Editorial Spring 2013

Although, I’m writing this in February it does really feel like Spring today. The sun has finally come out, there are some Daffodils starting to flower and I’ve managed to get to the top of the beacon without wearing my gloves! However, I feel a sense of impending doom as there are 8 nine year old girls soon arriving for a birthday sleep over. My husband has conveniently organised to do some RCGP examining in London instead of providing some moral support. If I didn’t know better I would think he may have engineered this absence. The same time last year he managed to find a job as the Doctor on the Trans Siberian Express…

As Stephen K has noted times are a wee bit grim in General Practice at the moment with increasing workload pressures and an imposed New Contract with impossible QOF targets just around the corner. I’m hoping to distract you all from this with this rivetting edition of the Bulletin. As usual we have some great regular articles. Julian has been beavering away through the journals to save us all time from reading them ourselves. Stephen Hayes has found a really interesting dermoscopy case, which, will make us all stop and think. There is News North of the Border and also an insight into life in Rwanda. There is also a fantastic article written by Frances Humphries, which manages to unravel the complexities of urticaria.

I would also like to welcome our three new Committee Members. They are Angelike Razzaque, Michelle Ralph and Robert Berry. I am really looking forward to working with them all. You will also see that I have managed to coerce them into writing something for the Bulletin already! If anyone has any interesting articles, cases or knowledge of authors, as always, I would be happy to receive them via pcds@pcds.org.uk.

Must fly to find some ear plugs and prepare for the descending chaos.

Helen Frow
No other emollients perform quite like them!
Doublebase™ – The difference is in the GELS

Original emollient Gel

- Emolliency like an ointment
- Cosmetic acceptability like a cream

Enhanced emollient Gel

- Highly emollient long lasting protection
- As little as twice daily application

Doublebase™ Gel
Isopropyl myristate 15% w/w, liquid paraffin 15% w/w

Doublebase Dayleve™ Gel
Isopropyl myristate 15% w/w, liquid paraffin 15% w/w

Rx by name for formulation of choice

Legal category: P
MA holder: Dermal Laboratories, Tatmore Place, Gosmore, Hitchin, Herts, SG4 7QR. Date of preparation: November 2012. ‘Doublebase’ and ‘Dayleve’ are trademarks.

Adverse events should be reported. Reporting forms and information can be found at www.mhra.gov.uk/yellowcard. Adverse events should also be reported to Dermal.

www.dermal.co.uk
Greetings to you all and commiserations for the huge stress and complexities produced by the current NHS changes. In my 40 years of NHS experience as a GP trainee, GP, Hospital Practitioner and GPwSI there has never been so much confusion and disquiet! I wonder who remembers a Conservative manifesto promise not to undertake major NHS reform. If this is minor what can we expect to be faced with in the future?

We cannot turn back the massive changes nor can we produce the trillions of national debt which have made the comments about ring fencing funding for the NHS a sad joke but that does not mean we should give up. We are all aware of the rocketing demands from an ageing population and the savings we are expected to achieve as the secondary to primary care shift in care arrives without the means to provide it.

In particular I am saddened by the dramatic reduction in skin surgery (note: it is not “minor”) with the strict requirements for skin cancer surgery and the local restrictions on benign nuisance lesions at the same time as we have a tidal wave of BCCs and ever increasing patient cosmetic demand. It is, however, still possible to gain experience and skills under the auspices of a dermatology department in secondary care (coupled with PCDS surgical training) although, at present, your partners will consider your work as charity, given the current pay scales! How many of you are offering a private service?

The next hurdle for some is the reluctance of some consultant colleagues to train us for fear of future changes allowing GPs to take the bread from their children’s mouths! There are fortunately many who see the benefit of an integrated service and they deserve our support and encouragement. Please make every effort to talk to your local secondary care colleagues, if possible face to face rather than through managerial go-betweens who may have a vested interest in the status quo. Maybe suggesting jointly funded posts between CCGs and District General Hospital departments when a consultant could be responsible for training, accreditation and appraisals for local GPSIs.

We have heard of bids for dermatology services which have no staff in place. When successful, which is easier when you have a skilled team of lawyers and accountants applying for contracts around the country, they then trawl local and other losing providers offering new, often worse, terms and conditions. Failing that they have been known to “train” up some ordinary GPs within 2 or 3 weeks and having accredited them “in house” call them GPSIs!

If you have knowledge of such local activities please let us know since it demeans the GPSI role and risks lowering dermatology services.

The Department of Health (DH) accreditation guidance (2007) specifies that accreditation should be by PCTs which as we know will cease to exist in April 2013! The DH has been repeatedly asked directly and by questions in the House of Commons prompted by the All Party Parliamentary Group on Skin (APPGS) for guidance as to who should be the accrediting body. Their vague view is that CCGs should perform this task not withstanding
the inevitable problems of provider/commissioner bias. No-one is keen to take on the expensive and difficult role but there is hope that the RCGP may be persuaded to take this on and the PCDS will do its best to help despite not being an accrediting organisation itself.

We have lost our dermatology responsible administrator at the DH and dermatology does not feature in the disease domains from NICE under the new organisation.

To guide us in our influential role at the centre of national discussions please contact the PCDS office with comments and evidence of local problems or great successes.

We are very pleased to report the 50th anniversary of one of our long standing sponsors. Dermal Laboratories, established by a doctor in 1963, is one of the few UK pharmaceutical companies that is truly independent. As the name suggests, Dermal was founded to research, develop and market dermatological products.

I was surprised to see the range of topical products for which they are responsible, in particular the range of topical ibuprofen gels and spray, dithranol and coal tar. They also produce wart paints and gel; anti-inflammatory acne treatment; ear wax drops as well as their extensive and increasing range of emollients representing 2 out of the 3 most commonly prescribed emollients in the UK which are more familiar to us as Dermal products. Congratulations and thank you.

I look forward to meeting more of you at our wide range of meetings this year all listed on the website www.pcds.org.uk

Angelike Razzaque
Applying for and accepting the invitation to join the PCDS Committee has been a natural progression in my interest in dermatology as a GP. Having personally experienced how little emphasis is paid to dermatology education at medical school and during GP training I was thrilled to learn about the PCDS. I became a member and regularly attended the meetings to build up my portfolio. Years later and additionally having completed the Cardiff Diploma for practical dermatology I am providing an in-house dermatology service in a busy inner city practice and act as the dermatology adviser for my local PCT/CCG. I can certainly testify that the PCDS has been the platform for my continuous development. I am now looking forward to contributing to the content and delivery of the educational events and meetings, to share my experiences, spread some of my enthusiasm for dermatology and engage in discussions about the provision of good patient care with members and fellow GP’s throughout the country.

Stephen Kownacki
Executive Chair
PCDS
Michelle Ralph
I am a part-time semi-rural GP working in the village of Thaxted famous for its connection with Dick Turpin and Morris dancing. On 2 days of the week to avoid highway men and dancing bells I work as aGPSI in dermatology running clinics and minor operative lists. I am extremely fortunate to work alongside a brilliant dermatology nurse and be under the clinical supervision of Cambridge Dermatology Department. In my spare time I love spending time with my teenage children, reading, catching up with friends and organizing the next holiday. I try (and fail miserably) to stay fit by jogging and am a ski addict.

I am passionate in trying to increase dermatology knowledge in primary care and sincerely hope I will be able to help the PCDS continue to build on their success.

Dr Robert Berry
I qualified from University of Wales, College of Medicine in Cardiff in 2003 before entering the Newport Basic Surgical Training scheme. After a brief spell with other specialties including Paediatrics, Psychiatry and Emergency Medicine, I decided General Practice was worth a shot. And it was a good bet, as I am relishing General Practice training, and I’m currently an ST3 due to complete training in Summer 2013.

I enjoyed all of the specialties I worked in, and have achieved Membership of the RCS, and Diplomas in Head and Neck Surgery, Obstetrics & Gynaecology, Child Health and Clinical Psychiatry.

I am on the trainee advisory board of the MHRA, and also represent the PCDS on the British Standards Institution Committee for Standards in Aesthetic Surgery and Aesthetic Non-Surgical Medical treatments.

I developed an interest in Dermatology during GP training, realising the importance of this under-taught specialty. I plan to undertake the Diploma of Practical Dermatology locally in Cardiff, and continue to develop my interest in Dermatology and Skin Surgery with the PCDS.

Iain Henderson
Scottish Representative, PCDS Executive Committee

News from North of the Border

Once again Spring is around the corner and whilst the rest of nature has “romance” on their minds, spare a thought for this old codger who has to turn his mind to organising the Scottish meeting programme. Not that I mind as it’s a chance to network with colleagues old and new in order to bring you a cornucopia of education. I am in fact still groaning from Christmas cracker jokes such as:

Doctor, doctor, I’ve swallowed a film!
Don’t worry. Let’s wait and see what develops.

Q. What do you get if you eat Christmas decorations?
A. Tinselitis

This year’s Scottish meeting is at the Harriott Dalmahoy Hotel near Edinburgh again on 9th/10th November. Those of you who have been there will know it is an excellent venue for what will be an excellent programme and weekend.

Not much has happened North of the Border since the last bulletin but I have found that London doesn’t cope with 3cm of snow when we regularly cope with 2 feet (picture 1 below).

PSALV and Psoriasis Scotland held a Parliamentary briefing on 20th February where our honorary member Danny Kemmett was speaking. Although he still lives in the Dark Side

PSALV and Psoriasis Scotland held a Parliamentary briefing on 20th February where our honorary member Danny Kemmett was speaking. Although he still lives in the Dark Side
(Edinburgh) he is back working in the west at Vale of Leven and clinics at Oban, Campbeltown etc. Any one interested in attending the next Parliamentary Cross Party on Psoriasis and Psoriatic Arthritis which will involve a wee trip to Holyrood on 17th April can send an email to the PSALV administrator, Mairi Maciver, at mairi.maciver@hotmail.co.uk and she will get you on the invite list.

The Skin Care Campaign Scotland (SCCS) has changed its name to Skin Conditions Campaign Scotland and has a new website www.skinconditionscampaignscotland.org. It is a “work in progress” but is worth looking at for the various details of the various Scottish and UK charities dealing with dermatological conditions.

I’m now out the loop of the Dermatology Council for Scotland but will endeavour to keep them abreast of PCDS activities.

My next clinical case from Rwanda shows that even patients with type VI skin can still get photosensitivity reactions (picture 2). The clue was the sparing under the chin (picture 3). We thought it was due to one of the patient’s anti-retrovirals.

Finally, as Dalmahoy is near Edinburgh, here are 2 jokes just to keep it even.

Q. Why isn’t the Hearts team allowed to own a dog?
A. Because they can’t hold on to a lead.

Q. What’s the difference between O J Simpson and Hibs
A. O J Simpson had a more credible defence.

That’s all for now.

Dr Tim Cuncliffe
GPSt Darlington & PCDS Executive Committee Member

PCDS Website

It has been a busy period for the website. All of the clinical chapters from the old site have now been updated and moved across. New chapters have also been added. Some areas to highlight are as follows:

- Changes to the lichen planus chapter
- A new chapter on follicular lichen planus including lichen planopilaris and frontal fibrosing alopecia
- A new chapter on vitiligo
- A video clip on emollient application, as well as advice on the fingertip unit method of steroid application - both of these can be found in the management section of atopic eczema
- Many new images have been added to the website, this is the pick of the best for this quarter

The next piece of work is alopecia, a big one, so don’t wait up!

Best wishes
Cosmetic Dermatology

The world of cosmetic dermatology is changing. There has been much recognition and criticism of the lack of standards and regulation to date in both the surgical and non-surgical cosmetics industry. The PIP scandal has brought this to light in recent months, and perhaps the bad publicity will bring about some change for the better.

There has also been much debate in recent years around the issues of who should and shouldn’t deliver cosmetic procedures, especially in the non-surgical injectables arena, where one may find a range of healthcare professionals offering similar cosmetic treatments, from Plastic Surgeons to Nurses, and GPs to Paramedics to mention but a few.

This is quite a unique area of therapy in the UK in terms of the healthcare setting. All non-surgical cosmetic work is performed as ‘private’ procedures, in the sense that they are paid for, and not offered on the NHS. There is understandably no NHS provision of non-surgical cosmetic work (unless your blepharospasm treatment yields cosmetically pleasing crow’s-feet as an unintended side-effect – and then you would hope the result was symmetrical!). But it leaves a situation where we have no benchmark from which to compare standards of care.

Currently, the training to set out on providing cosmetic treatments is ropey to say the least. A one-day training course is sufficient to secure Indemnity Insurance with recognised providers. The general requirements for attendance on cosmetic training courses available, state that delegates should be qualified Doctors, Dentists or Nurses. However, legally there is no restriction on who can and cannot administer cosmetic injectables, including Botox® and Dermal Fillers; which has led to a parallel industry of cosmetic injectable treatments provided by non-healthcare practitioners, i.e. beauticians and various other therapists.

There are two note-worthy processes in place at present with an intention of improving the standards of the Medical Cosmetics industry in the UK and Europe:

- The Department of Health Review into the Regulation of Cosmetic Interventions
- The CEN development of a standard into Aesthetic Surgery and Aesthetic Non-Surgical Medical Services

The first of these is due for completion in March 2013. It is led by Sir Bruce Keogh, Medical Director of the NHS in England. The aim of the review is to investigate the regulation of all aspects of the cosmetic industry, including organisations, practitioners and cosmetic products.

The second of these is a European-wide attempt to achieve a Standard into the provision of cosmetic procedures, including both surgical and non-surgical interventions. The purpose of the consultation process for ‘prEN16372’, the draft standard document, is to develop a European best practice standard for independent healthcare facilities offering aesthetic procedures to change physical appearance.

It’s aims are to define the requirements for the quality of aesthetics surgery, and non-surgical aesthetic medical services, offered in order to ensure patient safety and other factors which influence the overall quality of service.

The next step in the CEN process is a second Public Consultation into the draft standard document, which will be released in mid-March 2013. The UK has been granted a number of exceptions in the current document, based on UK legislation already in place, which would otherwise clash with recommendations in the standard. The most significant exception has been the term ‘practitioner’, which in the UK will include Registered Dentists and Registered Nurse Prescribers, as well as Medical Doctors.

The proposed standard offers a significant improvement on the current situation in...
the UK, although it is worth noting that it is not a legally binding document, and is unlikely to be harmonised as EU Law. It will therefore serve as a best practice document rather than a mandatory standard.

The BSI is leading the consultation process in the UK and interested parties from the aesthetics sector can register their comments online at http://drafts.bsigroup.com from mid-March 2013.

The BSI Committee will meet in June 2013, before the end of the consultation period, and the next CEN meeting will be in Vienna on 30th/31st August 2013. The final standard is likely to be complete in late 2014.

Any comments or opinions from PCDS members in regards to this topic and the processes in place would be very welcome. It would be helpful to have an idea if there is much interest in this field within the Society and how people feel we should be representing these issues as a group of professionals.

Please email pcds@pcds.org.uk with the subject “Cosmetic Dermatology” with any comments and feedback.

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A twentiesomething white female presented with a clear history of a mole on the upper arm that had either just appeared or become elevated (she wasn’t certain) over the last 9 months.

The appearance is fairly non-descript. It was thought to be somewhat pearly with abnormal fine vessels, hinting at a papular BCC. However, dermoscopy does not show typical BCC features. Despite the imperfect focus, brown globules can be clearly seen between 12 and 3 o’clock and at 5 o’clock. Some irregular vessels are visible, not the typical well focussed tapering vessels of a BCC nor the comma vessels of a benign intradermal naevus.

Going back to basics: a lesion is either melanocytic or non-melanocytic. There are no warty features and it certainly isn’t a haemangioma. It was too soft for a dermatofibroma and also lacks a central white scar. So that’s 3 common benign types of lesion ruled out on negative features (e.g. features which are NOT present). Turning to positive features, the brown globules point strongly to a melanocytic lesion. The next stage of diagnosis is to ask – if melanocytic, can we say for sure it’s benign? The globules are peripheral (indicating active growth) and asymmetrical (unsupportive of benignity).

Two experienced dermoscopists were undecided on the diagnosis but agreed on the management. Despite its bland appearance, it was excised as a new, growing melanocytic lesion in an adult female which could not be confidently designated as benign.

Histology was naevoid melanoma, Breslow 1.9 mm.

Naevoid melanoma are said to make up about 1% of melanomas and are often misdiagnosed at first due to their banal appearance. Prognosis is said to be comparable to regular melanomas, neither better or worse, but there are few studies due to rarity. Some studies suggest they may be slightly less dangerous, but recurrence and metastasis are well documented.

This case illustrates the need to always remember the ‘wolf in sheep’s clothing’, remember that rare things exist, follow the basic rules and take a systematic approach. It also illustrates the validity of the diagnostic aphorism s ‘A history of recent change trumps everything’ and ‘If in doubt, refer or excise for histology’. 

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**Dermoscopy**

**Stephen Hayes**

GPSI Southampton, PCDS Committee Member & Trustee
Colleagues often ask me which emollient they should prescribe. That’s a bit like asking which car they should buy. There are a lot of choices out there, they all have different advantages and disadvantages, but at the end of the day a lot really comes down to personal preference. The old adage, ‘the best emollient is the one the patient will use’, is paramount. There are, however some important considerations.

Emollients should be the first line treatment for all dry skin conditions (xeroses), especially the eczemas, psoriasis and the ichthytic conditions. The common ichthytic conditions are ichthyosis vulgaris (which probably affects about 1% of the UK population) and ichthyosis senilis, which is an almost inevitable consequence of ageing. I believe many patients with these dry skin conditions can control their disease with appropriate emollient therapy alone and avoid more potent, expensive treatments. Emollients not only restore the epidermal barrier, but also have anti-inflammatory effects on the skin.1,2

There is now overwhelming evidence that the regular use of emollients can control eczema and significantly reduce the need for steroids. Ideally, for the majority of patients in primary care, topical steroids or topical immunomodulators should then only be necessary for the control of flares. In my practice, I aim for a 10 to 1 ratio of prescribing emollients to topical steroids (gm:gm) and I encourage my patients to try to use up to about 500gms of emollient per week to achieve optimal benefit.

There is a wide spectrum of emollients. Sometimes, a patient is prepared to use a thicker, greasier emollient at bedtime, but during the day they may need a more ‘cosmetically acceptable’ preparation. Similarly, it can sometimes be helpful to supply smaller quantities for use during the day, for example in the car or after sport etc. (If this is prescribed at the same time as a larger quantity of the same emollient, there should only be one prescription charge to the patient).

Our ancestors didn’t use soap and rarely washed. Admittedly they probably smelt pretty awful, but our skin has not evolved to cope with the daily assault from detergents that is encouraged by today’s society.

Soaps, shampoos, shower gels and bubble baths are all detergents. All detergents work by degreasing the skin.
Dirt is trapped in the skin and by removing the grease dirt is lifted off, but only at the expense of degreasing the skin. Grease is essential on the surface of the skin to prevent trans-epidermal water loss (TEWL). Emollients can be used to wash with instead. These re-grease the skin and cleanse at the same time. Detergents do more harm than just de-greasing the skin though; they also raise the surface pH from about 4.5 to about 8 (that is almost a thousand fold change in acid ions!) and it takes healthy skin more than 48 hours to recover the normal surface pH after washing with soap.

Complete Emollient Therapy refers to the optimal use of a bath oil, wash emollients (soap substitutes) and leave-on emollients. I often hear the view that we should not prescribe bath oils ‘as there is no evidence of their efficacy’. Whilst that is true, lack of evidence of efficacy is not evidence of lack of efficacy and surely common sense must direct our prescribing until such evidence is available.

Our understanding of the extraordinary function of the top two layers of the epidermis has developed amazingly over the past few years (see diagram 1). These cell layers are from the ‘dead cells waiting to fall off’. Two main types of granules are produced in the stratum granulosa layer;

1. ‘Keratohyaline granules’ producing pro-filaggrin and keratin tonofilaments
2. ‘Lamellar granules’ producing ceramides, cholesterol and free fatty acids that form the lipid lamellare envelop – a fatty matrix that forms a seal around the corneocytes, rather like cement in a brick wall.

Pro-filaggrin is a protein that is broken down by proteases to filaggrin and further broken into fragments that act as ‘natural moisturising factors’ (NMF). These include urea and a number of acids (see Table 1), creating the normal surface acidity. An acidic skin surface is essential for controlling balanced protease activity in the outer few cell layers of the stratum corneum. They also hold moisture in the corneocytes and possibly even draw moisture from deeper layers or the environment, thereby ensuring the corneocytes are tightly packed one against another. Filaggrin also combines with the keratin tonofilaments in the stratum corneum concertining the cuboidal cells of the stratum granulosa cell layer into stratified squamous epithelium.

### Table 1: Natural moisturising factors by percentage of total

<table>
<thead>
<tr>
<th>NMF</th>
<th>% Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Free amino acids</td>
<td>40%</td>
</tr>
<tr>
<td>Pyrrolidone carboxylic acid</td>
<td>12%</td>
</tr>
<tr>
<td>Lactate</td>
<td>12%</td>
</tr>
<tr>
<td>Sugars, organic acid, peptides</td>
<td>9%</td>
</tr>
<tr>
<td>Urea</td>
<td>7%</td>
</tr>
<tr>
<td>Ammonia, uric acid, glucosamine creatine</td>
<td>1.5%</td>
</tr>
<tr>
<td>Others (chloride, sodium, potassium, calcium,</td>
<td>19%</td>
</tr>
<tr>
<td>phosphate, citrate, formate)</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from Harding CR et al
What we now know is that there are remarkably sophisticated and elaborate processes that have evolved to create the healthy skin barrier of the epidermis. These are easily disturbed by the regular use of detergents. Patients with dry skin conditions are genetically especially vulnerable. Furthermore, inflammation in the skin inhibits the production of filaggrin, so in inflammatory dry skin conditions (such as eczema), it is easy to produce a vicious cycle.

**Choices:**

1. **Simple ‘every day emollients’**
   These are generally cosmetically acceptable oil-in-water creams, well liked by patients (Table 2).

   Many contain petrolatum which forms a water impermeable layer on top of the skin, inhibiting TEWL, but within a couple of hours any therapeutic benefit has dissipated. They are all designed as ‘leave-on’ emollients but they are all easily miscible with water and can therefore be used as soap substitutes to wash with. Clearly, patients should be warned that the bath or shower tray could be rendered dangerously slippery and to avoid blocking the drains, boiling water or perhaps caustic soda crystals should be poured down the plug hole every week or so.

   E45 is the least expensive and remains a very popular everyday choice for patients and is often the chosen over-the-counter product.

   Cetraben is also popular, especially amongst my secondary care colleagues. However, it does contain Sodium Lauryl Sulphate (SLS, see below), albeit at only 0.5%.

   Diprobase cream is a straightforward, fairly basic emollient which patients like. It is, however, relatively expensive.

   Oilatum is a much more ‘intelligent’ emollient. It contains glycerol (which is a NMF), but probably not at a high enough concentration to offer much effect. It also contains povidone (see below), which forms a microscopic film on the surface of the skin, extending its TEWL benefit to up to 12 hours, which is very useful. It is remarkably competitively priced.

   I also like QV cream and Doublebase gel (which also contains some glycerol). Doublebase does have a rather gelatinous texture though which some people don’t like.

2. **Simple emollient ointments**
   These are thicker and greasier products of water-in-oil (see Table 3). As such they have significantly better rehydrating properties than the creams, but inevitably are less cosmetically acceptable. They are barely miscible in water so not really suitable as soap substitutes. Being very thick, they don’t come in pump dispensers.

   Epaderm ointment and Hydromol ointment contain exactly the same ingredients, in the same concentrations. Their process of manufacture however is different and consequently they do feel different and behave slightly differently. Nevertheless currently hydromol is more cost effective.

3. **Sprays**
   These can be very useful for difficult to reach areas, such as the back (Table 4). However, even spraying something onto your own back demands a degree of flexibility in the shoulder that many elderly patients don’t have.

   I like Emollin. It is simple 50:50 in a spray, with no fragrance or additives. It can even be used upside down and is a fabulous lubricant. I do warn patients that the spray mist can get onto the floor, which could be rendered dangerously slippery, if it is hard surface (e.g. tiled).
4. Emollients with additional properties

Some emollients have additional agents that offer significant benefits. These include:

a) Polyvinyl pyrodilline (Povidone)

This creates a film as described above.

There are only two on the market currently (Table 5).

Doublebase Dayleve also contains a higher concentration of glycerol and only costs marginally more than Doublebase, so that makes it even more attractive.

c) Natural moisturising factors

These could include urea, glycerol, lactic acid, sodium pyrrolidone carboxylate and a number of complex sugars. Generally though, when I consider an effective humectant emollient, I am looking for one that contains urea (Table 6).

Urea is an odourless crystal and is very unstable. It rapidly breaks down into ammonia and carbon dioxide, so needs to be ‘stabilised’. Ammonia of course, smells horrible.

The urea content in the skin of patients with atopic eczema is massively reduced (85% reduction in affected skin and 70% in unaffected skin). This is probably partly due to genetic factors, but further aggravated by the negative impact of inflammation on this deficient filaggrin production. It makes sense to try to replace this with a humectant emollient containing urea.

At just 5%, urea has anti-pruritic properties. As most of the very dry skin conditions are itchy, that is especially useful. At 10%, urea can almost overhydrate the stratum corneum and it begins to develop keratolytic properties, which would obviously be useful in conditions like psoriasis, but may be undesirable in eczema.

Most emollients should be applied every 3-4 hours to achieve optimal control of TEWL. One of the main advantages of urea is that it extends the duration of activity of the emollient to at least 12 hours. Not only is that really useful to patients who have a busy schedule (including school children) but it also means the product provides full 24-hour cover with just twice daily application. That balances some of the extra cost of these products and makes them considerably more cost-effective.

c) Lipid lamellar mimicking agents (usually ceramide)

Ceramides, cholesterol and free fatty acids make up the ‘cement’ between the corneocytes in the stratum corneum that is so essential for maintaining an effective skin barrier. All detergents wash these out very efficiently. In the elderly there is deficient production of these in the skin in the first place, so the epidermis is vulnerable to cracking and drying out (asteatotic eczema (figure1) is a typical example which is further aggravated by the dry environment of modern day living, especially as seen in hospital or in nursing homes).

Currently, the only emollient available on the NHS that contains a ceramide, is Balneum cream (contains 3%). The Cetaphil Restoraderm range also contains a therapeutically effective concentration of ceramide and are superb products, but unfortunately do not have an NHS tariff. Some Aveeno products also contain ceramide, but likewise are not available on the NHS.

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**Table 4: Spray emollients**

<table>
<thead>
<tr>
<th>Comment/active agents</th>
<th>BNF price 2013</th>
</tr>
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<tbody>
<tr>
<td>Emollin spray 240ml</td>
<td>£6.05</td>
</tr>
<tr>
<td>Evolve</td>
<td></td>
</tr>
</tbody>
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**Table 5: Emollients containing Povidone**

<table>
<thead>
<tr>
<th>Comment/active agents</th>
<th>BNF price 2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oiatatum cream 500gm</td>
<td>£4.99</td>
</tr>
<tr>
<td>Doublebase dayleve gel</td>
<td>£6.29</td>
</tr>
</tbody>
</table>

**Table 6: Emollients containing urea**

<table>
<thead>
<tr>
<th>Comment/active agent</th>
<th>BNF price 2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>Balneum cream 500gm</td>
<td>£9.84</td>
</tr>
<tr>
<td>Eucerin intensive lotion 240ml</td>
<td>£7.93</td>
</tr>
<tr>
<td>Hydromol intensive cream 100gm</td>
<td>£4.37</td>
</tr>
<tr>
<td>Flexitol heel balm 200gm</td>
<td>£9.90</td>
</tr>
</tbody>
</table>
d). Antiseptics

Infection plays a major role in atopic eczema flares (figure 2). Dry, cracked skin conditions are especially vulnerable to secondary infection too. The appropriate use of an emollient with antiseptic properties can therefore be very important. I also recommend their use as an option to prevent recurrent folliculitis, as for example following shaving (Table 7).

Oilatum Plus bath emollient (£6.98) contains 6% benzalkonium and 2% triclosan. A short soak in a bath with just two capfuls (15ml in 8 inches water), perhaps just once a week, can be very helpful at preventing or controlling repeated atopic eczema flares. It can, however, be quite irritant and there is no need to have a long soak as that only aggravates this irritation and offers no additional benefit.

I am not aware of any evidence that 1% chlorhexidine offers any additional antiseptic cover than the lower concentration combination used in the Dermol range. It is also potentially more irritant. For these reasons and also for the benefit of a wide portfolio of preparations offered by Dermol, I prefer the Dermol range.

e). Anti-pruritic agents

At 5% urea alone has useful antipruritic properties. The addition of lauromacrogols (as in Balneum Plus* and E45 itch relief creams) complements that. These two products are identical (and indeed used to be manufactured from the same source, they were just packaged and marketed by different companies) and each cost £16.40 for 500gm.

(*Balneum Plus cream contains 5% urea, but does not contain the ceramide in Balneum cream)

In my experience, roughly half my patients with pruritus enjoy good or considerable benefit from these products, but the other half see no additional benefit compared to a standard emollient.

Similarly Balneum Plus bath oil (£6.66) can offer some patients with intractable itch, remarkable soothing benefits.

Currently the only product with a cooling antipruritic effect is Dermacool, which is Aqueous cream with 10% menthol. This can be very helpful in some patients. However, aqueous cream contains a high concentration of SLS, so this product should be used with caution. Many of us believe that it should not be prescribed as a leave on cream.

5. Sodium lauryl sulphate

This agent is an emulsifier which is designed to render an emollient water miscible. It is markedly irritant and is damaging even to normal skin⁴⁵⁶. It is used sometimes as a control for skin irritation with patch testing! I agree with Professor Michael Cork from Sheffield who has described any emollient that contains SLS as ‘insane’.

Aqueous cream has been around since 1958. It is very cheap and therefore continues to enjoy being prescribed widely. It contains 2% SLS which is disastrous for the skin. Sometimes Health Care Professionals assume it can still be used safely simply as a soap substitute. This year, physicians in New Zealand have been advised by Medsafe (Information for Health Professions) never again to prescribe Aqueous cream BP. The evidence of the damaging effect of this emollient is overwhelming.

Epaderm and Hydromol contained SLS until it was removed from these two products last summer. Cetraben still contains SLS, but at only 0.5%.

Several products (such as the Sanex range OTC) contain sodium laureth sulphate, not lauryl. This is also damaging to the skin, but substantially less than SLS.

6. Flammability

Not all emollients contain paraffin, but those that do (see Table 8) are dangerous if there is any risk of fire. Patients prescribed these products must

<table>
<thead>
<tr>
<th>Table 7: Antiseptic emollients</th>
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<tbody>
<tr>
<td>Comment/active agent</td>
</tr>
<tr>
<td>Dermol cream 500gm</td>
</tr>
<tr>
<td>Dermol lotion 500ml</td>
</tr>
<tr>
<td>Dermol shower emollient 200ml</td>
</tr>
<tr>
<td>Eczmol cream</td>
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<table>
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<tr>
<th>Table 8: Paraffin containing emollients</th>
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<tbody>
<tr>
<td>Cetraben cream</td>
</tr>
<tr>
<td>Diprobase</td>
</tr>
<tr>
<td>Emulsifying ointment</td>
</tr>
<tr>
<td>50:50 wsp/ysp</td>
</tr>
<tr>
<td>Epaderm</td>
</tr>
<tr>
<td>Hydromol</td>
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</table>
be warned about this risk and I would make a note in their records every time I prescribed one of these emollients that I had issued that advice (not least, for medico-legal reasons).

Conclusions

There has been a worrying increase in the prevalence of atopic dermatitis (AD) year on year over the past 50 years. This cannot be attributed to increased reporting alone. The prevalence of AD in cohorts of children born in 1948, 1957 and 1970 were 5.1%, 7.3% and 12.2% respectively. That had risen to 16.5% in children aged 1-5 years in the UK by 1998. A change of this magnitude, over just one or two generations, can only be attributed to environmental factors.

During this time there were a number of dramatic changes in lifestyle in the developed world, including fridges in every home, widespread use of antibiotics and the availability of warm running water and soaps for washing in every home. I have no doubt that the regular use of detergents on our skin, especially that of babies and children, has had a massive impact on the integrity of the skin barrier and must be responsible for much of this disease.

Perhaps the so called ‘atopic march’ of eczema → asthma → hay fever, as well as the appearance in the last decade of common food allergies, can all be attributed to defects in the skin barrier allowing allergens to gain access to the stratum spinosum. This is where the Langherghans’s cells (tissue macrophages) trigger alterations in the immune response in vulnerable individuals and drive the abnormal T2 –IL-4 allergic pathways etc.

I have discussed how emollients can be used instead of detergents to help to restore and protect this skin barrier. That is even more essential in individuals with compromised filaggrin activity.

Healthy skin has 12 or so exons coding filaggrin on chromosome 1. In AD, one or two are typically missing. In ichthyosis vulgaris the deficiency is even more profound. With this sort of deficiency, we can now see how vulnerable the epidermis is and why our modern lifestyle is causing so much disease.

I confidently believe mild eczema can be controlled or even prevented altogether by education and the appropriate use of effective emollients. The long-term savings to society, not just in terms of drug costs, but also productivity and time off work would be staggering.

Similarly, the anti-inflammatory and anti-proliferative effects of emollients in psoriasis makes them the first line treatment for this condition. There is mounting evidence that plaques of psoriasis produce chemicals (such as fibronectin) which drive further progress of psoriasis. I have found that controlling mild psoriasis is considerably easier than efforts to re-gain control and in my experience, regular appropriate use of emollients, together with avoiding any contact with detergents, can keep psoriasis at bay completely.

It is ironic and tragic that as our understanding of these physiological processes has advanced so fantastically in the past decade, we are facing mounting pressure on prescribing this inexpensive, essential, safe and highly effective treatment.

It is up to us to insist that this safe, effective and indeed essential treatment remains at the forefront of our practice and we do not allow ignorance and false economies to interfere with this first line therapy.

I am enormously grateful to Professor Mike Cork, not only for allowing me to use his diagram, but also for his wisdom and advice in the preparation of this personal view.

References

The urticarias are a group of disorders, caused by release of vasoactive agents particularly histamine, resulting in vasodilatation and extravasation of plasma from dermal capillaries. Clinically this results in a rash consisting of weals (dermal oedema), Figure 1, and/or more deep seated areas which are termed angio-oedema, Figure 2. These symptoms are characteristically transient, resolving as the extravasated fluid is re-absorbed into the circulation.

Brief, mild urticaria is very common. The lifetime prevalence of urticaria is around 15%. A minority of patients have more severe symptoms or chronic disease, which can be difficult to manage. This is a practical guide to assist with the more difficult clinical problems which can occur. Urticaria can be associated with considerable distress and disability as shown by quality of life studies.

Firstly, make sure the patient does have urticaria

Perhaps 1 in 10 patients referred to an urticaria clinic do not have the condition at all. As the signs are transient it is common for the rash/swelling to be absent when the patient attends therefore the history is of paramount importance. Patients are now in the habit of photographing their rash and this can be helpful although the quality is sometimes not sufficient to confirm the diagnosis.

The main diagnostic features of urticaria are:
1. Individual lesions last hours
2. It resolves leaving normal skin
3. Lesions itch

The most important differential diagnosis is contact dermatitis, particularly of the face, which can result in severe swelling. Weeping and subsequent desquamation do not occur in urticaria and their presence should lead you to suspect contact dermatitis and organise patch testing. Other differentials are listed in Table 1. The diagnosis can usually be made on the history, but arranging to see the patient on an “SOS” basis when the rash is present or asking the patient to take photos can be helpful.

Secondly classify the urticaria

There are a number of differing classifications of urticaria. By definition urticaria lasting less than 6 weeks is
termed acute as opposed to chronic. A large proportion of patients with chronic urticaria are now known to have an auto-immune mechanism and these patients are classified with others who have no exogenous cause as having “spontaneous” urticaria (formally termed “ordinary” or “idiopathic”). A further group of well known urticarias are induced reproducibly by physical stimuli eg friction causing dermographism. See Table 2. In addition urticaria/angio-oedema can occur as part or all of an IgE mediated allergy, as a drug reaction eg to non-steroidal antiinflammatory (NSAID) drugs, as a contact phenomenon and rarely in association with systemic disease eg SLE, or inherited disorders eg C1 esterase inhibitor deficiency, auto-inflammatory syndromes. Around 10% of urticaria patients report that their symptoms are worsened by NSAIDs and it is helpful to ask specifically if this is the case. ACE inhibitors are now a well known cause of angio-oedema and this characteristically affects the oro-pharynx and may cause respiratory difficulties. This is thought to be a kinin-mediated problem and may first be manifest many months or even years after starting the drug.

During history taking you will be able to establish whether the patient has acute or chronic urticaria, whether physical factors induce the problem, relevant drugs or apparent allergen ingestion, family history, unusual characteristics such as systemic symptoms accompanying the outbreak or long lasting weals or swellings which suggest urticarial vasculitis. Table 3 shows a classification of urticarial disorders.

**Thirdly choose appropriate investigations**

If a physical urticaria is suspected then it is possible to perform confirmatory tests in the clinic for dermographism, cold urticaria and cholinergic urticaria. Other physical challenge tests may require additional facilities. See Table 2. Check the likely severity of the reaction before testing. If the patient is on a non-sedative antihistamine a negative challenge does not exclude the disorder.

If a young, healthy person presents with classical chronic urticaria it is likely that blood tests will not alter management or reveal any underlying cause. In acute urticaria tests to exclude an infection as a trigger may be indicated. Relevant blood tests should be done for systemic disease in patients who have other symptoms such as arthralgia, myalgia.

Any patient who presents with angio-oedema in the absence of urticaria should have their C3, C4, and C1 esterase inhibitor levels checked. If the C4 is low but the C1 esterase inhibitor level is normal then a functional assay of C1 esterase inhibitor needs to be done, for this to be accurate the blood sample has to arrive and be processed quickly at the laboratory.

There is now reasonable evidence that patients with confirmed *H. Pylori* infection and chronic urticaria have a

<table>
<thead>
<tr>
<th>Physical urticaria</th>
<th>Test</th>
<th>Other information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dermographism</td>
<td>scratch skin with non-sharp implement, appears in 5-10 minutes</td>
<td>Common, often co-exists with chronic urticaria. Figure 3</td>
</tr>
<tr>
<td>Delayed pressure urticaria</td>
<td>tested by hanging weight over limb, appears after hours</td>
<td>Less common, always co-exists with spontaneous symptoms. Figure 4</td>
</tr>
<tr>
<td>Cholinergic*</td>
<td>exercise until sweating occurs</td>
<td>Usually occurs alone, not very responsive to anti-histamines</td>
</tr>
<tr>
<td>Cold urticaria</td>
<td>ice cube test Figure 5</td>
<td>not always responsive to anti-histamines</td>
</tr>
<tr>
<td>Solar</td>
<td>needs monochromator testing</td>
<td>rare</td>
</tr>
<tr>
<td>Exercise induced anaphylaxis</td>
<td>do not test</td>
<td>could symptoms be food-dependent?</td>
</tr>
<tr>
<td>Aquagenic</td>
<td></td>
<td>rare</td>
</tr>
<tr>
<td>Localised heart urticaria</td>
<td></td>
<td>rare</td>
</tr>
<tr>
<td>Vibratory angio-oedema</td>
<td></td>
<td>rare</td>
</tr>
<tr>
<td>Adrenergic urticaria</td>
<td></td>
<td>rare</td>
</tr>
</tbody>
</table>

* cholinergic urticaria included for simplicity but is not considered strictly to be a physical urticaria as the cause is a rise in body temperature not an exogenous factor
greater chance of remission if the H. Pylori is treated and tests for this may be indicated if initial treatment is unsuccessful.

The autologous serum test which gives evidence of auto-immunity is not performed in routine practice.

Urticaria is associated statistically with positive thyroid auto-antibodies but the incidence of thyroid disease is not statistically increased.

In childhood urticaria there is some evidence of an increased prevalence of positive coeliac antibodies.

**Allergy tests in urticaria**

The vexed question of allergy tests in urticaria

Some patients develop a belief that their urticaria is an allergy even when it is not associated with a particular food. Occasionally this can lead to self-imposed severe dietary restriction. This belief can be promoted by discussion with medical personnel at times. The evaluation of the history is important to assess whether there could be an allergic component. It should also be borne in mind that false negatives and irrelevant positive specific IgE tests occur. Patients may have a positive blood test but no reaction to ingestion of the food at all if they are in a state of tolerance. The test results therefore have to be interpreted in light of the history.

Most centres only have a limited number of specific IgE tests available. I only send these if I suspect a food allergy or on the occasions when I think it is helpful for the patient to know that they are not allergic to a food. Other tests eg for house dust mite or pollen sensitivity can indicate if a patient is atopic, as can the total IgE but these will not determine the aetiology of the urticaria.

**Table 3: Practical classification of urticarias**

<table>
<thead>
<tr>
<th>1. By duration</th>
<th>2. By cause/mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute</td>
<td>Spontaneous</td>
</tr>
<tr>
<td>lasts less than 6 weeks</td>
<td>Systemic</td>
</tr>
<tr>
<td></td>
<td>Physical</td>
</tr>
<tr>
<td></td>
<td>Drugs</td>
</tr>
<tr>
<td></td>
<td>Allergic</td>
</tr>
<tr>
<td></td>
<td>Inherited</td>
</tr>
<tr>
<td>Chronic</td>
<td>May be acute or chronic, 40% thought to have an auto-immune mechanism</td>
</tr>
<tr>
<td>lasts over 6 weeks</td>
<td>Reproducibly associated with a physical change, can be tested for, see Table 2</td>
</tr>
<tr>
<td></td>
<td>May be allergic eg to penicillin or pharmacological eg to NSAIDs, ACE inhibitors</td>
</tr>
<tr>
<td></td>
<td>Eg in SLE, Schnitzler’s syndrome</td>
</tr>
<tr>
<td></td>
<td>Eg to foods, animal saliva includes some contact urticaria</td>
</tr>
<tr>
<td></td>
<td>C1 esterase inhibitor deficiency(angio-oedema alone) Autoinflammatory syndromes</td>
</tr>
</tbody>
</table>
Remember that there is a rare condition called food dependent exercise induced anaphylaxis in which a specific food taken before exercise results in an urticarial or anaphylactic response. This should be thought of in patients presenting with exercise induced problems. Such patients usually have a weak positive specific IgE to the food and develop symptoms only when the food and exercise are combined.

Fourthly initiate or change treatment to optimise control

Any causative factor such as an ACE inhibitor should be stopped.

The mainstay of treatment for urticaria/angio-oedema is with H1 antihistamines. If the patient seems to have poorly controlled urticaria consider the following:

1. Is a non-sedating antihistamine being taken regularly as opposed to as required?
2. Is the antihistamine being taken at the right time? eg if symptoms are worse first thing in the morning it should be taken at bedtime
3. Is the problem that the effect wears off before 24 hours if so either increase the frequency to twice daily (as advised in guidelines) or give 2 different non-sedating antihistamines 12 hours apart

If after doing this control it is still not good then the next steps would be to increase the dose of antihistamines eg desloratadine 5mg daily can be increased to 5mg tds, cetirizine 10mg od can be increased to 10mg bd. A change to a different non-sedating antihistamine can also be effective. The next stage would be to add a drug for which there is some evidence but which is not licensed for urticaria such as ranitidine 150mg bd or montelukast 10mg nocte.

Short courses of systemic steroids can be helpful in severe acute urticaria. I also use quite low doses of steroids eg prednisolone 10-20mg daily for 2-3 days for attacks of angio-oedema as this decreases the severity and shortens the time of an attack so that patients can return to work/normal activities sooner.

Learning points

- The diagnosis of urticaria is largely based on the history
- Investigations should be tailored to the patient and may not be necessary
- Patients with angio-oedema alone should be tested for C1 esterase inhibitor deficiency
- ACE inhibitors can cause angio-oedema and the onset may be months or years after the drug is commenced
- Changing to a different antihistamine, increasing the dose and altering the time of administration may lead to better control of symptoms

Acknowledgements

Figures 1 and 3-5 are reproduced with kind permission Cardiff University and are copyright.

Figure 2 is reproduced with kind permission Dr Niels Veien from the Danderm Atlas.

Further reading

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The definition and diagnostic testing of physical and cholinergic urticarias- EAACI/GA2LEN/EDF/UNEV consensus panel recommendations. Magerl M et al.
Allergy 2009 64, 1715-1721.
“I was crippling shy as a child and things only got harder to deal with as a teenager. I felt very angry most of the time. It was a painful emotion to live with, and I began to isolate myself. It was only at university that I developed the social skills to cope and not let other people’s attitudes dominate my life.” Woman in her 20’s with a port-wine stain.

People with skin conditions often experience depression, anxiety, embarrassment, lack of confidence, low self-esteem and, in extreme cases, contemplate suicide. They have to cope with staring, intrusive questions and unhelpful or inappropriate comments exacerbating a deterioration in quality of life impacting on schooling, employment, using public transport, making friends, forming relationships, social life, health care, shopping, swimming, intimacy and hygiene. Research confirms that despite the vast range of medical skin conditions, the psycho-social concerns of patients who require medical care are very similar (Rumsey and Harcourt, 2004).

Changing Faces, the UK’s leading charity that supports and represents people living with conditions that affect appearance are currently running a campaign targeting dermatology services seeking better integrated support and advice for people living with skin conditions, such as vitiligo, acne or eczema.

Often patients report that whilst their clinical needs are addressed, the impact of their condition on their appearance goes unrecognised or untreated. In addition they often experience lack of information provision, lack of joined-up care and long waiting lists. In a recent survey carried out by Changing Faces, 61% of those seeking help for their skin could not find any support in addition to their medical treatment.

“Health professionals need to understand the impact of watching helplessly as your appearance changes and also realise the social impacts that you go through, not being able to go outdoors or take part in normal social life. It affects way more than just your skin.”

It is also important for clinicians to appreciate that the extent or severity of a skin condition does not necessarily correlate with the degree of emotional and social distress caused. This means that treating it medically or surgically (even virtually removing it) is no guarantee that the patient will adjust well psychologically. Kleve and Robinson (1999) highlight the importance of ‘perceived severity’ or noticeability of disfigurement rather than objective severity as the critical indicator of good or poor outcome. In other words, it is how the individual interprets the condition that is critical to their long-term recovery i.e. their own self-appraisal, personality and coping style.

Patients need support and advice to cope in all areas of their lives and the best long-term, psycho-social adjustments come with help from appropriately resourced professionals able to offer an effective package of help and support designed to promote living confidently. The package designed by Changing Faces is essentially a cognitive-behavioural approach combining a mix of psycho-educational direction and psycho-therapeutic support, known as FACES.

- **F** – FINDING OUT – to gain realistic information about their condition and its treatment
- **A** – ATTITUDE – to develop a positive outlook and beliefs about the future
- **C** – COPING with feelings – to have access to a trained professional to talk to
- **E** – EXCHANGING – to share experiences with others in similar situations
- **S** – SOCIAL SKILLS – to learn the skills to handle the reactions of strangers, friends etc.
Its overall aim is to promote self-esteem by enabling people to examine their feelings, beliefs and behaviour and to explore alternatives where appropriate.

The FACES model can be adapted to a hospital or community setting and a graded approach is recommended, setting goals for those living with skin conditions. This helps them prepare and develop the number and variety of coping strategies with which they feel comfortable. All healthcare professionals, including primary care staff, need greater knowledge and skills to support their patients and help them cope.

The “Look at Me” campaign is seeking to ensure that people with a range of skin conditions receive appropriate psycho-social care in addition to medical treatment integrated into their care pathways. No dermatological consultation is complete without addressing the psychological component (BAD Working Party Report on minimum Standards for Psychodermatology Services 2012). The campaign is seeking:

1. To influence the development and implementation of quality standards in dermatology to include the provision of psycho-social care
2. To raise awareness and understanding of the psycho-social impact of disfiguring skin conditions amongst health and social care professionals and commissioners in the area of dermatology
3. To raise awareness amongst people with skin conditions to understand what service they should be able to expect from their health and social care professionals.

Primary care providers are key players in seeing patients with skin conditions and in the design and delivery of local services needs to ensure that patients’ psycho-social needs are addressed. Changing Faces is currently looking for examples of good practice within primary care and to influence future service delivery at local level.

A final campaign report will be published in autumn 2013 with recommendations on best models of care within primary care services, to promote best patient outcome for patients with skin conditions.

For further information, contact Henrietta Spalding at henrietta.spalding@changingfaces.org.uk

Julian Peace
GPSI Sheffield & PCDS Treasurer

Journal Watch
November 2012 – February 2013

As we shiver in the depths of winter, although anticipating the first green shoots of spring around the corner, it is always wise to be prepared for the future. Sunscreen may be the last thing on our minds at the moment, but a new paper from Japan1 provides us with the evidence that we need to assist our patients with their sun-protection. As we all know, sunscreens are graded by their sun protection factor (SPF). What you may not know is that the SPF is determined after the application of a standard amount of sunscreen to the skin. In practice, it is all but impossible to replicate the proscribed amount of sunscreen - 2mg/cm² – rendering most SPF values as purely aspirational. A double application of screen, however, approximates the true SPF value – it is this method we should recommend.

It suddenly struck me, in a moment of reverie, that we do not have enough references to eccentric German dermatologists in these pages. As Heinrich Koechner was both German, and eccentric, I propose to right this wrong forthwith. A team in Belgium have introduced a new assessment tool2 looking specifically at the Koechner phenomenon (KP) in vitiligo. Although it is well over 100 years since this association was first described, there is still precious little research as to exactly why some cutaneous diseases Koechnerise and others don’t. One hypothesis, although backed with little data as yet, is that KP can be a marker of disease activity. In this study, if the vitiligo was associated with KP, it was likely to have appeared at an earlier age, and there was an elevated risk of further depigmentation. A sub-group, who had artefactual KP (in scratches and scars, rather than in areas of friction), were also more likely to have co-existent thyroid disease. Early days, but interesting, nonetheless.

One of the curiosities of living in an Island nation, is the somewhat, well, insular view it encourages. A Europe wide perspective on the incidence of malignant melanoma (MM)3 is,
therefore, likely to be of great interest for those of us who look beyond these shores. In 2008, there were approximately 20,000 deaths from MM in Europe. Incidence was highest in Switzerland and lowest in Greece whilst death rates were highest in Norway, Greece again being the lowest. The UK has a slightly above average incidence, and a slightly below average death rate. The discrepancies are quite marked, with the Swiss incidence being nine times that of Greece. The death rate was highest in central and eastern Europe, possibly representing missed opportunities for early diagnosis. Whilst we strive to improve things in our own back yard, there may be more opportunities to improve the situation elsewhere...

Next, something remarkably topical. As the father of two teenage daughters, it was brought to my attention that it was possible to buy small ultra-violet A (UVA) units designed for home use for curing gel nails and setting more conventional nail varnishes. Naturally, I was concerned about this – after all, UVA can cause skin cancers, but more importantly, I might be expected to provide funds to purchase such a unit! Thankfully, the risk of neoplasia is extremely small, and the use of fingerless gloves prior to ‘curing’ virtually eliminates even this small risk. The risk of purchase was also successfully eliminated, without the need for gloves of any kind.

My reader will notice that I am now revisiting something already mentioned above. This may turn in to a theme for this bulletin...We have already commented on the association of KP in vitiligo with thyroid disease, it also appears that vitiligo is associated with thyroid disease, period. A systematic review of studies published between 1968 and 2012 show risk ratios of having both diseases varying between 1.9 and 5.2 (all statistically significant). This falls short of creating a recommendation of screening vitiligo sufferers, but does suggest that vigilance is needed for signs of co-existent conditions.

We still await the results of a large, prospective study looking at disease associations with psoriasis. Whilst we tap our feet and impatiently drum our fingers, meta-analyses of existent data suggests that psoriasis does indeed significantly increase the risk of both stroke and myocardial infarction. The effect is independent of conventional cardiovascular risk factors.

Surprisingly little data is available concerning the role of GPs in the diagnosis of melanoma. For this paper we look to our colleagues in France and their experience. It is remarkable how similar the situation appears on both sides of the Channel. GPs tended to diagnose thicker tumours, by definition those tumours with a poorer prognosis. However, there was a direct correlation between GPs with extra training in lesion recognition and thinner melanomas being diagnosed. In France, as in the UK, increasing patient awareness of melanoma has to be linked to training of GPs in lesion recognition. Now where have I heard that before?

Psoriasis is a disease with both physical and psychological components. It always comes as something of a surprise to see how poor adherence to treatment really is – some studies show adherence rates as low as 22%. Lifestyle changes can also have a significant role to play in disease activity and yet, as this literature review demonstrates, none of the 29 papers concerning adherence to treatment studied examine adherence to advice about lifestyle change. There is evidently much to be done to improve this situation. We forget, in this world we work in of targets and deadlines that we need to take a more holistic approach to many of our patients.

On to another bit of déjà vu (or is that déjà déjà vu?). Although vitiligo is often easy to diagnose, the pathogenic and aetiological mechanisms for its development are still unclear. This, almost inevitably, tends to hold back progress in treatment. Guidelines have, previously, been generated at national levels, but here is an attempt to create a set of guidelines at European level. Sadly, it is a disease with little definitive recommendations for successful treatment. As such, a void is created which can all too often be filled by treatments of limited clinical efficacy and vulnerable patients can be enticed by promises that have more in common with charlatanism than with sound, evidence based medicine. A stepwise approach to treatment is proposed. In segmental vitiligo, this would comprise avoidance of triggering factors, topical therapies (corticosteroids, calcineurin inhibitors) and then phototherapy followed by surgical techniques, if considered appropriate. Non-segmental vitiligo follows the same pathway, but followed by systemic therapies (steroids, immunosuppressants), grafting and finally, depigmenting techniques. Camouflage is an option at all stages of the disease and psychological therapies can also be helpful in both the short and long term. I would recommend seeking out this set of guidelines in their entirety, a summary such as this can only scratch the surface and the paper repays close examination.

As any fule kno, dermoscopy is now an essential tool for diagnosing pigmented lesions. Although recognised to be widely used in practice, there are only data available from Australia and USA of how many practitioners use this technique. No more, came the cry! We have a study from
France to wave from the top of the barricades. Uptake there has been 94.6% of respondents, the majority of whom (83%) use it several times a day for the diagnosis of both pigmented and non-pigmented lesions. This was a large study, of over 3000 dermatologists. In the UK we can only dream of such numbers, but as this is the first published European study it does show excellent penetration of this vital technique.

Back to the sunshine. The SunSmart campaign has been ongoing in Australia since 1988. A paper from Victoria takes a retrospective look at its success in changing behaviour in a range of Ozzie populations. There was an initial rapid improvement in behaviour – with more use of sunscreens, less unprotected exposure and less sunburn, with a subsequent slower improvement as the programme became ‘part of the furniture’. There are signs that there is some decline in behaviour being noted suggesting that regular intensive campaigns may engender greater long term benefit by reminding the target populations of the dangers lurking out there. What is evident is that the SunSmart programme continues to be an effective method of population education – if only the Department of Health in the UK could be convinced similarly...

Meanwhile, in Iceland, a team are looking at the role the tonsils play in both the pathogenesis of and the prognosis of psoriasis. Patients with psoriasis do, it appears, suffer more sore throats than non-psoriatic individuals. It is also well documented that streptococcal sore throats can be the trigger for the onset of psoriasis. What does appear also to be the case is that psoriatics who have their tonsils removed suffer less severe outbreaks of their disease and also require less treatment. A little early, perhaps, to advise tonsillectomy as part of standard treatment, but interesting nonetheless.

A brief therapeutic interlude. Hidradenitis suppurativa is, as I have mentioned before, one of our greatest treatment challenges. Anything that points towards a better management strategy is to be congratulated. In this systematic review both retinoids and immunosuppressive agents were studied – the best results coming from the use of acitretin, infliximab and adalimumab, although not, curiously, isotretinoin. Whilst this is still both experimental and off license, it does point towards a new hope for therapeutic options for this distressing disease.

So there you have it. Another cornucopia of condensed commentary. Until next time...

4. Diffey – The risk of squamous cell carcinoma in women from exposure to UVA lamps used in cosmetic nail treatment. BJD2012;167;1175-1178.
Zyclara 3.75% cream (imiquimod). Indications: Zyclara is indicated for the topical treatment of clinically typical, nonhyperkeratotic, nonhypertrophic, visible or palpable actinic keratosis (AK) of the full face or balding scalp in immunocompetent adults when other topical treatment options are contraindicated or less appropriate. Dosage: Treatment should be initiated and monitored by a physician. Apply up to 2 sachets, once daily, before bedtime to the skin of the affected treatment area for two treatment cycles of 2 weeks each separated by a 2-week no-treatment cycle or as directed by the physician. The treatment area is the full face or balding scalp. The safety and efficacy of imiquimod in AK in children and adolescents below the age of 18 years have not been established. For external use only. Contact with eyes, lips, and nostrils should be avoided. The treatment area should not be bandaged or otherwise occluded. Apply as a thin film to the entire treatment area and rub in until the cream vanishes. Partially-used sachets should be discarded and not reused. Leave on the skin for approximately 8 hours; after this time it is essential that the cream is removed by washing the area and the hands with mild soap and water. Contraindications: Hypersensitivity to the active substance or to any of the excipients. Warnings and precautions: Lesions clinically atypical for AK or suspicious for malignancy should be biopsied to determine appropriate treatment. Not recommended until the skin has healed after any previous medicinal products or surgical treatment. Use of sunscreen is encouraged; and patients should minimise or avoid exposure to natural or artificial sunlight. Not recommended for the treatment of AK lesions with marked hyperkeratosis or hypertrophy as seen in cutaneous horn. During therapy and until healed, affected skin is likely to appear noticeably different from normal skin. Local skin reactions are common but generally decrease in intensity during therapy or resolve after cessation of therapy. Rarely, intense local inflammatory reactions including skin weeping or erosion can occur after only a few applications. There is an association between the complete clearance rate and the intensity of local skin reactions. These local skin reactions may be related to the stimulation of local immune response. Imiquimod has the potential to exacerbate inflammatory conditions of the skin. If required by the patient’s discomfort or the intensity of the local skin reaction, a rest period of several days may be taken. Treatment can be resumed after the skin reaction has moderated. The intensity of the local skin reactions tend to be lower in the second cycle than in the first treatment cycle. Flu-like systemic signs and symptoms may accompany, or even precede, intense local skin reactions and may include fatigue, nausea, fever, myalgia, arthralgia, and chilli. An interruption of dosing or dose adjustment should be considered. Use with caution in patients with reduced haemalogic reserve. Patients with cardiac, hepatic or renal impairment were not included in clinical trials. Caution should be exercised in these patients. Use with caution in immunocompromised patients and/or patients with autoimmune conditions and consider balancing the benefit of treatment for these patients with the risk associated either with the possibility of organ rejection or graft versus-host disease or a possible worsening of their autoimmune condition. No data are available on re-treating AK that have cleared after two cycles of treatment and subsequently recur. Stearyl alcohol and cetyl alcohol may cause local skin reactions. Methyl parahydroxybenzoate (E 218), and propyl parahydroxybenzoate (E 217) may cause allergic reactions (possibly delayed). No interaction studies have been performed but use with caution in patients who are receiving immunosuppressive drugs. Avoid using with any other imiquimod creams in the same treatment area. No data are available on the use of Zyclara during pregnancy or breast-feeding and there are no data on the risk to human fertility. There is no or negligible influence on the ability to drive or use machinery. Side effects: Herpes simplex, skin infection, lymphadenopathy, haemoglobin, white blood cell and platelet counts decreased, anaemia, blood glucose increased, insomnia, depression, headache, dizziness, nausea, diarrhoea, vomiting, erythema, scab, skin exfoliation, skin oedema, skin ulcer, skin hypopigmentation, dermatitis, erythema multiforme, Stevens Johnson syndrome, cutaneous lupus erythematous, skin hyperpigmentation, myalgia, arthralgia, application site effects, including erythema, scabbing, exfoliation, dryness, oedema, ulcer; discharge, reaction, pruritus, swelling, burning, irritation and rash, fatigue, pyrexia, influenza like illness, pain, chest pain. Consult the Summary of Product Characteristics before prescribing, particularly in relation to side effects, precautions and contraindications. Legal Category: POM. Pack size and basic NHS price: Pack of 28 sachets £113.00. Product licence number: EU/1/2012/783/002. Marketing authorisation holder: Meda AB. Pipers väg 2A, 170 73 Solna, Sweden. Date of preparation of prescribing information: January 2013. UK/2YC/13/0003

Adverse events should be reported. Reporting forms and information can be found at www.mhra.gov.uk/yellowcard. Adverse events should also be reported to Meda Pharmaceuticals Ltd.

Forthcoming Meetings 2013

Dermoscopy for Beginners 2013
• 9th May BELFAST
• 16th May MILTON KEYNES
• 7th June LONDON
• 26th September BRISTOL
• 7th November EDINBURGH
• 21st November LEEDS

Advanced Dermoscopy 2013
• 3rd October LONDON

Essential Dermatology and Level 2 2013
• 7th March EXETER – EDL2
• 15th May LEICESTER – EDL2
• 13th June DUBLIN – ED
• 19th June WARRINGTON – ED
• 11th September NOTTINGHAM – ED
• 25th September LONDON – ED
• 17th October DURHAM – ED
• 20th November LEEDS – EDL2
• 28th November COLCHESTER – ED

PCDS 2013:
Spring Meeting
16th & 17th March, Manchester

Improvers Skin Surgery
26th & 27th April, Barnstaple

Summer Meeting
6th June, London

Autumn Meeting
12th September, Nottingham

Scottish Meeting
9th & 10th November, Edinburgh