreen product – one that does not exacerbate acne/cause spots and is pleasant to use (spreadable, acceptable smell) and is in an acceptable price range
- Check awareness of vitamin D requirements and advise on supplements
- Reinforce the importance of regular skin checks. Transplant patients should examine their skin from head to toe each month to check for changes, and see a dermatologist for a full-body skin examination at least every year, as it may aid in the early detection of potentially deadly melanomas, aggressive SCCs and other skin cancers
- Signpost to local services if necessary.

Supporting patients receiving treatment for AKs

- Patients need to know that whichever type of treatment is used, some inflammation is to be expected because the sun-damaged cells are being destroyed. This applies to cryotherapy and PDT as well as to pharmacological agents. The skin may weep, peel, crack or even blister and then crust or scab over. The area may be itchy, sore, painful, burning or a combination of these
- Reinforce the need for ongoing sun protection to prevent development of further AKs.

Reflection exercise 4

All topical treatments for AK destroy the sun-damaged cells so they will induce inflammation in the skin, which can end up oozing, crusting and eventually scabbing over. The treated area can be painful and look very unsightly. Reflect on how you would explain this to a patient.

References

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2. NICE. Skin cancer prevention: information, resources and environmental changes. NICE guidelines [PH32], January 2011
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10. O'Reilly Zwald F *et al.* Skin cancer in solid organ transplant recipients. *J Am Acad Dermatol.* 2011; 65(2):253-261
11. Nindl I *et al. Dis Markers* 2007; 23(4): 247-259
12. Ulrich C, Jürgensen JS, Degen A *et al.* Prevention of non-melanoma skin cancer in organ transplant patients by regular use of a sunscreen: a 24-month, prospective, case-control study. *Br J Dermatol* 2009 Suppl 3:78-84
13. NICE. Vitamin D: increasing supplement use among at-risk groups. NICE guidelines [PH56], November 2014
14. CMO letter: Vitamin D – advice on supplements for at-risk groups 2012. CEM/CMO/2012/04 Gateway reference: 17193

ACTINIC KERATOSES AND SKIN CANCER

assessment questions

1. Which statement is FALSE about skin cancers:
a. There are about 2,200 deaths from skin cancer in the UK annually
b. Squamous cell carcinoma is the commonest skin cancer
c. UV exposure is a contributory factor to the development of skin cancers
d. Non-melanoma skin cancer accounts for about a third of all cancers detected in the UK

2. Which is NOT a risk factor for development of malignant melanoma?
a. Genetic predisposition
b. Exposure to UV radiation including sunlight
c. Vitamin D deficiency
d. Immunosuppression

3. Which is NOT a feature of actinic keratosis?
a. Small area of rough dry skin
b. A bluish-purple raised spot
c. A hyperkeratotic plaque
d. A keratotic spike or horn

4. In immunocompetent individuals the risk of transformation of an AK to a SCC is:
a. Up to 16 per cent
b. Up to 25 per cent
c. Below 10 per cent
d. Below 1 per cent

5. Which statement is FALSE regarding seborrhoeic keratoses?
a. The colour can vary from light brown to nearly black
b. They have a characteristic 'stuck on' appearance
c. They have a rough surface
d. They can undergo malignant transformation

6. Find the FALSE statement. UV radiation:
a. Impairs local immunity
b. Causes programmed cell death (apoptosis)
c. Directly damages cellular DNA
d. Increases the expression of viral anti-apoptotic genes

7. Find the TRUE statement regarding organ transplant patients:
a. They have a 250-fold greater risk of developing AKs
b. BCCs are the commonest type of skin cancer found in this group
c. The risk of developing a BCC is increased 100-fold
d. They should have their skin checked by a dermatologist every two years

8. Which is NOT a topical treatment for AK?
a. 5-fluorouracil
b. Ingenol mebutate
c. Imiquimod
d. Ibuprofen

Pharmacy Magazine CPD record January 2015

Use this form to record your learning and action points from this module on actinic keratoses and skin cancer and include it in your CPD portfolio and record online at www.uptodate.org.uk. Any training, learning or development activities that you undertake for CPD can also be recorded as evidence as part of your RPS Faculty practice-based portfolio when preparing for Faculty membership. Start your Faculty journey today by accessing the portfolio and tools at www.rpharms.com/development/faculty.asp

Activity completed. (Describe what you did to increase your learning. Be specific)
(ACT)

Date: Time taken to complete activity:

What did I learn that was new in terms of developing my skills, knowledge and behaviours? Have my learning objectives been met?*
(EVALUATE)

How have I put this into practice? (Give an example of how you applied your learning). Why did it benefit my practice? (How did your learning affect outcomes?)
(EVALUATE)

Do I need to learn anything else in this area? (List your learning action points. How do you intend to meet these action points?)
(REFLECT & PLAN)

*** If as a result of completing your evaluation you have identified another new learning objective, start a new cycle. This will enable you to start at Reflect and then go on to Plan, Act and Evaluate. This form can be photocopied to avoid having to cut this page out of the module. Complete the learning scenarios at www.pharmacymag.co.uk**

ENTER YOUR ANSWERS HERE Please mark your answers on the sheet below by placing a cross in the box next to the correct answer. Only mark one box for each question. Once you have completed the answer sheet in ink, return it to the address below together with your payment of £3.75. Clear photocopies are acceptable. **You may need to consult other information sources to answer the questions.**

1.	2.	3.	4.	5.	6.	7.	8.
a. ☐	a. ☐	a. ☐	a. ☐	a. ☐	a. ☐	a. ☐	a. ☐
b. ☐	b. ☐	b. ☐	b. ☐	b. ☐	b. ☐	b. ☐	b. ☐
c. ☐	c. ☐	c. ☐	c. ☐	c. ☐	c. ☐	c. ☐	c. ☐
d. ☐	d. ☐	d. ☐	d. ☐	d. ☐	d. ☐	d. ☐	d. ☐

Name (Mr, Mrs, Ms) _____

Business/home address _____

Town _____ Postcode _____ Tel _____ GPhC/PSNI Reg no. ☐☐☐☐☐☐☐☐

I confirm the form submitted is my own work (signature) _____

Please charge my card the sum of £3.75 Name on card _____ ☐ Visa ☐ Mastercard ☐ Switch/Maestro

Card No. _____ Start date _____ Expiry date _____

Date _____ Switch/Maestro Issue Number _____

Processing of answers
Completed answer sheets should be sent to Precision Marketing Group, Precision House, Bury Road, Beyton, Bury St Edmunds IP30 9PP (tel: 01284 718912; fax: 01284 718920; email: cpd@precisionmarketing group.co.uk), together with credit/debit card/cheque details to cover administration costs. This assessment will be marked and you will be notified of your result and sent a copy of the correct answers. The assessors' decision is final and **no correspondence** will be entered into.

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