AUTUMN MEETING

Thursday 12 th & Friday 13 th October 2017

Farnham Estate, Co Cavan.

Enbrel® etanercept

Transforming lives[1]

1st approved anti-TNF in RA[1-7]

15 years of clinical trials and real world experience[1]

More than 400 trials[†8]

More than 6400 publications[†9]

5 Over million patient-years of collective clinical experience[†11]

1 Over million patients treated[†10]

over 15 years of partnership and experience[1]

Pfizer

ABBREVIATED PRESCRIBING INFORMATION
Enbrel®
etanercept

Before prescribing Enbrel® please refer to full Summary of Product Characteristics (SmPC).

Presentation: Enbrel Pre-filled Syringe: Enbrel 25 mg and 50 mg solution for injection in pre-filled syringe. Each pre-filled syringe contains either 25 mg or 50 mg etanercept. Enbrel Pre-filled Pen (MYCLIC®): Enbrel 25mg and 50 mg solution for injection in pre-filled pen. Each pre-filled pen contains either 25mg or 50 mg etanercept. Enbrel Powder: Enbrel 25 mg powder and solvent for solution for injection. Each vial contains 25 mg etanercept and each pre-filled syringe contains 1 ml water for injections. Enbrel Paediatric: Enbrel 10 mg powder and solvent for solution for injection for paediatric use. Each vial contains 10 mg etanercept and each pre-filled syringe contains 1 ml water for injections. **Uses:** *Adults:* Moderate to severe active rheumatoid arthritis (RA), in combination with methotrexate, when response to disease-modifying anti-rheumatic drugs DMARDs, including methotrexate (unless contraindicated), has been inadequate. Enbrel can be given as monotherapy in the case of intolerance to methotrexate or when continued treatment with methotrexate is inappropriate. Severe, active and progressive RA without prior methotrexate treatment. Enbrel alone or with methotrexate has been shown to reduce the rate of progression of joint damage measured by X-ray and to improve physical function. Patients with moderate to severe plaque psoriasis (PP) who failed to respond to, or who have a contraindication to, or are intolerant to other systemic therapy including ciclosporine, methotrexate or PUVA. Active and progressive psoriatic arthritis (PsA) when response to DMARDs has been inadequate. Enbrel has been shown to improve physical function in PsA patients, and to reduce the progression rate of peripheral joint damage as measured by X-ray in patients with polyarticular symmetrical subtypes of PsA. Severe active ankylosing spondylitis (AS) when response to conventional therapy has been inadequate. Non-radiographic axial spondyloarthritis (nr-axSpA).Treatment of adults with severe non-radiographic axial spondyloarthritis with objective signs of inflammation as indicated by elevated C-reactive protein (CRP) and/or magnetic resonance imaging (MRI) evidence, who have had an inadequate response to nonsteroidal anti-inflammatory drugs (NSAIDs). *Children aged 2-17 years:* Juvenile idiopathic arthritis (JIA). Polyarthritis (rheumatoid factor positive or negative) and extended oligoarthritis from the age of 2 years when inadequate response to, or intolerant of methotrexate. Psoriatic arthritis from the age of 12 years when inadequate response to, or intolerant of methotrexate. Enthesitis-related arthritis from the age of 12 years when inadequate response to, or intolerant of, conventional therapy. *Children aged 6-17 years:* Chronic severe psoriasis when inadequately controlled by, or intolerant to, other systemic therapies or phototherapies. **Dosage:** RA – 25 mg twice weekly or 50 mg once weekly PP - 25 mg twice weekly or 50 mg once weekly for up to 24 weeks, or 50 mg twice weekly for up to 12 weeks followed by 25 mg twice weekly or 50 mg once weekly for a further 12 weeks if needed. Continuous therapy may be appropriate for some adult patients. Discontinue if no response after 12 weeks. For re-treatment: 25 mg twice weekly or 50 mg once weekly for up to 24 weeks. AS, nr-axSpA and PsA – 25 mg twice weekly or 50 mg once weekly. *Children aged 2-17 years:* JIA – 0.4 mg/kg (maximum per dose 25 mg) twice weekly with an interval of 3 – 4 days or 0.8 mg/kg (maximum per dose 50 mg) once weekly. Discontinuation of treatment should be considered in patients who show no response after 4 months. *Children aged 6-17 years:* Plaque psoriasis in children aged 6-17 years – 0.8 mg/kg (maximum per dose 50 mg) once weekly for up to 24 weeks. Discontinue if no response after 12 weeks. For re-treatment: 0.8 mg/kg (maximum per dose 50 mg) once weekly for up to 24 weeks. **Contra-indications:** Hypersensitivity to any of the ingredients, sepsis or risk of sepsis, active infections. **Warnings and Precautions:** In order to improve the traceability of biological medicinal products, the trademark and the batch number of the administered product should be clearly recorded (or stated) in the patient file. Enbrel should be initiated and supervised by specialist physicians experienced in the diagnosis and treatment of RA, JIA, PsA, AS, PP or Paediatric PP. Patients treated with Enbrel should be given the Patient Alert Card. Use carefully in patients predisposed to, or with history of, infection due to underlying diseases other than RA (e.g. advanced or poorly controlled diabetes) or with history of blood dyscrasias, pre-existing or predisposition to demyelinating disease or congestive heart failure (CHF). There have been rare (< 0.1%) reports of new onset CHF, including CHF in patients without known pre existing cardiovascular disease, including patients under 50 years of age. Cases of active tuberculosis have been reported, therefore all patients should be evaluated for both active and inactive TB prior to being treated with Enbrel. If active TB is diagnosed, Enbrel should not be initiated. Caution should be used when administering Enbrel to patients previously infected with hepatitis B and there have been reports of worsening hepatitis C in patients receiving Enbrel. Use with caution in patients with a history of hepatitis C. Whether treatment with Enbrel might influence the development and course of active and/or chronic infections is unknown. Concurrent administration of Enbrel and anakinra has been associated with increased risk of serious infections and neutropenia, and is therefore not recommended. In clinical studies, concurrent administration of abatacept and Enbrel resulted in increased incidences of serious adverse events, and is therefore not recommended. Use caution when considering combination therapy with DMARDs other than methotrexate. Reports of various malignancies have been received in the post-marketing period, therefore with current knowledge, a possible risk for the development of lymphomas, leukaemia or other haematopoietic or solid malignancies in patients treated with a TNF-antagonist cannot be excluded. Malignancies, some fatal, have been reported among children, adolescents and young adults (up to 22 years of age) treated with TNF-antagonists (initiation of therapy ≤ 18 years of age) in the post marketing setting. Melanoma and non-melanoma skin cancer (NMSC) have been reported in patients treated with TNF-antagonists, including Enbrel. Post-marketing cases of Merkel cell carcinoma have been reported very infrequently in patients treated with Enbrel. Periodic skin examination is recommended for all patients, particularly those with risk factors for skin cancer. Enbrel has not been studied in combination with other systemic therapies or phototherapy for the treatment of psoriasis. Monitor closely if patient develops new infection during treatment. Discontinue treatment if serious infection or allergic reaction develops or if blood dyscrasias are confirmed. Caution should be used in patients who have moderate to severe alcoholic hepatitis and Enbrel should not be used in patients for the treatment of alcoholic hepatitis. Discontinue temporarily if significantly exposed to varicella virus. Live vaccines should not be given concurrently with Enbrel. Paediatric patients should have received all vaccines recommended in current immunization guidelines prior to starting Enbrel. Treatment with Enbrel may result in the formation of autoantibodies. Enbrel is not recommended for use in patients with Wegener's granulomatosis. There have been reports of hypoglycaemia in Enbrel patients receiving medication for diabetes, necessitating a reduction in anti-diabetic medication in some of these patients. There have been reports of Inflammatory Bowel Disease (IBD) and uveitis in JIA patients being treated with Enbrel. Caution should be exercised when treating the elderly and with particular attention to occurrence of infections. **Pregnancy & Lactation:** Enbrel is not recommended in pregnant or breast-feeding women. **Undesirable Effects:** *Adults:* The most commonly reported adverse reactions are injection site reactions, infections, allergic reactions, development of autoantibodies, itching, and fever. See SmPC for less commonly reported side effects. TNF-antagonists, such as Enbrel, affect the immune system and their use may affect the body's defences against infection and cancer. Serious infections affect fewer than 1 in 100 patients treated with Enbrel. Reports have included fatal and life threatening infections and sepsis. Various malignancies have also been reported with use of Enbrel, including cancers of the breast, lung, skin and lymphatic system (lymphoma). Serious infections and other adverse events such as uncommon reports of: thrombocytopenia, systemic vasculitis, uveitis and scleritis, interstitial lung disease, elevated liver enzymes, rare reports of tuberculosis, opportunistic infections, anaemia, leucopoenia, neutropenia, pancytopenia, seizures, heart failure, autoimmune hepatitis, Steven Johnson's syndrome, anaphylaxis, and very rare reports of: toxic epidermal necrolysis and aplastic anaemia have been reported. Reactivation of hepatitis B (a liver infection) and worsening of symptoms of dermatomyositis have also been reported. Central and peripheral demyelinating events have been seen rarely and very rarely, respectively, with Enbrel use. There have been rare reports of lupus, lupus-related conditions, and vasculitis. Rate of new malignancies was similar to that expected for the population studied. Fatalities associated with serious infections, pancytopenia, aplastic anaemia and interstitial lung disease have also been reported. *Paediatrics:* Generally as for adults, except the following were more common: headaches, nausea, vomiting and abdominal pain. In addition the following were reported as severe events: varicella, appendicitis, gastroenteritis, depression/personality disorder, cutaneous ulcer, oesophagitis/gastritis, group A streptococcal septic shock, type I diabetes mellitus and soft tissue and post-operative wound infection. There have been post-marketing reports of IBD and uveitis in JIA patients, including cases indicating a positive re-challenge. See section 4.8 of the SmPC for how to report adverse reactions. **Package Quantities:** Enbrel Pre-filled Syringe: Each carton contains 4 pre-filled syringes containing either 25 mg or 50 mg of Enbrel and 4 alcohol swabs. Enbrel Pre-filled Pen (MYCLIC): Each carton contains 4 pre-filled pens containing either 25mg or 50 mg of Enbrel and 4 alcohol swabs. Enbrel Powder: Each carton contains 4 vials of Enbrel 25 mg powder, 4 pre-filled syringes of water for injections, 4 needles, 4 vial adaptors and 8 alcohol swabs. Enbrel Paediatric (10 mg): Each carton contains 4 vials of Enbrel 10 mg powder, 4 prefilled syringes of water for injections, 4 needles, 4 vial adaptors and 8 alcohol swabs. **European Marketing Authorisation Numbers:** Enbrel Pre-filled Syringe 25 mg: EU/1/99/126/013 Enbrel Pre-filled Syringe 50 mg: EU/1/99/126/017 Enbrel Pre-filled Pen (MYCLIC) 25 mg: EU/1/99/126/023 Enbrel Pre-filled Pen (MYCLIC) 50 mg: EU/1/99/126/020 Enbrel Powder 25 mg: EU/1/99/126/003 Enbrel Paediatric 10 mg: EU/1/99/126/022. **Legal Category:** S1A. **European Marketing Authorisation Holder:** Pfizer Limited, Ramsgate Road, Sandwich, Kent, CT13 9NJ, UK. **For full prescribing information see the Summary of Product Characteristics. For further information on this medicine please contact:** Pfizer Medical Information on 1800 633 363 or at EUMEDINFO@pfizer.com. For queries regarding product availability please contact: Pfizer Healthcare Ireland, Pfizer Building 9, Riverwalk, National Digital Park, Citywest Business Campus, Dublin 24 + 353 1 4676500. **API Reference Number:** EN 11_0 **Pfleet number:** 2017-0024332. **Date of Prescribing Information:** May 2017.

† Across all indications.

References: 1. Scott LJ. Drugs. 2014;74:1379-1410. **2.** Enbrel Summary of Product Characteristics. **3.** Humira Summary of Product Characteristics. **4.** Remicade Summary of Product Characteristics. **5.** Cimzia Summary of Product Characteristics. **6.** Simponi Summary of Product Characteristics. **7.** Remicade EMA report **8.** www.clinicaltrials.gov. Date accessed: May 2016. **9.** http://www.ncbi.nlm.nih.gov/pubmed. Date accessed: May 2016. **10.** Data on File. January 2015. **11.** Data on File, February 2016.

Date of preparation: August 2017. PP-ENB-IRL-0163

Welcome Message from the President Dr Kevin McKenna

Welcome to the Autumn meeting of the IAD 2017 at Farnham Estate, County Cavan. I hope you all enjoyed our last meeting in Belfast and visit to Parliament Buildings, Stormont.

The theme of this meeting is of vasculitis, blistering disorders and Steven Johnson syndrome/TEN. We have a great line up of speakers including Dr Nick Levell, current President of the British Association of Dermatologists, Dr Richard Groves and Dr Daniel Creamer. We look forward to their presentations regarding these complex conditions which can be challenging to manage.

At the Registrars Symposium our junior colleagues will have the opportunity to compete for the prestigious Rogers Prize. I wish them well for all the hard work that is put into these presentations. I would like to thank the Scientific Committee for their invaluable contribution towards making this meeting a success. As always I would like to thank our pharmaceutical colleagues for their ongoing support of our meetings. This is my last meeting as President. I have been proud and honoured to have served the IAD in this capacity. Special thanks to Jacqui Carroll and the members of the Executive Committee for all their generous support.

Yours sincerely,

Dr Kevin McKenna
President
Irish Association of Dermatologists

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Further information is available from: AbbVie Limited, 14 Riverwalk, Citywest Business Campus, Dublin 24.

HCPs are asked to report any suspected adverse reactions via HPRA Pharmacovigilance, Earlsfort Terrace, IRL - Dublin 2; Tel: +353 1 6764971; Fax: +353 1 6762517. Website: www.hpra.ie; E-mail: medsafety@hpra.ie.

Reference: 1. Humira Summary of Product Characteristics, August 2017

Full prescribing information is available at medicines.ie | **Date of Preparation:** August 2017 | IREHUD170512

abbvie

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Dr Anne-Marie Tobin
Consultant Dermatologist
Tallaght Hospital, Dublin

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who was followed by:
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1999-01 Dr Clifford McMillan
2001-3 Prof Frank Powell
2003-5 Dr Raymond Fulton
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BIOLOGY AT THE SERVICE OF DERMATOLOGY

La Roche-Posay Sponsored Symposium

Irish Association of Dermatologists Autumn Meeting

12th October at the Farnham Estate Hotel

CONFIRMED FACULTY

Dr Suzanne Clements

Consultant Dermatologist, Belfast City Hospital

Dr Anne-Marie Tobin

Consultant Dermatologist, Tallaght Hospital

PROGRAMME

Symposium Title: The impact of the Skin Microbiome in managing skin disease

10.45am	Welcome & Opening remarks
11:00am	Advances in the understanding of the skin microbiome in Acne and Rosacea *Dr Suzanne Clements, Consultant Dermatologist, Belfast City Hospital*
11:30am	An update on the impact of the skin biome in Eczema management *Dr Anne-Marie Tobin, Consultant Dermatologist, Tallaght Hospital*
12:00pm	Questions & Answers and Close

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Biographical Sketches

Dr Suzanne Clements MBChB MRCP PhD

Education and experience

Dr Suzanne Clements graduated from the University of Manchester and began her training in dermatology at the Salford Royal Hospital in Manchester. She then trained at St John's Institute of Dermatology in London where she completed a clinical research fellowship in the field of genetic skin disease. She was awarded a PhD in Genetics and Molecular Medicine from the University of London. Dr Clements underwent her specialist training in Belfast and is a Consultant Dermatologist at the Belfast City Hospital.

Achievements

Dr Clements' research into ectodermal dysplasia syndromes led to several peer-reviewed publications and she has presented at national and international dermatology meetings. She was awarded the Burrows Cup for Best Laboratory Research at the Irish Association of Dermatologists meeting in 2012. Dr Clements recently completed a 2-year Postgraduate Diploma in Skin Ageing and Aesthetic Medicine from the University of Manchester in 2016.

Memberships

Member of the Irish Association of Dermatologists, the British Association of Dermatologists and the Royal College of Physicians.

Dr. Anne – Marie Tobin

Special Interests

Dr Tobin's clinical interests are in inflammatory skin disease (psoriasis and hidradenitis suppurativa) and skin cancer. She runs a systemic clinic for patients with psoriasis and eczema and a hidradenitis suppurativa clinic. She also provides skin cancer screening for renal transplant patients and a pigmented lesion clinic. Dr Tobin is involved in Clinical Research and Clinical Trials in psoriasis, hidradenitis suppurativa and eczema

Education

Dr Anne – Marie Tobin is dual-qualified in Medicine and Pharmacy from Trinity College Dublin.
She also completed a PhD in Translational Medicine from University College Dublin.

Experience

Following completion of Specialist Registrar training in Dermatology Dr Tobin undertook a Clinical Fellowship in Psoriasis in St Vincent's University Hospital and a PhD in Translational Medicine in UCD.
She then took up a post of Locum Consultant in the Mater in 2010 and commenced her Consultant Post in Tallaght and Naas Hospital in 2011. She is also a Senior Lecturer in Medicine in Trinity College Dublin
Achievements
Irish Association of Dermatologist Burrows Cup for Best Laboratory Research 2010
Royal Academy of Medicine Ireland Jacob's Medal for Best Clinical Research 2011
Dr Tobin was appointed Clinical Lead for the National Clinical Programme in Dermatology in 2014.
Other appointments:

- Vice Chair Research Ethics Committee St James and Tallaght Hospital 2012
- Member of Scientific Committee of the Irish Association of Dermatologists
- Member of the International Psoriasis Council
- Member of the European Hidradenitis Suppurativa Foundation

Professional Memberships

- Irish Association of Dermatologists

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INDICATIONS

- Oily, blemish-prone skin
- Comedones & blackheads
- Red & brown marks
- Excess sebum
- Inflammatory & non-inflammatory lesions
- Clogged pores
- Uneven skin texture

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IRISH ASSOCIATION OF DERMATOLOGISTS

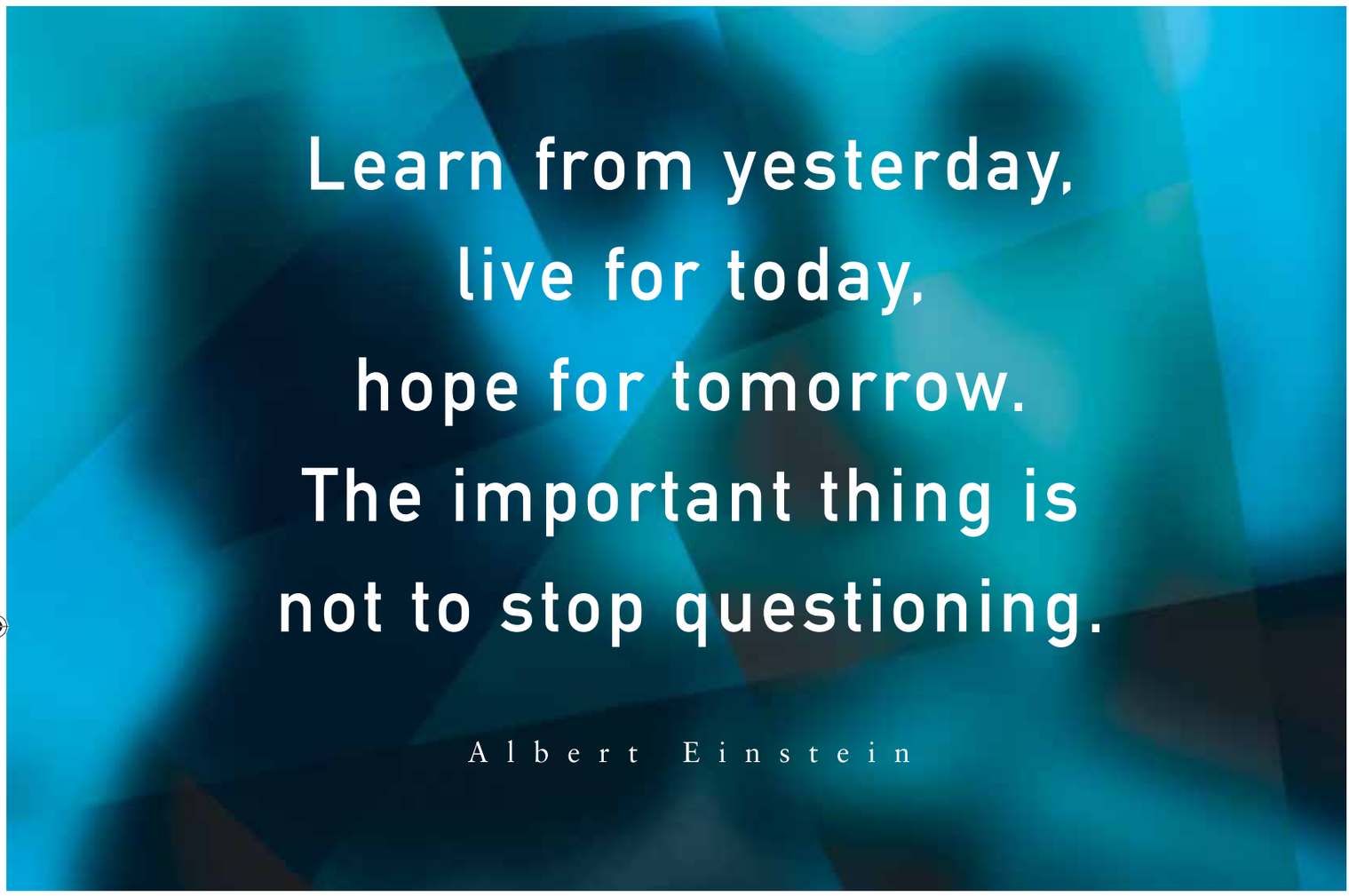
Autumn meeting
Thursday 12th & Friday 13th October 2017

Thursday 12th October 2017 - Farnham Estate, Co Cavan

10.00am	REGISTRATION, TEA / COFFEE
10.45am - 12.30pm	La Roche-Posay Sponsored Satellite Symposium *"The impact of the Skin Microbiome in managing skin disease"*
12.30pm - 2.00pm	LUNCH, EXHIBITION & REGISTRATION
	IAD Autumn Meeting – Theme "Internal Medicine"
2.00pm - 2.45pm	Dr Nick Levell Consultant Dermatologist, Norfolk & Norwich University Hospital *Cutaneous Vasculitis: the pipes are inflamed*
2.45pm - 3.30pm	Dr Richard Groves Consultant Dermatologist, St John's Institute of Dermatology, Guy's Hospital, London *"Tricky blisters: modern management of immunobullous disease"*
3.30pm - 4.00pm	COFFEE & EXHIBITION
4.00pm - 4.45pm	Dr Daniel Creamer Consultant Dermatologist King's College Hospital, London *Managing SJS/TEN – an update*
5.00pm - 6.00pm	IAD Business Meeting
7.30pm	IAD CONFERENCE DINNER

Friday 13th October 2017 Farnham Estate, Co. Cavan

9.30 - 10.50am	Registrars' Symposium - Rogers Prize
10.50am	COFFEE & EXHIBITION
11.30 - 1.00pm	Case presentations
1.00 - 1.15pm	Presentation of prizes
1.15pm - 2.00pm	LUNCH



Unmet needs require new solutions to old problems, which is why we push ourselves to see challenges from different perspectives, constantly questioning and forging new paths toward solutions, both in the lab and in our communities.

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UK-CELG160205
Date of Preparation: November 2016

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IAD Speakers Biographies

Dr Nick Levell

Nick Levell is a Dermatologist at the Norfolk and Norwich University Hospital and BAD President from 2016-18. He does a combined clinic with rheumatology colleagues enjoying medical dermatology, being a Past President of the British Society for Medical Dermatology. He co-authored the Vasculitis Chapter in the latest edition of Rook. His research is around health economics, cancer epidemiology, health service delivery, clinical trials and medical history. He is also national dermatology lead for the UK National Institute of Health Research and national clinical lead for the NHS Improvement GIRFT programme. He enjoys cycling and running very slowly. He also enjoys travel to meet people and enjoy good company, so he thanks you for this very kind invitation.

Dr Richard Groves.

Dr. Groves qualified in medicine from Guy's Hospital, London. Following a general medicine rotation at the Royal London Hospital he completed his dermatology training at Guy's and then spent three years at Harvard University, Boston, undertaking research in immunodermatology. He returned to the UK in 1996 as Senior Lecturer in Dermatology at University College London and in 2001 moved to the Foundation chair in Dermatology at Imperial College. In 2005 he was recruited to the St. John's Institute of Dermatology as Head of the Clinical Immunodermatology Unit.

Dr. Groves' clinical and research interests centre around the interplay between the immune system and the skin. He runs a tertiary referral service for patients with immunobullous disorders in addition to the St. John's diagnostic immunofluorescence laboratory. His research focuses on developing better diagnostic tests for blistering diseases and characterising ways in which cells in the skin initiate inflammatory responses. Additionally, Dr. Groves has a long-standing interest in medical education, particularly in methods of assessment.

Dr Daniel Creamer

Dr Daniel Creamer is a consultant dermatologist at King's College Hospital, London. He trained in general medicine at St Mary's Hospital, London and in dermatology at the St John's Institute of Dermatology. Dr Creamer's major research interest is drug-induced severe cutaneous adverse reactions (SCAR). He is the principal investigator of SCAR-UK, a collaborative project established to study clinical and pathogenetic aspects of the severe drug eruptions, and is the UK Lead for RegiSCAR, an international research consortium

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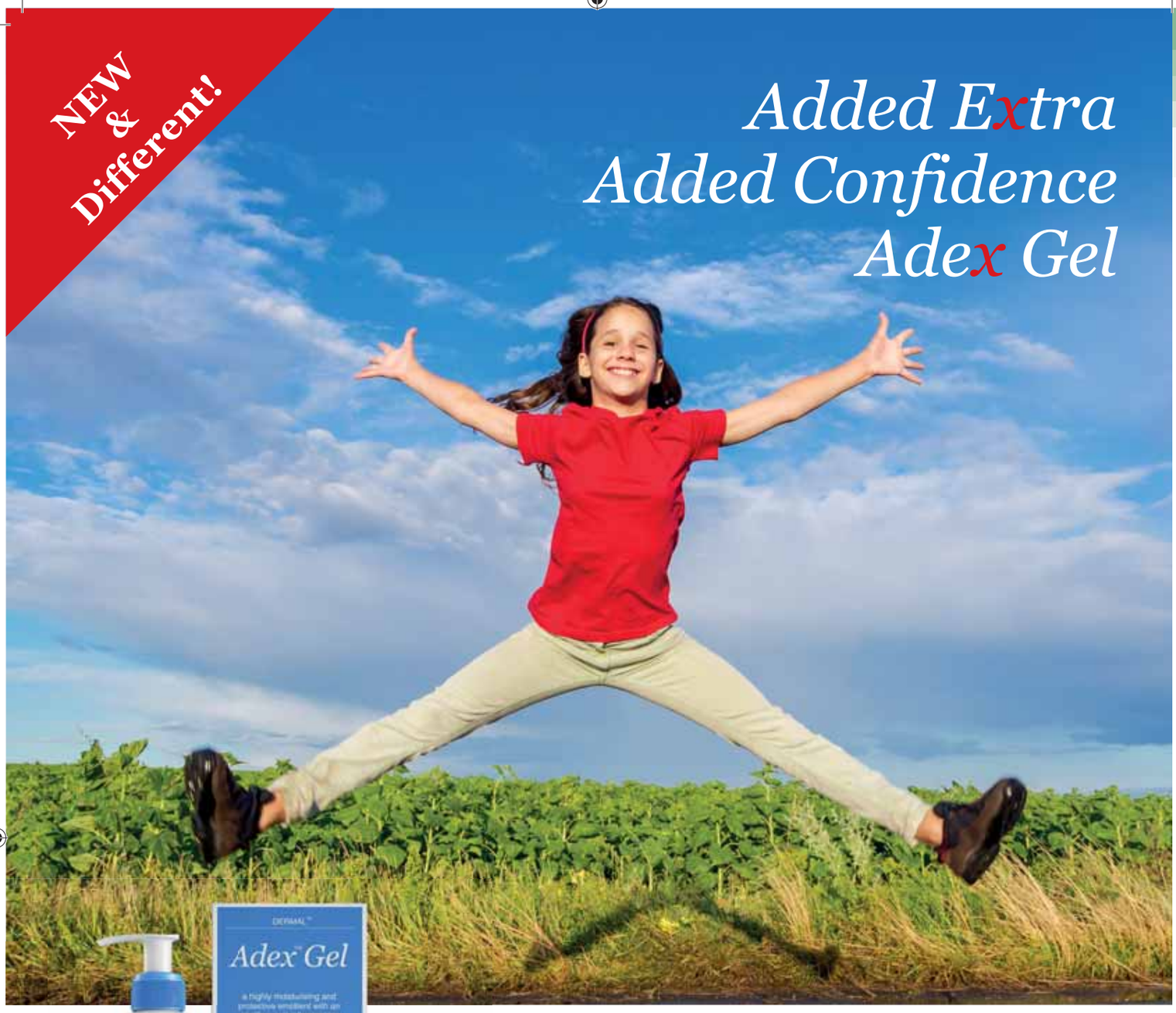

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Directions: Adults, the elderly and children from 1 year of age. For generalised all-over application to the skin. Apply three times daily or as often as needed. Adex Gel can be used for as long as necessary either occasionally, such as during flares, or continuously if the added anti-inflammatory action is beneficial. Seek medical advice if there is no improvement within 2-4 weeks.

Contra-indications, warnings, side effects etc: Do not use if sensitive to any of the ingredients. Keep away from the eyes, inside the nostrils and mouth. Temporary tingling, itching or stinging may occur with emollients when applied to damaged skin. Such symptoms usually subside after a few days of treatment, however, if they are troublesome or persist, stop using and seek medical advice. Rarely skin irritation (mild rashes) or allergic skin reactions can occur on extremely sensitive skin, these tend to occur during or soon after the first few uses and if this occurs stop treatment. As safety trials have not been conducted during pregnancy and breast-feeding, seek medical advice before using this product.

Care should be taken as emollients which soak into clothing, pyjamas, bedlinen etc. can increase the flammability of these items. Patients should avoid these materials coming into contact with naked flames or lit cigarettes etc. As a precaution, dressings and clothing, etc., should be changed frequently and laundered thoroughly.

Ingredients: Carbomer, glycerol, isopropyl myristate, liquid paraffin, nicotinamide, phenoxyethanol, sorbitan laurate, trolamine, purified water.

Pack sizes: 100g tube and 500g pump pack.

Legal category: Class III medical device with an ancillary medicinal substance.

Further information is available from the manufacturer: Dermal Laboratories, Tatmore Place, Gosmore, Hitchin, Herts, SG4 7QR, UK.

Date of preparation: August 2017.

'Adex' is a trademark.

Adverse events should be reported to Dermal.

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DATE FOR DIARY...

IAD Spring Meeting 2018

Thursday 26th, Friday 27th & Saturday 28th April

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Registrars' Symposium
Rogers Prize
Friday 13th October 2017

O1. 9.30am
The use of reflectance confocal microscopy in dermatoscopically atypical lesions helps to exclude and confirm lentigo maligna
J. Boggs, L. Griffin, K. Ahmad, C. Hackett, N. Leonard, B. Ramsay. University Hospital Limerick.

O2. 9.40am
The use of reflectance confocal microscopy in dermatoscopically atypical lesions helps to exclude and confirm lentigo maligna
L. Griffin, B. Ramsay. University Hospital, Limerick.

O3. 9.50am
Lipoprotein subclass in women with Hidradenitis Suppurativa does not differ from those without HS.
S. Kirthi1, P. Reilly3, G. Boran3, M. Connolly1, LA Behan2, J. Gibney2, AM Tobin1
1. Dermatology Department, Tallaght Hospital, Dublin
2. Endocrinology Department, Tallaght Hospital, Dublin
3. Biochemistry Department, Tallaght Hospital, Dublin

O4. 10.00am
Pilot project on electronic photo-triage of referrals for infantile haemangiomas
Background:
A. Flynn, M. Murphy. South Infirmary Victoria University Hospital, Cork.

O5. 10.10am
Factors Influencing Immediate and Delayed Pain Response to Cryotherapy of actinic Keratosis.
J. MacMahon, C. MacEochagain, C. Gillespie, H. Krudyshova, L. Killion, P. Lenane, N. Ralph, F. Moloney. Mater Misercordie University Hospital, Dublin.

O6. 10.20am
The effect of immunosuppression on patch testing
A. Flynn, M. Malik, J. Bourke. South Infirmary Victoria University Hospital, Cork.

O7. 10.30am
The Prevalence, Severity and Management of Pain in Patients with Hidradenitis Suppurativa: Results from an epidemiological study in Ireland
S. McCarthy, E. Delaney, G. Gormley, R. Hughes, S. Kirthi, T. Markham, AM. Tobin, M. Murphy, B. Kirby. South Infirmary Victoria University Hospital Cork, AbbVie Limited, St Vincent's University Hospital, Dublin, Adelaide & Meath Hospital, Dublin, University Hospital Galway.

O8. 10.40am
Cutaneous Signs of Insulin Resistance in Hidradenitis Suppurativa
C. Gallagher, S. Kirthi, K. Molloy, J. Clowry, L. Nestor, AM. Tobin. Tallaght Hospital, Dublin.

O9. 10.50am
Frontal Fibrosing alopecia: possible association with anti-aging skin care products
R. O'Connor, D. Roche, M. Murphy, J. Bourke. South Infirmary Victoria University Hospital, Cork.

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Men should seek advice about the possibility of sperm preservation before starting therapy. **Effects on ability to drive and use machines:** Metoject has minor or moderate influence on the ability to drive and use machines. **Undesirable effects**: The following headings are used to organise the undesirable effects in order of frequency: Very common (≥1/10), common (≥ 1/100 to < 1/10), uncommon (≥ 1/1,000 to < 1/100), rare (≥1/10,000 to < 1/1,000), very rare (<1/10,000), not known (cannot be estimated from the available data) The most relevant undesirable effects are suppression of the haematopoietic system and gastrointestinal disorders. **Very common:** Stomatitis, dyspepsia, nausea, loss of appetite, **common**: oral ulcers, diarrhoea.**Uncommon:** pharyngitis, enteritis, vomiting, **rare:** gastrointestinal ulcers, **very rare:** hematemesis, haematorrhea, toxic megacolon. *Skin and subcutaneous tissue disorders:* **common:** Exanthema, erythema, pruritus, **uncommon:** photosensitisation, loss of hair, increase in rheumatic nodules, herpes zoster, vasculitis, herpetiform eruptions of the skin, urticaria, **Rare:** increased pigmentation, acne, ecchymosis, **Very Rare:** Stevens-Johnson syndrome, toxic epidermal necrolysis (Lyell's syndrome), increased pigmentary changes of the nails, acute paronychia, furunculosis, telangiectasia *General disorders and administration site conditions*. **Rare:** Allergic reactions, anaphylactic shock, allergic vasculitis, fever, conjunctivitis, infection, sepsis, wound-healing impairment, hypogammaglobulinaemia,**Very Rare:** local damage of injection site following intramuscular or subcutaneous administration. *Metabolism and nutrition disorders:* **uncommon:** Precipitation of diabetes mellitus *Nervous system disorders:* Common: headache, tiredness, drowsiness, **uncommon:** dizziness, confusion, depression. **Very rare**: impaired vision, pain, muscular asthenia and paraesthisia in the extremities, changes in sense of taste, convulsions, meningism, paralysis, **unknown:** *Leukoencephalopathy* ***Eye disorders***: Rare: Visual disturbances. **Very rare:** retinopathy. ***Hepatobilary disorders:*** **Very common:** Elevated transaminasis. **Uncommon:** cirrhosis, fibrosis and fatty degeneration of the liver, decrease of serum albumin, **Rare:** acute hepatitis, **Very Rare:** hepatic failure. ***Cardiac disorders:*** **Rare:** Pericarditis, pericardial effusion, pericardial tamponade. ***Vascular disorders:*** **Rare:** Hypotension, thromboembolic events. ***Respiratory, thoracic and mediastinal disorders:*** **Common**: Pneumonia, interstitial alveolitis/ pneumonitis often associated with eosinophilia, Rare: pulmonary fibrosis, pneumocystis carinii pneumonia, shortness of breath and bronchial asthma, pleural effusion.***Blood and lymphatic system*** *disorders:* **Common:** Leukopenia, anaemia, thrombopenia. **Uncommon:** pancytopenia, **Very rare:** agranulocytosis, severe courses of bone marrow depression. ***Renal and urinary disorders:*** **uncommon:** Inflammation and ulceration of the urinary bladder renal impairment, disturbed micturition, **Rare:** renal failure, oliguria, anuria, electrolyte disturbances. ***Reproductive system and breast disorders: Uncommon:*** Inflammation and ulceration of the vagina **Very rare**: Loss of libido, impotence, gynaecomastia, oligospermia, impaired menstruation, vaginal discharge. ***Musculoskeletal and connective tissue disorders:*** **uncommon:** Arthralgia, myalgia, osteoporosis. ***Neoplasms benign malignant and unspecified (incl. cysts and polyps):*** Very rare: Reports of individual cases of lymphoma which subsided in a number of cases once treatment with methotrexate had been discontinued. **Overdose:** Calcium folinate is the specific antidote for neutralising the toxic undesirable effects of methotrexate. **Legal classification:** POM. **Marketing Authorisation Holder:** Medac Gesellschaft fur Klinische Spezialpraparate MbH, Theaterstr,6,22880 Wedel, Germany. **Marketing authorisation number:** PA 623/14/1. **Date of Revision of PI:** February 2017. **MARKETED IN IRELAND BY: FANNIN LTD, FANNIN HOUSE, LEOPARDSTOWN, DUBLIN 18.**

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REF: IE17/05/SmPC - APR2015

Orals - Abstracts

O1. Rogers Prize 9.30am

The use of reflectance confocal microscopy in dermatoscopically atypical lesions helps to exclude and confirm lentigo maligna

J. Boggs, L. Griffin, K. Ahmad, C. Hackett, N. Leonard, B. Ramsay. University Hospital Limerick.

With increasing referrals to all dermatology departments it is important to be able to accurately diagnose malignant from non-malignant lesions in a timely manner, ideally reducing the benign to malignant ratio. Dermoscopy was the big advance in recognition of cutaneous malignancy in the 80's and 90s. In recent years reflectance confocal microscopy (RCM) has become the bridge between dermoscopy and histopathology. Its laser light is safe, non invasive, gives cellular real time imaging and can be performed in the clinical setting of PLC and NMSC clinics. It is increasingly recognised as very helpful in atypical dermoscopic lesions.1 Differentiating benign pigmentary lesions from early LM/LMM can be problematic especially on photodamaged facial skin.

Our paper describes, for the first time in Ireland, the clinical, dermatoscopic, RCM and histologic changes in 8 patients (age range: 32-73 years, mean age: 55.6 years, sex: 6 female) and confirms how RCM can help differentiate between benign and malignant pigmented lesions.

6 of the patients had solar lentigo or pigmented actinic keratosis lesions with challenging dermatoscopic images but had distinctive findings on RCM. We illustrate 2 patients with LM to demonstrate their characteristic RCM features. Lentigo simplex has specific confocal appearances of polymorphous dermal papillae and branching tubular structures at the DEJ. These correlate with the cord like rete ridges seen histologically in LS & flat seborrhoeic keratoses. Pigmented actinic keratosis has atypical honeycomb pattern of the epidermis, inflammatory infiltrate and vascular canilicular changes. The best RCM determinant remains the major and minor CFM criteria to confirm LM.2 A key finding in our 6 patients with non malignant lesions was the absence of pagetoid cellular infiltrate. We were able to confirm LM in one of our 2 patients with LM at a geographically difficult site using the hand held 3000 vivascope.

In conclusion we illustrate the applicability of RCM in the clinical setting for difficult to diagnose pigmented lesions in particular differentiating lentigo maligna from lentigo simplex, pigmented actinic keratosis and lichenoid keratosis.

References:
1 Difficult-to-diagnose facial melanomas: Utility of reflectance confocal microscopy in uncovering the diagnosis ;Pellacani G . JAAD Case Reports 2017;3:379-83
2 Guitera P, Pellacani G, Crotty KA et al. The impact of in vivo reflectance confocal microscopy on the diagnostic accuracy of lentigo maligna and equivocal pigmented and nonpigmented macules of the face. J. Invest. Dermatol. 2010; 130:

O2. Rogers Prize 9.40am

Title: Reflectance confocal microscopy helps exclude melanoma in breast-milk line melanocytic naevi

L. Griffin1, J. Boggs1, K. Ahmad1, N. Leonard2, C. Hackett1, B. Ramsay1. 1 Dermatology department, University Hospital Limerick. 2 Pathology Department, St. James Hospital, Dublin 8.

Introduction: Pigmented lesions of the anatomic milk line often present as atypical melanocytic naevi with concerning features on dermoscopy. This cosmetically sensitive area can offer management dilemmas. In this retrospective study, we identified a cohort of milk line nevi (n=14) and compared our clinical impression from dermoscopy, to Reflectance Confocal Microscopy (RCM) criteria for melanoma and histology to see if diagnostic accuracy could be improved preoperatively.

Methods: 14 pigmented nevi from anterior chest /breast of both sexes were identified where a decision to remove the lesion had been made. Dermoscopy images had been examined by 2 clinicians pre-operatively for features of malignant melanoma (MM). RCM images for 12 lesions were examined using RCM diagnostic criteria for MM by Pellacani et al (1).

Results: The mean age of patients was 34 years, age range: 19-67 years and 10 were female. A clinical impression of MM was made in 5 and dysplastic nevus in 9 following dermoscopy. The dermoscopic findings included asymmetric pigment network, peripheral globules, pigmented streaks, dots and radial streaming. Confocal examination was variable. While most retained the regular meshwork epidermal structure we usually see in naevi, significant architectural atypia were noted. Cellular variation in melanocytic size and shape was noted. Pagetoid cells were noted in 6, yet only 2 lesions fulfilled the RCM criteria for MM pre-excision. One was a superficial spreading MM, Breslow 0.6mm and the other was other deemed to be an atypical special site breast nevus by a specialist dermatopathologist. In the remaining 12, significant regression was noted in 4 and dramatic dermal pigment incontinence in 3, but all were benign.

Conclusion: Histopathology literature has recognised a group of naevi in the milk line that are associated with atypical histological appearance with an atypical growth pattern of the loosely connecting variably sized melanocytic cells, prominent nests along sides of the rete ridges and an active junctional component (2). This, however, was only seen in one of our patients. The remaining histology was reassuring benign in 12 patients and a single malignant melanoma was diagnosed. With the spotlight now firmly on pigmented lesion clinic waiting times, it is helpful to be aware that naevi in the breast-milk line may have atypical clinical appearance but based on our series, MM is less likely in this group. RCM was helpful in determining whether patients had melanoma as the RCM criteria excluded MM in 10 out of 12 lesions (83%).

1. Pellacani G, et al. Reflectance-mode confocal microscopy of pigmented skin lesions--improvement in melanoma diagnostic specificity. J Am Acad Dermatol. 2005, Dec; 53(6):979-85.

2. Hosler, G. Et al. Nevi with site-related atypia: a review of melanocytic nevi with atypical histologic features based on anatomic site. J of Cut Path, 2008, 35: 889–898.

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REFERENCES

1. Taieb A *et al., Br J Dermatol* 2015;172:1103–10.
2. SOOLANTRA Summary of Product Characteristics. March 2015.
3. Stein Gold L *et al., J Drugs Dermatol* 2014;13(11):1380-86.

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O3. Rogers Prize 9.50am

Lipoprotein subclass in women with Hidradenitis Suppurativa does not differ from those without HS.

S. Kirthi1, P. Reilly3, G. Boran3, M. Connolly1, LA Behan2, J. Gibney2, AM Tobin1

1. Dermatology Department, Tallaght Hospital, Dublin

2. Endocrinology Department, Tallaght Hospital, Dublin

3. Biochemistry Department, Tallaght Hospital, Dublin

We have previously documented that patients with HS have raised Framingham Risk1. One of the main risk factors for cardiovascular disease is dyslipidaemia defined as hypertriglyceridemia, reduced high density lipoprotein (HDL) and increased low density lipoprotein (LDL).Increased concentrations of small LDL particles is associated with an increased risk of cardiovascular and cerebrovascular disease. In previous work carried out by the Department of Endocrinology, lipoprotein subclasses were characterised in 140 women and we sought to compare lipoprotein subclasses in our cohort of female patients with HS2 .

Sequential patients attending the dermatology outpatients department with a diagnosis of HS were invited to partake in this study. Physical characteristics such as Hurley Stage, height, weight and Blood Pressure were assessed and demographic data such as age, smoking status and sex recorded. Fasting bloods were drawn for measurement of glucose and insulin and the Hoemostasis Model was used to calculate Insulin Resistance (HOMA-IR). Lipoprotein subclasses were measured using the Quantimetrix Lipoprint System which measures LDL and HDL particle size by comparing particle electrophoretic mobility with the electrophoretic mobility of particles with known sizes. Differences between patients with HS and those without recruited by the Endocrinology Department were analysed using Students T test (Graph Pad Prism), a p value < 0.05 was considered significant.

Sixty-two patients were recruited with a mean age of 39 years (18-58) and Hurley Stage 2. There was no difference in the subfractions of HDL particles (Large HDL 20.6% vs 24.5 % $p > 0.05$, Intermediate HDL 55.8% vs 53.9 % $p > 0.05$ and small HDL 23.4% vs 18% $p > 0.05$) in HS patients and those without HS. There was also no difference in subfractions of LDL between both groups (Large LDL (1+2) 23.8% vs 27.6%, Small dense LDL (3 – 7), 2.2% vs 1.0, mean LDL particle size 266.4 vs 271.5, all p values > 0.05).

Our results show similar lipoprotein subclasses in those patients with and without HS, suggesting that other factors such as smoking and hypertension may be driving Framingham Risk in patients with HS.

O4. Rogers Prize 10.00am

Pilot project on electronic photo-triage of referrals for infantile haemangiomas

A. Flynn, M. Murphy. South Infirmary Victoria University Hospital, Cork.

Background: The discovery of propranolol as an effective treatment for infantile haemangiomas (IH) has been one of the highlights of paediatric dermatology.1 IH mainly develop between 4 weeks and 3 months of age. There is evidence that a period of accelerated growth occurs between 5.5 and 7.5 weeks of life and that those treated earlier have more favourable outcomes.2 Thus, early IH need to be recognized promptly and infants with high-risk IH should be referred to specialists urgently for either initiation of treatment or close clinical observation. In an era of prolonged waiting lists, consultant dermatologists need to have mechanisms in place for urgent evaluation of infants with high-risk IH and a triage system to determine what is high-risk in order to optimise timing of consultation and management.

Methods: The aim of this project was to provide a fast-track approach for general practitioners to send a photograph of the haemangioma with the child's date of birth via Healthmail to a paediatric dermatologist. The photographs were reviewed within five working days and the general practitioner was contacted with an outcome.

Results: The project initiated November 2016. To date there have been 88 referrals. Eight of the referrals did not have a photograph attached. At photo-triage 84% (67/80) were infantile haemangioma, 10% (8/80) port-wine stain, 1% (1/80) vascular malformation and 5% (4/80) could not have a definitive diagnosis made. Age varied from less than 1 week to 57 weeks, with a mean age of 16 weeks. 31% were located on the face, 28% on the trunk, 19% limbs, 11% scalp, 5% ear, 4% genital and 2% on the neck. 46% (31/67) of the IH did not require treatment, 36% (24/67) had a routine review and 18% (12/67) required urgent review.

Conclusion: This novel photo-triage study has shown to be effective at identifying high-risk infantile haemangiomas. This allowed appropriate allocation of resources, with urgent clinical reviews arranged for infants who would benefit from early intervention. Patients with low-risk IH and port-wine stains were triaged appropriately, obliterating the need for urgent clinical review and saving resources.

References:

1. Leaute-Labreze C, Dumas de la Roque E, Hubiche T et al. Propanolol for severe haemangiomas of infancy. N Eng J Med. 2008 Jun 12; 358(24): 2649-51

2. Megha M. Tollefson, Ilona J. Frieden. Early growth of infantile haemangiomas: what parents' photographs tell us. Pediatrics 2012; 130(2) e314-320

O5. Rogers Prize 10.10am

Factors Influencing Immediate and Delayed Pain Response to Cryotherapy of actinic Keratosis.

J. MacMahon, C. MacEochagain, C. Gillespie, H. Krudyshova, L. Killion, P. Lenane, N. Ralph, F. Moloney. Mater Misercordie University Hospital, Dublin.

Study Aim: Assessment of the tolerability of cryotherapy in the treatment of actinic keratosis (AK) and of factors which influence pain during and in the 24 hour period following treatment.

Overview: Cryotherapy represents a first line treatment of AK. This study aimed to identify patient and lesional factors which contribute to poor tolerability and to document and stratify the intensity of side effects.

Methods: 71 patients were prospectively recruited to this observational study by consecutive convenience sampling. Patients with ≤4 AKs, for whom cryotherapy was indicated, were invited to participate.

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Along with baseline demographics, enrolled patients were questioned regarding history of chronic pain, baseline analgesic use, immunosuppressive therapy, previous experience of cryotherapy, pain perception prior to initiation of treatment and smoking status. Patients were asked to complete a Hospital Anxiety and Depression (HADS) questionnaire.

Location and grading of treated AKs was recorded. Cryotherapy was delivered using a standardised protocol.
Visual analogue scores (VAS) for pain were documented prior to, immediately after, and again at 10 minutes following treatment. Further pain scores and post-treatment analgesia requirement / blistering status were assessed by telephone at 2-3 hours and again at 24 hours.

Findings: 71 patients (31 males; 40 females) were recruited. Median age was 78 (IQR 10). Mean number of areas treated was 1.97 (=1.01) Cryotherapy was generally well tolerated, with 66.1% of patients no/mild pain at all timepoints, and 18.3% reporting severe pain at any timepoint assessed. Blistering reaction was self-reported in 25.4% of patients.

Correlation was observed between participants with a history of chronic pain and highest reported pain score during the 24 hour period (Cochrane-Armitage, p=0.039). Furthermore, it was observed that a history of prior analgesia use correlated positively with reported pain scores at the 2-3 hour interval (Ordinal Regression, p=0.042), but not at other timepoints.

No correlation was observed between pain scores and number or size of areas treated, skin type, grade of AK, HADS score, body site, age or gender.

In conclusion, cryotherapy was well tolerated by the cohort with most patients reporting mild pain not requiring of additional analgesia. Analysis identified higher pain scores in those suffering with chronic pain/pain syndromes and those taking prior analgesia.

O6. Rogers Prize 10.20am

The effect of immunosuppression on patch testing

A. Flynn, M. Malik, J. Bourke. South Infirmary Victoria University Hospital, Cork.

Background: Patch testing in patients receiving immunosuppressants is problematic. It is assumed that there is a significant risk of false negative results although there are no randomised studies published which assess this. Several case series indicate that positive patch tests are seen in immunosuppressed patients1 but the sensitivity in this situation has not been assessed. We assessed a random sample of patients with inflammatory bowel disease attending a regional centre to look for steroid allergy.[2] As some of those patients were receiving immunosuppressants, we felt it would be worth comparing the patch test positivity of those patients with the rest of the sample to determine the effect of immunosuppression on patch test reactivity.

Methods: One hundred and eighty four patients with inflammatory bowel disease were patch tested to the british standard, steroid and bakery series. Thirty-eight of these patients were on oral immunosuppression including 6-mercaptopurine, prednisolone, infliximab, azathioprine, adalimumab, methotrexate, mycophenylate mofetil and ciclosporin.

Results: When comparing demographic details between the immunocompetent and immunosuppressed groups, the age and gender profiles were similar (mean age 39 years and 45 years respectively; 53% female in both groups).
15.8% (6/38) of patients on immunosuppression were positive to nickel. The remaining 146 patients (who were not taking oral immunosuppression) had a prevalence of 21.9% (32/146) positivity to nickel (p=0.405). Interestingly, 6.2% (9/146) and 4.1% (6/146) of the immunocompetent cohort had allergic contact dermatitis to fragrance mix and balsalm of peru respectively. No patients in the immunosuppressed cohort had a positive patch test to either of these (p=0.21 and p=0.35 respectively)
Patients with crohns disease who were not immunosuppressed had higher rates of positive patch tests than those immunosuppressed (51% and 32% respectively) but this fell short of significance (p=0.13). There was not as significant a difference found in patients with ulcerative colitis.

Conclusion: Although the number of positive patch tests in immunosuppressed patients was smaller than in immunocompromised patients, the differences were not significant. It is therefore worthwhile patch testing patients on oral immunosuppressants.

1. Wentworth AB, David MD. Patch testing with the standard series when receiving immunosuppressive medications. Dermatitis. 2014 Jul-Aug; 25(4): 195-200

2. M Malik et al. Steroid allergy in patients with inflammatory bowel disease. Contact Dermatitis and Allergy. BJD 2007; 967-969

O7. Rogers Prize 10.30am

The Prevalence, Severity and Management of Pain in Patients with Hidradenitis Suppurativa: Results from an epidemiological study in Ireland

S. McCarthy, E. Delaney, G. Gormley, R. Hughes, S. Kirthi, T. Markham, AM. Tobin, M. Murphy, B. Kirby. South Infirmary Victoria University Hospital Cork, AbbVie Limited, St Vincent's University Hospital, Dublin, Adelaide & Meath Hospital, Dublin, University Hospital Galway.

Introduction & Objectives: Hidradenitis suppurativa (HS) is a chronic inflammatory disease that affects apocrine-gland bearing skin and has a substantial impact on quality of life. To date, few studies have described the pain experienced by patients with HS and the management of pain associated with HS.
This was a cross-sectional, epidemiological study of HS in an Irish population. The primary objective was to determine the number of patients with HS attending selected dermatology clinics in Ireland. Secondary objectives included the assessment of disease characteristics, and the collection of patient reported outcomes to determine disease burden, including pain.

Materials & Methods: Data were collected from four dermatology clinics in a hospital setting in Ireland over a 6-month period in 2015. A total of 15,547 patients attended the selected sites during the recruitment period, of whom 150 formed the full analysis set (FAS). Patients in the FAS were aged ≥18 years and had a diagnosis of HS confirmed by a consultant dermatologist. The level of pain experienced by patients was measured using the visual analogue scale (VAS; where 0=no pain and 10=worst pain possible) and EQ-5D-5L questionnaires. Additional information was collected on the use of analgesics in this population.

continued >>

Stelara® (ustekinumab)

PSORIASIS

YOUR 1ST LINE BIOLOGIC THAT WILL STAY THE COURSE

REAL-WORLD EVIDENCE:
MORE PATIENTS STAY ON STELARA® OVER THE LONG TERM THAN ANY 1ST LINE ANTI-TNF THERAPY [1-5]

janssen Immunology

PHARMACEUTICAL COMPANIES OF Johnson&Johnson

STELARA® 45 mg and 90 mg solution for injection and 130 mg concentrate for solution for infusion PRESCRIBING INFORMATION. ACTIVE INGREDIENT(S): Ustekinumab. Please refer to Summary of Product Characteristics (SmPC) before prescribing. **INDICATION(S): Plaque psoriasis adults:** Treatment of moderate to severe plaque psoriasis in adults who failed to respond to, or who have a contraindication to, or are intolerant to other systemic therapies including ciclosporin, methotrexate or PUVA. **Plaque psoriasis paediatrics:** Moderate to severe plaque psoriasis in adolescent patients from 12 years of age, who are inadequately controlled by, or are intolerant to, other systemic therapies or phototherapies. **Psoriatic arthritis:** Alone or in combination with methotrexate for treatment of active psoriatic arthritis in adult patients when response to previous non-biological disease-modifying anti-rheumatic drug (DMARD) therapy has been inadequate. **Crohn's Disease:** Treatment of adult patients with moderately to severely active Crohn's disease who had inadequate response with/lost response to/were intolerant to either conventional therapy or TNFα antagonist or have contraindications to such therapies. **DOSAGE & ADMINISTRATION:** ***Adults:*** Under guidance and supervision of a physician experienced in diagnosis and treatment of psoriasis/psoriatic arthritis/Crohn's disease. **Psoriasis or psoriatic arthritis**. Subcutaneous (s.c.) injection. Avoid areas with psoriasis. Self-injecting patients or caregivers ensure appropriate training. Physicians are required to follow-up and monitor patients. **Plaque psoriasis, adults & elderly:** Patients ≤ 100kg, 45 mg at week 0 followed by a 45 mg dose at week 4, then every 12 weeks. Patients >100 kg, 90 mg at week 0 followed by a 90 mg dose at week 4, then every 12 weeks (45 mg was less effective in these patients). **Plaque psoriasis paediatrics (12 years and older):** Patients <60 kg, 0.75 mg/kg at week 0, followed by 0.75 mg/kg at week 4 then every 12 weeks thereafter. Patients ≥60 - ≤100kg, 45 mg at week 0 followed by 45 mg at week 4, then every 12 weeks. Patients >100 kg, 90mg at week 0, followed by 90mg at week 4, then every 12 weeks. **Psoriatic arthritis, adults & elderly:** 45 mg at week 0 followed by a 45 mg dose at week 4, then every 12 weeks. Alternatively, 90 mg may be used in patients with a body weight >100 kg. Consider discontinuation if no response after 28 weeks. **Crohn's Disease** Initial single intravenous infusion dose based on body weight (260 mg or 390 mg or 520 mg) diluted in 0.9% w/v sodium chloride solution and given over at least one hour. At week 8 after intravenous dose, 90 mg s.c. dose is given; followed by every 12 weeks (or 8 weeks based on clinical judgement). Consider discontinuation if no response at 16 weeks. Immunomodulators and/or corticosteroids may be continued but consider reducing/discontinuing corticosteroids if responding to Stelara. If therapy interrupted, resume s.c. every 8 weeks if safe/effective. **Children: <12 years** - Not recommended for psoriasis. **<18 years** - Not recommended for psoriatic arthritis and Crohn's disease. **Renal & Hepatic impairment:** Not studied. **CONTRAINDICATIONS:** Hypersensitivity to product; clinically important, active infection. **SPECIAL WARNINGS & PRECAUTIONS:** ***Infections:*** Potential to increase risk of infections and reactivate latent infections. Caution in patients with a chronic infection or history of recurrent infection, particularly TB. Patients should be evaluated for tuberculosis prior to initiation of STELARA. Consider anti-tuberculosis therapy prior to initiation of STELARA in patients with past history of latent or active tuberculosis. Patients should seek medical advice if signs or symptoms suggestive of an infection occur. If a serious infection develops, closely monitor and STELARA should not be administered until infection resolves. ***Malignancies:*** Potential to increase risk of malignancy. No studies in patients with history of malignancy or in patients who develop malignancy while receiving STELARA. Monitor all patients, in particular those older than 60, patients with a medical history of prolonged immunosuppressant therapy or those with a history of PUVA treatment for non-melanoma skin cancer. ***Concomitant immunosuppressive therapy:*** Caution, including when changing immunosuppressive biologic agents. ***Hypersensitivity reactions:*** Serious hypersensitivity reactions (anaphylaxis and angioedema) reported, in some cases several days after treatment. If these occur appropriate therapy should be instituted and STELARA discontinued. **Latex sensitivity:** Needle cover contains natural rubber (latex), may cause allergic reactions. ***Immunotherapy:*** Not known whether STELARA affects allergy immunotherapy. ***Serious skin conditions:*** Exfoliative dermatitis reported following treatment. Discontinue STELARA if drug reaction is suspected. **SIDE EFFECTS:** ***Common:*** upper respiratory tract infection, nasopharyngitis, dizziness, headache, oropharyngeal pain, diarrhoea, nausea, vomiting, pruritus, back pain, myalgia, arthralgia, fatigue, injection site erythema, injection site pain. ***Other side effects:*** cellulitis, serious hypersensitivity reactions (including anaphylaxis, angioedema), skin exfoliation, exfoliative dermatitis. Studies show adverse events reported in ≥12 year olds with plaque psoriasis were similar to those seen in previous studies in adults with plaque psoriasis. **Refer to SmPC for other side effects. FERTILITY:** The effect of ustekinumab has not been evaluated. **PREGNANCY:** Should be avoided. Women of childbearing potential: Use effective contraception during treatment and for at least 15 weeks post-treatment. **LACTATION:** Limited data in humans. **INTERACTIONS:** In vitro, STELARA had no effect on CYP450 activities. ***Vaccinations:*** Live vaccines should not be given concurrently with STELARA, and should be withheld for at least 15 weeks after last dose of STELARA. STELARA can resume at least 2 weeks after such vaccinations. No data on secondary transmission of infection by live vaccines in patients receiving STELARA. ***Concomitant immunosuppressive therapy:*** Psoriasis: Safety and efficacy of STELARA in combination with other immunosuppressants, including biologics, or phototherapy have not been evaluated. Psoriatic arthritis: concomitant MTX did not appear to affect STELARA. Crohn's disease: concomitant immunosupressive or corticosteroid therapy did not appear to affect STELARA. **Refer to SmPC for full details of interactions. LEGAL CATEGORY:** Prescription Only Medicine. **PRESENTATIONS, PACK SIZES, MARKETING AUTHORISATION NUMBER(S):** 45 mg, 1 x vial, EU/1/08/494/001. 45 mg, 1 x 0.5 ml pre-filled syringe, EU/1/08/494/003, 90 mg, 1 x 1.0 ml pre-filled syringe, EU/1/08/494/004. 130 mg, 1 x vial, EU/1/08/494/005. **MARKETING AUTHORISATION HOLDER:** JANSSEN-CILAG INTERNATIONAL NV, Turnhoutseweg 30, B-2340 Beerse, Belgium. **FURTHER INFORMATION IS AVAILABLE FROM:** Janssen-Cilag Limited, 50 – 100 Holmers Farm Way, High Wycombe, Buckinghamshire, HP12 4EG, UK. Prescribing information last revised: 11/2016

Adverse events should be reported. Healthcare professionals are asked to report any suspected adverse events via: HPRA Pharmacovigilance, Earlsfort Terrace, IRL - Dublin 2, Tel: +353 1 6764971, Fax: +353 1 6762517, Website: www.hpra.ie, E-mail: medsafety@hpra.ie. Adverse events should also be reported to Janssen-Cilag Limited on +44 1494 567447 or at dsafety@its.jnj.com.

* Psoriasis Longitudinal Assessment and Registry

† Outcome and Retention Rate of Biologic Treatments for Psoriasis

‡ British Association of Dermatologists Biologic Interventions Register

§ Continuous Assessment of Psoriasis Treatment Use REgistry with biologics

References: 1. Menter A, *et al.* J Eur Acad Dermatol Venerol 2016;30:1148–58. **2.** Vilarrasa E, *et al.* J Am Acad Dermatol 2016;74:1066–72. **3.** Warren RB, *et al.* J Invest Dermatol 2015;135:2623–40. **4.** Gniadecki R, *et al.* Br J Dermatol 2015;172:244–52. **5.** van den Reek J, *et al.* Br J Dermatol 2014;171:1189–96.

PHIR/STE/0417/0009 | Date of Preparation: May 2017

Results: Pain was widely reported in the FAS. Of 134 responders to the VAS questionnaire, 110 (82.1%) reported some level of pain (score ≥1) and 48 (35.8%) reported a score ≥5. In the EQ-5D-5L questionnaire, 118/145 (81.4%) responders indicated that they felt pain or discomfort, and 45/145 (31.0%) expressed "severe" or "extreme" pain/discomfort. The relationship between Hurley stage and EQ-5D-5L response on pain/discomfort was statistically significant ($p<0.001$). Patients who had no pain/discomfort were exclusively Hurley stages I and II, while those reporting extreme pain/discomfort were exclusively stages II and III. 61/150 (40.7%) patients reported taking analgesics. A total of 99 pain medications were reported, of which 61 (61.6%) were over-the-counter medications

Conclusions: Many patients with HS experience considerable pain, which has a substantial impact on their quality of life. In general, patients with more severe disease reported greater pain and discomfort. As such, it is crucial that physicians and researchers be aware of the pain experienced by patients with HS, and that pain be included as a key outcome in future clinical trials.

O8. Rogers Prize 10.40am

Cutaneous Signs of Insulin Resistance in Hidradenitis Suppurativa

C. Gallagher, S. Kirthi, K. Molloy, J. Clowry, L. Nestor, M. Connolly, AM. Tobin. Tallaght Hospital, Dublin.

Aim of the study: Hidradenitis Suppurativa (HS) is a debilitating chronic inflammatory skin condition associated with obesity. Chronic inflammation in other dermatological diseases such as psoriasis is also associated with obesity and an association with PASI and BMI has been shown. Raised BMI incurs metabolic consequences particularly insulin resistance (IR), and an association between PASI and IR has been found. However, there are limited studies looking at the relationship between metabolic alterations such as insulin resistance in HS. Insulin has an important role in homeostasis and physiology of the skin and has protean cutaneous manifestations (acanthosis nigricans, acrochorda, granuloma annulare and tinea incognito), interestingly recent observations suggest HS may be a cutaneous manifestation of IR. Our objective therefore was to determine the prevalence of IR in an Irish HS cohort and the cutaneous manifestations of same. Female patients were assessed for cutaneous signs of Polycystic Ovarian Syndrome (PCOS); acne, hirsutism and androgenetic alopecia another manifestation of IR.

Methods: A pilot prospective cohort study. Following ethical approval and informed consent, patients with a diagnosis of HS attending Tallaght Hospital HS clinic, who were not previously diagnosed with IR, were assessed for cutaneous signs of IR. Anthropometric measurements were taken and bloods drawn for measurement of fasting insulin and glucose, HOMA-IR was calculated using the formula insulin x glucose /22.5. > 2.5 was defined as insulin resistant.

Results: 45 patients have been recruited so far. A shocking 80% (n=36) were insulin resistant. Of these patients, the majority (92%,n=33) were female, only 8% (n=3) were male . Notably none had a healthy BMI; 3%(n=1) were overweight (BMI 25-30), 64%(n=23) obese (BMI 30-40) and 31%(n=11) were severely obese (BMI >40). 11% (n=4) had a family history of HS. Regards cutaneous signs of IR, the majority had signs of PCOS; 58% (n=21) had acne, 44% (n=16) had hirsuitism and 36% (n=13) had androgenic alopecia. In addition, 36% (n=13) had acrochorda, 17% (n=6) had acanthosis nigricans and 3% (n=1) had psoriasis. Interestingly we did not find any patients with granuloma annulare or tinea incognito.

Conclusion: Our results indicate a very high prevalence of IR in patients with HS and confirm recent observations that HS may be a cutaneous manifestation of IR. Other cutaneous signs were less prevalent, nevertheless this study highlights how close observation of cutaneous signs may be an indicator of internal disease.

O9. Rogers Prize 10.50am

Frontal Fibrosing alopecia: possible association with anti-aging skin care products

R. O'Connor, D. Roche, M. Murphy, J. Bourke. South Infirmary Victoria University Hospital, Cork.

Background: Frontal Fibrosing alopecia is an inflammatory scarring alopecia that involves the frontal hairline and the eyebrows. It mainly affects postmenopausal women although it also occurs in younger women and occasionally men. Since first reported in 1994 it has become increasing common in the past 10-15 years suggesting involvement of environmental factors in its aetiology.

Aim: To identify possible causative environmental factors in FFA.

Methods: 12 female and 1 male patient with a clinical diagnosis of frontal fibrosing alopecia were recruited from dermatology clinics in the South Infirmary University Hospital Cork to participate in the study. 9 patients completed a detailed questionnaire enquiring about lifestyle, social, medical and cosmetic factors. Detailed information on the use of facial skin care and hair care products and styling methods was taken. 13 patients underwent patch testing to an extended British standard series of allergens including standard, facial, fragrance, cosmetics, sunscreen and anti-aging products.

Results: The mean age of onset of hair loss in the patients with FFA was 55 (range 41-70). 7 (87.5%) patients had hair loss from the frontal and frontotemporal hairline, 6 (66%) patients from the eyebrows and 4 (44%) from the limbs. 9 (100%) patients reported use of sunscreens and 8 (88%) patients reported use of facial moisturisers. 4 (44%) patients reported use of anti-aging skin care products. Of the 13 patients tested, 11 (84.6%) patients had at least one positive patch test and 6 (46.1%) had more than one positive reaction. The majority of positive reactions were to fragrances (limonene, n=4; lanolin, n=1; lyral, n=1; hydroxycitronellal, n=1). 3 patients (23%) had a positive result to sodium metabisulphite and one had a positive reaction to parabens, which was present in their hair products. 4 patients who tested positive to fragrance also reported using facial moisturiser, sunscreen and foundation.

Conclusion: Our results support a previous study by Aldorri et al1 suggesting an association between FFA and the use of leave-on facial skincare products and sunscreens.

References

1. Aldoori N, Dobson K, Holden CR et al. Frontal fibrosing alopecia: possible association with leave-on facial skin care products and sunscreens; a questionnaire study. Br J Dermatol 2016; 175:762–7.

Abbreviated Prescribing Information for Enstilar® 50 micrograms/g + 0.5 mg/g cutaneous foam Please refer to the full Summary of Product Characteristics (SmPC) (www.medicines.org.uk/emc) before prescribing.
Indication: Topical treatment of psoriasis vulgaris in adults.
Active ingredients: 50 µg/g calcipotriol (as monohydrate) and 0.5 mg/g betamethasone (as dipropionate).
Dosage and administration: Apply by spraying onto affected area once daily. Recommended treatment period is 4 weeks. The daily maximum dose of Enstilar should not exceed 15 g, i.e. one 60 g can should last for at least 4 days. 15 g corresponds to the amount administered from the can if the actuator is fully depressed for approximately one minute. A two-second application delivers approximately 0.5 g. As a guide, 0.5 g of foam should cover an area of skin roughly corresponding to the surface area of an adult hand. If using other calcipotriol-containing medical products in addition to Enstilar, the total dose of all calcipotriol-containing products should not exceed 15 g per day. Total body surface area treated should not exceed 30%. Safety and efficacy in patients with severe renal insufficiency or severe hepatic disorders have not been evaluated. Safety and efficacy in children below 18 years have not been established. Shake the can for a few seconds before use. Apply by spraying, holding the can at least 3 cm from the skin, in any orientation except horizontally. Spray directly onto each affected skin area and rub in gently. Wash hands after use (unless Enstilar is used to treat the hands) to avoid accidentally spreading to other parts of the body. Avoid application under occlusive dressings since systemic absorption of corticosteroids increases. It is recommended not to take a shower or bath immediately after application.
Contraindications: Hypersensitivity to the active substances or any of the excipients. Erythrodermic and pustular psoriasis. Patients with known disorders of calcium metabolism. Viral (e.g. herpes or varicella) skin lesions, fungal or bacterial skin infections, parasitic infections, skin manifestations in relation to tuberculosis, perioral dermatitis, atrophic skin, striae atrophicae, fragility of skin veins, ichthyosis, acne vulgaris, acne rosacea, rosacea, ulcers and wounds.
Precautions and warnings: Adverse reactions found in connection with systemic corticosteroid treatment, e.g. adrenocortical suppression or impaired glycaemic control of diabetes mellitus, may occur also during topical corticosteroid treatment due to systemic absorption. Application under occlusive dressings should be avoided since it increases the systemic absorption of corticosteroids. Application on large areas of damaged skin, or on mucous membranes or in skin folds should be avoided since it increases the systemic absorption of corticosteroids. Due to the content of calcipotriol, hypercalcaemia may occur. Serum calcium is normalised when treatment is discontinued. The risk of hypercalcaemia is minimal when the maximum daily dose of Enstilar (15 g) is not exceeded. Enstilar contains a potent group III-steroid and concurrent treatment with other steroids on the same treatment area must be avoided. Skin on the face and genitals are very sensitive to corticosteroids. Enstilar should not be used in these areas. Instruct the patient in the correct use of the product to avoid application and accidental transfer to the face, mouth and eyes. Wash hands after each application to avoid accidental transfer to these areas. When lesions become secondarily infected, they should be treated with antimicrobiological therapy. However, if infection worsens, treatment with corticosteroids should be discontinued. When treating psoriasis with topical corticosteroids, there may be a risk of rebound effects when discontinuing treatment. Medical supervision should therefore continue in the post-treatment period. Long-term use of corticosteroids may increase the risk of local and systemic adverse reactions. Treatment should be discontinued in case of adverse reactions related to long-term use of corticosteroid. There is no experience with the use of Enstilar in guttate psoriasis. During Enstilar treatment, physicians are recommended to advise patients to limit or avoid excessive exposure to either natural or artificial sunlight. Topical calcipotriol should be used with UVR only if the physician and patient consider that the potential benefits outweigh the potential risks. Enstilar contains butylhydroxytoluene (E321), which may cause local skin reactions (e.g. contact dermatitis), or irritation to the eyes and mucous membranes.
Pregnancy and lactation: There are no adequate data from the use of Enstilar in pregnant women. Enstilar should only be used during pregnancy when the potential benefit justifies the potential risk. Caution should be exercised when prescribing Enstilar to women who breast-feed. The patient should be instructed not to use Enstilar on the breast when breast-feeding.
Side effects: There are no common adverse reactions based on the clinical studies. The most frequently reported adverse reactions are application site reactions. *Calcipotriol*: Adverse reactions include application site reactions, pruritus, skin irritation, burning and stinging sensation, dry skin, erythema, rash, dermatitis, psoriasis aggravated, photosensitivity and hypersensitivity reactions, including very rare cases of angioedema and facial oedema. Systemic effects after topical use may appear very rarely causing hypercalcaemia or hypercalciuria. *Betamethasone (as dipropionate)*: Local reactions can occur after topical use, especially during prolonged application, including skin atrophy, telangiectasia, striae, folliculitis, hypertrichosis, perioral dermatitis, allergic contact dermatitis, depigmentation and colloid milia. When treating psoriasis with topical corticosteroids, there may be a risk of generalised pustular psoriasis. Systemic reactions due to topical use of corticosteroids are rare in adults; however, they can be severe. Adrenocortical suppression, cataract, infections, impaired glycaemic control of diabetes mellitus, and increase of intra-ocular pressure can occur, especially after long-term treatment. Systemic reactions occur more frequently when applied under occlusion (plastic, skin folds), when applied on large areas, and during long-term treatment.
See SmPC for a full list of side effects.
Precautions for storage: Do not store above 30°C. Extremely flammable aerosol. Pressurised container. May burst if heated. Protect from sunlight. Do not expose to temperatures exceeding 50°C. Do not pierce or burn, even after use. Do not spray on an open flame or other ignition source. Keep away from sparks/open flames. No smoking.
Legal category: POM.
Marketing authorisation number and holder: PL 05293/0008. LEO Pharma A/S, Ballerup, Denmark.
Basic NHS price: £39.68/60 g
Last revised: May 2016
Further information can be found in the Summary of Product Characteristics or from: LEO Pharma, Horizon, Honey Lane, Hurley, Berkshire SL6 6RJ. e-mail: medical-info.uk@leo-pharma.com
® Registered trademark
UK 1070/00024g
Date of preparation: May 2016

Reporting of Suspected Adverse Reactions
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 Drug Safety at LEO Pharma by calling +44 (0)1844 347333 or e-mail medical-info.uk@leo-pharma.com

LEO®

Case Presentations
Friday 13th October 2017

01. 11.30am
Head, Shoulders, Knees and Toes
O. Molloy, A. Flynn, I. Timoney, E. Waldon, M. Azhar, N. Moriarty. University Hospital Galway.

02. 11.39am
Xanthelasmoid variant of urticaria pigmentosa - a rare presentation of cutaneous mastocytosis
W. Abdelrahman, D. Watkins, C. Grattan, C. Devereux. Antrim Area Hospital & St John's Institute, London.

03. 11.48am
Pregnancy related pyoderma faciale, a challenging dermatoses with limited treatment options
I. McDonald, N. Mansoor, M. O'Connell. University Hospital Waterford.

04. 11.57am
Successful surgical management of two patients with severe toxic epidermal necrolysis
I. Timoney, L. Nestor, M. Sadlier, D. Wall, B. Wynne, O. Shelly, L. Barnes. St James's Hospital.

05. 12.06pm
An unusual radiotherapy related rash
W. Abdelrahman, M. Walsh, O. Dolan. Royal Victoria Hospital, Belfast.

06. 12.15pm
Recognising a familiar dermatoses in an unfamiliar site , erosive pustular dermatoses of the leg, a diagnostic challenge.
I. McDonald, N. Mansoor, L. Paul. University Hospital Waterford.

07. 12.24pm
An Aggressive Dermatophytosis In A Cardiac Transplant Patient
J. MacMahon, L. Cunningham, A. O'Connell, D. Brady, N. Ralph. Mater Misericordiae Hospital, Dublin.

08. 12.33pm
A rare case of giant myxoid Dermatofibrosarcoma Protuberans with focal fibrosarcomatous change.
L. Timoney, I. Timoney, N. Walsh. Sligo University Hospital, St James's Hospital, Blackrock Clinic, Dublin.

9. 12.42pm
Atypical lower limb ulceration in a patient with rheumatoid arthritis on long term methotrexate – a rare cause
C. Harnett, N. Kearney, C. Feighery. Our Lady of Lourdes Hospital, Drogheda.

10. 12.51pm
A case of primary cutaneous mammary analog secretory carcinoma: a rare entity
S. McCarthy, M McMenamin, C. Heffron, M. McDermott, C. Hackett, M. Lynch. University Hospital Limercik, St James's Hospital, Dublin, Cork University Hospital.

5 reasons HUMIRA®

HUMIRA® product label

NAILS

Significant mNAPSI improvement in nail psoriasis at week 26

47% vs 3%, HUMIRA® and placebo respectively (p<0.001)[1]

SKIN

Sustained PASI 90 long-term efficacy results supported by real-world data

59% of biologic-naïve patients achieved PASI 90 at 6 months[2]

74% of those PASI 90 responders, sustained PASI 90 at 12 months[2]

JOINTS

Rapid and sustained efficacy in joint symptoms

58% of HUMIRA® patients achieved ACR20 by week 12[3]

57% of HUMIRA® patients achieved ACR20 at 2 years[4]

*** Please see the Summary of Product Characteristics for more information**

ACR: American College of Rheumatology | **PASI:** Psoriasis Area Severity Index | **TNF:** Tumor Necrosis Factor | **mNAPSI:** Modified Nail Psoriasis Severity Index

Prescribing Information: Humira (adalimumab) 40mg solution for injection in pre-filled pen or pre-filled syringe or Humira 40mg/0.8ml solution for injection for paediatric use. Refer to Summary of Product Characteristics (SmPC) for full information. Presentation: Each 0.4 ml single dose pre-filled pen or pre-filled syringe contains 40mg of adalimumab. Each 0.8 ml single dose vial contains 40mg of adalimumab. **Indications:** Rheumatoid arthritis (RA), adults: In combination with methotrexate (MTX) for moderate to severe, active RA with inadequate response to disease-modifying anti-rheumatic drugs (DMARDs) including MTX. In combination with MTX for severe, active and progressive RA when not previously treated with MTX. Can be given as monotherapy if intolerance to or when continued treatment with MTX is inappropriate. Reduces rate of progression of joint damage on X-ray and improves physical function, in combination with MTX. Polyarticular juvenile idiopathic arthritis (pJIA), paediatrics 2 years and above: In combination with MTX, for active pJIA, with inadequate response to one or more DMARDs; or monotherapy if intolerance to or when continued treatment with MTX is inappropriate. Enthesitis-related arthritis (ERA), paediatrics 6 years and above: For active ERA with inadequate response or intolerance to, conventional therapy. Psoriatic arthritis (PsA), adults: For active and progressive PsA with inadequate response to DMARDs. Reduces rate of progression of peripheral joint damage on X-ray in polyarticular symmetrical subtypes of the disease and improves physical function. Ankylosing spondylitis (AS), adults: For severe active AS with inadequate response to conventional therapy. Axial spondyloarthritis without radiographic evidence of AS (nr-axSpA), adults: For severe nr-axSpA with objective signs of inflammation (elevated CRP and / or MRI), and an inadequate response to, or intolerance to nonsteroidal anti-inflammatory drugs (NSAIDs). Crohn's disease (CD), adults: For moderately to severely, active CD with inadequate response, contraindication or intolerance to corticosteroid and/or an immunosuppressant therapy. Crohn's disease (CD), Paediatrics 6 years and above: For moderately to severely active CD with inadequate response, contraindication or intolerance to conventional therapy including primary nutrition therapy and a corticosteroid, and/or an immunomodulator. Psoriasis (Ps), adults: For moderate to severe chronic plaque psoriasis who are candidates for systemic therapy. Psoriasis, paediatrics 4 years and above: For severe chronic plaque psoriasis with inadequate response, or if topical therapy and phototherapies are inappropriate. Hidradenitis suppurativa (HS), adults and adolescents from 12 years of age: For active moderate to severe hidradenitis suppurativa (acne inversa) in patients with an inadequate response to conventional systemic HS therapy. Ulcerative colitis (UC), adults: For moderately to severely active UC with inadequate response, contraindication or intolerance to conventional therapy including corticosteroids and 6-mercaptopurine (6-MP) or azathioprine (AZA). Uveitis, adults: For non-infectious intermediate, posterior and panuveitis with inadequate response to corticosteroids, in need of corticosteroid-sparing, or in whom corticosteroid treatment is inappropriate. **Dosage and administration:** Specialist physicians experienced in the diagnosis and treatment of the condition, to initiate and supervise treatment. Ophthalmologists to consult with an appropriate specialist before initiation of treatment. Provide patients with special alert card. Patients may self-inject after proper injection training, with physician approval and appropriate medical follow-up. Optimise other concomitant therapies. RA, adults: 40mg dose every other week. Concomitant MTX should be continued. During monotherapy patients may require 40 mg each week if they experience a decrease in clinical response. Treatment beyond 12 weeks should be reconsidered if no clinical response in that time. Consider need for dose interruption, e.g. before surgery or if serious infection occurs. Re-introduction after 70 days dose interruption gave same magnitudes of clinical response and similar safety profile as before dose interruption. pJIA, paediatrics 2 years and above: Treatment beyond 12 weeks reconsidered if no clinical response in that time. pJIA, paediatrics 2-<4 years: 24mg/m² body surface area up to 20mg maximum single dose every other week (see vial SmPC for height/weight dosing chart). pJIA, paediatrics 4-12 years: 24mg/m² body surface area up to 40 mg maximum single dose every other week (see vial SmPC for height/weight dosing chart). pJIA, paediatrics 13 years and above: 40mg every other week regardless of body surface area. ERA, paediatrics 6 years and above: 24mg/m² body surface area up to a maximum single dose of 40mg every other week (see vial SmPC for height/weight dosing chart). PsA, AS and nr-axSpA, adults: 40 mg every other week. Treatment beyond 12 weeks should be reconsidered if no clinical response in that time. CD: Adults: Induction: 80mg at Week 0 followed by 40mg at Week 2. For a more rapid response, 160mg at Week 0 (either as 4 injections in 1 day or 2 injections/ day for 2 consecutive days), 80mg at Week 2; risk of adverse events higher during induction. Maintenance: 40mg every other week. If decrease in clinical response, can increase dose to 40 mg weekly. Corticosteroids may be tapered in maintenance phase in accordance with clinical guidelines. Patients with no response by Week 4 may benefit from continued therapy to Week 12. Treatment beyond 12 weeks should be reconsidered if no clinical response in that time. CD, paediatrics 6 years and above<40Kg: Induction: 40mg at Week 0, 20mg at Week 2. For a more rapid response: 80mg at Week 0 (2 injections in 1 day), 40mg at Week 2; risk of adverse events higher during induction. Maintenance: 20mg every other week. If insufficient response, consider 20mg every week. Treatment beyond 12 weeks should be reconsidered if no clinical response in that time. CD, paediatrics 6 years and above ≥40Kg: Induction: 80 mg Week 0, 40 mg at Week 2. For a more rapid response: 160 mg at Week 0 (4 injections in 1 day or 2 injections/day for 2 consecutive days), 80 mg at Week 2; risk of adverse events higher during induction. Maintenance: 40 mg every other week. If insufficient response, consider 40 mg every week. Treatment beyond 12 weeks should be reconsidered if no clinical response in that time. Psoriasis, adults: 80mg induction dose at week 0, 40mg every other week from week 1. Treatment beyond 16 weeks should be reconsidered if no clinical response in that time. Beyond 16 weeks, patients with inadequate response can increase dosing frequency to 40 mg every week. If adequate response is achieved with an increased dosing frequency, dose may subsequently be reduced to 40 mg every other week. If there is inadequate response to the increased frequency, carefully reconsider treatment. Psoriasis, Paediatrics 4 years and above: 0.8 mg per kg body weight (maximum of 40 mg/dose) weekly for the first 2 doses and then every other week (see vial SmPC for weight dosing chart). Treatment beyond 16 weeks should be reconsidered if no response in that time. HS: Adults: 160mg initially at Day 1 (four 40mg injections in one day or two 40mg injections per day for two consecutive days), followed by 80 mg two weeks later at Day 15 (two 40mg injections in one day). Two weeks later (Day 29) continue with a dose of 40 mg every week. Antibiotics may be continued if necessary. Concomitant topical antiseptic wash on HS lesions should be used on a daily basis. Treatment beyond 12 weeks should be reconsidered if no clinical response in that time. Reintroduction after interruption: 40 mg every week. Evaluate periodically the benefit and risk of continued long-term treatment. HS, adolescents from 12 years of age weighing at least 30 kg: 80 mg initially at week 0 (given as two 40 mg injections on day one). 40 mg injection in week 1 followed by 40mg every other week. In adolescent patients with inadequate response to Humira 40 mg every other week an increase in dosing frequency to 40 mg every week may be considered. Antibiotics may be continued if necessary. Concomitant topical antiseptic wash on HS lesions should be used on a daily basis. Treatment beyond 12 weeks should be reconsidered if no clinical response in that time. UC: Adults: Induction: 160mg at week 0 (4 injections in 1 day or 2 injections / day for 2 consecutive days) and 80mg at week 2. Maintenance: 40mg every other week. During maintenance, corticosteroids may be tapered in accordance with clinical practice guidelines. If insufficient response,

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- Paediatric Plaque Psoriasis (from age 4)
- Psoriatic Arthritis (PsA) in adults
- Hidradenitis Suppurativa in adults and adolescents (from age 12)

consider 40mg every week. Treatment beyond 8 weeks should be reconsidered if no clinical response in that time. Uveitis: Adults: 80 mg induction dose at week 0, 40 mg every other week from week 1. Experience of initiating treatment with Humira alone is limited. Treatment can be initiated in combination with corticosteroids and/or other non-biologic immunomodulatory agents. Two weeks after initiating treatment, concomitant corticosteroids may be tapered in accordance with clinical guidelines. Evaluate on a yearly basis, the benefit and risk of continued long term treatment. **Contraindications:** Active tuberculosis (TB), severe infections (e.g. sepsis), and opportunistic infections; moderate to severe heart failure (NYHA class III/IV); hypersensitivity to adalimumab or any of the excipients. **Precautions and Warnings:** Clearly record trade name and batch number of administered product to improve traceability of biological medicinal product. ***Infections:*** Patients are more susceptible to serious infections especially if impaired lung function. Monitor for infections, including TB, before, during and for 4 months after treatment. Do not initiate treatment with an active infection, until it is controlled. Consider risk/benefit prior to treatment in patients exposed to high risk of TB or endemic mycoses. Evaluate new infections during treatment and monitor closely. Stop treatment if new serious infection or sepsis, and treat appropriately. Exercise caution in patients with a history of recurring infections or who are predisposed to infections. ***Serious infections:*** Serious infections, including those with hospitalisation or death reported in patients receiving treatment. ***TB:*** Consult SmPC for details. Reactivation and new onset TB, both pulmonary and extra-pulmonary (disseminated) reported. Screen all patients before therapy initiation for active or latent TB. If active TB is diagnosed Humira therapy must not be initiated. If latent TB is suspected, consult a physician with appropriate expertise and follow local treatment recommendations for prophylaxis prior to initiation of Humira. Despite prophylaxis TB reactivation has occurred on Humira. ***Other opportunistic infections:*** Opportunistic infections observed in patients receiving Humira. Stop treatment in patients with signs and symptoms of such infections. Consult with physician with appropriate expertise for diagnosis and administration of empiric antifungal therapy in these patients. ***Hepatitis B Reactivation:*** Reactivation has occurred in chronic carriers i.e. surface antigen positive) tested for HBV infection before initiating treatment. HBV carriers should consult with a specialist physician and be closely monitored for reactivation of HBV infection throughout therapy and for several months following termination of Humira. If reactivation occurs stop treatment and initiate appropriate anti-viral and supportive treatment. ***Neurological events:*** Caution in patients with pre-existing or recent-onset central or peripheral nervous system demyelinating disorders and consider stopping treatment if these disorders develop. Rare association with new onset or exacerbation of symptoms and/or radiographic evidence of central and peripheral demyelinating disease. Known association between intermediate uveitis and central demyelinating disorders. Evaluate patients with noninfectious intermediate uveitis before therapy initiation and regularly during treatment to assess for pre-existing or developing central demyelinating disorders. ***Allergic reactions:*** Reports of serious allergic reactions including anaphylaxis received. For serious allergic or anaphylactic reaction stop Humira immediately and initiate appropriate therapy. ***Malignancies and lymphoproliferative disorders:*** A possible risk of malignancy, including lymphoma and leukaemia, in all patients including paediatric patients, treated with TNF antagonists. Monitor all patients, especially those with a medical history of extensive immunosuppressant or PUVA treatment for non-melanoma skin cancer prior to and during Humira therapy, caution in COPD patients, and in patients with increased risk of malignancy due to heavy smoking. Consider the potential risk with the combination of AZA or 6-MP and Humira (hepatosplenic T-cell lymphoma has occurred). Risk of hepatosplenic T-cell lymphoma cannot be excluded. Caution in patients with a history of malignancy. Risk for developing dysplasia or colon cancer is unknown. Patients with UC, prior history of dysplasia or colon carcinoma to be screened for dysplasia before therapy and during treatment. ***Haematologic reactions:*** Adverse events of the haematologic system reported with Humira. Patients should seek immediate medical attention if signs and symptoms of blood dyscrasias. ***Vaccinations:*** Patients may receive concurrent vaccinations, except for live vaccines. Bring paediatric patients up to date with all immunisations prior to Humira treatment. ***Congestive heart failure:*** See contraindications. Caution is advised in mild heart failure (NYHA class I/II). Discontinue treatment for new or worsening symptoms of congestive heart failure. ***Autoimmune processes:*** Autoimmune antibodies may form. Stop treatment if development of a lupus-like syndrome with positive antibodies against doublestranded DNA. Surgery: Consider the long half-life of Humira for planned surgical procedures. Closely monitor for infections. ***Small bowel obstruction:*** Failure to respond to treatment for CD may indicate the presence of fixed fibrotic stricture requiring surgical treatment. ***Elderly:*** Serious infections were higher in patients over 65 years of age, some of whom had fatal outcomes. Consider risk of infection. **Interactions:** Combination of adalimumab with other biologic DMARDS (e.g. anakinra and abatacept) or other TNF-antagonists is not recommended. **Fertility, pregnancy and lactation:** Not recommended during pregnancy. Women of childbearing potential should use adequate contraception and continue its use for at least five months after the last Humira treatment. Women must not breast-feed for at least five months after the last treatment. **Side Effects:** ***Very common ≥ 1/10:*** Infections, leukopenia, anaemia, lipids increased, headache, abdominal pain, nausea and vomiting, elevated liver enzymes, rash (including exfoliative rash), musculoskeletal pain, injection site reaction. **Serious, including fatal, side effects have been reported** including infections/sepsis, intestinal perforation, opportunistic infections, TB, endemic mycoses, demyelinating disease, malignancies including lymphoma (including hepatosplenic T-cell lymphoma), leukaemia and skin cancer (including melanoma and merkel cell carcinoma), cytopenias, worsening heart failure, myocardial infarction, pulmonary embolism, pleural effusion, pulmonary fibrosis, cerebrovascular accident, interstitial lung disease, lupus, Stevens-Johnson syndrome, angioedema, anaphylaxis, sarcoidosis, hepatitis, liver failure and worsening of symptoms of dermatomyositis. Prescribers should consult the SmPC for the complete list of reported side effects. **Legal Category:** POM. **Marketing Authorisation Numbers/Presentations:** Vial: EU/1/03/256/001; Pre-filled Syringe: EU/1/03/256/013; Pre-filled Pen: EU/1/03/256/017. Further information is available from AbbVie Limited, 14 Riverwalk, Citywest Business Campus, Dublin 24.

HCPs are asked to report any suspected adverse reactions via HPRA Pharmacovigilance, Earlsfort Terrace, IRL - Dublin 2; Tel: +353 1 6764971; Fax: +353 1 6762517. Website: www.hpra.ie; E-mail: medsafety@hpra.ie.

Date of revision of PI: January 2017, PI/256/018

References: 1. Humira Summary of Product Characteristics, available at www.medicines.ie. **2.** Data on file, AbbVie Ltd. **3.** Mease PJ, et al. Adalimumab Effectiveness in Psoriatic Arthritis Trial Study Group. Adalimumab for the treatment of patients with moderately to severely active psoriatic arthritis: results of a double-blinded, randomized, placebo controlled trial. Arthritis Rheum. 2005;52(10):3279-3289. **4.** Mease PJ, et al. Adalimumab for long-term treatment of psoriatic arthritis: 2 year data from the Adalimumab Effectiveness in Psoriatic Arthritis Trial (ADEPT). Ann Rheum Dis. 2009;68(5):702-709. **5.** Burmester GR, et al. Adalimumab: long-term safety in 23 458 patients from global clinical trials in rheumatoid arthritis, juvenile idiopathic arthritis, ankylosing spondylitis, psoriatic arthritis, psoriasis and crohn's disease. Ann Rheum Dis. 2013;72(4):517-524. **6.** Menter A, et al. Five-year analysis from the ESPRIT 10-year postmarketing surveillance registry of adalimumab treatment for moderate to severe psoriasis. J Am Acad Dermatol. 2015;73(3):410-419. **7.** Data on file, AbbVie Ltd.

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References: 1. Bissonnette R et al. *Br J Dermatol* June 2017. **2.** Thaçi D et al. *J Am Acad Dermatol* 2015; 73(3): 400-409.
3. Bissonnette R et al. Seminars in Cutaneous Medicine and Surgery 2016: 35(Supplement 7); PA-30.

Date of Preparation: August 2017
IE02/COS17-CNF041a

NOVARTIS

Case Presentations

Case presentation 01. 11.30am

Head, Shoulders, Knees and Toes

O. Molloy, A. Flynn, I. Timoney, E. Waldon, M. Azhar, N. Moriarty. University Hospital Galway.

Introduction: Classic hand foot and mouth disease is caused by the Coxsackie A16 or EV71 virus. It predominantly affects children under 5 years with only symptomatic and supportive treatment required (1, 2). An emerging trend which started in South East Asia (3) and recently the United States (4) implicates the Coxsackie A6 viral strain in an atypical form of hand foot and mouth that affects both children and adults.
We observed a trend in vesiculo-bullous eruptions presenting to the paediatric dermatology service. We questioned whether these cases were eczema herpeticum or could they represent an alternative entity. We postulated that patients with a vesiculo-bullous eruption in the absence of significant symptoms could represent Eczema coxsackium.

Methods: Patients presenting with vesiculo-bullous eruptions from July 2016 – June 2017 were assessed for infection by HSV 1 and 2, enterovirus and bacterial culture.
Skin swabs were taken in all cases, throat swabs were taken in some. Patients were treated with acyclovir and topical emollients. Retrospective analysis of these cases was carried out. Basic demographics and patients characteristics were obtained.

Results: 8 patients (2 female, 6 male) with atypical vesiculobullous eruption were identified. All presented with crops of vesicles involving the face and acral sites and systemic symptoms without significant morbidity. One patient developed Onychomadesis and another developed oral lesions, causing odynophagia.
71.4% of patients tested positive for Coxsackie A6 on viral skin swabs, 62.5% of the total cohort. None of the blister cultures isolated HSV I or II. In addition Staphylococcus aureus was cultured from skin swabs in two cases.
Although our numbers are small, there was a predilection for the colder months which is at odds with reports of clustering of the Coxsackie A6 strain in warmer months (2).

Conclusion: We describe a case series of paediatric eczema patients presenting with vesiculo-bullous eruptions, secondary to the Coxsackie A6 strain. This represents a paradigm shift in the presentation of HFMD in children, presenting to our hospital department. We discuss the implications of these findings in our eczema population, with reference to recent literature on the subject.
While eczema herpeticum is a serious sequela of herpes infection, eczema coxsackium has significantly less morbidity attached and heralds a new pattern of viral exanthems that the clinician must be aware of (5).

Case presentation 02. 11.39am

Xanthelasmoid variant of urticaria pigmentosa - a rare presentation of cutaneous mastocytosis

W. Abdelrahman, D. Watkins, C. Grattan, C. Devereux. Antrim Area Hospital & St John's Institute, London.

A 5 month old infant of Polish origin was referred to Dermatology with a rash that started at two months of age in the groin, progressing slowly to involve face/scalp/trunk/limbs. Aside from episodic flushing when warm associated with itch, she was otherwise asymptomatic. Examination revealed multiple asymptomatic papulonodular lesions with a yellow hue in an otherwise well appearing infant. Skin biopsy showed features in keeping with cutaneous mastocytosis supported by immunohistochemistry (positive CD43, tryptase, CD117, CD68; negative CD1a, S100, CD5). The clinical findings supported by histological features were in keeping with the xanthelasmoid variant of urticaria pigmentosa. Given the absence of organomegaly clinically and radiologically and that she was well, a bone marrow biopsy was not indicated. Serum mast cell tryptase elevated (43.9microgram/l). This continued to increase until 20 months of age (58.6microgram/l- corresponding to development of new lesions) followed by a marked reduction (25.7microgram/l- corresponding to improvement in clinical appearance) at 28 months of age. Apart from a stable lymphocytosis, blood tests were otherwise unremarkable. An insignificant improvement in frequency of flushing was noted with non-sedating antihistamines (cetirizine, desloratidine), ranitidine and montelukast. Mastocytosis can be classified as cutaneous or systemic. Cutaneous mastocytosis of the xanthelasmoid variant is rare and usually presents with yellow papulonodular lesions resembling xanthomas. The term 'xanthelasmoidea' was coined by Fox in 1875 and between 1875-1883, 19 cases were reported (1)(2). Treatment is the same as for other patients with cutaneous mastocytosis, including the avoidance of drugs that cause mast cell degranulation. As with other childhood mastocytosis, gradual improvement is anticipated, but may take longer (3). Interestingly, Husak et al. stated that compared to childhood variants of mastocytosis, there were no differences reported regarding development of systemic involvement or malignant transformation (4).

References:
1.Fox T. On xanthelasmoidea (an undescribed eruption). Trans Clin Soc London 1875;8:53-7.
2.Fox TC. On urticaria pigmentosa or xanthelasmoidea. Med Chir Trans 1883;66:329-47.
3. Stein DH. Mastocytosis: a review. Pediatr Dermatol 1986; 3: 365-75.
4. Husak R, Blume-Peytavi U, Pfrommer C, Geilen CC, Goerdt S, Orfanos CE. Nodular and bullous cutaneous mastocytosis of the xanthelasmoid type: case report. Br J Dermatol 2001; 144: 355-358.

Case presentation 03. 11.48am

Pregnancy related pyoderma faciale, a challenging dermatoses with limited treatment options

I. McDonald, N. Mansoor, M. O'Connell. University Hospital Waterford.

We report the case of a 37year old pregnant lady who presented at 15 weeks gestation to the dermatology department with a severe centrofacial pustular eruption. The patient gave a history of severe acne as a teenager requiring treatment with systemic Isotretinoin. Four weeks prior to presentation she reported the abrupt onset of painful papules, pustules and nodules over her forehead, cheeks and chin. She denied any ocular symptoms, fever or joint pains but complained of significant facial pain and discomfort. On examination she had centrofacial erythema and oedema affecting her forehead, cheeks, nose and chin. This was associated with multiple papules, pustules and nodules. There were no comedomes on examination. A diagnosis of pyoderm faciale was made. She was commenced on azithromycin 500mgs once daily for 3 days a week

in combination with zinc 20mgs once daily. At review, 4 weeks later she reported significant improvement with reduced nodules and pustules and less facial discomfort. On examination she had centrofacial erythema but less papules and pustules and no nodules. Her dose of azithromycin was reduced to 250mgs for 3 days per week for a further month in combination with zinc 20mgs. She was reviewed again at 26 weeks gestation. At this stage she was off antibiotics for 3 weeks. She denied new pustules or nodules. On examination she had mild background erythema. The papulopustular and nodular eruption had resolved. As part of ongoing treatment she was maintained on zinc throughout her pregnancy with no relapse.

Rosacea fulminans also known as pyoderma faciale was originally described in 1940 by O'Leary and Kierland and further classified in 1992 by Plewig et al. as a severe variant of rosacea. It is a rare cutaneous disorder characterized by the sudden onset of painful papules, pustules and cysts associated with centrofacial erythema. It occurs most commonly in women aged 15-46. As in this case it has also been associated with pregnancy. Recommended treatment options include high potency topical or systemic corticosteroids in association with isotretinoin, dapsone and oral tetracyclines . However many treatments are contraindicated in pregnancy. Previous case reports have shown azithromycin to be an effective agent in this setting. Oral zinc has also been proposed as an additional treatment for rosacea. Its combination with azithromycin in this case resulted in the effective, safe and sustained treatment of this rare and distressing skin condition.

Case presentation 04. 11.57am

Successful surgical management of two patients with severe toxic epidermal necrolysis

I. Timoney, L. Nestor, M. Sadlier, D. Wall, B. Wynne, O. Shelly, L. Barnes. St James's Hospital.

Steven-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are severe mucocutaneous reactions characterised by blistering and epithelial sloughing. SJS/TEN is a disease spectrum, with TEN involving epidermal detachment of greater than 30% of total body surface area and associated higher mortality. UK guidelines for the management of SJS/TEN advocate a multi-disciplinary approach with dermatologists often leading care. One strategy to protect and preserve the dermis in TEN is to cover it with a temporary skin substitute. Surgical debridement followed by wound closure using biosynthetic dressings, xenograft or allograft could be considered following failure of conservative management.(1) We present two cases that illustrate this multidisciplinary approach to care, and in particular the surgical management of TEN. Both patients were treated with aggressive debridement and allograft use, with good effect.

A 36 year-old-man self-referred to the emergency department with a 3 day history of fever and a 2 day history of odynophagia. On the day of admission, he developed an erythematous rash localised to his chest. No obvious culprit drug was identified. There was rapid progression with diffuse painful blistering, ocular and mucocutaneous involvement and a SCORTEN of 3. Biopsies showed full thickness epidermal necrosis consistent with the clinical diagnosis of TEN. Despite intensive nursing care in the burns unit and IV immunoglobulins, he had significant skin loss (>60% BSA). On day 3 of admission, all necrotic skin was debrided and allograft and Biobrane® dressings applied.

Patient 2, a 40-year-old man, was admitted 2 days after onset of a widespread erythematous papular rash. He had started oral prednisolone and omeprazole four weeks previously for interstitial pneumonitis. At initial assessment, a clinical diagnosis of SJS/TEN overlap was made. SCORTEN was 3. By day 4 of admission bullae had spread to involve 60-70% of BSA with almost 100% skin loss by day 10. Deterioration despite optimal medical management, along with intractable pain, prompted surgical intervention. Debridement of the lower limbs, chest and left arm with application of meshed 2:1 allograft using staples was performed on day 12.

Both patients were discharged within five weeks of surgical intervention. We present these cases to highlight the successful surgical treatment of TEN.

References:
1. UK guidelines for the management of Stevens-Johnson syndrome/toxic epidermal necrolysis in adults

Case presentation 05. 12.06pm

An unusual radiotherapy related rash

W. Abdelrahman, M. Walsh, O. Dolan. Royal Victoria Hospital, Belfast.

An elderly gentleman was referred following a diagnosis of Merkel cell carcinoma (MCC) on histology of an isolated right groin node swelling removed by general surgeons. Meticulous examination and PET CT imaging did not identify a malignant primary focus. A final diagnosis of primary nodal MCC was made. He underwent a radical groin dissection and adjuvant radiotherapy was delivered to right pelvic/ inguinal nodes over 6 weeks with volumetric arc therapy. One week prior to completing treatment he developed an intensely itchy, burning, erythematous, pustular eruption over the right buttock. Histologic examination revealed inflammation extending into the epidermal/ dermal junction predominantly lymphocytic in type as well as a perivascular infiltrate composed of small mature lymphocytes and histiocytes but with significant number of eosinophils in keeping with eosinophilic, polymorphic and pruritic eruption associated with radiotherapy (EPPER). This settled with topical corticosteroids. EPPER, a rare complication of radiotherapy, was first described in 1999. Clinical findings are polymorphic and include papules, urticaria, vesicles and sub epidermal bullae. Most cases have been in relation to breast and cervical cancer, to our knowledge this is the first reported case in the literature of EPPER related to MCC and the second associated with a pustular eruption.

Case presentation 06. 12.15pm

Recognising a familiar dermatoses in an unfamiliar site , erosive pustular dermatoses of the leg, a diagnostic challenge.

I. McDonald, N. Mansoor, L. Paul. University Hospital Waterford.

We report the case of a sixty eight year old man referred to the dermatology department with a persistent, painful rash affecting his right lower limb. The patient was immobile with right-sided hemiplegia and expressive dysphasia following a Cerebrovascular accident (CVA) in 2007. Although he had intermittent skin rashes on his right shin since the CVA it deteriorated significantly over a period of eight months prior to presentation. He developed a worsening pustular eruption with discharge, bleeding and painful erosions on the skin of his right shin. Skin swabs

taken from the site repeatedly grew Staphylococcus aureus. Despite four separate courses of antibiotics and twice weekly dressing changes it continued to deteriorate requiring opioid analgesia for pain control. On examination of his right lower limb there was a sharply demarcated area of erythema extending from his right shin to his distal thigh. There were extensive erosions with skin fragility and bleeding. There were also multiple discrete and coalescing pustules and flaccid pus filled bullae. A skin biopsy was performed and skin swabs were taken from the area. Histology showed epidermal spongiosis with neutrophil infiltration of the epidermis and papillary dermis. There were no features of vasculitis and fungal stains were negative. Skin swabs again grew Staphloccus aureus. Given the clinical and histopathological findings a diagnosis of erosive pustular dermatoses of the leg was made. The patient was treated with daily potassium permanganate soaks and clobetasol propionate 0.05% ointment. He was also commenced on oral flucloxacillin 500mgs six hourly for seven days. After four weeks there was marked clinical improvement with mild post inflammatory erythema of the skin and minimal residual pustules and erosions. He was continued on this topical treatment regime for a further two weeks resulting in further clinical improvement and resolution. Erosive pustular dermatosis of the leg (EPDL) is an uncommon, idiopathic inflammatory dermatoses first described by Lanigan and Cotteril in 1987. It is an amicrobial pustuloses typically affecting the lower limbs of elderly patients. It is sometimes misdiagnosed as cellulitis given clinical features of pain and erythema delaying effective treatment. Clinical and histopathological features help point to the diagnosis and treatment with potent topical steroids can result in prompt clinical improvement and resolution. Prophylaxis with tacrolimus as an alternative to potent topical steroids has also been reported.

Case presentation 07. 12.24pm

An Aggressive Dermatophytosis In A Cardiac Transplant Patient

J. MacMahon, L. Cunningham, A. O'Connell, D. Brady, N. Ralph. Mater Misericordiae Hospital, Dublin.

A 63 year old gentleman was admitted to hospital, November 2016 with an LRTI and was noted to have painful lesions of his lower legs for 7 months. following cardiac transplantation for non-ischaemic cardiomyopathy . He had multiple other co-morbidities including insulin-dependent diabetes mellitus and stage IV chronic kidney disease requiring dialysis. He was immunesuppressed on tacrolimus 3mg twice daily and prednisolone 10mg daily.

Initial examination revealed multipleviolaceous palpable lesions some of which were bullous and some eroded on the lower legs and feet. the appearance of which was consistent with a bullous vasculitis. Biopsy showed changes suggestive of same and the patient initially responded to a tapering course of oral prednisolone. However he represented three weeks later with clinical deterioration and worsening pain. Examination revealed annular lesions spreading from the initial sites which were studded with areas of ulceration, and a malodourous exudate along with new lesions appearing. A second set of biopsies were taken including a tissue culture which revealed fungal spores and histological features consistent with a fungal infection.

Given his advanced kidney disease, the decision was made to commence Voriconazole therapy instead of Amphotericin B. Tacrolimus unfortunately had to be reduced due to the interaction with Voriconazole which put the patient at risk of transplant rejection. Multiple cardiac biopsies were performed to monitor same. Scrapings and nail clippings were taken for mycology and a sample sent to the UK for fungal PCR.

Once available, PCR identified the pathogen as Trichophyton rubrum. TR is a common skin commensal but it is uncommon to see such aggressive resultant disease. Immunosuppression and advanced diabetes are comorbidities which increased this patient's susceptibility to an aggressive dermatophytosis from what is commonly a benign commensal. In this case however, the size and severity of the lesions caused a vulnerable patient to become very unwell and resulted in a prolonged hospital course.

Though dermatophytosis is common, deep cutaneous infection by dermatophyte fungi is a rare occurrence seen in immunesuppressed individuals (1). This case represents an unusual appearance of dermatophytosis and a therapeutic challenge but with a positive treatment outcome.

1.A Unique Clinicopathological Manifestation of Fungal Infection: A Case Series of Deep Dermatophytosis in Immunosuppressed Patients. Kershenovich R, Sherman S, Reiter O, Huss SR, Didkovsky E, Mimouni D, Hodak E, Segal R.Am J Clin Dermatol. 2017 Apr

Clinical photography & histology slides available.

Case presentation 08. 12.33pm

A rare case of giant myxoid Dermatofibrosarcoma Protuberans with focal fibrosarcomatous change.

L. Timoney, I. Timoney, N. Walsh. Sligo University Hospital, St James's Hospital, Blackrock Clinic, Dublin.

A 64-year-old woman presented with a large indurated plaque consisting of exophytic, sausage shaped nodules, affecting the lower abdomen and groin. The mass had grown rapidly over a period of twelve months. It measured more than 20cm in maximum diameter at initial presentation.
A biopsy was taken from one of the nodules. Histology showed characteristic features of the fibrosarcomatous (higher grade) variant of dermatofibrosarcoma protuberans (DFSP) with strikingly prominent whorls of eosinophilic myoid cells. This nodule was subsequently excised. The bulk of the tumour (>50%) had a myxoid appearance. Surprisingly, there were no remaining high grade areas of DFSP seen histologically. Overall, diagnosis was consistent with dermatofibrosaroma protuberans, myxoid variant with focal fibrosarcomatous change. In keeping with this diagnosis, immunohistochemistry was positive for CD34 and negative for S100 protein.
MRI abdomen showed extension of some of the nodules deep into subcutaneous tissue. There was no evidence of pathologically enlarged lymph nodes or distant metastasis.
The patient was referred for surgical excision of the entire plaque. The wound defect was reconstructed using a combination of local tissue advancement and skin grafting. The tumour was excised, however the deep margin was 1.5mm and further wider excision was subsequently performed.

Discussion: DFSP is a rare, cutaneous malignancy. It is a slow growing but locally aggressive tumour.
DFSP has an annual incidence of 0.8-4.2 cases per million. Histological variants of DFSP include myxoid, pigmented, atrophic,

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giant cell fibroblastoma (GCF) and DFSP with fibrosarcomatous change. Classic DFSP consists of spindle cells in the dermis arranged in a storiform pattern with infiltration into adjacent tissue. The myxoid variant has spindle cells embedded haphazardly in a pale myxoid stroma. The fibrosarcomtous variant is distinguished from the classic variant by increased cellularity, cytologic atypia and mitotic activity.
We present a case of giant, myxoid DFSP with focal fibrosarcomatous change. In addition, nodules of eosinophilic myoid cells were seen histologically. The concomitant presence of these variants of DFSP in one tumour is particularly rare and, to our knowledge, has only been reported on one occasion in the literature. In addition, the large size of this DFSP and striking morphology is noteworthy. The presence of a higher grade fibrosarcomatous component confers a higher risk of distant metastases.

Case presentation 09. 12.42pm
Atypical lower limb ulceration in a patient with rheumatoid arthritis on long term methotrexate – a rare cause
C. Harnett, N. Kearney, J. Thorne, J. Sargent, C. Feighery. Our Lady of Lourdes Hospital, Drogheda.

A 74 year old female presented with a six month history of multiple atypical ulcers on her left lower limb and papulonecrotic lesions affecting both legs. Her past medical history included rheumatoid arthritis (RA) and hypertension. No prior dermatological history. She had been prescribed Methotrexate 10mg weekly for RA and folic acid for several years. Other longterm medications included Furosemide/Amiloride hydrochloride, Nifedipine, Atenolol, and Lanzoprazole.

She had been systemically well prior to presentation. On examination there were multiple ulcers on her left leg which were punched out, with an erythematous base. They were not painful. There were also multiple smaller papulonecrotic lesions on both legs which had appeared prior to the onset of the ulcers. Skin exam was otherwise unremarkable. No palpable lymphadenopathy.

Given the atypical appearance and recent onset of the ulcers on a background of longterm immunosuppression; the differential diagnosis included atypical infection such as mycobacterium or viral/fungal aetiology, vasculitis, or an atypical skin lymphoma.

Full blood count was normal (WCC 7.6 with normal differential), CRP elevated at 50, renal and liver function tests within normal range. Swabs showed no fungal/viral growth, AFB negative. Vasculitic screen was negative. Hepatitis B and C, HIV and VZV negative. HSV 1+2 negative. Skin biopsy showed infiltrate of atypical lymphoid cells, many large, some showing prominent nucleoli. The infiltrate appeared to be angiocentric. Immunophenotyping: CD20 and CD30 positive, EBV strongly positive, EBER positive. Features consistent with lymphomatoid granulomatosis. CT thorax abdomen and pelvis showed a large left adrenal mass with calcification and a well circumscribed lesion in the anterior abdomen with a calcific rim. Lungs appeared normal.

Lymphomatoid granulomatosis (LG) is a rare EBV-driven angiodestructive B cell lymphoproliferative disorder. It has been reported in patients with RA likely due to the immunosuppressive state induced by immunomodulating agents. Several studies have demonstrated the relationship between EBV reactivation and the pathogenic mechanism of lymphomatoid granulomatosis. LG cases often involve the lungs (>90%), skin (25- 50%), kidneys (32%), liver (29%) and brain (32%).

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Case presentation 10. 12.51pm
A case of primary cutaneous mammary analog secretory carcinoma: a rare entity
S. McCarthy, M. McMenamin, C.C.B.B Heffron, M. McDermott, C. Hackett, M. Lynch. University Hospital Limerick, St James's Hospital, Dublin, Cork University Hospital.

An 82-year-old male presented with a 30-year history of a left shoulder lesion. He complained of irritation associated with the lesion for several years. On examination there was a 10 x 7 mm erythematous papule, paler centrally, with peripheral arborising vessels on dermoscopy. Our differential diagnoses included dermatofibroma, basal cell carcinoma and an adnexal tumour.

Histopathological examination of the excision biopsy demonstrated a dermal glandular neoplasm forming cords and tubular structures with vacuolated cytoplasm and abdundant eosinophilic proteinaceous contents. These findings were most supportive of a diagnosis of primary cutaneous mammary analog secretory carcinoma (MASC) although an S100 immunostain was negative. The ETV6-NTRK3 gene translocation was not detected by recombinant polymerase chain reaction. The mucin immunostain, MUC-4, was multifocally positive which has been described in MASC. A 10mm wide local excision was undertaken to minimise the risk of recurrence.

Mammary analog secretory carcinoma is a rare salivary carcinoma first described in 2010, that shares histologic, immunohistochemical and genetic features with secretory carcinoma of the breast. MASC is generally a solitary, well-circumscribed nodule, that most often presents in the parotid gland. MASC presenting as a primary tumour in the skin has only been reported in 11 cases to date. The most common site reported is the axillary skin. Features on histology include cystic, tubular, solid and/or papillary architecture with eosinophilic vacuolated cytoplasm. The ETV6-NTRK3 fusion transcript is seen in most but not all cases. Recombinant polymerase chain reaction can be negative but evidence of a break in ETV6 may be detected by fluorescent in-situ hybridisation. Immunoreactivity for mammaglobin and the S100 protein is usually positive. Most patients have an indolent course. There is a moderate risk of local recurrence, however a low risk of distant metastasis.

We report this case to raise awareness of this recently described rare entity with distinct histopathology that may present as carcinoma in the skin. This case is not classical; it shows histological features of MASC but only focal mammaglobin positivity and was S100 negative. The spectrum may be wider than has been described to date.

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Poster 01.

"The Benefits of a Combined Dermatology/Rheumatology Connective Tissue Disease Clinic".

L. McDonald, C. McCourt, E. Ball, C. Riddell. Belfast Health & Social Care Trust.

Introduction: Comprehensive management of Connective Tissue Disease (CTD) often requires input from both Dermatology and Rheumatology with successful management of complex cases requiring a holistic, multi-disciplinary approach. In Northern Ireland suspected CTD patients have historically been assessed independently by both specialties despite both receiving secondary and tertiary referrals.

Aims: A quality improvement project was designed with the aim of improving care for patients with suspected CTD. Pilot clinics involving four clinicians with an interest in CTD were designed to allow immediate clinician interaction, earlier consensus diagnosis and commencement of optimal treatment and improved staff education. Additional benefits proposed were avoidance of duplication of clinic appointments and enhanced inter-disciplinary co-operation.

Methods: 34 patients were identified to attend seven pilot clinics between October 2014 and February 2017. Suitability was determined by the presence of a dermatological issue in the context of a possible or known diagnosis of CTD. Following a team de-briefing after the initial clinic a standardised proforma was developed to capture data and inform patient assessment during each clinic. Patient clinical, immunological and previous treatment data was collated prior to attendance. Two consultant rheumatologists and two consultant dermatologists assessed the patient at the clinic and agreed a combined management plan. Patients were then given anonymised patient satisfaction ques tionnaires.

Results: 20/34 (59%) of patients had an already established diagnosis of CTD. The remaining 14/34 (41%) had a suspected diagnosis of CTD. Median (range) disease duration was 6(0.5,23) years.

Of those with a previous CTD diagnosis 9/20 (45%) had this diagnosis revised. 6/20 (30%) received a new additional diagnosis following attendance. 25/34 (74%) patients with either known or suspected CTD had this diagnosis confirmed. 11/34 (32%) had their treatment plan adjusted and 2/34 (6%) of patients were able to be discharged completely. 28/34 (82%) of patients were previously known or referred to Dermatology services. 8/28 (29%) did not require Dermatology follow-up after combined assessment, avoiding unnecessary appointments.
100% of patient satisfaction questionnaires received (28/34 (82%)) rated helpfulness of combined assessment 5 or above on a Likert scale (1=not helpful, 7=very helpful). All said they would attend again.

Conclusion: Our data is promising in terms of both clinical outcomes and patient experience with very encouraging positive patient feedback. The combined clinic created an environment conducive to shared learning, staff development and ultimately improved patient care.

Poster 02.

Undergraduate dermatology teaching: an un-met need

L. O'Higgins, R. Hughes. University College Dublin, Belfield; Dublin 4, St Vincent's University Hospital, Dublin 4.

Background: Skin conditions are one of the most frequent reasons for consultation in general practice. Malignant melanoma is the fifth most common form of cancer in the UK and Ireland and its incidence is projected to increase. Studies from other countries suggest that there is insufficient dermatology teaching provided in the undergraduate medical curriculum. However we are not aware of any data from Ireland, which might help inform the content of the undergraduate curriculum in dermatology.

Aim: The aim of this study was to assess interest in dermatology and perception of the current dermatology teaching curriculum among Irish medical students.

Method: An on-line survey was sent to all medical students in an Irish university in both undergraduate and graduate entry medical programmes (n=1364). Google Forms was used to collect and analyse the data.

Results: The response rate was 16.6% (227/1364). Of the responders 20.3% (46/227) said they were very interested in dermatology, 66.5% (151/227) were somewhat interested and 13.2% (30/227) had no interest in dermatology.

Asked whether interested in pursuing a career in general practice 24.2% (55/227) said yes, 48.9% (111/227) said maybe and 26.9% (61/227) said no.

In relation to dermatology teaching 94.7% (215/227) of students felt they had not had sufficient teaching and 88.9% (202/227) said they would like more.

Regarding the form that such teaching should take, clinical skills workshops were the preferred form of teaching (52.6%, 119/227), followed by tutorials (19.1%, 43/227), problem based learning (17.2%, 39/227) and lectures (11.2%, 25/227).

Conclusion: As far as we are aware, this is the first study to assess students' perception of dermatology teaching in Ireland. Despite an increasing number of resources for undergraduate dermatology teaching including on-line teaching programmes, handbooks from the British Association of Dermatology, Annual DermSchool programmes and a recently launched Dermatology Medical Student App, there is a dearth of teaching on the undergraduate curriculum.

Our results show an un-met need for dermatology teaching in Ireland at an undergraduate level. The first step in instigating change is to identify a need. We believe the results of this survey should form the basis for a renewed focus on the provision of dermatology teaching to medical students.

Poster 03.

Practical experience of secukinumab in the treatment of psoriasis: experience from a single centre

L. Griffin, B. Ramsay, C. Hackett, K. Ahmad, M. Lynch. University Hospital Limerick.

Background: Secukinumab is a novel anti-interleukin-17A agent that has achieved a 75% decrease from baseline in Psoriasis Area and Severity Index (PASI 75) in 77-93% of patients treated in clinical trials. There are limited data on the use of secukinumab outside of clinical trials.

Objectives: To assess the efficacy and safety of secukinumab in patients with severe psoriasis attending a dermatology service in Ireland.

Methods: A retrospective case-note review of 22 patients with psoriasis treated with secukinumab.

Results: At baseline the mean duration of psoriasis was 20.6±sd12.6 years, mean body mass index 33.9±10.7 kgm-2, mean age 41.6±13.5 years and 17 (77%) were female. The mean PASI was 14.5±7.9 and mean Dermatology Life Quality Index 16.9±7.1. Nineteen (86%) patients had chronic plaque psoriasis, two (9%) patients had palmoplantar psoriasis and one (4%) patient had predominantly scalp disease. Prior to secukinumab therapy 95% of patients had been treated with a biologic (mean number of biologics 2.4±0.8), 95% had been treated with a systemic agent (mean number of systemic agents 1.1±0.9) and 73% had received phototherapy. Eleven patients (50%) had psoriatic arthritis. Twenty patients were commenced on secukinumab (300mg monthly maintenance dose) by dermatology and two patients were commenced by rheumatology (150mg monthly maintenance dose).
After 16 weeks of treatment with secukinumab PASI 75 was observed in 9/20 (45%) patients; a physician global assessment (PGA) score of almost clear (AC) was recorded in one further patient (50% of patients achieved PASI 75 or AC on PGA, data unavailable in two patients). A decision to discontinue therapy was made in four patients (18%) due to lack of efficacy. In the remaining six patients, treatment with secukinumab was continued at 16 weeks as they were achieving improvements in PASI but had not achieved PASI 75.
Secukinumab therapy was well tolerated. A serious adverse event was observed in one patient (4.5%) and was considered unlikely to be secukinumab treatment-related (one death secondary to acute coronary syndrome). Adverse events that were considered possibly treatment-related, noted in 4 patients (18%), were of mild severity, and did not require hospital admission (candidiasis (n=2), respiratory tract infections (n=2), and grade 1 neutropaenia (n=2).

Conclusions: In this single unit retrospective study secukinumab demonstrated acceptable levels of short-term therapeutic efficacy and safety in everyday clinical practice.

Poster 04.

Skin biopsies conducted by non-specialist services are an inefficient use of services

S. O'Sullivan, S. Bowe, M. Murphy, C. Heffron, J. Bourke. South Infirmary Victoria University Hospital, Cork University Hospital, Cork

We conducted an audit of excision biopsies referred to the regional pathology department in Cork University Hospital primarily to assess the appropriateness of procedures carried out by hospital and general practitioners. We compared the number of malignant to benign specimens referred to pathology during the month of October 2016. The department of pathology at Cork University Hospital serves County Cork with some spill over from adjacent counties of Kerry, Limerick, Tipperary and Waterford. We included 900 specimen results from this period and sorted them into the following categories: Melanomas (invasive and in situ), squamous cell carcinoma (invasive and in situ), basal cell carcinoma, benign pigmented lesions and warts, tags and cysts. We excluded scar revisions, punch biopsies and biopsies looking for residual malignancy. Overall we found that dermatology/plastics had the highest ratio for number of malignant to benign excisions and biopsies at 1 malignant for every 6 benign lesions. The ratio for general practitioners was 1 malignant for every 91 benign lesions. In conclusion we feel it would be most appropriate for excisions and biopsies to be conducted in a hospital setting by the skin cancer specialist service rather than in the community.

Poster 05.

Local radio and newspaper best methods to reach male population for Euromelanoma campaign in Ireland

L. Griffin1, D. Roche2, L. Roche1, M. Murphy2. 1 University Hospital Limerick, 2 South Infirmary Victoria, University Hospital, Cork.

Introduction: Euromelanoma is a pan-European prevention campaign against skin cancer and promotes an annual walk-in screening day. Dermatology consultants from the South Infirmary Victoria University Hospital, Cork and Waterford Hospital held a screening clinic in May 2017 in South Tipperary Hospital, Clonmel, Co. Tipperary. The event was advertised with Euromelanoma posters (1) displayed in Clonmel town and hinterland, local print media (the 'Tipperary Star' and 'The Nationalist' newspapers) and the local radio station ('Tipp FM'). It was also publicised on Facebook and twitter. 264 people attended. We examined how they became aware of the event.

Methods: Each attendee completed a standardised Euromelanoma questionnaire, which included demographics. They were also asked to fill a questionnaire to ascertain how they had heard of the campaign and these were matched and results analysed via SPSS.

Results: Complete data is available for 245 attendees. 66% were female. The average age of male attendees was 58.6 years and female was 54.43 years. 79.31% of the male attendees and 63.29% of the females were >50 years. Over half of those who attended became aware of the event from the 2 local newspapers (the 'Tipperary Star' and 'The Nationalist') and the local radio station ('Tipp FM'). Social media (Facebook and twitter), posters, and word of mouth were less important methods.

Men>50's were even more responsive to traditional methods with 73.9% reporting hearing about the event via local newspapers and local radio stations combined. Social media use was only 7% in this demographic.

Conclusion: The incidence of melanoma and non-melanoma skin cancer (NMSC) increases with age. Men have a higher mortality rate from NMSC (2) and present later with melanoma with worse prognosis (3). One third (34%) of those screened in Clonmel were male, in keeping with international pooled Euromelanoma data (4). Our campaign shows the success of local newspaper and local radio in reaching males over 50 years for health promotion.

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Poster 06.

Giant Cell Arteritis presenting as scalp necrosis

L. Griffin1, JP. Doran2, N. Leonard3, S. Ryan1, B. Ramsay1. 1 Dermatology department, University Hospital Limerick. 2 Rheumatology department, University Hospital Limerick. 3 Pathology Department, St. James Hospital, Dublin 8.

Giant cell arteritis (GCA) is a systemic inflammatory vasculitis affecting medium to large vessels, most commonly seen in the elderly. The aetiology is unknown and it has a predilection for the arteries of the head and neck. Prompt diagnosis can be challenging yet important to prevent complications such as visual loss by treatment with systemic steroids. Introduction of steroid sparing agents may be considered to reduce side effects of systemic corticosteroids (1).

A 79-year-old man presented with a one month history of painful scalp ulceration. This was preceded by a low-grade headache and neck pain for 3 weeks for which he applied a non-steroidal anti-inflammatory gel. He denied other symptoms. He had a history of coronary artery disease, hypertension, chronic obstructive airway disease and gastritis. On physical examination, he had a large area of ulceration of the vertex of his scalp measuring 13.5 x 9.5cm with necrotic eschar. The remainder of clinical examination was unremarkable. His erythrocyte sedimentation rate was 59 mm/hour.

Incisional skin biopsy of his scalp showed ulceration and acute inflammation. Muscular blood vessels present at the junction between the dermis and subcutaneous fat distant to the area of ulceration showed evidence of acute inflammation indicating vasculitis. Giant cells were not seen.

Despite topical therapy with clobetasol and oral minocycline and dapsone his ulceration failed to improve. A temporal artery biopsy was performed showing classic granulomatous inflammation with mononuclear infiltration of medium sized vessels, confirming the diagnosis of Giant Cell Arteritis. He commenced on oral corticosteroids and was referred for ophthalmological assessment.

His ulceration has improved slowly. Some weeks into treatment, he developed severe bilateral leg pain which was confirmed as ruptured achilles tendons on ultrasound scanning. He has been commenced on fortnightly tocilizumab and his steroid therapy is being gradually reduced.

Scalp ulceration and necrosis are rare cutaneous complications of GCA and present a diagnostic challenge for the physician in the absence of other typical symptoms. It suggests multi vessel involvement as the scalp is a well vascularised area with 4 supplying arteries and is associated with a higher incidence of irreversible visual loss and tongue gangrene, than those with GCA and no scalp necrosis (2). Adequate and prompt immunosuppression is therefore important. Our case demonstrates the need to consider GCA in patients who present with scalp ulceration and the benefit of temporal artery biopsy in cases of uncertainly.

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Poster 07.

A case of postoperative pyoderma gangrenosum

L. Timoney1, A. Drummond2, F. Campbell2. Sligo University Hospital1, Queen Elizabeth University Hospital, Glasgow2.

A 33-year-old woman was delivered by emergency Caesarean for reduced foetal movements at 36 weeks' gestation. She was well post-operatively till day 10 when she developed pyrexia of 39°C. Erythema was noted round the wound which enlarged rapidly and ulcerated over a few days. The edges became necrotic. White cell count was raised at 35 with predominant neutrophilia and C-reactive protein (CRP) was 346.

A presumptive diagnosis of necrotising fasciitis was made. Broad-spectrum antibiotics were commenced and emergency wound debridement was done as far as healthy tissue. Histology showed a prominent acute inflammatory infiltrate from the epidermis down to the deep fascial planes and it was reported that this could be consistent with the clinical history of necrotising fasciitis.

Following the debridement, necrotic tissue rapidly re-appeared at the wound margins. The wound progressed despite parenteral antimicrobials (linezolid, clindamycin, meropenem, gentamicin and fluconazole) and wound debridement on eight further occasions. Gram-staining and specimen culture were consistently negative. Mycobacterial and fungal stains were also negative.

During this period, a pustule noted at the site of previous cannula insertion on her right forearm progressed to ulceration.

She was intubated, ventilated and required inotropic support in the Intensive Care Unit. Dermatology specialist advice was sought due to the deterioration in her condition despite successive wound debridement.

On examination she had a large abdominal wound extending from the umbilicus to the mons pubis and across the anterior abdominal wall. The wound extended as deep as the abdominal cavity in areas. A faint purple rim noted at the top of the wound raised the possibility of pyoderma gangresnosum (PG). Histopathology was reviewed. The non-specific suppurative inflammation and necrosis throughout the dermal, fat and muscle layers was consistent with either NF or PG but histological distinction was impossible.

Following multi-disciplinary review, PG was diagnosed based on the following characteristic features:
1. Lack of improvement with broad-spectrum antibiotics and surgical debridement
2. Negative investigations for bacterial, fungal and mycobacterial infection
3. Rapidly-progressing necrotic lesions with irregular violaceous borders
4. Pathergy phenomenon noticed on her forearm

She was commenced on IV methylprednisolone. Over the following five days, CRP decreased from 346 to 22mg/L. Wound progression ceased. Striking clinical improvement allowed extubation. IV steroids and antibiotics were discontinued and oral ciclosporin 100mg b.d and prednisolone 75mg daily were commenced. The patient was discharged and remains on immunosuppressive therapy.

Poster 08.
"The Perils of Pyoderma"
C. Maguire, L. McDonald, S. Hoey. Royal Victoria Hospital, Belfast.

Introduction: We present a case of refractory pyoderma gangrenosum developing in a 24-year old lady with inflammatory bowel disease and its successful management with Adalimumab.
Case Description
This 24-year old lady was referred to Dermatology with a 1-month history of a painful itchy rash on both shins. An diagnosis of erythema nodosum was made based on the initial clinical appearance and over 6-months she was treated with varying combinations of topical and oral steroids, emollients, antibiotics, dapsone and colchicine without success.
Ongoing evolution of affected areas with ulceration then led to a revised diagnosis of pyoderma gangrenosum (PG) and a further course of high dose prednisolone was trialled at a maximal dose of 50mg daily. During this same time period the patient had reported ongoing abdominal cramping and diarrhoea. Subsequent gastroenterology (GI) investigation led to a diagnosis of Crohn's disease. The dual diagnoses of inflammatory bowel disease and refractory PG prompted a trial of Azathioprine 250mg, equating to 2mg/kg. Subsequently the patient suffered vomiting and this had to be stopped. Combined management discussion between Dermatology and Gastroenterology (GI) advocated Adalimumab 40mg every fortnight and improvement was gradually noted in the PG. 2-months post-commencement this lady became pregnant. Both Dermatology and GI teams recommended continuing Adalimumab until the third trimester, resulting in complete healing of the leg lesions.

Both the PG and Crohn's disease remained stable until delivery. Obstetric complications necessitated an emergency Caesarean section. The wound did not heal and active rapidly progressing PG developed at the site, indicative of pathergy. Adalimumab was re-introduced with good response and full wound healing was achieved over a 3 month period. She remains on Adalimumab therapy and both the PG and Crohn's disease have remained stable.

Discussion: PG is a complex neutrophilic dermatosis with significant potential morbidity and it remains a therapeutic challenge. Whilst it is well-reported in the context of inflammatory bowel disease (IBD) the variety of clinical presentations and lack of agreed diagnostic criteria can sometimes lead to misdiagnosis and treatment delay. Rapid escalation beyond standard oral systemic immune-suppressants to anti-TNF therapies for severe PG have been reported and this case illustrates the therapeutic benefit of Adalimumab as a first-line anti-TNF for both PG in the context of IBD and in the post-traumatic context. Our case also highlights the benefit of multi-disciplinary discussion and management of such complex cases to ensure optimal patient care.

Poster 09.
Scalp necrosis: a rare cutaneous manifestation of giant cell arteritis.
L. McDonald, O. Kerr. Belfast Health & Social Care Trust.

Introduction: We present a case of scalp necrosis as a rare manifestation of giant cell arteritis (GCA).

Case description: An 81-year old lady presented with an 8-week history of an enlarging, tender ulcerated area on her scalp. She had no history of skin malignancy and had attributed the ulcer to a minor graze with garden shears. Further questioning revealed an episode of ipsilateral jaw claudication two months prior to presentation. Examination revealed ulceration extending from hair margin to parieto-occipital junction. White cell count was normal but C-reactive Protein and Erythrocyte Sedimentation Rate (ESR) were elevated at 87.7 mg/L and 112mm/hr respectively. Skin swabs were negative for bacteriology and virology.

Incisional biopsies taken from anterior and superior poles of the lesion demonstrated ulceration, vascularization and necrosis extending to subcutaneous fat with no evidence of malignancy. Vasculitis was suggested as a likely cause of the extensive necrosis. Subsequent temporal artery biopsy demonstrated transmural inflammation, disruption of elastic lamina and multinucleated giant cells consistent with GCA.

A diagnosis of extensive scalp necrosis secondary to GCA was made. The patient was treated with intravenous methylprednisolone for three days followed by a reducing course of oral predni-

solone commencing at a dose of 1mg/kg/day, with twice weekly debridement and dressings. Although ESR normalized within one month and her scalp re-epithelialized within 5 months she required 24-months of prednisolone therapy to achieve complete healing.

Discussion: Scalp necrosis is a rare cutaneous complication of GCA developing as a result of multi-vessel occlusion of all four main arteries supplying the temporal scalp. The presence of scalp necrosis is associated with a higher incidence of visual loss and other visual defects and an increased standardized mortality ratio compared to cases without scalp necrosis. This potentially life-threatening complication reflects extensive vascular involvement with mortality often resulting from thrombosis of cerebral or coronary arteries.

Misdiagnosis is common, resulting in delayed treatment of underlying GCA. A high index of suspicion in this case ensured a focused history eliciting jaw claudication and recognition that, whilst the minor trauma reported was a possible precipitating factor in ulceration, the degree of ensuing necrosis suggested a grossly compromised arterial supply. This case highlights the importance of considering scalp necrosis as a manifestation of GCA, especially in the context of a raised ESR. Prompt diagnosis and treatment is necessary to prevent significant morbidity and mortality.

Poster 10.

Plane Xanthoma associated with Symptomatic Myeloma: A Case Series

G. Callaghan, M. Coyne, P. O'Gorman, F. Moloney. Mater Misericordiae Hospital, Dublin.

Xanthoma can be associated with lymphoproliferative disorders and monoclonal gammopathies. The appearance of plane xanthomas in particular, characterised by symmetrical sheets of yellow-orange macules on the face, neck and upper trunk, should prompt a screen for underlying myeloma.

A two patient case series with plane xanthoma in association with an Ig G Kappa paraprotein, running an uncharacteristically benign course over several decades is discussed.

Case 1 : A female patient with high-risk myeloma, lost to follow up, presented seventeen years post-diagnosis, with biopsy proven planar xanthomatous deposits, extensively involving her upper neck and chest, and a mildly elevated lipid profile. The paraprotein amount and restaging assessments were unchanged from her initial diagnosis.

Case 2: A male patient with high risk myeloma was noted to have developed striking plane xanthoma six years after diagnosis. Similarly, normal lipids and a stable paraprotein level with no progression of end-organ parameters was documented.

Both cases demonstrate a discordant clinical course to their predicted prognosis at diagnosis. The appearance of plane xanthoma may suggest an anti-myeloma effect. Xanthomatosis may suggest a role of the macrophage endothelial system in achieving disease stability. Although sample size is small, the theory of xanthomatosis mediated disease amelioration is worthy of future exploration.

Poster 11.

Challenges in CTCL; a spectrum of difficult clinical cases

N. Byrne, R. Barry, N. Swan, P. Collins, B. Moriarty. St Vincent's University Hospital, Dublin, St James's Hospital, Dublin.

The 2011 consensus statement from the ISCL, USCLC and EORTC provides a framework for standardised evaluation and management of cutaneous T cell lymphoma (TCL). This optimises individual patient care and facilitates essential clinical trials. The spectrum of disease presentation is vast however, with diagnosis almost universally delayed. We present 3 recent cases from our service highlighting challenges in achieving these goals.

A 64-year-old man presented with a 1-year history of an erythematous plaque on the nasal tip diagnosed clinically and histologically as sarcoid. Intralesional steroid, plaquenil and methotrexate failed to control his disease with extension of cutaneous lesions. Adalimumab and doxycycline were added leading to a severe flare with markedly photodistributed accentuation. Repeat skin biopsies during and after this episode demonstrated an atypical lymphocytic infiltrate diagnostic of TCL. T-cell rearrangement studies demonstrated a T-cell clone. PET CT showed parotid and cervical lymph node uptake raising the possibility of systemic TCL. Staging investigations are currently in progress. Cutaneous T cell lymphoma rarely exhibits extreme photosensitivity which in this case is likely to have been modulated by therapy.

A 59-year-old man presented wide-spread pruritic erythematous patches, clinically suspicious for mycosis fungoides. Initial histology was non-confirmatory. In 2016 he developed hip pain leading to the diagnosis of atypical systemic marginal zone B-cell lymphoma which was successfully treated with 6-cycles of R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, prednisolone) chemotherapy. His rash, which cleared with R-CHOP, recurred after 6 months with increasing area of involvement. Skin histology and TCR confirmed TCL, mycosis fungoides type, overall clinical stage III (T4N0M0B0). He had an initial response to PUVA and is currently maintained with skin directed therapy. Cutaneous TCL, particularly erythrodermic mycosis fungoides is associated with an increased risk of secondary systemic malignancies, including B-cell lymphoma.

A 31-year-old Croatian lady, known to be Hepatitis B positive, presented at 39+4/40 gestation with a 7-year history of a persistent rash. Examination revealed folliculotropic patches and plaques clinically and histologically typical of TCL, mycosis fungoides type, affecting proximal limbs and trunk; overall clinical stage 1B (T2aN0M0B0). She is currently maintained with skin directed therapy. Prognosis in CTCL may be usefully stratified using CLIPi (Cutaneous Lymphoma International Prognostic Index). Treatment decisions should be guided by disease risk.

We present these fascinating cases from our service to illustrate challenges in achieving now well-defined goals in cutaneous TCL.

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Poster 12.

Lichen aureus masquerading as acral melanoma

G. Callaghan, C. O'Keane, F. Moloney. Mater Misericordiae Hospital, Dublin.

A 62 year old lady was referred for evaluation of a left palmar pigmented lesion, increasing in size over eight months. Her general practitioner's referral indicated the presence of a parallel ridge dermoscopic pattern. Her background history was significant for allergic rhinitis and osteopenia. She worked as a sales assistant in the luggage section of a large department store.

The patient was left-handed, with Fitzpatrick Skin Type II. A 12mm x 7mm area of asymmetrical macular pigmentation on her left palm dermoscopically demonstrated non-homogeneous golden brown pigmentation favouring the acral ridges centrally, and becoming petechial towards the periphery. There were no other positive dermoscopic features of melanoma. Further examination revealed pigmented pupura on her lower limbs bilaterally, but no atypical melanocytic lesions or palpable lymphadenopathy.

An incisional biopsy demonstrated a dense superficial perivascular lichenoid infiltrate, associated with epidermal hyperplasia, and abundant iron pigment. No melanocytic lesion was identified. Lichen aureus was diagnosed following clinicopathological correlation. Review six months later demonstrated a gradual fading of the pigmentation.

Lichen aureus is a localised variant of the pigmented purpuric dermatoses, referring to a group of inflammatory purpuric disorders, without any evidence of vasculitis. Classically it affects the lower limbs, with asymptomatic areas infiltrated by localised yellow or bronze-coloured macules, papules and plaques, however it can affect other parts of the body.

On volar sites, the parallel ridge pattern has a sensitivity of 86%, and a specificity of 99% for the diagnosis of acral melanoma, both invasive and in-situ. In this case, general skin examination and clinicopathological correlation out-ruled melanoma and aided diagnosis.

Poster 13.

Drug Induced Cutaneous Lupus Erythematous Secondary to Pirfenidone Therapy

Aine Kelly, A. Lally. St Vincent's University Hospital, Dublin.

Drug related cutaneous lupus erythematous is characterised by clinical and immunopathological findings similar to lupus but which is temporarily related to drug exposure. Pirfenidone is a novel therapeutic agent for idiopathic pulmonary fibrosis, a chronic, progressive and fatal lung disease of unknown aetiology. Pirfenidone reduces the activity of transforming growth factor beta and has been shown to improve patient survival . To date there have been no documented cases of pirfenidone induced cutaneous lupus erythematous, although photosensitive reactions to this drug are well recognised.

We describe the case of a 54 year old woman with a 6 month history of idiopathic pulmonary fibrosis. The patient commenced pirfenidone (Esbriet™) 8 weeks prior to her acute presentation to dermatology with a painful intensely itchy rash in a photosensitive distribution. She had associated general malaise. Physical examination revealed tender, erythematous, oedematous plaques on the dorsal upper limbs, V of the neck, back and face. She had ragged cuticles and dilated capillary loops at the nail fold on dermoscopy. The mucous membranes were uninvolved. She had a 30-year history of chronic plaque psoriasis treated successfully on 2 occasions with narrowband UVB phototherapy, most recently 12 months prior to acute presentation and her psoriasis remained in remission. The presumed diagnosis at time of acute presentation was a phototoxic reaction secondary to pirfenidone.

Histopathology from affected skin showed interface dermatitis with basal vacuolar change and intraepidermal and junctional civatte bodies. There was mild superficial perivascular dermal inflammation including lymphocytes and histiocytes. There was significant mucin deposition, which was highlighted with alcian blue stain. Blood tests showed a mild lymphopenia of 0.9 10^9 (1-4) and strongly positive anti ds DNA of 32 IU/ml (0-9.9) but were otherwise normal. She was treated with topical clobetasol propionate 0.05% ointment and cessation of pirfenidone treatment. There was an improvement in symptoms within 14 days. By 4 weeks the rash had almost completely resolved with no residual scarring.

This patient had clinical, serological and histopathological findings consistent with an acute cutaneous lupus. Given the temporal association with her pirfenidone treatment and improvement upon cessation, this presentation was deemed most likely to be a drug induced cutaneous lupus. To our knowledge, this is the first reported case of cutaneous lupus erythematous secondary to pirfenidone therapy. It is unclear if previously reported photosensitivity reactions to this drug may actually have been in keeping with drug induced cutaneous lupus.

Poster 14.

Giant apocrine hidrocystoma of the forehead

I. Timoney, L. Timoney, N. Walsh. Sligo University Hospital, St James's Hospital, Blackrock Clinic, Co. Dublin.

A 35-year-old man presented with a seven year history of a slowly growing, well-defined, dome-shaped nodule on his forehead, abutting the left eyebrow. The nodule was predominantly flesh coloured. Erythematous and blue tinted areas were confined to discrete portions of the lesion, which had an irregular surface and resultant multi-loculated appearance.

The nodule measured approximately 5 x 6.5 cm and had arisen in previously healthy skin. The patient was asymptomatic. Punch biopsies were taken. Histopathology demonstrated fragments of skin containing benign apocrine hidrocysttoma. The patient declined definitive surgical excision.

Discussion:
Hidrocystomas (or cystadenomas) are benign, cystic lesions of the sweat glands which can be classified into apocrine and eccrine subtypes. Apocrine hidrocystomas are cystic lesions that arise from the apocrine secretory coil, while eccrine hidrocystomas represent retention cysts of the eccrine duct.
Both types of hidrocystoma have a similar clinical appearance and are best differentiated by histological examination. Apocrine hidrocystomas demonstrate multiple cystic spaces, frequently with papillary projections into the lumen. A layer of myoepithelial cells lies peripheral to the inner layer of secretory cells. By contrast, eccrine hidrocystomas have a single cystic cavity and lack papillary projections into the lumen.
Apocrine hidrocystadenomas were first differentiated from simple retention cysts by Mehregan in a clinicopathological study of 17 cases published in 1964.
Apocrine hidrocystomas usually present clinically as a solitary, soft, dome-shaped papule, most commonly occurring on the face, particularly the peri-ocular region.
Hidrocystomas are typically 1-3mm in diameter. Rarely , giant apocrine hidrocystomas (measuring more than 20mm) occur, with less than fifteen cases identified in the literature.
The differential diagnosis of apocrine hidrocystomas includes eccrine hidrocystomas, molluscum contagiosum, mucoid cysts , sebaceous cysts and atypical basal cell carcinoma.
Apocrine hidrocystomas tend to grow slowly and persist indefinitely without treatment. Surgical excision with complete cyst wall removal is the treatment of choice to prevent recurrence. Various other treatment modalities have been used, particularly for multiple lesions, including pulsed-dye laser, thermoablation, electrodessication and trichloroacetic acid.
In summary, we present a rare case of giant solitary apocrine hidrocystoma. It is unusual due to its size and striking clinical appearance.

Poster 15

Vitamin D status in patients with frontal fibrosing alopecia

E. Gilhooley, A. Kelly, R. Crowley, A. Lally. St Vincent's University Hospital, Dublin

Frontal fibrosing alopecia (FFA) is a primary scarring alopecia that was first described by Kossard in 1994. This relatively new condition, a clinical variant of lichen planopilaris, is postulated to be an immune-mediated disorder. Vitamin D is a secosteroid hormone involved in a number of metabolic processes. 1,25 (OH)2 D3 is a modulator of the innate and adaptive immune system and Vitamin D receptors (VDRs) are expressed on B cells, T cells and antigen presenting cells. Many autoimmune processes have been linked with low vitamin D levels. Vitamin D receptors are present within the hair follicle and the lack of VDRs is associated with reduced epidermal differentiation and hair follicle growth.
Frontal fibrosing alopecia has become increasingly recognised, leading to suggestions that environmental processes may play a significant role. The aim of this study was to evaluate vitamin D status in patients with FFA.

Methods
Patients with FFA were recruited consecutively from outpatient dermatology clinics. Pertinent clinical information was collected on participants and 25(OH) vitamin D levels were measured in an extended summer period between May and September 2017.

Results
Twenty female patients were identified. The diagnosis of FFA had been made as an incidental finding when patients were attending for another skin diagnosis in 9/20 cases and half of the patients had been diagnosed 1-5 years previously. Seventeen (17/20) participants were post or perimenopause. Treatments included topical clobetasol propionate 0.05%, hydroxychloroquine, pioglitazone and mycophenolate mofetil. Ten (10/20) were vitamin D deficient with serum 25(OH) vitamin D levels <50nmol/L; including 1 with levels <30nmol/L (range 17.9- 143.7). Eight (8/10) patients were receiving prescription Vitamin D supplementation at time of recruitment, including 3/10 patients with deficient vitamin D levels. Ten patients (10/20) reported wearing daily sun protection factor (spf) and a further six (6/20) reported that spf was contained in their daily moisturiser.

Discussion
Vitamin D deficiency in an Irish population, defined as <50nmol in an extended summer, has a prevalence of 35.4%. A high frequency of sunscreen use has been demonstrated in FFA, raising the possibility of a causative role of the chemicals within sunscreens. This also raises the question as to whether the increasing trends in photoprotection may render FFA patients deficient in Vitamin D. This small pilot study suggests further work looking at FFA and Vitamin D deficiency is required.

IAD
IRISH ASSOCIATION OF DERMATOLOGISTS

Spring Meeting

Galderma symposium

Paula Oliver, Michelle O'Loghlen,Lesley Ramsden & Maria McElwee. Galderma Symposium

Michelle O'Loghlen, Kevin Duffy, Dr Sandra Minor, Sandy Richardson, Dr Geraldine Morrow, Paula Oliver & Maria McElwee, Galderma Symposium

Dr Sandra Minor, Dr David Alderdice, Dr Geraldine Morrow & Paula Oliver Galderma

Dr Colin Fleming & Dr Kevin McKenna

Dr Fergal Moloney IAD & Guest speaker Prof Julia Newton-Bishop

Dr Fergal Moloney, Prof Julia Newton-Bishop & Dr Anne-Marie Tobin

Dr Hilary Jenkinson, Dr Sinead Field, Dr Veronique Bataille & Dr Catherine Harwood

Dr Kevin McKenna IAD President & Guest speaker Dr Judith Carser

Dr Kevin McKenna, IAD President & Dr Catherine Harwood, Guest speaker

Prof Colin Fleming Guest speaker & Dr Olivia Dolan IAD

Dr Kevin McKenna, IAD President & Dr Veronique Bataille

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PHARMA EXHIBITION

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PHARMA EXHIBITION

PHARMA EXHIBITION

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IAD SPRING MEETING WINNERS

Burrows Cup winner Dr Laura Nestor & Dr Anne-Marie Tobin

Dr Kevin McKenna IAD President & Burrows Cup winner Dr Laura Nestor

Dr Kevin McKenna IAD President & Dr Catherine Quinlan,
Burrows Cup runner-up

Burrows Cup Spring 2017 - Winning Abstract

Burrows Cup presentation, Spring 2017 winning abstract

Circulating Mucosal Associated Invariant T cells are depleted in melanoma and show an exhausted prof

L. Nestor, K. Molloy, J. Clowry, C. Gallagher, A.Salim, M. Connolly, A.M. Tobin.
Tallaght Hospital, Dublin.

"Circulating mucosal associated invariant T cells are depleted in melanoma and show an exhausted profile with increased expression of PD1: a prospective cohort study"

Manipulation of the immune response invoked by melanoma by the CTLA-4 inhibitor (Ipilimumab) lead to the development of one of the first effective treatments for melanoma. Ipilimumab blocks the inhibitory receptor CTLA-4 expressed on cytotoxic T cells activated by antigen presenting cells, facilitating their function to destroy melanoma cells. Less is known of innate cytotoxic T cells in the setting of melanoma including invariant NKT cells (iNKT), Mucosal Associated Invariant T cells (MAIT) and gamma/delta T cells. We undertook a prospective study of circulating iNKT, MAIT and gamma/delta T cells in patients diagnosed with melanoma.

Following ethical approval, patients undergoing excision of a suspicious pigmented lesion were invited to take part.Controls were also recruited who did not have melanoma or dysplastic lesions. Blood samples were drawn and peripheral blood monocytes extracted and analyzed at the Institute of Molecular Medicine, Trinity College Dublin by flow cytometry. Samples were analyzed for numbers of the following CD3, CD8, MAIT, NK, INKT, gamma delta T cells. We also looked at the activation and exhaustion profiles of all of these subsets. Differences between patients diagnosed with melanoma and dysplastic lesions and controls were analyzed using Graph Pad Prism (Mann-Whitney Test for non-parametric data ($p < 0.05$)

In total 8 patients were recruited, six diagnosed with melanoma and two with dysplastic naevi and 15 controls. Circulating MAIT cells were significantly reduced in patients with dysplastic lesions compared to controls (2.1% vs 2.5%, $p = 0.04$). Levels of NK, iNKT and gamma/delta T cells were similar among both cohorts. Circulating MAIT cells had significantly increased surface expression of PD1 in patients with melanoma/dysplastic lesions compared to controls (55% vs 11%, $p < 0.0001$) and significantly increased expression of TIMMAIT- PD1 (55% vs 10%, $p < 0.0001$).

Our results indicate defects in the innate immune system of patients with early stage melanoma. Reduced circulating MAIT cells have previously being described in patients with mucosal- associated cancer and our study is the first to describe this phenomenon in melanoma. Therapeutic blockade of PD-1 is one of the most effective therapeutic strategies in metastatic melanoma and our results indicate that blockade may have protective effects on innate immune cells.

Burrows Cup Spring 2017 - Runner-up

Burrows Cup Presentation, Spring 2017 Runner-up abstract

Missed Opportunities for Melanoma Detection in Secondary Care

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3. Department of Histopathology, Cork University Hospital, Cork
4. Department of Medical Oncology, Bon Secours Hospital, Cork

Introduction: Early detection of melanoma is associated with improved survival.[1,2] There has been significant emphasis on the role of general practitioners in melanoma screening strategies.[3-6] However, the role of secondary care providers in the detection of melanoma has been rarely explored.

Aim: To identify inpatient and outpatient episodes in patients with intermediate and thick melanomas in the 5 years and 1 year prior to their diagnosis.

Methods: A multicentre, retrospective case review was conducted at Cork University Hospital, South Infirmary Victoria University Hospital Cork, Mercy University Hospital Cork, Bon Secours Hospital Cork and University Hospital Kerry. Databases at the five hospitals were reviewed. All patients with a Cork/Kerry address with primary cutaneous melanomas of greater than or equal to 1mm Breslow depth from January 2013 to December 2014 diagnosed or reviewed by CUH pathology department were included. Data from the patient record enquiry for the 5 years prior to diagnosis was collected for each patient at each clinical site. This included inpatient admissions, day case admissions, outpatient clinics and emergency department attendances.

Results: 106 patients were included with a mean age of 63 years. The median Breslow depth was 2.3mm. 32 (30%) of the melanomas were located on the head/neck region. Of the 106 patients, 67% (n=71) had a secondary care interaction in the 5 years prior to their melanoma diagnosis and 42.5% (n=45) in the year prior to diagnosis. Most of these hospital encounters were in the outpatient clinic (57.5%), but almost one third (31%) had an inpatient admission in the five years prior to diagnosis and 10%(n=11) in the year prior to diagnosis.
The three specialties with the most interactions in the year prior to diagnosis were ophthalmology (n=20), orthopaedics (n=18) and emergency medicine (n=16).

Discussion: A significant opportunity exists to improve early detection of intermediate and thick melanomas in secondary care. Patients with intermediate and thick melanomas are being seen in secondary care facilities in the years prior to their diagnosis. Education and awareness campaigns directed at secondary care providers should be implemented to encourage them to perform skin assessment as part of clinical examination.

Spring 2017 - Poster Winner

Spring posters 2017 1st prize winner abstract

An Audit of Compliance with TB Screening Procedures prior to treatment of Psoriasis with Biologic.

Dr. Aine Kelly, Dr. Aoife Lally, Prof. Brian Kirby
St. Vincent's University Hospital, Dublin.

The BAD guidelines for treatment of psoriasis with anti- TNF, IL 12/23 and IL17 recommend screening for latent TB. It is common practice in Dermatology centres to perform both a chest Xray and an Interferon Gamma Release Assay (IGRA) at the time of screening. The IGRA is aproximately 96% sensitive and 99% specific for the diagnosis of latent TB. A chest X-ray is approximately 73-79% sensitive and 60 -63% specific.
We assessed clinician compliance with TB screening in 107 psoriasis patients prior to commencing biologic therapy. We used the BAD guidelines on biologic therapy in psoriasis as our audit standard. We also looked at the incidence of latent TB, incidence of positive chest X ray results in those who were negative for latent TB (as per respiratory physician) and the consequence of these positive findings.
Of the one hundred and seven patients, the charts were unavailable for six. Thirty patients had a tuberculin skin test and seventy-one patients had IGRAs. Eleven patients (10.2 %) had documented TB risk factors. Five patients (4.6%) had a positive TST or IGRA (4 TST and one IGRA). All five patients with a positive TST or IGRA received chemoprophylaxis for latent TB. Sixteen patients (15 %) had granulomas of unknown significance on chest X-ray. Ten of these sixteen patients were referred to respiratory medicine for an opinion. Twelve of these sixteen patients went on to have further imaging and procedures.
Compliance with tuberculosis screening with chest X-ray and IGRA was one hundred per percent at our hospital. Compliance with documentation of TB risk factors was poor at 10.2%. Five percent of psoriasis patients screened were diagnosed with latent TB. There was no diagnosis of latent TB made from a positive chest X-ray finding. All latent TB diagnoses were based on a TST or IGRA positivity. There were seven C.T. thoraces, bronchoscopies and repeat chest X-rays done due to a false positive chest X-ray. No additional diagnoses of latent TB were made after these procedures.
The audit suggests that chest X-ray is both insensitive and non- specific as a screening tool for latent TB. In our study, chest X-ray screening led to unnecessary expensive tests that are potentially hazardous. Further studies should be performed to delineate the safest and most cost efficient screening algorithm for TB infection prior to biologic therapy.

STORMONT CASTLE - SPRING 2017