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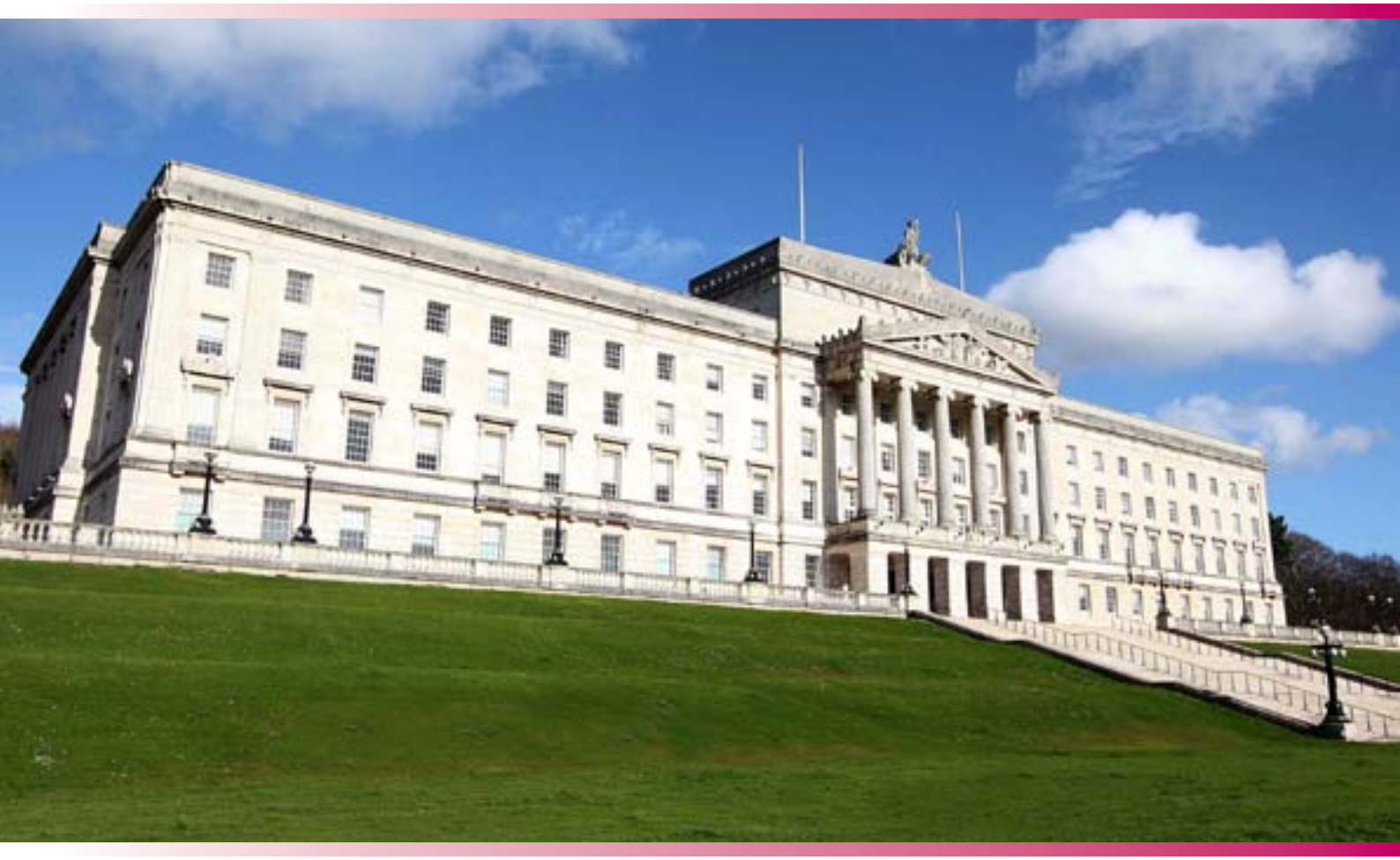

IAD
IRISH ASSOCIATION OF DERMATOLOGISTS

SPRING MEETING

Thursday 27th, Friday 28th & Saturday 29th April 2017

Stormont Hotel, Belfast & Belfast City Hospital

Welcome Message from the President Dr Kevin McKenna

Welcome to the Spring meeting of the IAD 2017 at the Stormont Hotel, Belfast and Belfast City Hospital. It gives me particular personal pleasure to host this meeting in my home city and hospital. I hope you find the meeting both stimulating and educational.

The theme of this meeting is malignant melanoma. We have the Thursday afternoon dedicated to a Dermoscopy session under the direction of Professor Colin Fleming. Guest speakers for the Friday include Professor Julia Newton-Bishop, Professor Catherine Harwood, Dr Veronique Bataille and Dr Judith Carser. We look forward to the presentations from these international leaders in the field of melanoma.

Our junior colleagues will have the opportunity to compete for the prestigious Burrows Cup at the Registrars Symposium. I would like to thank our Scientific Committee for all their hard work towards organising the programme for this meeting.

I would like to take the opportunity to thank our secretary Dr Art O'Hagan and Jacqui Carroll for all their hard work and support. Special thanks to Dr Gillian Gibson who is stepping down a treasurer for keeping our finances in such good shape.

I hope you enjoy the meeting and our opportunity to visit Parliament Buildings, Stormont for our IAD Conference Dinner.

Yours sincerely,

Dr Kevin McKenna
President
Irish Association of Dermatologists

IAD Past Presidents

1965/7 Dr R. Hall, Belfast,
who was followed by:
1967/9 Dr D.O'C Donelan
1969/71 Dr J.M. Beare
1971/3 Dr D.M. Mitchell
1973/5 Dr D.B. Buckley
1975/7 Prof D. Burrows
1977/9 Dr F.O.C. Meenam
1979/81 Dr Agnese M.T. Kelly
1981/3 Dr Count H. Viani
1983/5 Dr Grace Allen
1985/7 Dr Marjory Young
1987/9 Dr Roddy Matthews
1989/91 Dr David O'Gorman
1991/3 Dr Rory Corbett
1993/5 Prof Sarah Rogers
1995/7 Dr E.A. Bingham
1997-9 Dr. Fergus Lyons
1999-01 Dr Clifford McMillan
2001-3 Prof Frank Powell
2003-5 Dr Raymond Fulton
2005-7 Prof Louise Barnes
2007-9 Dr Hilary Jenkinson
2009-11 Dr Gillian Murphy
2011-13 Dr Pat Podmore
2013-2015 Dr Rosemarie Watson
2015-Present Dr Kevin McKenna

IAD Irish Association of Dermatologists

GALDERMA

Galderma Sponsored Symposium

Stormont Hotel, Belfast
Thursday 27th April 2017

5.30pm	Registration & Light refreshments
6.30pm	Welcome *Chaired by: Dr David Alderdice* *Consultant Dermatologist, Ulster Hospital Dundonald, Belfast*
6.40pm	Acne management in an era of antibiotic stewardship *Dr Sandra Minor* *Consultant Dermatologist, St Richards Hospital, Chichester*
7.10pm	Rosacea management in an era of antibiotic Stewardship *Dr Geraldine Morrow* *Consultant Dermatologist, Beacon Hospital, Dublin*
7.40pm	Daylight PDT: "A walk in the park?" *Dr Sandra Minor* *Consultant Dermatologist, St Richards Hospital, Chichester*
8.10pm	Questions & Discussion
8.30pm	Close & Fork Supper

This meeting has been sponsored by Galderma (UK) Ltd

IAD
Irish Association of Dermatologists

SPRING MEETING

Thursday 27th, Friday 28th & Saturday 29th April 2017
Stormont Hotel, Belfast & Belfast City Hospital

THURSDAY 27th APRIL: Dermoscopy Session

Prof Colin Fleming, Consultant Dermatologist, Clinical Director. Specialist Services, Oncology, Haematology and Renal. Honorary Reader, Ninewells Hospital and Medical School Dundee.

2.00pm	Quiz
2.25pm	Refresher; key concepts
2.50pm	Update on terminology
3.20pm	Coffee
3.35pm	Real life workshop
4.00pm	Lentigo maligna
4.25pm	Acral dermoscopy
4.45pm	Unusual melanomas workshop
5.05pm	Last man stands
5.15pm	Finish

Galderma Sponsored Symposium – Theme 'ACNE'

5.30pm	Registration & Light Refreshments
6.30-8.30pm	Galderma Symposium
8.30pm	Fork Supper

FRIDAY 28th APRIL: IAD Spring Meeting – Theme 'Melanoma'

8.00am	**Registration**
9.00-10.30am	**Registrars' Symposium – Burrows Cup**
10.15-11.15am	**Exhibition**
11.15am-12.00pm	**Prof Julia Newton-Bishop, Professor of Dermatology, University of Leeds** *"What determines survival from melanoma?"*
12.00-12.45pm	**Prof Catherine Harwood, Consultant Dermatologist, Barts and the London School of Medicine & Dentistry** *"Melanoma and immunosuppression"*
12.45-2.15pm	**Exhibition**
2.15-3.00pm	**Dr Veronique Bataille, Consultant Dermatologist, West Hertfordshire Trust** *"Melanoma susceptibility, what is the trade off?"*
3.00-3.45pm	**Dr Judith Carser, Consultant Medical Oncologist, Cancer Centre, Belfast Health & Social Care Trust** *"Current Systemic therapies in metastatic melanoma"*
3.45-5.00pm	**Exhibition**
4.30pm	**Presentation of Burrows Cup & Poster Prizes**
5.00-6.00pm	**IAD Business Meeting**
7.30pm	**IAD CONFERENCE DINNER,** Parliament Buildings, Stormont Estate

SATURDAY 29th APRIL 2017: Clinical Meeting
Belfast City Hospital, Out Patients Department

Hosted By: Dr Suzanne Clements, Dr Andrea Corry, Dr Helen Hunter, Dr Olga Kerr, Dr Collette McCourt, Dr Kevin McKenna.

8.30am	Patients arrive
9.00am	Review of Patients
10.15am	Coffee
10.45am	Discussion of Cases
12.15pm	Lunch

Biographical Sketches

Prof Colin Fleming

Prof Colin Fleming is a Consultant Dermatologist, Mohs' Surgeon, Honorary Reader and Clinical Director in the dermatology department at Ninewells Hospital and Medical School, Dundee. He was an undergraduate at Glasgow University where he also studied for a BSc(Honours) in Immunology, and subsequently developed an interest in skin cancer research and treatment through working in Australia, Glasgow and Lisbon.

He has been a Consultant in Tayside since 1999 and has set up multiple services for skin cancer patients, including the NOSCAN Macmillan Mohs service. He has research interests in diagnosis and treatment of skin cancer, and has over 80 publications in skin cancer, skin surgery and general dermatology. He has been a leading proponent of dermoscopy in the UK for the last 20 years, and has taught dermoscopy in numerous courses and lectures. He is a ex-board member of the European Association of Dermato-Oncology, a fellow of the American Society for Mohs Surgery, and former President of the British Society of Dermatological Surgery.

Dr Sandra Minor

Dr Sandra Minor graduated from Queen's University, Belfast and subsequently trained in dermatology in Belfast, St John's Institute, London and Portland, Oregon. Soon after gaining a consultant post in Antrim she moved to Carson City ,Nevada where she lived and worked for 18 years. She returned to UK in 2001 and since then has worked at St Richard's hospital,
Chichester and in private practice.

Dr Geraldine Morrow

Dr. Geraldine Morrow is a Consultant Dermatologist at Beacon Hospital, Dublin. She is a Graduate of UCD in 1985. She trained in Dermatology at the Mater Hospital and St. Anne's Skin and Cancer Hospital, Dublin. Her special areas of interest include Acne, Rosacea and Occupational Dermatology. She has lectured in Dermatology in UCD on the Higher Diploma courses in Occupational Health and the Masters in Sports Medicine and has also examined on these courses. She also lectures regularly for ICGP CME groups.

Professor Julia Newton-Bishop

Professor Julia Newton-Bishop is a dermatologist who worked for many years (till 2015) in the Leeds Specialist Melanoma Multidisciplinary Team which manages poor prognosis melanoma for the Yorkshire region of the UK. She is also a clinician scientist and Professor of Dermatology at the University of Leeds, UK. She leads the melanoma research group within the Section of Epidemiology and Biostatistics, Leeds Institute of Cancer and Pathology, University of Leeds, lead by Tim Bishop. The research group uses genetics to understand susceptibility to melanoma and survival from melanoma and for the last year Julia has been working in melanoma research full time. She is a fellow of the Academy of Medical Sciences in the UK.

Irish Association of Dermatologists Spring Meeting 2017

Biographical Sketches

Dr Catherine Harwood

Catherine Harwood is a Consultant Dermatologist in the Centre for Cell Biology and Cutaneous Research, Blizard Institute, Barts and the London School of Medicine and Dentistry, Queen Mary University of London and Barts Health NHS Trust. She qualified in Medicine from the University of Cambridge and St Thomas's Hospital Medical School, London. Her training in dermatology was at Westminster Hospital, St John's Institute of Dermatology, St George's Hospital and Barts and the London NHS Trust. Her main clinical and research interests are related to skin cancer, particularly in immunosuppressed individuals.

Dr Veronique Bataille

Dr Veronique Bataille trained at the Louvain Medical School in Brussels and graduated in July 1985 with magnum cum laude. She then worked in many teaching hospitals in London and started her dermatology training at St John's Institute of Dermatology at St Thomas Hospital in 1989. She then moved to the Imperial Cancer Research Fund in Holborn and the Royal London Hospital as a clinical research fellow where she completed her PhD on the genetic epidemiology of skin and eye melanoma in 1995 under the supervision of Professors Julia Newton Bishop, Jack Cuzick and Tim Bishop.

Dr Bataille became an accredited consultant dermatologist in 1996. In 1996, she was appointed Senior Lecturer and Honorary Consultant Dermatologist at Barts and the London School of Medicine and Dentistry where she continued her interest in the genetics of melanoma and other skin cancers. Since 2004, Dr Bataille has been working at the West Hertfordshire Trust where she is also providing specialised care for patients with skin cancers where she works with Dr Paul Nathan in the multi-disciplinary melanoma clinic at the Mount Vernon Cancer Centre. Dr Bataille is also in charge of the skin programme at the Twin Research Unit at Kings College London looking at the genetics of common skin diseases.

Dr Bataille has published extensively in many dermatology, genetic and cancer journals over the last 20 years and has presented many abstracts at national and international meetings. She regularly writes reviews and book chapters on skin cancer and reviews manuscripts for many dermatology journals. She is assistant editor for the Acta Dermatologica and Venereologica, European Journal of Cancer, BMJ case reports dermatology and Research Notes for BIOMED Central.

Dr Judith Carser

Dr Judith Carser graduated from Queens University, Belfast and trained in medical oncology in Northern Ireland. Since completing training in 2010 I have taken up consultant posts at Clatterbridge Cancer Centre, Wirral as well as Southern Health & Social Care Trust, Northern Ireland. I currently provide a regional service at the Cancer Centre, Belfast Health & Social Care Trust for melanoma and testicular cancers as well as leading the Trust Acute oncology service.

Sponsors & Date for Your Diary

IAD would like to thank the following sponsors and exhibitors for their generous support

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DATE FOR DIARY...

IAD Autumn Meeting 2017

'INTERNAL MEDICINE'

Thursday 12th & Friday 13th October

Radisson Blu Hotel, Farnham Estate, Cavan

IDNA
Irish Dermatology Nurses Association Ltd.

Friday 28th April 2017
14th IDNA Annual Meeting

8.30- 9.00 am	Registration
9.00- 9.45	IDNA BUSINESS MEETING
9.45-10.30 am	P. COWAN (BHSCT) "DOWN UNDER"
10.30-11.15am	Coffee & Exhibition
11.15am-12.00pm	Carrie Wingfield MOHS SURGERY FOR NURSES
12.00-12.45pm	IAD Bursary Presentations Bernie Finneran " Dermoscopy for Beginners" Eilish Ryan " Phototherapy: Lessons Learnt'
12.45-2.15pm	Lunch & Exhibition
2.15- 3.00 PM	Speaker to be confirmed
3.00-3.45 pm	MICHELE MCCALLUM (BHSCT) DERMATOLOGY WOUNDS CASE STUDIES
3.45-4.30pm	Coffee & Exhibition
4.30pm- 5.00	DR EMMA MACK Development of an Outpatient APP

Registrars' Symposium
Oral Case Presentations Burrows Cup
Friday 28th April 2017

Oral 01. 9.00am
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.

Oral 02. 9.10am
Missed Opportunities for Melanoma Detection in Secondary Care
Authors: C. Quinlan1, S. McCracken2, E.Tierney1, C. Heffron3, J. Fitzgibbon3, C. Murphy2,4, J.F. Bourke1, M. Murphy1,2

Oral 03. 9.20am
Myositis-specific antibodies and immunotype-phenotype correlation in Irish dermatomyositis patients:
Authors: Fatima Awdeh , Rebecca Hellen , Qamar Rhazali , Marina O'Kane.
Instutution: Beaumont Hospital

Oral 04. 9.30am
Photoprotective behaviours in an Irish at risk Inflammatory Bowel Disease Population
C. Gallagher1+3, A. Ridge2, D. Kevans2, D. McNamara3, AM Tobin1
Dermatology Department Tallaght Hospital. Gastroenterology Department St James's Hospital/ Trinity Academic Gastroenterology Group

Oral 05. 9.40am
Hidradenitis suppurativa is driven by insulin resistance rather than hyperandrogenism in the setting of polycystic ovarian syndrome
S Kirthi1, M Connolly1, C Gallagher1 ,LA Behan2, J Gibney2, AM Tobin1
1.Department of Dermatology, Tallaght Hospital
2.Department of Endocrinology, Tallaght Hospital

Oral 06. 9.50am
Medication adherence among psoriasis patients on systemic and biologic treatment
Roisin Hambly,1 Aine Kelly,1 Eimear Gilhooley,1 Eilis Nic Dhonncha,1 Aizuri Murad,1 Rosalind Hughes,1 Aoife Lally,1,2 Brian Kirby.1,2
1. The Charles Centre, Department of Dermatology, St Vincent's University Hospital, Elm Park, Dublin.
2. University College Dublin School of Medicine and Medical Sciences, Dublin.

Oral 07. 10.00am
A re-evaluation of teenage sunbed use following the introduction of banning legislation for under 18 year olds.
Stephanie Menzies, Selene Daly, Miriam Fitzgerald, Dermot McKenna.
Department of Dermatology, Sligo University Hospital

Orals - Abstracts

Oral 01. 9.00am

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.

Oral 02. 9.10am

Missed Opportunities for Melanoma Detection in Secondary Care

Authors: C. Quinlan1, S. McCracken2, E.Tierney1, C. Heffron3, J. Fitzgibbon3, C. Murphy2,4, J.F. Bourke1, M. Murphy1,2

Affiliations:
1. Department of Dermatology, South Infirmary Victoria University Hospital, Cork
2. School of Medicine, University College Cork
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.

References:

1. Marks. R. Prevention and Control of Melanoma; The Public Health Approach. Ca Cancer J Clin. 1996:46; 199-216

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2. Schuchter. L. et al. A prognostic model for predicting 10-year survival in patients with primary melanoma. Annals of Internal medicine. 1996;125(5):369-75
3. Baade. P.D. et al The relationship between melanoma thickness and time to diagnosis in a large population-based study. Archives of dermatology. 2006;142(11):1422-7
4. Richard. M.A. et al Delays in diagnosis and melanoma prognosis (II):the role of doctors. International journal of cancer. 2000;89(3):280-5
5. Argenziano. G. et al. Total body skin examination for skin cancer screening in patients with focused symptoms. J Am Acad Dermatol. 2012;66(2):212-9
6. Aitken. J.F. et al. Clinical whole body skin examination reduces the incidence of thick melanomas. International journal of cancer. 2010;126(2):450-8

Oral 03. 9.20am

Myositis-specific antibodies and immunotype-phenotype correlation in Irish dermatomyositis patients

Authors: Fatima Awdeh , Rebecca Hellen , Qamar Rhazali , Marina O'Kane. Instutution: Beaumont Hospital

Myositis autoantibodies are categorized as either myositis-specific antibodies (MSA); or myositis-associated antibodies (MAA; mostly occurring in myositis-overlap syndromes). There is growing interest in the prognostic role of MSA in dermatomyositis (DM)1. In addition newer immune targets for MSAs have been identified in distinct phenotypes of DM1. Current understanding of their significance in dermatomyositis is incomplete due to small numbers of studied patients. This pilot is part of a larger study aiming to evaluate relationships between clinical phenotype, MSA serotypes and prognosis, in Irish DM patients.

Patients with DM attending dermatology at a university hospital were identified from a database. We included patients aged 18 and over with diagnosis of DM, meeting Bohan and Peter criteria for DM or Sontheimer's criteria for clinically amyopathic DM (CADM). Chart review, extended MSA panel, and skin examination were performed at subsequent review appointments. Clinical data was collected as part of routine medical care; all were examined by a consultant dermatologist for the presence/ absence of 13 cutaneous signs, to confirm documented or emerging clinical phenotype. Cutaneous signs were divided into those specific to DM (eg Gottrens papules/ sign, heliotrope) and those occurring in DM but not specific to the disorder. Extended MSA panel included anti-Mi-2 (directed against chromatin remodeler enzyme, Mi-2), anti-transcription intermediary factor-1gamma (TIF-1gamma), anti-small ubiquitin like modifier enzyme (SAE), anti- nuclear matrix protein-2 (NXP-2) and anti-melanoma differentiation associated gene-5 (MDA-5).

Evaluation is complete in 14 patients (2 men,12 women, mean age 60). 11/14 patients had DM; 3/14 had CADM. Three DM patients had positive MSAs; no CADM patient had positive MSAs. The most frequently occurring MSAs were anti-TIF-1γ and NXP-2, accounting for 21% of antibody positive patients (7% anti–NXP-2; 14% anti–TIF-1γ). The patients positive for anti–TIF-1**γ** and/or anti–NXP-2 did not express any other MSA or MAA; 2 patients expressed both antibodies. The two patients with classical DM who were anti–TIF-1γ positive both had small cell lung cancer. Patients with anti-TIF-1γ antibodies had severe and extensive cutaneous involvement in addition to typical DM features. Despite small numbers in this pilot study, the above findings are consistent with recent MSA literature 1 Dermatomyositis is rare. Extended MSA panel testing in the assessment of all patients presenting with DM may facilitate better understanding of immunotype-phenotype correlations and prognostic implications.

(These are interim results)

1 Daly ML, Gordon PA, Creamer D. Cutaneous Features of Dermatomyositis associated with Myositis Specific Antibodies. Br J Dermatol 2016 DOI: 10.1111/bjd.15020

Oral 04. 9.30am

Photoprotective behaviours in an Irish at risk Inflammatory Bowel Disease Population

C. Gallagher1+3, A. Ridge2, D. Kevans2, D. McNamara3, AM Tobin1 Dermatology Department Tallaght Hospital. Gastroenterology Department St James's Hospital/ Trinity Academic Gastroenterology Group

Azathioprine and TNF-α inhibitors are widely used immunosuppressants in Inflammatory Bowel Disease (IBD). It has been reported that such treatments increase the risk of developing all types of skin cancer. Patients on combination therapy have been shown to have up to five times the relative risk of non- melanoma skin cancer. The British Association of Dermatology (BAD) has published preventative guidelines for patients on immunosuppression, but specific gastroenterology recommendations are lacking.
Our aims were to examine skin cancer risk factors, attitudes towards sun exposure and preventative strategies adopted in an Irish at risk IBD cohort.

A prospective pilot cohort study. Following ethical approval and informed consent, a self-assessment questionnaire was given out to patients attending our IBD clinic over a twelve week period. Clinical data was recorded including diagnosis, immunosuppressants, skin cancer risk factors and photoprotective behaviours.
To date, 244 patients completed the questionnaire. Patients were excluded as follows; 23 (9%) with indeterminate colitis or an unconfirmed diagnosis and 79 (32%) not on azathioprine or TNF-inhibitors.

The majority of patients were a high risk phenotype with light coloured eyes (51%(n=73)), ›30 freckles (52% (n=75)), fair skin (50% (n=71)), or had blonde/red hair (31% (n=45)). Of interest, ten (7%) patients had a personal history and 15 (10%) gave a family history of any type of skin cancer. With regard to other risk factors; 54 (38%) had previous blistering sunburn, 31 (22%) used sunbeds and 37 (26%) worked outdoors.

With reference to BAD recommended preventative measures, the majority of our cohort (63% (n=90)) wore sun cream, but failed to take other important measures; staying in the shade at high risk times (59%(n=84)), re-applying sun cream every 2 hours (50%(n=71)), and wearing a hat (26% (n=38)).
In addition, while 51% (n=73) knew what changes to look for in a suspicious mole, only 43% (n=62) performed regular self-skin checks.

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Our pilot study highlights gaps in our at risk IBD cohort's education regarding skin cancer risk and prevention associated with immunosuppression therapy and warrants further investigation. Ideally educational interventions to enhance patient awareness should be undertaken and assessed.

Oral 05. 9.40am
Hidradenitis suppurativa is driven by insulin resistance rather than hyperandrogenism in the setting of polycystic ovarian syndrome
S Kirthi1, M Connolly1, C Gallagher1 ,LA Behan2, J Gibney2, AM Tobin1
1.Department of Dermatology, Tallaght Hospital
2.Department of Endocrinology, Tallaght Hospital

Studies have documented higher rates of polycystic ovarian syndrome in patients with HS, suggesting that hyperandrogenism may drive HS. Polycystic ovarian syndrome is also associated with insulin resistance and in study of our patients with HS, approximately 78% of female patients were insulin resistant. In order to elucidate the role of insulin - resistance and hyperandrogenism in patients with PCOS and HS, we carried out a prospective cohort study in our outpatient department.

Following ethical approval, successive female patients attending the HS clinic were screened for clinical signs of polycystic ovarian syndrome (acne, hirsutism, androgenetic alopecia) and fasting blood samples were drawn for measurement of testosterone, dihydroepiandrostenedione (DHEAS), androstenedione and sex hormone binding globulin (SHBG). Insulin and glucose were also measured to generate the HOMA-IR as a measure of insulin resistance. Patients diagnosed with HS and PCOS according to the Rotterdam criteria were compared to HS only patients with age (+/- 3 years) and BMI (+/- 3 kg/m2) matched, and also patients with PCOS only, recruited from the endocrinology outpatient department. Data were analysed using GraphPad Prism, a p value ‹ 0.05 was considered significant. Fifteen patients diagnosed with HS and PCOS (mean age 30, mean Hurley stage 2, mean BMI 35.6 kg/m2) were compared with 30 patients with HS (mean age 31 years, mean Hurley 2, mean BMI 34) who had no signs of PCOS. Patients with HS and PCOS had significantly higher HOMA-IR compared to their counterparts with HS only (6.3 vs 1.7, p = 0.007). They also had higher levels of testosterone (1.3 nmol/L vs 0.9nmol/L, p = 0.04). Levels of DHEAS, androstenedione and SHBG were similar, p = 0.3, 0.08 and 0.9 respectively. When these patients were compared to patients diagnosed with PCOS without signs of HS, HOMA-IR were 6.3 vs 3.8, p = 0.6. Patients with HS and PCOS had significantly lower levels of testosterone than those with PCOS only (1.3nmol/L vs 3.4nmol/L, p = 0.006) and adrostenedione (19.6nmol/L vs 14.1nmol/L , p = 0.03) and similar levels of DHEAS and SHBG (p = 1.0 for both).

Our data suggest that the key driver of HS in patients with PCOS is not hyperandrogenism but insulin resistance. Our study also suggests that hormone measurements in our patients appear superfluous compared to gauging patients' insulin status.

Oral 06. 9.50a
Medication adherence among psoriasis patients on systemic and biologic treatment
Roisin Hambly,1 Aine Kelly,1 Eimear Gilhooley,1 Eilis Nic Dhonncha,1 Aizuri Murad,1 Rosalind Hughes,1 Aoife Lally,1,2 Brian Kirby.1,2
1. The Charles Centre, Department of Dermatology, St Vincent's University Hospital, Elm Park, Dublin.
2. University College Dublin School of Medicine and Medical Sciences, Dublin.

Medication adherence is "the extent to which a patients' behaviour in taking their medication corresponds to agreed recommendations by their healthcare provider". Patients with high adherence to medications have better outcomes. Systemic treatments for psoriasis are expensive and knowledge on adherence would be beneficial.
The aim of our study was to assess adherence to systemic agents in psoriasis and to identify predictors of adherence.

Following ethical approval, validated self-report questionnaires were completed by patients with moderate/severe psoriasis on systemic therapy, including Patient Global Assessment (PtGA), Hospital Anxiety and Depression Scale (HADS), Dermatology Life Quality Index (DLQI) and the 8-item Morisky Medication Adherence Scale (MMAS). There were 106 participants, 59% male and mean age 47.9 years (range 18-80). Thirty-two percent had psoriatic arthritis. Twelve percent were smokers and the mean disease duration was 24 years (range 1-51). Thirteen percent reported that psoriasis was clear; 37% almost clear; 26% mild; 21% moderate, and 3% severe.

The medications were adalimumab (30%), etanercept (23%), methotrexate (20%), fumaric acid esters (18%) and others (9%). Fifty-one percent of participants were also taking prescribed medications for other conditions (mean 3.6, range 1-12). The mean anxiety score on the HADS was 6.3 (range 0-18), the mean depression score was 3.4 (range 0-16) and the mean DLQI was 3.2 (range 0-27).
Fifty percent reported never missing a dose of their psoriasis medication for any reason. The reasons for missing doses included forgetting (49%), unwell (26%), too busy (15%), running out of medication (11%), psoriasis under control (13%), medication too expensive (8%) and side-effects (10%).

Ninety-one percent had taken the most recent dose of their psoriasis medication. Twenty-seven percent reported not taking their medication on one or more occasions over the previous 3 months. Using the MMAS, 76% of participants were classified as high adherers, 17% medium and 7% low adherers. In a study of 1,367 patients with hypertension using the MMAS, 15.9% were high adherers, 52% medium and 32.1% low adherers.

There was no significant difference in adherence based on gender (p=0.105) or age (p=0.146). There was an inverse relationship between adherence and anxiety scores (r=-0.267, p=0.008, n=97) and depression scores (r=-0.217, p=0.033, n=97). There was no correlation between adherence and DLQI values (p=0.15, n=97)
Adherence to systemic medication for psoriasis appears higher than for other chronic conditions. High levels of anxiety and depression may be a negative predictor of adherence to these medications.

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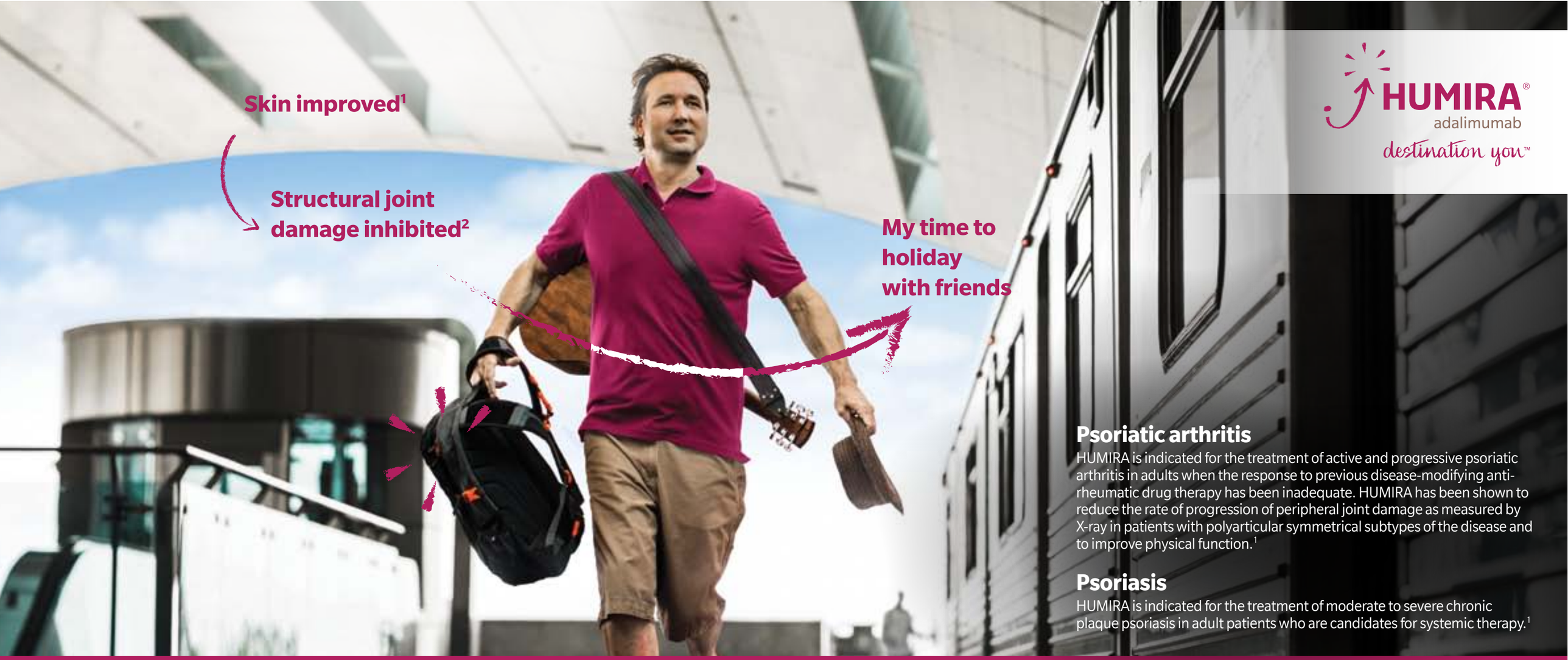

HUMIRA®
adalimumab
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Psoriatic arthritis

HUMIRA is indicated for the treatment of active and progressive psoriatic arthritis in adults when the response to previous disease-modifying anti-rheumatic drug therapy has been inadequate. HUMIRA has been shown to reduce the rate of progression of peripheral joint damage as measured by X-ray in patients with polyarticular symmetrical subtypes of the disease and to improve physical function.[1]

Psoriasis

HUMIRA is indicated for the treatment of moderate to severe chronic plaque psoriasis in adult patients who are candidates for systemic therapy.[1]

Important safety information for HUMIRA: Some patients may not be suitable for HUMIRA. You are strongly advised to read the prescribing information (PI) below.

Humira (adalimumab) 40 mg solution for injection in pre-filled pen or pre-filled syringe or paediatric vial containing 40 mg solution for injection. Refer to Summary of Product Characteristics (SmPC) for full information. Presentation: Each single dose pre-filled pen (0.4 ml), pre-filled syringe (0.4 ml) or vial (0.8 ml) contains 40 mg 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. 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). 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. Psoriasis, 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. 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. 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 the treatment of non-infectious intermediate, posterior and panuveitis in adult patients who have had an inadequate response to corticosteroids, in patients in need of corticosteroid-sparing, or in whom corticosteroid treatment is inappropriate. **Dosage and administration:** A specialist physician experienced in diagnosis and treatment of the indicated condition, to initiate and supervise 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: 40 mg dose every other week. Concomitant MTX should be continued. During monotherapy, patients may require 40 mg each week if they have experienced 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. Reintroduction after 70 day 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: 24 mg/m² body surface area up to 20 mg maximum single dose every other week (see vial SmPC for height/weight dosing chart). pJIA, paediatrics 4-12 years: 24 mg/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: 40 mg every other week regardless of body surface area. ERA, paediatrics 6 years and above: 24 mg/m² body surface area up to 40 mg maximum single dose every other week (see SmPC for height/weight dosing chart). AS, nr-axSpA and PsA, adults: 40 mg every other week. Treatment beyond 12 weeks should be reconsidered if no clinical response in that time. Psoriasis, adults: 80 mg induction dose at week 0, 40 mg 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, the 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/kg body weight (maximum 40 mg/dose) weekly for the first 2 doses 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: 160 mg initially at Day 1 (given as four 40 mg injections in one day or as two 40 mg injections per day for two consecutive days), followed by 80 mg two weeks later at Day 15 (given as two 40 mg 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 in a patient with no improvement 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 40 mg 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. If no improvement after 12 weeks refer to SmPC for guidance. CD, adults: Induction: 80 mg Week 0 and 40 mg at Week 2. For a more rapid response: 160 mg at Week 0 (either as 4 injections in 1 day or 2 injections/day for 2 consecutive days) and 80 mg at Week 2; risk of adverse events higher during induction. Maintenance: 40 mg 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 < 40 kg: Induction: 40 mg Week 0, 20 mg at Week 2. For a more rapid response: 80 mg Week 0 (2 injections in 1 day), 40 mg at Week 2; risk of adverse events higher during induction. Maintenance: 20 mg every other week. If insufficient response, consider 20 mg every week. Treatment beyond 12 weeks should be reconsidered if no clinical response in that time. CD, paediatrics 6 years and above ≥ 40 kg: 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. UC, adults: Induction: 160 mg at Week 0 (as 4 injections in 1 day or 2 injections/day for 2 consecutive days) and 80 mg at Week 2. Maintenance: 40 mg every other week. During maintenance, corticosteroids may be tapered in accordance with clinical guidelines. If insufficient response, consider 40 mg every week. Treatment beyond 8 weeks should be reconsidered if no clinical response in that time. Uveitis, adults: 80mg induction dose at week 0, maintenance dose; 40 mg every other week starting at week 1. Treatment can be initiated in combination with corticosteroids and/or with other non-biologic immunomodulatory agents. Concomitant corticosteroids may be tapered in accordance with clinical practice starting two weeks after initiating treatment with Humira. **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 excipients. **Warnings and precautions:** Clearly record trade name and batch number of administered product to improve traceability of biological products. ***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 latent TB suspected, consult physician with appropriate expertise and follow local treatment recommendations for prophylaxis prior to initiation of Humira. Despite prophylaxis, TB reactivation has occurred on Humira. If active TB is diagnosed, do not initiate treatment. ***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 (surface antigen positive) tested for HBV infection before initiating treatment. HBV carriers should consult a specialist physician and be closely monitored for reactivation of HBV infection throughout therapy and for several months following termination of treatment. 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. Rare association with new onset or exacerbation of symptoms and/or radiographic evidence of central and peripheral demyelinating disease. Discontinuation of treatment should be considered if any of these disorders develop. Neurologic evaluation should be performed in patients with non-infectious intermediate uveitis prior to initiation of treatment and regularly during treatment, to assess for 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 lymphomas and leukaemia, in all patients, including paediatric patients, treated with TNF antagonists. Examine all patients, especially those with a medical history of extensive immunosuppressant or PUVA treatment, for non-melanoma skin cancer prior to and during treatment; caution in COPD patients, and in patients with increased risk for 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 of developing dysplasia or colon cancer is unknown. Patients with UC, history of dysplasia or colon carcinoma to be screened for dysplasia before 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 with 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 double- stranded DNA. ***Surgery:*** Consider the long half-life of Humira for planned surgical procedures. 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 patients:*** Serious infections were higher in patients over 65 years of age, some of whom had a fatal outcome. Consider risk of infections. **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 age to use adequate contraception, and continue its use for at least 5 months after the last treatment. Women must not breast feed for at least 5 months after the last treatment. **Side effects:** *Very common ≥ 1/10*: Infections, leucopaenia, anaemia, lipids increased, headache, abdominal pain, nausea and vomiting, elevated liver enzymes, rash (including exfoliative rash), musculoskeletal pain, injection site reaction. *Common ≥ 1/100 to < 1/10*: skin cancer excluding melanoma (including basal cell carcinoma and squamous cell carcinoma), benign neoplasm, leucocytosis, thrombocytopaenia, hypersensitivity, allergies (including seasonal allergy), hypokalaemia, uric acid increased, blood sodium abnormal, hypocalcaemia, hyperglycaemia, hypophosphatemia, dehydration, mood alterations (including depression), anxiety, insomnia, paraesthesias, migraine, nerve root compression, visual impairment, conjunctivitis, blepharitis, eye swelling, vertigo, tachycardia, hypertension, flushing, haematoma, asthma, dyspnoea, cough, GI haemorrhage, dyspepsia, gastroesophageal reflux disease, sicca syndrome, worsening or new onset of psoriasis (including palmoplantar pustular psoriasis), urticaria, bruising (including purpura), dermatitis (including eczema), onychoclasis, hyperhidrosis, alopecia, pruritus, muscle spasms (including blood creatine phosphokinase increased), renal impairment, haematuria, chest pain, oedema, pyrexia, coagulation and bleeding disorders, autoantibody test positive, blood lactate dehydrogenase increased, impaired healing. **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, Stevens-Johnson syndrome, angioedema, anaphylaxis, sarcoidosis, hepatitis, liver failure and worsening of symptoms of dermatomyositis. **Other less common and rarely reported side effects are listed in the SmPC. Basic NHS price:** £704.28 (for 2 pens or 2 syringes or 2 vials). **Legal category:** POM. **Marketing Authorisation numbers**: EU/1/03/256/001, EU/1/03/256/013, EU/1/03/256/017. **Further information**: available from AbbVie Ltd., Maidenhead, SL6 4UB, United Kingdom. **Date of revision of PI**: December 2016, PI/Humira(combined)/37.

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 AbbVie on ukadverseevents@abbvie.com

For the Republic of Ireland adverse events should be reported to HPRA Pharmacovigilance, Earlsfort Tce, Dublin 2. Tel. +353 16764971; Fax +353 16762517; Email medsafety@hpra.ie. Adverse events should also be reported to AbbVie on IREpharmacovigilance@abbvie.com

References: 1. Humira Summary of Product Characteristics, AbbVie Limited **2.** Mease. P.J. et al Adalimumab for the treatment of patients with moderately to severely active psoriatic arthritis. 2005 *Arthritis and Rheumatism*, 52, 3279-3289.

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Oral 07. 10.00am

A re-evaluation of teenage sunbed use following the introduction of banning legislation for under 18 year olds.

Stephanie Menzies, Selene Daly, Miriam Fitzgerald, Dermot McKenna. Department of Dermatology, Sligo University Hospital

Indoor tanning is associated with an increased risk of skin cancer. The risk is higher in frequent users and in those using sunbeds at a younger age. In a previous study of Irish teenagers, we showed that 7.5% of respondents had used a sunbed with a higher rate in Dublin (11.2%). In 2014, legislation was introduced in Ireland to ban the use of sunbeds in teenagers <18 years old1.

The purpose of the present study was to reassess sunbed usage among teenagers 14-18 years old two years after the introduction of the ban. We assessed (i) sunbed usage rates; (ii) sunburn history; (iii) sun-protection habits; (iv) attitudes towards sun and sunbed exposure. The same secondary schools as before were visited and the teenagers completed an anonymous questionnaire. Numbers completing the questionnaire, age and male/female ratio were similar to the original study.

The results showed that more teenagers are using sunbeds following the ban (8.8%). Teenagers in Dublin continue to have a higher rate of sunbed use (14%) compared to other regions. After the ban there was an increase in sunbed usage in tanning shops (pre-ban, 21%; post-ban, 54%) and at home (pre-ban, 18%; post-ban, 41%). Only a minority of teenagers report being consented prior to treatment (pre-ban, 9%; post-ban, 15%). The majority did not wear eye protection (65%), resulting in eye problems in 11%. In the present study, 50% had experienced burning due to sunbed use, most frequently occurring on the face (33%) and chest (22%). A minority (2%) of teenagers used Melanotan.

Sunburn due to ambient exposure was reported by 91% of teenagers with 43% experiencing ≥5 burns. Sunscreen was seldom used at home (34%) compared to when holidaying abroad (84%). The majority of teenagers believe that a tan looks healthy (66%) and makes you look more attractive (67%). Conversely, the majority also acknowledged that tanning is harmful (82%), dangerous (72%) and associated with wrinkles (74%).

Our findings show that there has been no reduction of sunbed use in teenagers following the introduction of legislation banning its use in under 18's. This may be due to lack of enforcement of legislation, in addition to an increasing trend for sunbed use at home.

References:

1. Fitzgerald et al. Ambient and sunbed ultraviolet radiation exposure: exposure rates, protection habits and attitudes of Irish teenagers aged 14-18 years before introduction of national sunbed legislation. Br J Dermatol (2015) 173(Suppl. S1), pp3-10

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Referral of invasive melanoma to a Melanoma Multidisciplinary Team Meeting in Cork University Hospital
R. O'Connor1, C.M.R Fahy2, S. McCarthy1, J. Bourke1, M. Murphy1
1Department of Dermatology - South Infirmary Victoria University Hospital Cork
2Department of Dermatology – Royal United Hospital NHS Foundation Trust, Bath, UK

Poster Presentations

Poster 1

Introducing a Dermatology Handover system within the Belfast and South Eastern Health and Social Care Trust.

Skillen LA, Brennan R, O'Kane D, Hoey S.
Department of Dermatology, Belfast Health and Social Care Trust.

Background: Handover of care is one of the most perilous procedures in medicine, and when carried out improperly can be a major contributory factor to subsequent error and harm to patients. Handover theory to date has been centred round hospital at night care for acute specialties. Dermatology as a specialty involves complex chronic disease processes and acute emergency presentations, therefore requiring a handover system that allows for this.

Within the trusts, there are 8 registrars providing 24 hour on call to 6 hospitals. This provides a challenge for patient review and follow up arrangements. Handover within dermatology had been informal with phone call or an informal chat to handover patient care. This was flagged by the GMC as an issue needing addressed within the department.

Aim: To introduce a specialty specific, time efficient handover system for the dermatology on call service within the Belfast/ South Eastern Trusts within 4 months.
Method: An electronic survey questionnaire was sent to all CMTs, SPRs and Consultants involved in out of hour cover to get baseline data on how the current system worked and what improvements could be made. 44% of staff had been in a situation on call that could have been avoided through better communication between medical staff.

Changes made to the handover process include introduction of formal handover meetings, a template was created to allow access to "at risk" patients available to all on call staff through PDSA cycles. This was approved by the trusts available through a shared computer drive to ensure data protection.

A further electronic survey was sent out to review the implementation of the new handover procedures.

Results: 86% of staff preferred the new system that was introduced. 88% of the weekly handover were updated. Only 27% of updated emails were sent out on a Monday after on call weekend. Access to the shared drive to view and update the handover template was 77%.

Discussion: The new handover system has proved to be efficient and favoured by the medical staff involved in handover. Figures show that the new system is working. Improvements can be made in access to the shared drive for staff rotating into the department and ensuring that the update template is emailed to all staff after weekend on call. Effective communication lies at the heart of good medical practice. This Quality Improvement project shows that small changes can improve communication and efficiency.

Poster 02

A review of all patients recruited to BADBIR prior to 2014 in the Belfast Trust.

Skillen LA, McKenna K.
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Background: The British Association of Dermatologists' Biologic Interventions Register (BADBIR) was set up to follow up all patients receiving biologic therapy for psoriasis in the UK and ROI. Its aim is to investigate the long-term safety for patients treated with biologic agents, compared with conventional systemic therapy. The Belfast Trust has been recruiting patients since 2010.

Aim: To review all patients recruited to BADBIR until 31/12/2013 with an aim to determine key demographics in our local cohort of biologic patients and compare to national and international data. Also to assess baseline disease severity, response to biologic therapy with PASI 75 and compare to UK data and international data.

Method: A retrospective review of data for patients recruited to BADBIR prior to 31/12/13. This included BADBIR review, and medical chart review.

Results: 93 patients were identified. 62% were male, 38% female. Mean age was 48.5yrs. 30% of patients suffered from hypertension, 16% hypercholesterolaemia, 4% T2 Diabetes, 3% IHD, 19% Psoriatic Arthropathy and 20% depression. 84% were on adalimumab, 7% etanercept and 9% ustekinumab. 66.25% achieved PASI 75 at 3 months. 77% achieved PASI 75 at 6 months. There were no serious adverse events.

Discussion: Psoriasis affects 2-3% of the UK population. A small proportion of those patients require systemic and biological therapy to control their disease. Recruiting patients to BADBIR offers a system by which population data and outcomes can be collected, analysed and then compared with UK and international data.

Poster 03

The development of lentigines in chronic plaque psoriasis

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Psoriasis vulgaris is a common T cell mediated cutaneous disorder. In 20% of patients it follows a severe, recalcitrant course. Previous studies have reported the development of lentigines as an unusual form of post inflammatory hyperpigmentation in psoriasis.(1). It has been suggested that the proposed mechanism of this pigmentation is an abnormal reaction to UV light (2,3). There are reports of lentigines arising within psoriatic plaques in the absence of phototherapy. We report a case series [n=12] of patients with longstanding severe chronic plaque psoriasis.

A retrospective study was carried out on twelve patients over a three month period. The study was approved by the Medical

Poster Presentations

Research Ethics Committee of St Vincent's University Hospital. Written informed consent was obtained. Selected patients who were found to have lentigines confined to post inflammatory psoriatic plaques were asked to participate. The lesions were photographed and punch biopsies of the lentigines were taken. The specimens were stained by Haematoxylin and eosin and analyzed by a consultant histopathologist. Histology showed increased melanocyte proliferation with pigment incontinence in the dermal epidermal junction.

We describe the development of lentigines within resolving psoriasis plaques in twelve patients with severe disease. There are only three reports of this to date in the literature. Similar to our study, all had severe psoriasis of long duration that required systemic therapy. Histology was not however, performed in these case reports. This study was carried out on patients who had not had phototherapy for over ten years, and lentigines were present on non-sun exposed sights in some of our patients. They were also confined to the area of skin affected by the now resolved psoriatic plaque.

Our study suggests that this change is a form of post inflammatory hyperpigmentation. A recent study by Prinz et al in 2015 reports that melanocytes are target cells of the HLA-C*06:02 molecule that mediates T cell responses in psoriasis. We speculate that the formation of these "lentigines" may reflect potential autoimmunity directed against melanocytes in these patients. It would not explain, however, why this phenomenon appears uncommon.

Poster 04

What Is Your Skin Type?

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Introduction: The Fitzpatrick Skin Type Classification is the most commonly used measure of skin type. It is a scale of six skin types based on individuals' self-reported tanning and burning propensities after moderate sun exposure (fig.1). Clinically, it is used to estimate minimum erythema dose for phototherapy. It is also used as a standard for self–assessment of sun sensitivity in questionnaire based surveys.

Objectives:
1. To determine the accuracy of self-reported skin types.
2. To determine whether we could improve patients' estimation of their skin type.

Methods: Data was obtained from 74 patients attending pigmented lesion clinics over a four week period. All patients filled out a questionnaire requiring them to categorize their skin type (I-VI). Group 1 (33 patients) based their decision on their tanning and burning propensities only (Fig1). Group 2 (41 patients) were shown images of individuals with different skin types and their corresponding skin colour, ethnicity, tanning and burning propensities.

Results: Of the 74 patients assessed, only 24 (32%) selected their correct skin type. The correlation between physicians' assessments and self-reported skin types was higher for Group 2 (18/41)(44%) than Group 1 (6/33) (18%). The vast majority of patients (66%) underestimated their skins' sensitivity to the sun.

Conclusion: Patients are not aware of their skin type. The use of additional descriptions and visual aids improved the accuracy of patients' self assessment but the results remain suboptimal. This could have public health implications as self-reported questionnaires are commonly used to collect data. Skin type is an important risk factor for the development of skin cancers. Patients with skin types I- III are at an increased risk of sun damage, melanoma and non-melanoma skin cancers. The majority of patients under-estimated their skins' sensitivity to UV light. This alarming finding highlights the need to increase skin type awareness and the associated UV risk among the Irish population.

Fig.1 Fitzpatrick Skin Types I-VI:
Type I Always burn and never tan
Type II Burn easily and then tan
Type III Tan after initial burn
Type IV Burn minimally, tan easily
Type V Rarely burn, tan darkly
Type VI Never burns, always tan darkly

Poster 05

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.

Poster 06
Laboratory Monitoring during Isotretinoin Treatment of Acne: How much is enough?

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Dermatology Department, Our Lady of Lourdes Hospital Drogheda, Co. Louth, Ireland

Introduction: Isotretinoin is gold standard therapy for severe acne. Due to teratogenicity pregnancy prevention measures are required. Requirement for laboratory monitoring is less clear. The manufacturer's SPC recommends monitoring lipids and liver function at weekly or biweekly intervals until the response to isotretinoin has been established. The European Dermatology Forum recommends monitoring liver enzymes and lipids, before treatment, one month after starting and every three months thereafter. Recent studies including a large meta-analysis propose that monitoring of FBCs is not required and that a baseline LFT and lipid profile and once more during therapy is adequate (1,2). The aim of this study was to establish laboratory monitoring practices in our centre, determine the rate of blood abnormalities and ascertain the clinical consequence of any abnormalities.

Methods: A retrospective review was conducted of 50 patients who received at least one course of isotretinoin for the treatment of acne from January 2015 to January 2017. Patient demographics, laboratory results, interval of testing and isotretinoin dose were recorded.

Results: The mean age was 21yrs (12 -53 yrs), 58% were male. In total, 35 patients (70%) had an FBC measured at baseline, 24 (48%) had at least one FBC measured during treatment. There were no abnormalities in FBC detected in any patient. Lipid abnormalities occurred most frequently, 5 patients (10%) had a lipid abnormality detected at baseline and 14 (28%) developed an abnormality during treatment. No lipid abnormality required isotretinoin adjustment or discontinuation. One patient developed a raised ALT [94 IU/l (normal range: 24 - 68)], which normalized within one month. Four patients (8%) developed a raised GT [mean: 57 IU/l (normal range: 6 – 30)]. Three patients (6%) had an isolated raised fasting bilirubin level [mean: 23 µmol/l (normal range: 8.4 – 20.5)].

Discussion: A standardized approach to laboratory monitoring of isotretinoin would eliminate unnecessary testing and reduce costing. Our study demonstrates that laboratory monitoring of isotretinoin in our department would benefit from rationalisation. Hansen et al recommend measuring a baseline lipid profile and LFTs and repeating once while on the target dose (1). Our data suggest that adopting this approach would be safe and cost effective.

[1]Hansen TJ, Lucking S, Miller JJ et al. Standardized laboratory monitoring with use of isotretinoin in acne. JAAD. 2016 Aug 31;75(2):323-8.
[2]Lee YH, Scharnitz, Muscat J et al. Laboratory monitoring during isotretinoin therapy for acne: a systematic review and meta-analysis. JAMA derm. 2016 Jan 1;152(1):35-44.

Poster 07
Narrowband UVB phototherapy outcomes – a single-centre retrospective review

J Clowry, L Nestor, K Molloy, A Salim, M Connolly, AM Tobin

Systemic and biologic therapies are rapidly evolving areas of research and development in cutaneous inflammatory diseases including psoriasis and atopic dermatitis (AD). We sought to determine whether narrowband ultraviolet B phototherapy (NBUVB; 311 - 313nm) remains an effective treatment option, 30 years following its introduction.

We present a retrospective review of outcomes with in-office NBUVB phototherapy for AD in a tertiary-referral centre. We reviewed records for all AD patients treated with NBUVB phototherapy from 2006 - 2016. Data collected included age, sex, minimal erythema dose (MED), cumulative dose, number of treatment courses, adverse events and outcome. All patients were 18 years or older with generalised skin involvement and had failed first-line topical regimens.

A total of 36 patients, 15 females (42%) and 21 males (58%) were included. Twenty-two patients (61%) had a single treatment course, 9 patients (25%) had two courses and 5 (14%) patients had three courses. Seventy-eight percent (n = 28) used topical corticosteroids during treatment. An MED was recorded in 81% (n = 29) ranging from 0.2 - 0.77 J/cm2. Treatment limiting adverse events included herpes simplex (n = 1), polymorphic light eruption (n = 3), photo-aggravated atopic dermatitis (n = 2) and grade 3 erythema (n = 1). Two patients did not complete therapy for personal reasons. There were no reported melanoma or non-melanoma skin cancers.

Seventy-two percent (n = 26) were clear or almost clear following their first treatment course, reflecting previously reported outcomes1. The median number of exposures for the first course was 28 (range 6-40) and the median cumulative dose was 25.78 J/cm2 (range: 1.39 – 110.67 J/cm2). The median interval between patients' first and second treatment courses was 310 days (range: 140 – 1384) and 963 days (range: 86 - 3577) between courses 2 and 3. Twenty-two percent (n = 8) progressed to systemic therapy at a median of 273 days post completion of phototherapy (range: 5 – 1385).

Emerging therapies in AD offer great hope for refractory disease, however this tudy serves as a reminder of the impressive treatment outcomes with NBUVB phototherapy. The results reflect the commitment to maintaining the highest standards in phototherapy units and support the continued funding of these departments in a challenging era for the health service.

1. George SA et al. Narrow-band (TL-01) UVB air-conditioned phototherapy for chronic severe adult atopic dermatitis. Br J Dermatol 1993; 128: 49–56.

Poster 08
A single-centre review of methotrexate in the management of severe adult atopic dermatitis

J Clowry, L Nestor, K Molloy, A Salim, M Connolly, AM Tobin

Methotrexate is commonly prescribed in the management of atopic dermatitis. This is a single-centre retrospective review of treatment outcomes amongst a patient cohort ›18 years old. Cases were identified from laboratory records from January 2014 – December 2016.

Twenty-five patients with severe atopic dermatitis were included; thirteen females (52%) and twelve males (48%). Median age was 36 years (range 22 - 64). Previous treatments included topical therapies, antihistamines, narrow-band ultraviolet B phototherapy (n = 17, 68%), azathioprine (n = 4, 16%), mycophenolate mofetil (n = 1, 4%) and ciclosporin (n = 4, 16%). Prior oral prednisolone was documented in nineteen cases (76%) and hospital admissions for atopic dermatitis were recorded in ten (40%). Four patients (16%) had attended tertiary psychiatry services in the context of their cutaneous disease.

Methotrexate was commenced at 5-10mg weekly with folic acid supplementation and titrated up to a maximum 25mg. Median treatment duration was 13 months (range 1.5 – 44). Adjunctive oral therapies included prednisolone, acyclovir and antibiotics. Six patients (24%) required tapering dose prednisolone at the outset of treatment, n = 2 (8%) required short course prednisolone for an acute flare and n = 1 (4%) required maintenance prednisolone.

A four-point Physician's Global Assessment score (PGA) was used to assess outcome. A PGA score of 1 indicated disease clearance and PGA 4 corresponded with treatment failure. Two patients were lost to follow-up and nineteen cases (76% overall) had a PGA ≤3. Sixteen patients (64%) remained on methotrexate, of whom four (16% overall) had a PGA score of 1. Reasons for discontinuation included family planning (n = 1), lack of efficacy (n = 2), combined lack of efficacy and adverse effects (n = 3), neutropenic sepsis (n = 1) and remission (n = 2). Methotrexate was otherwise well tolerated, however 32% (n = 8) reported nausea. Transient mild elevations in liver function tests were recorded in nine cases (36%).

This review demonstrates favourable response rates to methotrexate in atopic dermatitis. Given the psychological impact of severe disease, this well-established systemic therapy should be considered early in the management of moderate-to-severe and monitored using patient and clinician-based assessments.

Poster 09
Remission of refractory cutaneous Crohn's disease with combination ertapenem and biologic therapy

J Clowry, L Nestor, K Molloy, A Salim, M Connolly, AM Tobin

We present two cases of severe cutaneous Crohn's disease, both refractory to standard treatments. These patients had a history of prolonged hospital admissions, but responded dramatically to an extended course of community administered intravenous (IV) ertapenem, added to their biologic regimens.

The first case is a thirty-three year old female with a lifelong history of Crohn's. She had intractable vulval disease with persistent pain, discharge, lymphoedema and fissuring. She had failed multiple courses of combination oral and intravenous antibiotics, dapsone, adalimumab and infliximab. The severity of her condition necessitated long-term oral steroid use, resulting in secondary Cushing's syndrome. Ustekinemab 90mg every two weeks was beneficial as a steroid-sparing agent however did not adequately control her symptoms. Ertapenem 1g daily was added with an immediate clinical response resulting in reduced pain, oedema and discharge. It was withdrawn four months later when the patient's pain had fully resolved, fissures had almost completely healed and discharge was minimal. She remains in remission on maintenance rifampicin and ustekinemab.

The second case, is also a thirty-three year old female with a ten year history of severe Crohn's disease. She was reviewed with perianal fistulating disease from the vagina to natal cleft with recurrent cellulitis. This had a profound impact on her quality of life and was unresponsive to a similarly exhaustive list of immunmodulatory therapies. Vedolizumab 300mg IV eight weekly was introduced with some benefit, but her cutaneous fistulating disease remained problematic. Ertapenem 1g daily was added with a significant reduction in pain and discharge and was withdrawn after a twelve week course.

Ertapenem is an intravenous beta-lactam antibiotic indicated for use against gram-positive, gram-negative and a wide range of anaerobic pathogens. Its pharmacokinetic profile favours convenient community-based once daily dosing and its antimicrobial spectrum against anaerobes is of particular benefit in discharging, fistulating Crohn's. In inflammatory bowel disease, it has only been reported to date as an oral therapy for chronic antibiotic-refractory pouchitis1 (CARP), given its poor oral absorption and selective gastrointestinal activity. Optimal duration of therapy is unclear, and necessitates balancing disease control with concerns regarding clostridium difficile infections and antibiotic resistance. Nevertheless, it is a potentially transformative agent in a select group of refractory cases.

1.Madirrala V et al Successful faecal coliform sensitivity-based oral ertapenem therapy for chronic antibiotic-refractory pouchitis: a case series. Eur J Gastroenterol Hepatol. 28(3), 277-80.

Poster 10

Referral of invasive melanoma to a Melanoma Multidisciplinary Team Meeting in Cork University Hospital

R. O'Connor1, C.M.R Fahy2, S. McCarthy1, J. Bourke1, M. Murphy1
1Department of Dermatology - South Infirmary Victoria University Hospital Cork
2Department of Dermatology – Royal United Hospital NHS Foundation Trust, Bath, UK

Introduction: The incidence of melanoma is rapidly rising in Ireland with over 700 new cases diagnosed each year. The NCCP guidelines suggest that all melanomas are referred for discussion to a Melanoma MDT. The Southwest Melanoma MDT meeting is a dynamic meeting held twice monthly at Cork University Hospital (CUH).

Aim: In this study we examined how many primary invasive melanomas diagnosed at CUH in 2014 were discussed at the melanoma MDT. We compared our results to a similar study performed in 2012.

Methods: Reports were obtained from all cases of primary invasive melanoma diagnosed at the Department of Pathology, CUH from January – December 2014. By analyzing records of MDT minutes, these cases were compared to those referred for discussion at the Melanoma MDT from January 2014 – June 2015.

Results: In 2014, 129 cases of primary invasive melanoma were diagnosed at CUH. 120 (93%) cases were discussed at the MDT. In 2012, 123 cases of invasive melanoma were diagnosed at CUH and 106 (86%) cases were discussed.

737 cases of melanoma were discussed at the Melanoma MDT in CUH from Jan – Dec 2014. Some cases were reviewed on multiple occasions as clinical situations evolved.

Dermatology had 66 cases of IM of which 65/66 were referred to the MDT. This reflects an improvement of 2% from 2012. Plastic Surgery referred 18/22, an improvement of 8% from 2012. Numbers of IM from other specialties include general surgery, N=9, 9/9 referred, GP N=14, 12/14 referred; maxillofacial surgery N=1, 1 referred; ENT N=1, 0 referred.

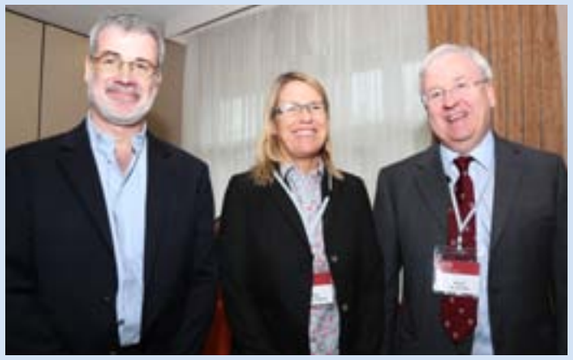
9 cases were never referred for discussion to the MDT from various specialties including Plastic Surgery, N=4, ENT N=1, GP N=2, Dermatology N=1 and Colorectal surgery N=1.
36 cases of melanoma were referred from external institutions for review at the Southwest Melanoma MDT.

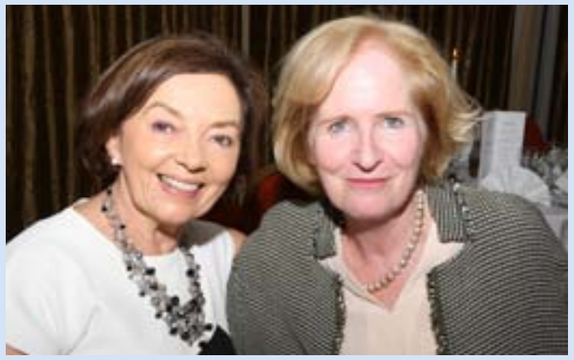
Discussion: Although the above results reflect an overall improvement in the number of cases referred for discussion to the melanoma MDT at Cork University Hospital, not all invasive melanomas diagnosed at this institution are being referred for discussion. It is likely that the percentage of melanomas referred to MDTs from institutions without an in-house MDT is even lower. The mechanism for referring melanomas to MDTs nationally needs to be evaluated and streamlined.

IAD
Irish Association of Dermatologists

Autumn Meeting


Dr Garry Meenagh & Dr Ian Coulson


Prof Eli Sprecher, Prof Catherine Nelson-Piercy Guest speakers & Dr Kevin McKenna, IAD President


Dr Rosemarie Watson & Prof Louise Barnes IAD


Dr Art O'Hagan, Honorary Secretary IAD & Guest speaker Dr Ian Coulson

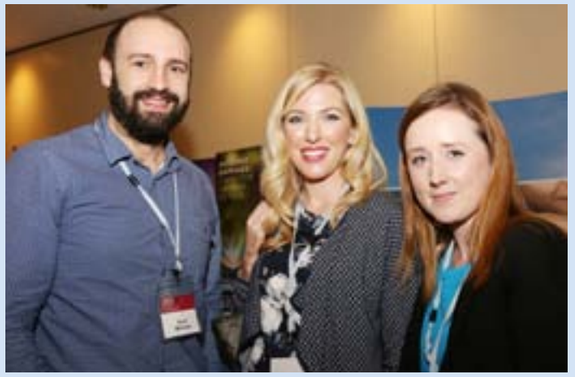

Roisin O'Connor, Ian McDonald, Conor McGrath, Laoise Griffin, Eimear Gilhooly, Jennifer Boggs & Veronika Dvorakova


Dr Maeve Lynch & Dr Oonagh Molloy


Dr Nicola Cooke, Dr Collette McCourt & Dr Helen Hunter


Dr Catherine Quinlan & Dr Lisa Roche


Dr's Louise Cunningham, Julianne Clowry & Alison Havelin

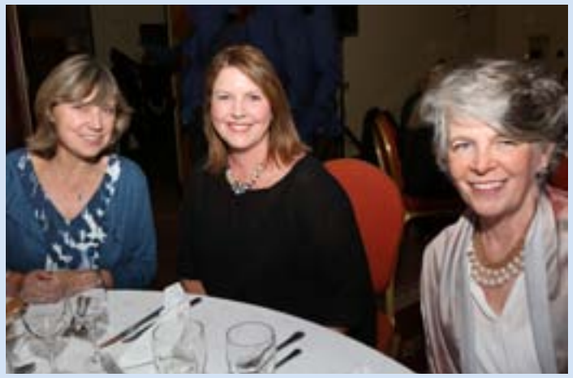

Dr's David Middleton, Victoria Campbell & Emma Mack


Dr Laura Skillen & Dr Donal O'Kane

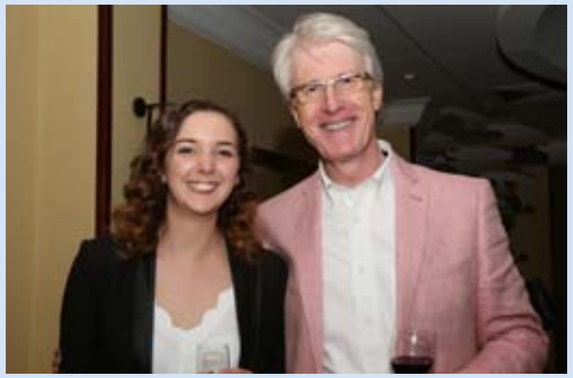

Dr Helen Hunter & Dr Julia Tolland


Dr Fergal Moloney, Dr Anousha Yazdabai & Dr Patsy Lenane


Suzanne Clements, Cliona Feighery, Louise Barnes, Rose-marie Watson & Muireann Roche



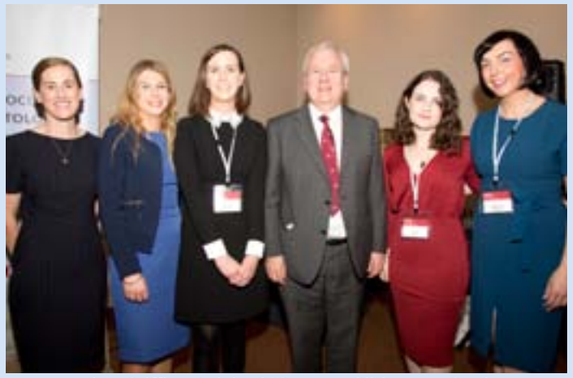
Christine McMillan, Julie Handley & Rosemary Black



Ms Clémence Rudolph Bioderma & Dr Colin Buckley IAD

Mr Richard Best & Dr Olivia Dolan

Dr Laoise Griffin, Dr Siobhan McCarthy & Dr Lisa Roche



Laoise Griffin, Roisin O'Connor, Sibhan McCarthy,
Kevin McKenna, Lisa Roche & Catherine Quinlan

Dr Nazish Mansoor, Dr Conor Broderick &
Dr Catherine Quinlan

Celgene Group Dr Conor Broderick IAD, Dr Kevin McKenna
President IAD, Celgene guest speakers Prof Kingston Mills
& Dr Gary Meenagh

Celgene
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Autumn 2016

Dr Kevin McKenna addresses the Symposium

Dr Gary Meenagh, Celgene symposium Guest Speaker

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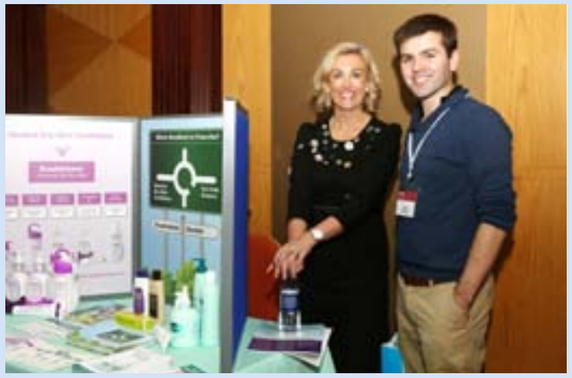


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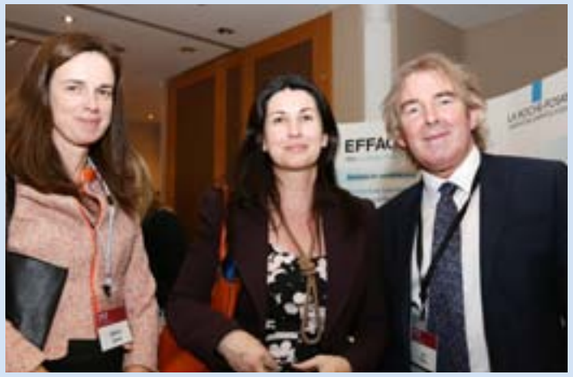

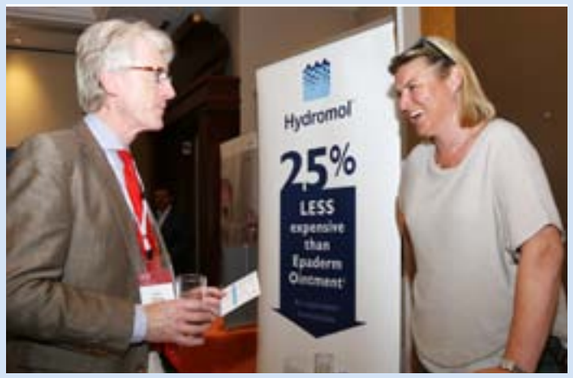
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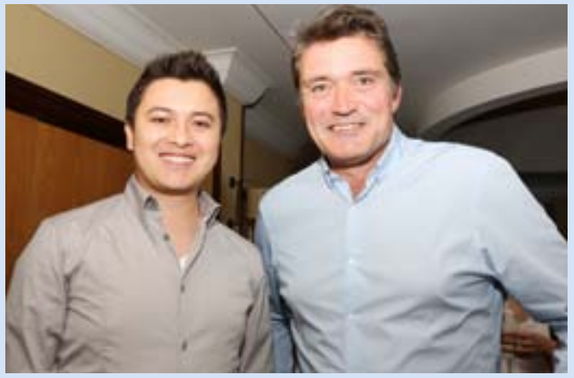

IAD AUTUMN MEETING WINNERS

1st Poster Prize, Winner Dr Conor Broderick & IAD President Dr Kevin McKenna

1st Prize Case Presentation winner Dr Victoria Campbell & IAD President Dr Kevin McKenna

1st Rogers prize winner Dr Laoise Griffin & IAD President Dr Kevin McKenna

2nd Rogers prize winner Dr Veronika Dvorakova & IAD President Dr Kevin McKenna

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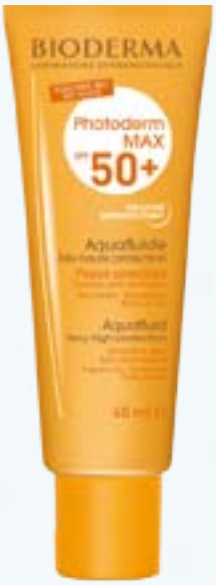

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