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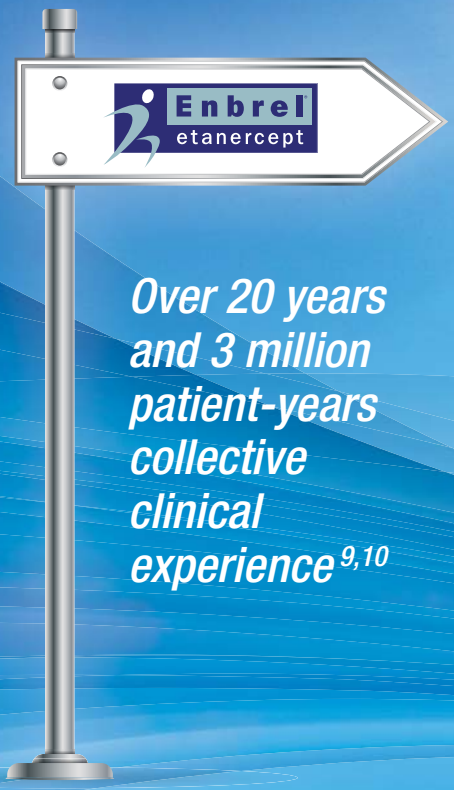
Irish Association of Dermatologists

Autumn Meeting

26th – 27th September 2013
Radisson Blu Hotel, Galway.



The ENBREL way

Indicated for RA, PsA, JIA, AS and PsO#

Over 20 years and 3 million patient-years collective clinical experience [9,10]

A unique mechanism of action

- Enbrel is the only fully human soluble tumour necrosis factor (TNF) receptor [1,2,3,4,5,6]
- It works differently than MAB's [1]

No neutralising antibodies [1]

- Enbrel is not associated with the production of neutralising antibodies in humans

Enbrel has a short half life (<3 days) [1]

- The half-life of anti-TNF agents should be taken into account if a treatment break is required

Efficacy

- Registry data and Cochrane Review data support efficacy & safety of Enbrel [7,8]

Enbrel (etanercept)
Abbreviated Prescribing Information

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 50 mg solution for injection in pre-filled pen. Each pre-filled pen contains 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. 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: By subcutaneous injection. Adults: 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 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: 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. 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 identified as carriers of hepatitis B virus 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 postmarketing 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 antidiabetic 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 breastfeeding 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: thrombocytopaenia, systemic vasculitis, uveitis and scleritis, interstitial lung disease, rare reports of tuberculosis, opportunistic infections, anaemia, leucopaenia, neutropaenia, pancytopenia, seizures, worsening of heart failure, autoimmune hepatitis, Steven Johnson's syndrome and very rare reports of : anaphylaxis, toxic epidermal necrolysis and aplastic anaemia have been reported. Reactivation of hepatitis B (a liver infection) has 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. Legal Category: POM. 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 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) 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. S1B: Product subject to a prescription which may be renewed. 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 363 633 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 6_1. Date of Prescribing Information: December 2012.

References:
1. Enbrel Summary of Product Characteristics August 2013. **2.** Remicade Summary of Product Characteristics. **3.** Humira Summary of Product Characteristics. **4.** Orencia Summary of Product Characteristics. **5.** Mabthera Summary of Product Characteristics. **6.** Simponi Summary of Product Characteristics. **7.** Singh J et al. CMAJ:2009 DOI;10.1503. **8.** Hetland ML et al. Arthritis & Rheumatism. Vol 62, no 1, January 2010. **9.** Data on File Pfizer Inc.10 Data on File Amgen

Rheumatoid Arthritis, Psoriatic Arthritis, Juvenile Idiopathic Arthritis, Ankylosing Spondylitis and Psoriasis.
For full prescribing information see the Summary of Product Characteristics.

ENB/2013/192/1
Date of preparation: September 2013

Pfizer

Welcome Message from the President Dr Pat Podmore

On your behalf I extend a warm welcome to our colleagues in Primary care.

A lot of effort has gone into devising this mutually interesting programme. This and our venue I am sure will provide a rich ground for us to interact, exchange views and experiences. I think in our busy professional lives we often do not have this luxury of time to discuss our mutual cases.

The satellite symposium does lengthen our meeting but I feel it is a worthwhile addition allowing us to focus on specific topics. As always we have to appreciate the support of our friends in the pharmaceutical industry and I do not have to remind you to visit the technical exhibits.

This being my last Presidential address I would like to take the opportunity to thank the Executive Members that I have served with, who have made this Office a treat rather than a task. It has been an honour and a pleasure to serve as your President.

I hope you enjoy this meeting.

Pat Podmore
President IAD

TRUST in HUMIRA – An unmatched legacy.

10 YEARS OF EFFICACY DATA FOR RA IN LABEL[1]

9 INDICATIONS - THE MOST OF ANY SELF-ADMINISTERED BIOLOGIC[1]

MORE THAN **85** COUNTRIES[2]

15 YEARS OF CLINICAL TRIAL EXPERIENCE, BEGINNING WITH RHEUMATOID ARTHRITIS (RA)[2]

71 CLINICAL TRIALS IN THE LARGEST PUBLISHED ANTI-TUMOR NECROSIS FACTOR (TNF) CROSS-INDICATION SAFETY DATABASE[3]

MORE THAN **670,000** PATIENTS CURRENTLY TREATED WORLDWIDE[2]

MORE THAN **23,000** PATIENTS IN GLOBAL STUDIES[3]

HUMIRA adalimumab 10 YEARS

Humira (adalimumab) 40mg solution for injection in pre-filled pen or pre-filled syringe and Humira 40mg/0.8ml solution for injection for paediatric use Refer to Summary of Product Characteristics for full information. Presentation: Each 0.8ml single dose pre-filled pen, pre-filled syringe or vial contains 40mg of adalimumab. **Indications:** Rheumatoid arthritis (RA): Humira in combination with methotrexate is indicated for the treatment of moderate to severe, active RA in adult patients when the response to disease-modifying anti-rheumatic drugs (DMARDs) including methotrexate has been inadequate. Humira is also indicated for the treatment of severe, active and progressive RA in adults not previously treated with methotrexate. Humira can be given as monotherapy in case of intolerance to methotrexate or when continued treatment with methotrexate is inappropriate. Humira has been shown to reduce the rate of progression of joint damage as measured by X-ray and to improve physical function, when given in combination with methotrexate. Polyarticular juvenile idiopathic arthritis (JIA): Humira in combination with methotrexate is indicated for the treatment of active JIA, in children and adolescents aged 2 to 17 years who have had an inadequate response to one or more DMARDs. Humira can be given as monotherapy in case of intolerance to methotrexate or when continued treatment with methotrexate is inappropriate. Psoriatic arthritis (PsA): Humira is indicated for the treatment of active and progressive PsA 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. Ankylosing spondylitis (AS): Humira is indicated for the treatment of adults with severe active AS who have had an inadequate response to conventional therapy. Axial spondyloarthritis (SpA) non-radiographic: Humira is indicated for the treatment of adults with severe axial spondyloarthritis without radiographic evidence of AS but with objective signs of inflammation by elevated CRP and / or MRI, who have had an inadequate response to, or are intolerant to nonsteroidal anti-inflammatory drugs. Crohn's disease (CD): Humira is indicated for treatment of moderate to severe, active CD, in patients who have not responded despite a full and adequate course of therapy with a corticosteroid and/or an immunosuppressant; or who are intolerant to or have medical contraindications for such therapies. Paediatric Crohn's Disease: Humira is indicated for the treatment of severe active Crohn's disease in paediatric patients (6 to 17 years) who have had an inadequate response to conventional therapy including primary nutrition therapy, a corticosteroid, and an immunomodulator, or who are intolerant to or have contraindications for such therapies. Psoriasis (Ps): Humira is indicated for the treatment of moderate to severe chronic plaque psoriasis in adult patients who failed to respond to or who have a contraindication to, or are intolerant to other systemic therapy including cyclosporine, methotrexate or PUVA. Ulcerative colitis (UC): Humira is indicated for treatment of moderate to severe active ulcerative colitis in adult patients who have had an inadequate response to conventional therapy including corticosteroids and 6-mercaptopurine (6-MP) or azathioprine (AZA), or who are intolerant to or have medical contraindications for such therapies. **Dosage and administration:** Humira treatment should be initiated and supervised by specialist physicians experienced in the diagnosis and treatment of conditions for which Humira is indicated. Patients treated with Humira should be given the special alert card. After proper training in injection technique, patients may self-inject with Humira if their physician determines that it is appropriate and with medical follow-up as necessary. During treatment with Humira, other concomitant therapies (e.g., corticosteroids and/or immunomodulatory agents) should be optimised. RA, PsA, AS or SpA non-radiographic: 40mg administered every other week as a single dose via subcutaneous injection. RA: In monotherapy some patients who experience a decrease in their response to Humira may benefit from an increase in dose intensity to 40mg every week. There may be a need for dose interruption, for instance before surgery or if a serious infection occurs. Available data suggest that re-introduction of Humira after discontinuation for 70 days or longer resulted in the same magnitudes of clinical response and similar safety profile as before dose interruption. For RA, JIA, PsA and AS, available data suggest that the clinical response is usually achieved within 12 weeks of treatment. Continued therapy should be carefully reconsidered in a patient not responding within this time period. JIA: Age 2 to 12 years: 24mg/m^2 body surface area to a maximum single dose of 40mg administered every other week via subcutaneous injection. The volume for injection is based on the patients' height and weight (see SmPC for height and weight dosing chart). A 40mg paediatric vial is available for patients who need to administer less than the full 40mg dose. Age 13 to 17 years: 40mg administered every other week via subcutaneous injection regardless of body surface area. CD: The recommended Humira induction dose regimen for adult patients with moderate to severe CD is 80mg at Week 0 followed by 40mg at Week 2. In case there is a need for a more rapid response to therapy, the regimen 160mg at Week 0 (dose can be administered as four injections in one day or as two injections per day for two consecutive days), 80mg at Week 2, can be used with the awareness that the risk for adverse events is higher during induction. After induction treatment, the recommended dose is 40mg every other week via subcutaneous injection. Alternatively, if a patient has stopped Humira and signs and symptoms of disease recur, Humira may be re-administered. There is little experience from re-administration after more than 8 weeks since the previous dose. During maintenance treatment, corticosteroids may be tapered in accordance with clinical practice guidelines. Some patients who experience decrease in their response may benefit from an increase in dosing frequency to 40mg Humira every week. Some patients who have not responded by Week 4 may benefit from continued maintenance therapy through Week 12. Continued therapy should be carefully reconsidered in a patient not responding within this time period. Paediatric CD: patients <40Kg: recommended induction dose regimen of 40mg at Week 0 followed by 20mg at Week 2 via SC injection. In case of need for a more rapid response to therapy, the regimen 80mg at Week 0 (dose can be administered as two injections in one day), 40mg at Week 2, can be used with the awareness that the risk for adverse events is higher during induction. After induction treatment, the recommended dose is 20mg every other week via SC injection. Some patients who experience insufficient response may benefit from 20mg every week; patients >40Kg: double the dose regimen for those patients <40Kg.Continued therapy should be carefully considered in a subject not responding by week 12. Psoriasis: The recommended dose of Humira for adult patients is an initial dose of 80mg administered subcutaneously, followed by 40mg subcutaneously given every other week starting one week after the initial dose. Continued therapy beyond 16 weeks should be carefully reconsidered in a patient not responding within this time period. UC: The recommended Humira induction dose regimen for adult patients with moderate to severe UC is 160mg at week 0 (dose can be administered as four injections in one day or as two injections per day for two consecutive days) and 80mg at week 2. After induction treatment, the recommended dose is 40mg every other week via subcutaneous injection. During maintenance treatment, corticosteroids may be tapered in accordance with clinical practice guidelines. Some patients who experience decrease in their response may benefit from an increase in dosing frequency to 40mg Humira every week. Available data suggest that clinical response is usually achieved within 2-8 weeks of treatment.Continued therapy is not recommended in patients not responding within this time period. **Contraindications:** Active tuberculosis or other severe infections such as sepsis, and opportunistic infections; moderate to severe heart failure (NYHA class III/IV) and hypersensitivity to adalimumab or any of the excipients. **Precautions and Warnings:** *Infections:* Patients taking TNF-antagonists are more susceptible to serious infections. Impaired lung function may increase the risk for developing infections. Patients must therefore be monitored closely for infections, including tuberculosis, before, during and for 4 months after treatment with Humira. Treatment with Humira should not be initiated in patients with active, chronic or localised infections until infections are controlled. In patients who have been exposed to tuberculosis and patients who have travelled in areas of high risk of tuberculosis or endemic mycoses, such as histoplasmosis, coccidioidomycosis, or blastomycosis, the risk and benefits of treatment with Humira should be considered prior to initiating therapy (see *Opportunistic infections*). Patients who develop a new infection while undergoing treatment with Humira should be monitored closely and undergo a complete diagnostic evaluation. Administration of Humira should be discontinued if a patient develops a new serious infection or sepsis, and appropriate antimicrobial or antifungal therapy should be initiated until the infection is controlled. Physicians should exercise caution when considering the use of Humira in patients with a history of recurring infection or with underlying conditions which may predispose to infections, including the use of concomitant immunosuppressive medications. *Serious infections:* Serious infections, including sepsis, due to bacterial, mycobacterial, invasive fungal, parasitic, viral, or other opportunistic infections such as listeriosis, legionellosis and pneumocystis have been reported in patients receiving Humira. Other serious infections seen in clinical trials include pneumonia, pyelonephritis, septic arthritis and septicaemia. Hospitalisation or fatal outcomes associated with infections have been reported. *Tuberculosis:* Tuberculosis, including reactivation and new onset of tuberculosis, has been reported in patients receiving Humira. Reports included cases of pulmonary and extra-pulmonary i.e. disseminated. Before initiation of therapy with Humira, all patients must be evaluated for both active or inactive ('latent') tuberculosis infection. Appropriate screening tests, should be performed in all patients, local recommendations may apply. If active tuberculosis is diagnosed, Humira therapy must not be initiated. If latent tuberculosis is suspected, a physician with expertise in the treatment of tuberculosis should be consulted and the benefit/risk balance of therapy with Humira should be considered. If inactive ('latent') tuberculosis is diagnosed, appropriate treatment must be started with anti-tuberculosis prophylaxis treatment before the initiation of Humira, and in accordance with local recommendations. In patients who have several or significant risk factors for tuberculosis despite a negative test for tuberculosis, anti-tuberculosis prophylaxis treatment should also be considered before the initiation of Humira and in patients with a past history of latent or active tuberculosis in whom an adequate course of treatment cannot be confirmed. Some patients who have previously received treatment for latent or active tuberculosis have redeveloped tuberculosis while being treated with Humira. *Other opportunistic infections:* Opportunistic infections, including invasive fungal infections have been observed in patients receiving Humira. These infections have not consistently been recognised in patients taking TNF-antagonists and this resulted in delays in appropriate treatment, sometimes resulting in fatal outcomes. For patients who develop the signs and symptoms such as fever, malaise, weight loss, sweats, cough, dyspnoea, and/or pulmonary infiltrates or other serious systemic illness with or without concomitant shock, an invasive fungal infection should be suspected and administration of Humira should be promptly discontinued. Diagnosis and administration of empiric antifungal therapy in these patients should be made in consultation with a physician with appropriate expertise. *Hepatitis B Reactivation:* Reactivation of hepatitis B (HBV) has occurred in patients receiving a TNF-antagonist including Humira, who are chronic carriers of this virus (i.e. surface antigen positive), with some fatal outcomes. Patients should be tested for HBV infection before initiating treatment. Patients that test positive should have a consultation with a physician. In patients who develop HBV reactivation, Humira should be stopped and effective anti-viral therapy with appropriate supportive treatment should be initiated. Carriers of HBV should be closely monitored for signs and symptoms of active HBV infection throughout therapy and for several months following termination of Humira. *Neurological events:* Humira has been associated, in rare cases, with new onset or exacerbation of clinical symptoms and/or radiographic evidence of central nervous system demyelinating disease including multiple sclerosis and optic neuritis, and peripheral demyelinating disease, including Guillain-Barré syndrome. Caution should be exercised when considering Humira in patients with pre-existing or recent-onset central or peripheral nervous system demyelinating disorders. *Allergic reactions:* Postmarketing serious allergic reactions including anaphylaxis have been received following Humira administration. If an anaphylactic reaction or other serious allergic reaction occurs, administration of Humira should be discontinued immediately and appropriate therapy initiated. *Malignancies and lymphoproliferative disorders:* In clinical trials, more cases of malignancies including lymphoma and leukaemia have been observed among patients receiving a TNF-antagonist compared with control patients. These data cannot exclude a possible risk of malignancy in patients including children and adolescents treated with TNF antagonists. Furthermore, there is an increased background lymphoma risk in RA patients. Rare postmarketing cases of hepatosplenic T-cell lymphoma have been identified in patients treated with adalimumab. Some of these cases have occurred in young adult patients on concomitant treatment with azathioprine or 6-mercaptopurine used for inflammatory bowel disease. The potential risk with the combination of azathioprine or 6-mercaptopurine and Humira should be carefully considered. A risk for the development of hepatosplenic T-cell lymphoma in patients treated with Humira cannot be excluded. Caution should be exercised in considering Humira treatment of patients with a history of malignancy. All patients, and in particular patients with a medical history of extensive immunosuppressant therapy or psoriasis patients with a history of PUVA treatment should be examined for the presence of non-melanoma skin cancer prior to and during treatment with Humira. Caution should also be exercised when using TNF-antagonists in COPD patients, as well as in patients with increased risk of malignancies due to heavy smoking. With current data it is not known if adalimumab treatment influences the risk for developing dysplasia or colon cancer. All patients with UC who are at increased risk for dysplasia or colon carcinoma (e.g. patients with long-standing UC or primary sclerosing cholangitis), or who had a prior history of dysplasia or colon carcinoma should be screened for dysplasia at regular intervals before therapy and throughout their disease course. This evaluation should include colonoscopy and biopsies per local recommendations. *Haematologic reactions:* Pancytopaenia including aplastic anaemia has rarely been reported with TNF blocking agents. Adverse events of the haematologic system, including cytopaenia (eg thrombocytopaenia, leucopaenia) have been reported with Humira. Patients should be advised to seek immediate medical attention if they develop signs and symptoms suggestive of blood dyscrasias. *Vaccinations:* Patients on Humira may receive concurrent vaccinations, except for live vaccines. It is recommended that JIA patients, if possible, be brought up to date with all immunisations in agreement with current immunisation guidelines prior to initiating Humira therapy. *Congestive heart failure:* Humira should be used with caution in patients with mild heart failure (NYHA class I/II) and discontinued in patients who develop new or worsening symptoms of congestive heart failure. *Autoimmune process:* Humira may result in the formation of autoimmune antibodies. If a patient develops symptoms suggestive of a lupus-like syndrome following treatment with Humira and is positive for antibodies against double-stranded DNA, further treatment with Humira should not be given. *Surgery:* There is limited safety experience of surgical procedures in patients treated with Humira. The long half life of Humira should be taken into consideration when a surgical procedure is planned, and the patient should be monitored for infections. *Small bowel obstruction:* Failure to respond to treatment for CD may indicate the presence of fixed fibrotic stricture that may require surgical treatment. Available data suggest that Humira does not worsen or cause strictures. *Elderly population:* The frequency of serious infections among Humira treated subjects over 65 years of age was higher than those under 65 years of age. Some of those had a fatal outcome. Particular attention regarding the risk for infection should be paid when treating the elderly. **Interactions:** Combination of adalimumab with other biologic DMARDS (e.g. anakinra and abatacept) or other TNF-antagonists is not recommended. **Pregnancy and lactation:** Administration of adalimumab is 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. Administration of live vaccines to infants exposed to adalimumab in utero is not recommended for 5 months following the mother's last adalimumab injection during pregnancy.Women must not breast-feed for at least five months after the last Humira treatment. **Driving and machinery:** Humira may have a minor influence on the ability to drive and use machines. **Side Effects:** From clinical trials unless marked '1' which indicates spontaneous reporting data.Very common ≥ 1/10: Respiratory tract infections (including lower and upper respiratory tract infection, pneumonia, sinusitis, pharyngitis, nasopharyngitis and pneumonia herpes viral), leucopaenia (including neutropaenia and agranulocytosis), anaemia, lipids increased, headache, abdominal pain, nausea and vomiting, elevated liver enzymes, rash (including exfoliative rash), musculoskeletal pain, injection site reaction (including injection site erythema). Common ≥ 1/100 to < 1/10: Systemic infections (including sepsis, candidiasis and influenza), intestinal infections (including gastroenteritis viral), skin and soft tissue infections (including paronychia, cellulitis, impetigo, necrotising fasciitis and herpes zoster), ear infections, oral infections (including herpes simplex, oral herpes and tooth infections), reproductive tract infections (including vulvovaginal mycotic infection), urinary tract infections (including pyelonephritis), fungal infections, joint infections, benign neoplasm, skin cancer excluding melanoma (including basal cell carcinoma and squamous cell carcinoma), thrombocytopaenia, leucocytosis, hypersensitivity, allergies (including seasonal allergy), hypokalaemia, uric acid increased, blood sodium abnormal, hypocalcaemia, hyperglycaemia, hypophosphotaemia, dehydration, mood alterations (including depression), anxiety, insomnia, paraesthesias (including hypoaesthesia), migraine, nerve root compression, visual impairment, conjunctivitis, blepharitis, eye swelling, vertigo, tachycardia, hypertension, flushing, haematoma, cough, asthma, dyspnoea, GI haemorrhage, dyspepsia, gastroesophageal reflux disease, sicca syndrome, worsening and new onset of psoriasis (including palmplanter pustular psoriasis)[1], alopecia[1], pruritis, urticaria, bruising (including purpura), dermatitis (including eczema), onychoclasis, hyperhydrosis, muscle spasms (including blood creatine phosphokinase increased), haematuria, renal impairment, chest pain, oedema, pyrexia[1], coagulation and bleeding disorders (including activated partial thromboplastin time prolonged), autoantibody test positive (including double stranded DNA antibody), blood lactate dehydrogenase increased, impaired healing. Uncommon ≥ 1/1000 to < 1/100: Opportunistic infections and tuberculosis (including coccidioidomycosis, histoplasmosis and mycobacterium avum complex infection), neurological infections (including viral meningitis), eye infections, bacterial infections, diverticulitis[1], lymphoma, solid organ neoplasm (including breast cancer, lung neoplasm and thyroid neoplasm), melanoma, idiopathic thrombocytopaenic purpura, sarcoidosis[1], cerebrovascular accident[1], tremor, neuropathy, diplopia, deafness, tinnitus, myocardial infarction[1], arrhythmia, congestive heart failure, pulmonary embolism[1], chronic obstructive pulmonary disease, interstitial lung disease, pneumonitis, pleural effusion[1], pancreatitis, dysphagia, face oedema, cholecystitis and cholelithiasis, bilirubin increased, hepatic steatosis, night sweats, scar, rhabdomyolysis, systemic lupus erythematosus, nocturia, erectile dysfunction, inflammation, vascular arterial occlusion, aortic aneurysm, thrombophlebitis. Rare ≥ 1/10,000 to < 1/1,000: Leukaemia[1], anaphylaxis[1], demyelinating disorders (e.g. optic neuritis, Guillain-Barré syndrome)[1], pancytopaenia, multiple sclerosis, cardiac arrest, pulmonary fibrosis[1], intestinal perforation[1], reactivation of hepatitis B[1], autoimmune hepatitis[1], erythema multiforme[1], cutaneous vasculitis[1], Stevens-Johnson syndrome[1], angioedema[1], lupus-like syndrome[1]. Very rare < 1/10,000: Liver failure[1]. Not known: Hepatosplenic T-cell lymphoma[1], Merkel Cell Carcinoma[1] **Prescribers should consult the summary of product characteristics for further information on side effects. Overdose:** No dose-limiting toxicity was observed during clinical trials. The highest dose level evaluated has been multiple intravenous doses of 10 mg/kg (approximately 15 times the recommended dose). **Legal Category:** POM **Marketing Authorisation Numbers/Presentations**: Vial : EU/1/03/256/001; 1 pack contains 2 cartons each containing 1 single use vial and empty sterile injection syringe, needle and vial adapter. Pre-filled Syringe : EU/1/03/256/003: Each carton contains 2 single use pre-filled syringes in a blister. Pre-filled Pen: EU/1/03/256/008: Each carton contains 2 single use pre-filled pens in a blister. **Further information is available from AbbVie Limited, Block B, Liffey Valley Office Campus, Quarryvale, Co. Dublin. Date of revision of PI:** February 2013 PI/256/008. **References: 1.** HUMIRA [summary of product characteristics]. AbbVie Limited; Available on www.medicines.ie **2.** Data on file, AbbVie Inc. 2012. **3.** Burmester GR, Panaccione R, Gordon KB, 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 [published online ahead of print May 5, 2012]. Ann Rheum Dis. doi:10.1136/annrheumdis-2011-201244.

IREHUM130069b Date of Preparation: June 2013

abbvie

abbvie **sponsored Symposium**

Management of psoriatic disease – aiming for goal

Irish Association of Dermatologists Autumn Meeting
26th September 2013 - Radisson Blu Hotel, Galway

10.30 - 10.45	Welcome and Introduction **Prof Brian Kirby**
10.45 - 11.15	Implementing treatment goals and guidelines in daily clinical practice: opportunities and challenges **Dr Richard Warren**
11.15 - 11.45	Management of patients with psoriasis and psoriatic arthritis: a Joint approach **Dr Ann-Marie Tobin**
11.45 - 2.20	Is there a significant association between psoriasis and cardiovascular disease? A debate *In support of the motion:* ***Prof Brian Kirby, Dublin*** *Opposing the motion:* ***Dr Richard Warren, Manchester*** *Rebuttals and discussion:* ***All***
12.20 - 12.30	Summary and close **Professor Brian Kirby**

Professor Brian Kirby, Consultant Dermatologist, St Vincent's University Hospital, Dublin
Professor Brian Kirby MD FRCPI is a Consultant Dermatologist at St Vincent's University Hospital and Associate Clinical Professor at University College Dublin, Ireland. His clinical and research special interest is in psoriasis. Together with Dr Aoife Lally he runs the largest psoriasis clinic in Ireland. Professor Kirby has more than 110 Medline quoted publications the majority of which are on psoriasis. His current research interests include genetics of psoriasis, immunology, psoriatic arthritis and lifestyle aspects of psoriasis including psychological distress and obesity. Brian Kirby is a member of the International Psoriasis Council and the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA).

Dr Richard Warren, Salford Royal Foundation Hospital, UK
Dr Richard Warren is a Clinical Senior Lecturer and honorary Consultant Dermatologist funded by the National Institute for Health Research (NIHR). Dr Warren graduated from Liverpool University with a first class honours degree in pharmacology and gained his medical degree, with honours, one year later. Dr Warren's work in dermatology has focused on pharmacogenetics (forming the basis of his PhD thesis), the genetic susceptibility to psoriasis and, more recently, biological therapies and their use in the treatment of Psoriasis. He has received national and international awards for his work into the pharmacogenetics of methotrexate. Dr Warren has published widely in dermatology with numerous abstracts, papers and book contributions. He is current EU Editor-in Chief for the journal Dermatology and Therapy. He has been an invited plenary speaker at major national and international dermatology meetings and was recently elected onto the International Psoriasis Council. Dr Warren is a member of the BAD biologics committee, BAD Biologics Research Sub-committee and recently served on the National Institute for Health and Clinical Excellence (NICE) guideline group for psoriasis.

Dr Anne-Marie Tobin, Consultant Dermatologist, AMNCH Hospital
Dr Anne – Marie Tobin is a Consultant Dermatologist and Clinical Lecturer at Tallaght Hospital and Trinity College Dublin with a subspecialty interest in psoriasis. She completed a PhD studying the effect of obesity and insulin resistance in psoriasis and has published on cardiovascular risk in patients with psoriasis. Dr Tobin also has a degree in pharmacy.

Stelara®
(ustekinumab)

365
−4*
361

That's 361 days to focus on life... not psoriasis

Clearance

- Early onset of action with visible efficacy by week 2†[1,2,3]
- Superior efficacy to etanercept 50mg twice weekly at 12 weeks[4]
- Efficacy response maintained through 5 years in Week 40 PASI 75 responders[5]**

Convenience

- Four doses per year, after 2 induction doses[6]

Confidence

- In 3,104 patient years of follow-up in the Phoenix 1 trial, Stelara® was generally well tolerated, with rates of serious infection remaining low and stable through 5 years of treatment[5]

*4 = Doses maintenance therapy after 2 induction doses.

**Week 40 responders comprised of patients who were randomised to Stelara® at baseline, were PASI 75 responders at both Weeks 28 and Week 40, and were re-randomised at Week 40 to continue 12-weekly maintenance treatment with Stelara®.

†PASI 50

STELARA® solution for injection in pre-filled syringe PRESCRIBING INFORMATION
ACTIVE INGREDIENT(S): Ustekinumab. Please refer to Summary of Product Characteristics (SmPC) before prescribing. **INDICATION(S):** 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 and PUVA. **DOSAGE & ADMINISTRATION:** Under the guidance and supervision of a physician experienced in diagnosis and treatment of psoriasis. Subcutaneous injection. Avoid areas with psoriasis. For self-injecting patients ensure appropriate training, follow-up and monitoring during treatment. **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). Consider discontinuation if no response after 28 weeks. **Children <18 years:** Not recommended. **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, they should be closely monitored and STELARA should not be administered until infection resolves. ***Malignancies:*** Potential to increase the risk of malignancy. No studies in patients with a history of malignancy or in patients who develop malignancy while receiving STELARA. ***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, discontinue STELARA immediately and institute appropriate therapy. ***Immunotherapy:*** Not known whether STELARA affects allergy immunotherapy. ***Latex sensitivity:*** Needle cover contains natural rubber (latex), may cause allergic reactions. **SIDE EFFECTS:** ***Serious side effects:*** Serious infections, malignancies. **Very common:** upper respiratory tract infection, nasopharyngitis. ***Common:*** hypersensitivity reactions (rash, urticaria), cellulitis, viral upper respiratory tract infection, depression, dizziness, headache, pharyngolaryngeal pain, nasal congestion, diarrhoea, pruritus, back pain, myalgia, arthralgia, fatigue, injection site erythema, antibodies to ustekinumab. ***Uncommon:*** herpes zoster infection, injection site reactions. ***Rare:*** serious hypersensitivity reactions (including anaphylaxis, angioedema), facial palsy ***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 witheld 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: The safety and efficacy of STELARA in combination with other immunosuppressants, including biologics, or phototherapy have not been evaluated. **LEGAL CATEGORY:** Prescription Only Medicine. **PRESENTATIONS, PACK SIZES, PRODUCT LICENCE NUMBER:** STELARA 45mg: 1 x 0.5ml pre-filled syringe. EU/1/08/494/003. STELARA 90mg: 1 x 1.0ml pre-filled syringe. EU/1/08/494/004 **MARKETING AUTHORISATION HOLDER:** JANSSEN-CILAG INTERNATIONAL NV, Turnhoutseweg 30, B-2340 Beerse, Belgium. **FURTHER INFORMATION IS AVAILABLE FROM:** Janssen-Cilag Ltd, 50 – 100 Holmers Farm Way, High Wycombe, Buckinghamshire, HP12 4EG UK.

Prescribing information last revised: 20/03/2012
PIVER: 200312

For further information on STELARA®, contact Janssen Medical Information at medinfo@janssen.ie or call 1800 709 122.

References:

1. Rich et al, Ustekinumab demonstrates rapid onset of efficacy in the treatment of moderate-to-severe psoriasis. Poster 040 presented at Psoriasis 2010, July
2. Leonardi C et al. Efficacy and safety of ustekinumab, a human interleukin-12/23 monoclonal antibody, in patients with psoriasis: 76-week results from a randomised, double-blind, placebo-controlled trial (PHOENIX 1). Lancet 2008: 371: 1665-1674
3. PAPP KA et al. Eicacy and safety of ustekinumab, a human interleukin-12/23 monoclonal antibody, in patients with psoriasis: 52-week results from a randomised, double-blind, placebo-controlled trial (PHOENIX 2) Lancet 2008: 371: 1675-1684
4. Griffiths C et al. Comparison of Ustekinumab and Etanercept for Moderate to Severe Psoriasis. N Eng J Med 2010; 362: 118-128
5. Kimball A et al. Spring EADV 2012. P582
6. Stelara® (ustekinumab) Summary of Product Characteristics

PHIR/STE/1112/0106
Date of preparation: November 2012

janssen
PHARMACEUTICAL COMPANIES OF Johnson & Johnson

Irish Association of Dermatologists
Primary Care Dermatology Joint Meeting
Thursday 26th & Friday 27th September 2013

Thursday 26th September 2013
Radisson Blu Hotel, Galway

9.30am	Registration
10.30am - 12.30pm	Abbvie Satellite Symposium *'Optimal management of psoriasis – aiming for goal'*
12.30pm - 2.00pm	LUNCH & EXHIBITION

IAD Programme

2.00pm - 2.45pm	Professor Frank Powell *'Primary Care and Dermatology: The Future in Ireland'* Consultant Dermatologist, Mater Misericordiae University Hospital, Dublin
2.45pm - 3.30pm	Dr Johnny Loughnane *'Hyperandrogenism in the female patient'* General Practitioner, Newcastle West, Co. Limerick
3.30pm - 4.00pm	COFFEE & EXHIBITION
4.00pm - 4.45pm	Prof Tony Ormerod *'Tele Dermatology'* Professor in Dermatology, Division of Applied Medicine, University of Aberdeen
5.00pm - 6.00pm	IAD Business Meeting
7.30pm	IAD GALA DINNER

Friday 27th September
Radisson Blu Hotel, Galway

9.30 - 11.00am	Registrars' Symposium - Rogers Prize
11.00am	COFFEE & EXHIBITION
11.30 - 1.00pm	Case presentations
1.00pm - 2.00pm	LUNCH

Etrivex®

500 micrograms/g Shampoo
(clobetasol propionate)

GALDERMA
Committed to the future of dermatology

BEAUTIFULLY SIMPLE

Etrivex Shampoo Abbreviated Prescribing Information UK & IRE

Presentation: 500 micrograms/g clobetasol propionate shampoo. **Indications:** Topical treatment of moderate scalp psoriasis in adults. **Dosage and Administration:** For cutaneous use on the scalp. Applied directly on to dry scalp once daily and left for 15 minutes before rinsing. Treatment duration should be limited to 4 weeks. Not recommended for use below 18 years of age. **Contraindications:** Hypersensitivity to active substance or any of the excipients. Skin areas affected by bacterial, viral, fungal or parasitic infections and specific skin diseases (skin tuberculosis, skin diseases caused by lues). Must not be applied to eyes or to ulcerous wounds. Not for use in children under 2 years. **Precautions and warnings:** Topical corticosteroids should be used with caution due to post treatment rebound relapses, tachyphylaxis & local or systemic toxicity. Abrupt discontinuation can lead to acute adrenal insufficiency, especially in children. If Etrivex shampoo is used in children and adolescents below 18 years of age, treatment should be reviewed weekly. Etrivex shampoo is only intended for the treatment of scalp psoriasis and should not be used to treat other skin areas. In particular, the face, eyelids, intertriginous areas (axillae and genitoanal regions) and other erosive skin surfaces. If Etrivex shampoo does enter the eye, the affected eye should be rinsed with copious amounts of water. **Pregnancy and lactation:** Should not be used during pregnancy or lactation unless clearly necessary. **Undesirable effects:** During clinical development the most commonly reported adverse drug reaction was skin discomfort (5% incidence), with no serious drug-related adverse events reported. Adverse events related to treatment were: Common (≥1/100, <1/10): Skin discomfort, acne/folliculitis, eye stinging/burning. Uncommon (≥1/1000, <1/100): Local signs of irritation, pruritus, urticaria, telangiectasia, skin atrophy. Prolonged use of topical corticosteroids/treatment of extensive areas/use of large amounts can result in sufficient systemic absorption to produce the features of hypercortisolism (Cushing syndrome) or of Hypothalamus-Pituitary-Adrenal (HPA) axis suppression. Risk of HPA axis suppression is low due to short contact nature of Etrivex shampoo. No HPA axis suppression was observed during clinical trials. Prolonged and/or intensive treatment with potent corticosteroids may cause local atrophic changes, such as local skin atrophy, striae, telangiectasia, erythema, purpura, contact dermatitis. During development of Etrivex Shampoo no skin thinning was observed. When applied to the face, very potent corticosteroids can induce perioral dermatitis, skin atrophy or worsen rosacea. There are reports of pigmentation changes, acne, pustular eruptions and hypertrichosis with topical corticosteroids. **Packaging Quantities and Cost:** 125ml UK £15.43, IRE €16.08. **MA Number:** PL 10590/0052 PA 590/23/1. **Legal Category:** POM. **Full Prescribing Information is available from:** Galderma (UK) Ltd, Meridien House, 69-71 Clarendon Road, Watford, Herts, WD17 1DS, Telephone: +44 (0) 1923 208950 Fax: +44 (0) 1923 208998
Date of Revision: February 2009.

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 Galderma (UK) Ltd.

 CLO/003/0212c March 2012

Biographical Sketches

Professor Frank Powell

Professor Frank Powell is a dermatologist and Chairman of the Board of Directors of the Charles Institute and acting Director. He is currently the President of the European Academy of Dermatology and Venereology and is a Past-President of the Irish Association of Dermatologists. He is a member of several international dermatologic societies and honorary member of the American Dermatologic Association, and the Hungarian and Romanian Dermatology Associations.

Dr Johnny Loughnane

Is a graduate of UCC. After house jobs in Limerick he completed general practice vocational training in Manchester. Attending dermatology clinics with Dr Robert Chalmers, at Manchester Royal Infirmary, stimulated a special interest in dermatology that has grown over the years.

He has been a general practitioner in Newcastle West since 1986. Following years as a trainer he is now an assistant programme director with the Mid Western General Practitioner Training Scheme.

He also works as a locum consultant in palliative medicine at Milford Hospice and University Hospital Limerick.

He has been involved with the Primary Care Dermatology Society of Ireland since its foundation, helping to organise the annual educational meeting.

Prof Tony Ormerod

Prof Tony Ormerod has published 160 peer reviewed articles and reviews covering a wide range of clinical and experimental dermatology including inflammatory allergic and malignant disease, immunological basis of disease and all aspects of psoriasis. He has a wide range of active research interests in clinical and experimental dermatology. Disease areas of special interest include inflammatory dermatoses psoriasis, eczema, acne, pyoderma gangrenosum and bullous disease and contact dermatitis. Allied to this is an interest in the immunological basis of the inflammatory diseases and modification by novel therapy. He conducted a study supporting a remote rural GP with special interest during his training via teledermatology and done a number of studies of teledermatology as surveillance for occupational hand dermatitis.

NEW

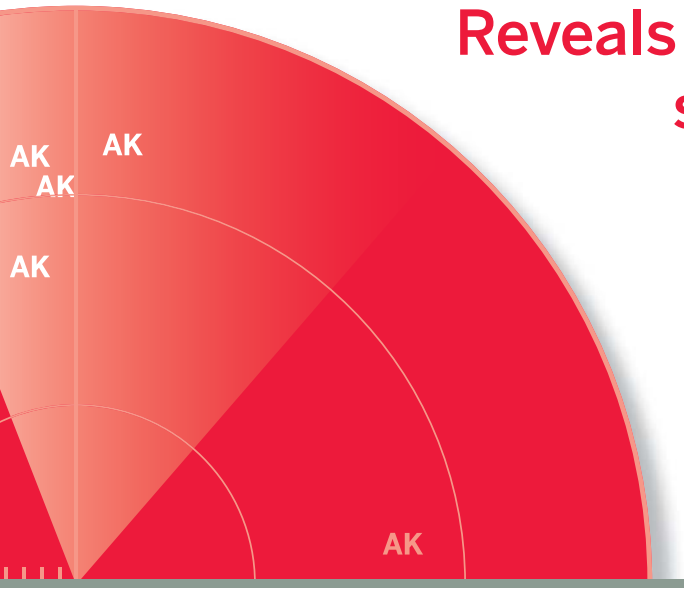
For Actinic Keratosis

ZYCLARA®

3.75% imiquimod cream

An Lmax product

Reveals and treats clinical and subclinical AK lesions[1-4]



Zyclara 3.75% cream (imiquimod). Indications: Zyclara is indicated for the topical treatment of clinically typical, nonhyperkeratotic, nonhypertrophic, visible or palpable actinic keratosis (AK) of the full face or balding scalp in immunocompetent adults when other topical treatment options are contraindicated or less appropriate. **Dosage:** Treatment should be initiated and monitored by a physician. Apply up to 2 sachets, once daily, before bedtime to the skin of the affected treatment area for two treatment cycles of 2 weeks each separated by a 2-week no-treatment cycle or as directed by the physician. The treatment area is the full face or balding scalp. The safety and efficacy of imiquimod in AK in children and adolescents below the age of 18 years have not been established. For external use only. Contact with eyes, lips, and nostrils should be avoided. The treatment area should not be bandaged or otherwise occluded. Apply as a thin film to the entire treatment area and rub in until the cream vanishes. Partially-used sachets should be discarded and not reused. Leave on the skin for approximately 8 hours; after this time it is essential that the cream is removed by washing the area and the hands with mild soap and water. **Contraindications:** Hypersensitivity to the active substance or to any of the excipients. **Warnings and precautions:** Lesions clinically atypical for AK or suspicious for malignancy should be biopsied to determine appropriate treatment. Not recommended until the skin has healed after any previous medicinal products or surgical treatment. Use of sunscreen is encouraged, and patients should minimise or avoid exposure to natural or artificial sunlight. Not recommended for the treatment of AK lesions with marked hyperkeratosis or hypertrophy as seen in cutaneous horns. During therapy and until healed, affected skin is likely to appear noticeably different from normal skin. Local skin reactions are common but generally decrease in intensity during therapy or resolve after cessation of therapy. Rarely, intense local inflammatory reactions including skin weeping or erosion can occur after only a few applications. There is an association between the complete clearance rate and the intensity of local skin reactions. These local skin reactions may be related to the stimulation of local immune response. Imiquimod has the potential to exacerbate inflammatory conditions of the skin. If required by the patient's discomfort or the intensity of the local skin reaction, a rest period of several days may be taken. Treatment can be resumed after the skin reaction has moderated. The intensity of the local skin reactions tend to be lower in the second cycle than in the first treatment cycle. Flu-like systemic signs and symptoms may accompany, or even precede, intense local skin reactions and may include fatigue, nausea, fever, myalgias, arthralgias, and chills. An interruption of dosing or dose adjustment should be considered. Use with caution in patients with reduced haematologic reserve. Patients with cardiac, hepatic or renal impairment were not included in clinical trials. Caution should be exercised in these patients. Use with caution in immunocompromised patients and/or patients with autoimmune conditions and consider balancing the benefit of treatment for these patients with the risk associated either with the possibility of organ rejection or graft-versus-host disease or a possible worsening of their autoimmune condition. No data are available on re-treating AK that have cleared after two cycles of treatment and subsequently recur. Stearyl alcohol and cetyl alcohol may cause local skin reactions. Methyl parahydroxybenzoate (E 218), and propyl parahydroxybenzoate (E 216) may cause allergic reactions (possibly delayed). No interaction studies have been performed but use with caution in patients who are receiving immunosuppressive drugs. Avoid using with any other imiquimod creams in the same treatment area. No data are available on the use of Zyclara during pregnancy or breast-feeding and there are no data on the risk to human fertility. There is no or negligible influence on the ability to drive or use machinery. **Side effects:** Herpes simplex, skin infection, lymphadenopathy, haemoglobin, white blood cell and platelet counts decreased, anorexia, blood glucose increased, insomnia, depression, headache, dizziness, nausea, diarrhoea, vomiting, erythema, scab, skin exfoliation, skin oedema, skin ulcer, skin hypopigmentation, dermatitis, erythema multiforme, Stevens Johnson syndrome, cutaneous lupus erythematosus, skin hyperpigmentation, myalgia, arthralgia, application site effects, including erythema, scabbing, exfoliation, dryness, oedema, ulcer, discharge, reaction, pruritus, swelling, burning, irritation and rash, fatigue, pyrexia, influenza like illness, pain, chest pain. Consult the Summary of Product Characteristics before prescribing, particularly in relation to side effects, precautions and contraindications. **Legal Category:** POM. **Package quantity and basic NHS price:** Pack of 28 sachets £113.00. **Product licence number:** EU/1/12/783/002. **Marketing authorisation holder:** Meda AB, Pipers väg 2A, 170 73 Solna, Sweden. **Date of preparation of prescribing information:** January 2013. UK/ZYC/13/0003

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

References: 1. Zyclara 3.75% Cream, Summary of Product Characteristics; Meda, August 2012. **2.** Stockfleth E and Alomar A. Poster Presented at 6th World Meeting of Interdisciplinary Melanoma Skin Cancer Centres & 8th EADO Congress 2012, Barcelona. **3.** Swanson N *et al.* J Am Acad Dermatol 2010; **62**(4): 582-590. **4.** Hanke CW *et al.* J Drugs Dermatol 2011; **10**(2): 165-170.

UK/ZYC/13/0005e Date of preparation: March 2013.

MEDA

IRISH SKIN FOUNDATION

SKIN AWARENESS WEEK

MONDAY 28th OCTOBER - SATURDAY 2nd NOVEMBER

including

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SATURDAY, 2nd NOVEMBER 2013

St. Stephen's Green Shopping Centre (Top Floor)
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- Psoriasis Talk Prof. Brian Kirby, St. Vincent's Hospital
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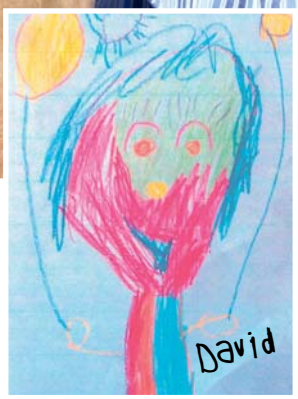

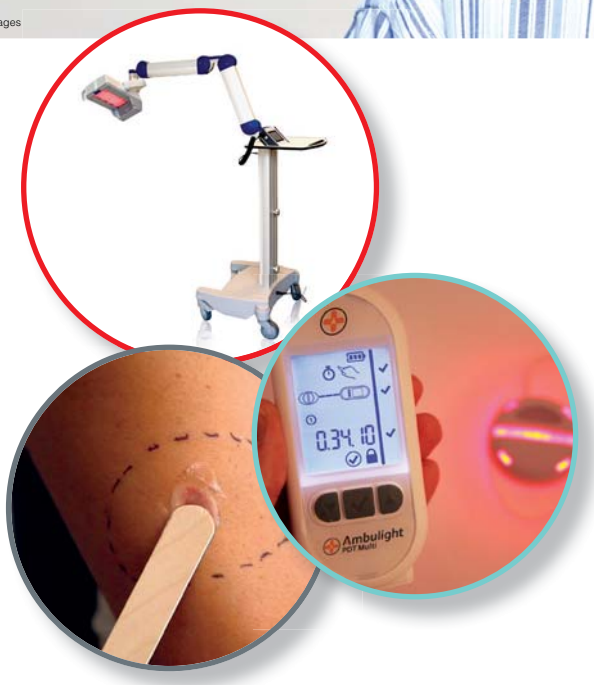

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Item no: SH/AMLZ/299/APR13 Date of preparation: 10th April 2013

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Royal Victoria Hospital, Belfast

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who was followed by:

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1973/5 Dr D.B. Buckley

1975/7 Prof D. Burrows

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Rogers Symposium

01. 11.30am **Psychological distress and quality of life in hidradenitis suppurativa.**
G Kelly1, CM Sweeney1, A Lally1, AM Tobin2, B Kirby1.
1Dermatology Research Group, St. Vincent's Hospital, Dublin 4.
2Adelaide and Meath Hospital, Tallaght, Dublin 24.

02. 11.40am **What is the relevance of contact allergy to sodium metabisulfite and which concentration of the allergen should we use?**
Nicola Ralph, Saroj Verma, Subha Merry, Brian Kirby, Aoife Lally, Paul Collins.
St Vincent's University Hospital, Dublin.
St Vincent's University Hospital, Dublin

03. 11.50am **Case mix and referral patterns of general practitioner graduates of the UCD Diploma in Dermatology.**
McDonald I, Fahy C, Powell F, Moloney F.
Dermatology Dept, Mater Misericordiae University Hospital, Dublin.

04. 12noon **First experiences using in vivo reflectance confocal microscopy on melanocytic lesions in Ireland – the bridge between dermoscopy and histopathology.**
M Sadlier, A Alani, K Ahmad, B Ramsay. Department of Dermatology
University Hospital Limerick

05. 12.10pm **A study of the Dermatology Consult Service at a Dublin Teaching Hospital.**
Verma S, Reid C, Eustace K, Hughes R, Keating C, Lally A, Kirby B, Collins P.
St Vincent's University Hospital, Dublin 4.

06. 12.20pm **Drug survival of fumaric acid esters for psoriasis: A retrospective study.**
Nuriah Ismail MB, Paul Collins MD, Sarah Rogers MD, Brian Kirby MD &
Aoife Lally MD. Department of Dermatology, St. Vincent's University Hospital,
Elm Park, Dublin 4, Ireland.

07. 12.30pm **Audit of serum vitamin D3 levels in patients with cutaneous lupus erythematosus.**
Eoin Storan, Bairbre Wynne.
Department of dermatology, St. James's Hospital, Dublin 8.

08. 12.40pm **Methylisothiazinoline sensitization: recent patch test results from North Leinster.**
R. Hellen, L. Jennings, CA. Egan.
Our Lady of Lourdes Hospital, Drogheda, Co Louth.

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Source: "IMS HEALTH – Pharmatrend Database including France, Germany, Italy, Spain, Switzerland, Austria, Belgium and Portugal – Dermo-cosmetic market (including beauty products for men, beauty products for women, unisex beauty products, personal hygiene products, hair products, baby hygiene and care products) – December 2012 MAT – In volume and Value."

Oral Presentations

ORAL ABSTRACT 01

A rare adverse event in TL – 01 phototherapy: A case series of 4 patients. Fahy, C.M.R., McDonald I, O'Donnell B, Gaynor L, Murphy G.M, Lenane P, Moloney F. National Photobiology Unit, Mater Misericordiae University Hospital, Dublin.

Narrow – band UVB (nb – UVB) phototherapy or TL – 01 is often used as a first line treatment in psoriasis. The emitted range (311 – 313 nm) has been shown to be optimal for anti – psoriatic activity. An unusual side - effect of nb – UVB is the development of blisters on psoriatic lesions during a treatment course. We report the occurrence of 4 such cases.

Two female and 2 male patients, ranging in age from 23 – 59 years, were affected. All developed psoriatic lesional bullae during treatment with TL – 01 phototherapy. Bullae developed from the 14th to the 37th treatment. Surrounding skin of all the affected lesions remained normal. Histopathology of a bulla from Case 2 demonstrated an intraepidermal vesicular dermatitis with suprabasilar epidermal necrosis and mild peri – vascular inflammation. The bullae resolved spontaneously and phototherapy was successfully recommenced.

TL - 01 phototherapy is more effective than broadband UVB in the treatment of psoriasis but higher levels of radiation are required with a steeper curve of phototoxicity when therapeutic doses are exceeded. This rare side - effect of blistering, occuring on solely the psoriatic plaques, was initially described by George and Ferguson in 1992.

All cases since described, including this case series, have common features such as: blistering occurring on the psoriatic plaques or papules, sparing of surrounding skin, negative auto - immune serology; negative porphyrin screens; negative direct immunofluorescence and similar histopathology demonstrating epidermal necrosis and mild perivascular inflammation. All have had a benign course of bullous eruption after resolution of which phototherapy was successfully resumed at lower doses. All reported cases of these eruptions occurred in Europe under a minimal erythema dosing, triweekly treatment protocol.

Suggested hypothesis includes loss of photoprotection where changes such as the increase in stratum corneum thickness and pigmentation, which occurs in the surrounding skin during the treatment course have not developed in the healing psoriatic plaque. It is thought that the incidence of such blistering may be higher than reported, but are often ignored by patients due to the benign course and rapid resolution of the bullae. Awareness of this adverse effect is important among both dermatologists and primary care physicians alike.

ORAL ABSTRACT 02

A rare case of Adiposis Dolorosa presenting in an acute setting in young male.

Q. Razali, C. Kiely, M. Connolly, R. Hughes, AM. Tobin. Adelaide and Meath Hospital, Tallaght

Adiposis dolorosa(AD) is a rare disorder of fatty tissue of unknown aetiology characterised by multiple painful lipomas. It usually occurs in obese postmenopausal females aged between 45 and 60 years. We report an unusual case of AD presenting acutely in an otherwise healthy man.

A 39 year old man presented to the emergency department with severely painful subcutaneous nodules. The nodules had become apparent 18 months previously but had only become painful in the past 3 months. The lesions restricted movement and caused difficulty sleeping. He also complained of bilateral upper limb weakness occurring intermittently in the past month. He was not overweight and had no significant past medical history. He had no family history of cutaneous disease.

On examination, there were multiple deforming swellings of the upper limbs, trunk and thighs varying in size from 2cm to 10cm. Over the left flank and lumbar region there was marked fullness caused by subcutaneous masses. The nodules were tense and exquisitely tender with normal overlying skin. Incidentally the patient also had vitiligo on the dorsal aspect of the hands and eyelids which was of recent onset. The nodules spared the face, neck, hands and lower legs. His neurological exam and laboratory investigations including blood glucose were normal. Opiate analgesia was commenced but has not been effective in alleviating his pain and the patient continues to be functionally disabled by enlarging nodules.

The diagnosis of Adiposis dolorosa is based on the clinical features of painful lipomas and neurological symptoms consistent with our patients presentation. Magnetic Resononence Imaging and Ultrasound with histopathology may be useful in supporting the diagnosis. It most often occurs in overweight postmenopausal women. Our case is an unusual acute presentation of this rare syndrome in an otherwise healthy 39 year old man. AD was first recognised in 1892 by Francis Xavier Dercum. There are still no known treatments that will halt the progression of the disease. Treatments that have been used include steroids, lidocaine, interferon, metformin and liposuction surgery. In addition depression and sleep disturbance can complicate care. Adiposis dolorosa presents numerous clinical challenges due to nerve impingement, joint mobility and pain that is refractory to standard therapies.

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ORAL ABSTRACT 03

Treatment resistant Nodular purigo which resolved during coincidental treatment with a glucagon-like peptide-1 agonist.

R Hughes1, C Kiely1, M Connolly1, J Gibney2, AM Tobin1Departments of Dermatology1 and Endocrinology2, AMNCH, Tallaght.

A forty-eight year old lady was referred to the dermatology service with a two-month history of widespread excoriated papules and nodules, with associated intense pruritus. A biopsy confirmed a diagnosis of nodular purigo and she was treated with a potent topical steroid but failed to respond adequately. She completed a one-month course of narrow band UVB phototherapy. Her pruritus improved however her symptoms returned within several weeks of stopping. She continued to use topical steroids with limited effect. Shortly after her purigo had returned to its full intensity, the patient was commenced on Victosa® for the treatment of longstanding type 2 diabetes. Within one month, and with no other change to her treatment regimen, the patient's nodular purigo cleared completely. She remained on victosa for 5 months and during this time her nodular purigo remained completely clear. Unfortunately, after 5 months of treatment, victosa was discontinued due to nausea. Approximately 3 weeks later, her symptoms of pruritus recommenced and within one month the nodular purigo had returned.

Liraglutide, marketed under the brand name Victoza®, is a long-acting glucagon-like peptide-1 agonist (GLP-1 agonist) developed for the treatment of type 2 diabetes. Two cases of improvement in psoriasis in patients treated with GLP-1 agonists, for type 2-diabetes have been reported. In both cases, an improvement in puritus was the intial indication of effect. There are no reported associations between GLP-1 agonists and nodular purigo.

We report a case of treatment resistant nodular purigo which appeared to resolve completely during coincidental treatment with Victosa and relapsed upon stopping the drug. We feel this case indicates a need for further investigation of the anti-pruritic effect of GLP-1 analogues.

ORAL ABSTRACT 04

Eccrine hidrocystomas of the nose treated with intralesional botulinim toxin.

R Hughes1, C Keating1, R Barry2, A Lally1.
1.Department of dermatology, St Vincent's University hospital ,
2. Department of dermatology , St/ James' Hospital.
R Hughes1, K Sweeney2, S Foley1, A Ormonde1, A Irvine2, R Watson1

Our Lady's Children's Hospital Crumlin1, St James's Hospital2

A forty three year old gentleman presented with a twelve-year history of multiple lesions affecting the nose. The lesions developed over 12-24 months and had subsequently remained unchanged. They were asymptomatic. He denied any history of trauma to the area and was unaware of any seasonal variation in the lesions. Clinically, there were multiple (more than 20) discrete, blue-grey cystic papules affecting the nasal bridge, alae and lateral nasal walls. A biopsy from a representative lesion showed a benign hidrocystoma of the eccrine type.

Eccrine hidrocystomas are rare, benign tumors produced by mature, deformed, eccrine sweat units. They are usually found on the face, most often in a periorbital distribuition. Treatment of solitary lesions can be easily achieved surgically however multiple lesions are more problematic due to sheer number and location. Our patient had attempted treatment by various cosmetic procedures, including facial peels and dermabrasion. None had been successful. We treated him with 0.1 ml of intralesional botulinum toxin (Botox®) per lesion. This process was repeated 3 months later. Two months after his first treatment the lesions were noticeably smaller.

Reported non-surgical treatment options for hidrocystomas include intralesional scopolamine, topical 1% atropine and pulsed dye laser. Each of these treatments has its limitations. Scopolamine and atropine are limited by side effects including nausea and photophobia. Pulsed dye laser carries a risk of scarring. To date there are 4 reported cases of hidrocystoms treated with intralesional botulin toxin. Botox® is easy to administer, carries no risk of scarring and is well tolerated. We present a case of effective treatment of eccrine hidrocystoma with local injection of botox® with excellent results.

ORAL ABSTRACT 05

Subacute Cutaneous Lupus Erythematosus: A Paraneoplastic Phenomenon in Oropharyngeal Squamous Cell Carcinoma.

A. Murad1, H. Rowley2, N. Mulligan3, B. O Donnell1
Department of Dermatology1, Ear Nose and Throat2 and Histopathology3, Mater Misericordiae University Hospital, Dublin, Ireland.

A 74-year-old man presented with a 1 month history of a gradually enlarging mass on his right supraclavicular region and weight lost. He also had a pruritic rash with papules and plaques involving his forehead, chest and back. Mild periungual erythema was noted on his fingers. Imaging of his neck revealed a 6.5 x 4.8 x 8.5 cm enhancing mass with a necrotic centre. The tumour arose from the base of the tongue and involved the right masticator space, parapharyngeal space and right pyriform fossa.

Histology of biopsy sample showed a squamous cell carcinoma (SCC). Skin histology showed vacuolar degeneration of the basal layer, papillary oedema with marked periadnexal and perivascular lymphocytic infiltrate in the dermis. There were also scale crust formation, prominent epidermal atrophy and necrotic keratinocytes. Blood investigations showed nucleolar pattern antinuclear antibod-

ies and a positive extractable nuclear antigen, anti-RO antibody. Creatine kinase, anti-JO-1 and immunofluorescence studies were negative. His clinical presentation and histological features were consistent with paraneoplastic subacute cutaneous lupus erythematosus (SCLE).

Paraneoplastic syndrome in head and neck cancers and in particular, oropharyngeal SCC is very rare. Only endocrine and dermatological manifestations have been reported as paraneoplastic syndrome in association with oral cancers. Amongst these, hypercalcemia is the most common and is associated with less favourable prognosis. Several cases of paraneoplastic SCLE have been reported in relation to malignancy of the lung, breast, stomach, uterus, liver and also lymphoma. Laryngeal SCC was the first reported case of head and neck malignancy with this association1. Paraneoplastic SCLE is less responsive to conventional therapy but usually resolves with treatment of the underlying malignancy. This patient underwent palliative radiotherapy for his locally advanced SCC. His rash was controlled with topical steroid treatment and photoprotection without requiring systemic treatment.

Reference:
1) Chaudhry SI, Murphy LA, White IR. Subacute cutaneous lupus erythematosus: a paraneoplastic dermatosis? Clin Exp Dermatol 2005;30(6):655-8.

ORAL ABSTRACT 06
Biologics induced bullous pemphigoid.
C. Kiely, M. Connolly, Q. Razali, R. Hughes, AM. Tobin.
Adelaide and Meath Hospital, Tallaght

Biologic agents are being used to treat an increasing array of inflammatory diseases. Auto-immune diseases attributable to these agents have been rarely reported. We report a case of bullous pemphigoid (BP) secondary to two different classes of anti-TNF in a patient with inflammatory bowel disease.

A forty-two year old woman presented with multiple 2 -15 mm violaceous non-pruritic plaques on the trunk and limbs. She had Crohn's disease and Ankylosing spondylitis and was treated with adalimumab 40mg subcutaneously fortnightly, mesalasine 2 grams daily and budesonide 9mg daily.

Three weeks prior to presentation her inflammatory bowel disease flared, adalimumab was increased to weekly dosing and high dose IV steroids were started. The rash became apparent as the steroids were weaned. A moderate perivascular lymphocytic infiltrate with numerous eosinophils was seen on histology from a lesion. The initial clinical impression was erythema multiforme secondary to adalimumab. Adalimumab was held and steroids increased. Azathioprine was introduced as an alternative immunosupressive agent. During the next 2 weeks the skin eruption settled, however her bowel symptoms continued to flare. Infliximab infusions were commenced. Azathioprine was discontinued due to abnormal liver function tests. Three Infliximab infusions were given over the following 6 weeks and steroids were weaned. After the third infliximab infusion her rash recurred. It was clinically indistinguishable from the initial eruption however this time it evolved within 2 days into a bullous eruption. Histology from a lesion demonstrated sub-epidermal bullae containing eosinophils, consistent with bullous pemphigoid. Serum indirect immunoflourescence was positive for bullous pemphigoid antibodies. Infliximab was stopped as the likely causal agent. The patients predinisolone was increased to 1mg/kg and minocycline 100mgs daily was commenced. The bullous eruption settled and steroids were slowly weaned by 2.5mg weekly over the next 6 weeks. Her bullous pemphigoid remains quiescent.

Bullous pemphigoid (BP) is an autoimmune disease characterised by subepidermal blistering most often affecting the elderly typically between the ages of 60–80 years. BP initially can present as urticarial lesions and may take weeks to months to erupt into bullae. More than 1500 cases of autoimmune diseases induced by biologics have been reported. The most frequent are lupus, vasculitis and sarcoidosis. There have been 2 previously reported cases of BP caused by adalimumab. This is the first reported case of BP due to infliximab.

ORAL ABSTRACT 07
Cutaneous T-cell lymphoma in a 13-year-old boy
S.J. O'Shea, B. O'Donnell
Department of Dermatology, Children's University Hospital, Temple Street, Dublin.

A 13-year-old boy presented in 2008 with a one-year history of a rash. His parents described the first lesion as a small, itchy, red, discoid patch on his back that later faded to a yellow/brown colour. The rash gradually spread to involve the arms and legs. He had a past history of asthma and oesophagitis.

On presentation, there was a yellow patch in the left infrascapular area and scattered lesions on the mid-back with minimal scale. The differential diagnosis at the time included pigmented purpuric dermatosis, clinically most suggestive of lichen aureus, and pityriasis versicolor. Skin scrapings were negative for fungus. Histology revealed a band-like chronic inflammatory infiltrate in the superficial dermis with interface change and vacuolar degeneration of keratinocytes. A repeat biopsy one month later was most consistent with pityriasis lichenoides chronica, although this was at variance with the clinical picture.

The patient was later seen at joint consultation and subacute eczema was suspected, however, the rash failed to respond to clobetasone butyrate 0.05%, betamethasone 0.025%, mometasone furoate ointment and emollients.

Repeat histology 5 years after the initial presentation, showed hyperkeratosis, parakeratosis, multiple foci of epidermotropism without spongiosis and Pautrier microabscesses were noted, suggesting the diagnosis of mycosis fungoides.

FBC, blood film, renal, liver profile, TFTs, CRP, ESR and LDH were normal. ANA, ENA, HTLV I/II and EBV IgM negative. CT neck, thorax, abdomen and pelvis showed subcentimetre lymph nodes in both axillary and inguinal regions. T-cell receptor gene rearrangements showed a polyclonal T cell population, failing to provide molecular support for mycosis fungoides. The patient has commenced a course of oral 8-MOP PUVA for presumed cutaneous T-cell lymphoma.

Mycosis fungoides is rare in children but the incidence is rising. Studies suggest that about 60% children present with patch stage disease, sometimes with unusual variants e.g. hypopigmented and purpuric types.1, 2 In children, there is often a delay in diagnosis and the rash tends to slowly evolve, with a better prognosis.1

References

Yazganoglu KD, Topkarci Z, Buyukbabani et al. Childhood mycosis fungoides: a report of 20 cases from Turkey. JEADV 2013;27:295-300.
Pope E, Weitzman S, Ngan B et al. Mycosis fungoides in the pediatric population: report from an international Childhood Registry of Cutaneous Lymphoma. J Cutan Med Surg. 2010;14(1):1-6.

ORAL ABSTRACT 08

An unusual purpuric eruption in a young girl

S.J. O'Shea, B. O'Donnell
Department of Dermatology, Children's University Hospital, Temple Street, Dublin.

A 5-year-old girl presented with a purpuric eruption of a few weeks' duration. Her mother had first noted a single, bruise-like patch on her neck/upper chest. Within two weeks, a generalized eruption developed consisting of several, smaller purpuric macules on the trunk and limbs. Apart from one episode of vomiting a fortnight before, the girl was otherwise well. There was no past medical history and no medications.

On examination, a purpuric macular rash was noted on the neck, trunk, upper and lower limbs. The rash followed skin cleavage lines on the trunk but lacked a collarette of scale. Mucous membranes were spared.

Urinalysis and MSU were normal. Baseline full blood count, bioprofile, C-reactive protein and coagulation screen were normal. Immunoglobulins, anti-streptolysin O titre, C_3, C_4, ANA, ENA and ANCA were normal or negative.

Histology showed mild orthokeratosis. There was a dermal, perivascular, lymphocytic infiltrate and surrounding red cell extravasation without vasculitis. Iron and mast cell stains are awaited.
This case was unusual in presentation. The differential diagnoses considered were the purpuric variant of pityriasis rosea and generalized pigmented purpuric dermatosis.

A number of atypical presentations of pityriasis rosea have been described including papular, vesicular, purpuric, inverse, limb-girdle type and pityriasis rosea circinata et marginata of Vidal.1 Comparison of our histology with other cases of suspected purpuric pityriasis rosea is limited as most are short-lived and a skin biopsy is not routinely done. Hyperkeratosis, patchy parakeratosis, spongiosis and a perivascular lymphocytic infiltrate involving the upper dermis are features of purpuric pityriasis rosea.

A generalized pigmented purpuric dermatosis was also considered. Schamberg's disease is often limited to the lower limbs but a generalized form responsive to phototherapy has been seen in children.2 In this case, the classical collarette of scale associated with pityriasis rosea was absent. This would explain the lack of epidermal involvement on histology. The history of a herald patch and the distribution along skin cleavage lines is suggestive of purpuric pityriasis rosea. As the epidermis was normal, a generalized form of Schamberg's disease would also be possible. We present an unusual purpuric eruption in a young girl that presented a diagnostic challenge.

References

Chuh A, Zawar V, Lee A. Atypical presentations of pityriasis rosea: case presentations. JEADV 2005;19:120-6.
Can B, Turkoglu Z, Kavala M et al. Successful treatment of generalized childhood Schamberg's disease with narrowband ultraviolet B therapy. Photodermatol Photoimmunol Photomed 2011:27(4):216-8.

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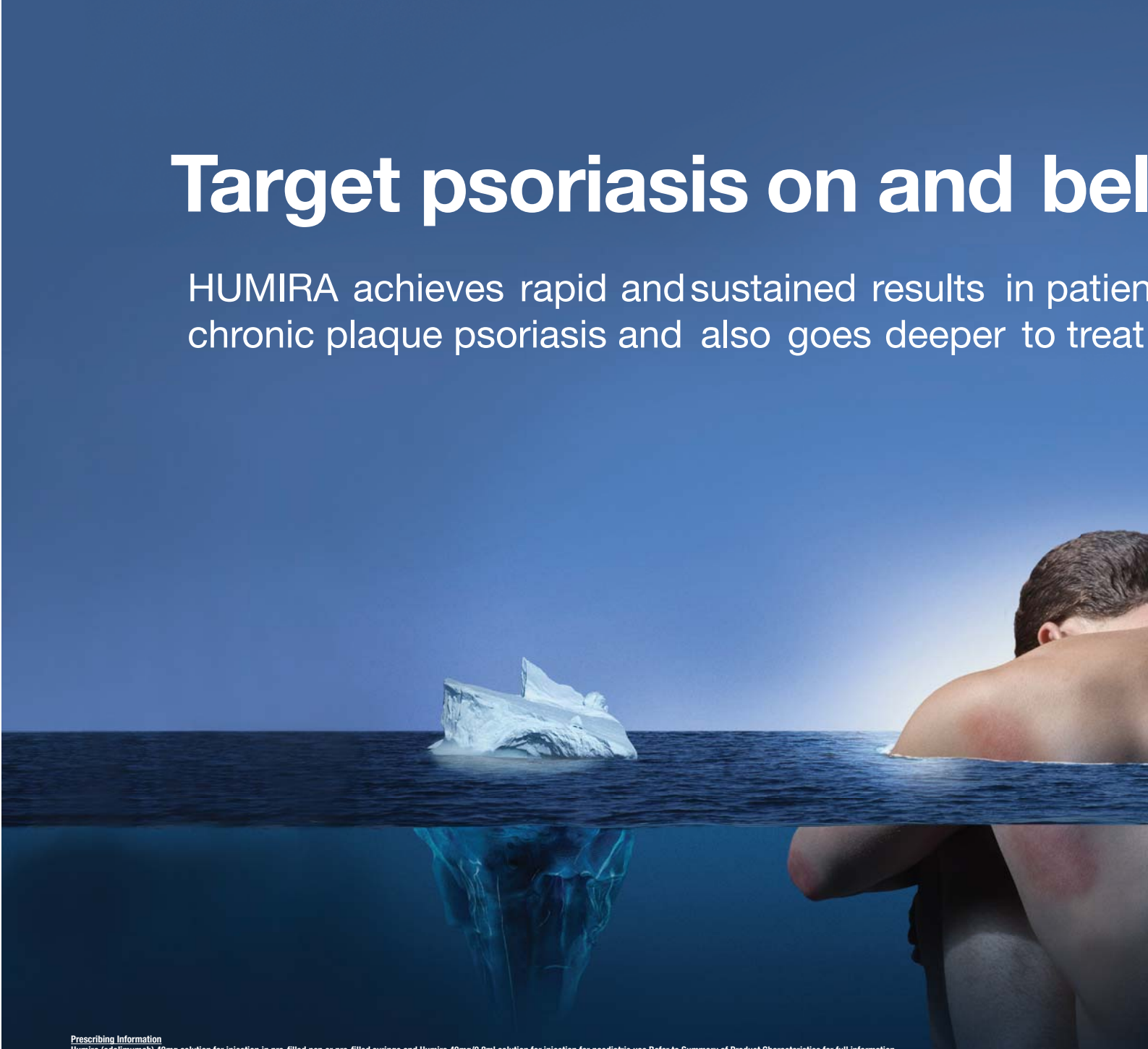

Prescribing Information

Humira (adalimumab) 40mg solution for injection in pre-filled pen or pre-filled syringe and Humira 40mg/0.8ml solution for injection for paediatric use Refer to Summary of Product Characteristics for full information.

Presentation: Each 0.8ml single dose pre-filled pen, pre-filled syringe or vial contains 40mg of adalimumab. **Indications:** Rheumatoid arthritis (RA): Humira in combination with methotrexate is indicated for the treatment of moderate to severe, active RA in adult patients when the response to disease-modifying anti-rheumatic drugs (DMARDs) including methotrexate has been inadequate. Humira is also indicated for the treatment of severe, active and progressive RA in adults not previously treated with methotrexate. Humira can be given as monotherapy in case of intolerance to methotrexate or when continued treatment with methotrexate is inappropriate. Humira has been shown to reduce the rate of progression of joint damage as measured by X-ray and to improve physical function, when given in combination with methotrexate. Polyarticular juvenile idiopathic arthritis (JIA): Humira in combination with methotrexate is indicated for the treatment of active JIA, in children and adolescents aged 2 to 17 years who have had an inadequate response to one or more DMARDs. Humira can be given as monotherapy in case of intolerance to methotrexate or when continued treatment with methotrexate is inappropriate. Psoriatic arthritis (PsA): Humira is indicated for the treatment of active and progressive PsA 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. Ankylosing spondylitis (AS): Humira is indicated for the treatment of adults with severe active AS who have had an inadequate response to conventional therapy. Axial spondyloarthritis (SpA) non-radiographic: Humira is indicated for the treatment of adults with severe axial spondyloarthritis without radiographic evidence of AS but with objective signs of inflammation by elevated CRP and / or MRI, who have had an inadequate response to, or are intolerant to nonsteroidal anti-inflammatory drugs. Crohn's disease (CD): Humira is indicated for treatment of moderate to severe, active CD, in patients who have not responded despite a full and adequate course of therapy with a corticosteroid and/or an immunosuppressant; or who are intolerant to or have medical contraindications for such therapies. Paediatric Crohn's Disease: Humira is indicated for the treatment of severe active Crohn's disease in paediatric patients (6 to 17 years) who have had an inadequate response to conventional therapy including primary nutrition therapy, a corticosteroid, and an immunomodulator, or who are intolerant to or have contraindications for such therapies. Psoriasis (Ps): Humira is indicated for the treatment of moderate to severe chronic plaque psoriasis in adult patients who failed to respond to or who have a contraindication to, or are intolerant to other systemic therapy including cyclosporine, methotrexate or PUVA. Ulcerative colitis (UC): Humira is indicated for treatment of moderate to severe active ulcerative colitis in adult patients who have had an inadequate response to conventional therapy including corticosteroids and 6-mercaptopurine (6-MP) or azathioprine (AZA), or who are intolerant to or have medical contraindications for such therapies. **Dosage and administration:** Humira treatment should be initiated and supervised by specialist physicians experienced in the diagnosis and treatment of conditions for which Humira is indicated. Patients treated with Humira should be given the special alert card. After proper training in injection technique, patients may self-inject with Humira if their physician determines that it is appropriate and with medical follow-up as necessary. During treatment with Humira, other concomitant therapies (e.g., corticosteroids and/or immunomodulatory agents) should be optimised. RA, PsA, AS or SpA non-radiographic: 40mg administered every other week as a single dose via subcutaneous injection. RA: In monotherapy some patients who experience a decrease in their response to Humira may benefit from an increase in dose intensity to 40mg every week. There may be a need for dose interruption, for instance before surgery or if a serious infection occurs. Available data suggest that re-introduction of Humira after discontinuation for 70 days or longer resulted in the same magnitudes of clinical response and similar safety profile as before dose interruption. For RA, JIA, PsA and AS, available data suggest that the clinical response is usually achieved within 12 weeks of treatment. Continued therapy should be carefully reconsidered in a patient not responding within this time period. JIA: Age 2 to 12 years: 24mg/m² body surface area to a maximum single dose of 40mg administered every other week via subcutaneous injection. The volume for injection is based on the patients' height and weight (see SmPC for height and weight dosing chart). A 40mg paediatric vial is available for patients who need to administer less than the full 40mg dose. Age 13 to 17 years: 40mg administered every other week via subcutaneous injection regardless of body surface area. CD: The recommended Humira induction dose regimen for adult patients with moderate to severe CD is 80mg at Week 0 followed by 40mg at Week 2. In case there is a need for a more rapid response to therapy, the regimen 160mg at Week 0 (dose can be administered as four injections in one day or as two injections per day for two consecutive days), 80mg at Week 2, can be used with the awareness that the risk for adverse events is higher during induction. After induction treatment, the recommended dose is 40mg every other week via subcutaneous injection. Alternatively, if a patient has stopped Humira and signs and symptoms of disease recur, Humira may be re-administered. There is little experience from re-administration after more than 8 weeks since the previous dose. During maintenance treatment, corticosteroids may be tapered in accordance with clinical practice guidelines. Some patients who experience decrease in their response may benefit from an increase in dosing frequency to 40mg Humira every week. Some patients who have not responded by Week 4 may benefit from continued maintenance therapy through Week 12. Continued therapy should be carefully reconsidered in a patient not responding within this time period. Paediatric CD: patients <40Kg: recommended induction dose regimen of 40mg at Week 0 followed by 20mg at Week 2 via SC injection. In case of need for a more rapid response to therapy, the regimen 80mg at Week 0 (dose can be administered as two injections in one day), 40mg at Week 2, can be used with the awareness that the risk for adverse events is higher during induction. After induction treatment, the recommended dose is 20mg every other week via SC injection. Some patients who experience insufficient response may benefit from 20mg every week; patients >40Kg: double the dose regimen for those patients <40Kg.Continued therapy should be carefully considered in a subject not responding by week 12. Psoriasis: The recommended dose of Humira for adult patients is an initial dose of 80mg administered subcutaneously, followed by 40mg subcutaneously given every other week starting one week after the initial dose. Continued therapy beyond 16 weeks should be carefully reconsidered in a patient not responding within this time period. UC: The recommended Humira induction dose regimen for adult patients with moderate to severe UC is 160mg at week 0 (dose can be administered as four injections in one day or as two injections per day for two consecutive days) and 80mg at week 2. After induction treatment, the recommended dose is 40mg every other week via subcutaneous injection. During maintenance treatment, corticosteroids may be tapered in accordance with clinical practice guidelines. Some patients who experience decrease in their response may benefit from an increase in dosing frequency to 40mg Humira every week. Available data suggest that clinical response is usually achieved within 2-8 weeks of treatment.Continued therapy is not recommended in patients not responding within this time period. **Contraindications:** Active tuberculosis or other severe infections such as sepsis, and opportunistic infections; moderate to severe heart failure (NYHA class III/IV) and hypersensitivity to adalimumab or any of the excipients. **Precautions and Warnings:** *Infections:* Patients taking TNF-antagonists are more susceptible to serious infections. Impaired lung function may increase the risk for developing infections. Patients must therefore be monitored closely for infections, including tuberculosis, before, during and for 4 months after treatment with Humira. Treatment with Humira should not be initiated in patients with active, chronic or localised infections until infections are controlled. In patients who have been exposed to tuberculosis and patients who have travelled in areas of high risk of tuberculosis or endemic mycoses, such as histoplasmosis, coccidioidomycosis, or blastomycosis, the risk and benefits of treatment with Humira should be considered prior to initiating therapy (see ***Opportunistic infections***). Patients who develop a new infection while undergoing treatment with Humira should be monitored closely and undergo a complete diagnostic evaluation. Administration of Humira should be discontinued if a patient develops a new serious infection or sepsis, and appropriate antimicrobial or antifungal therapy should be initiated until the infection is controlled. Physicians should exercise caution when considering the use of Humira in patients with a history of recurring infection or with underlying conditions which may predispose to infections, including the use of concomitant immunosuppressive medications. *Serious infections:* Serious infections, including sepsis, due to bacterial, mycobacterial, invasive fungal, parasitic, viral, or other opportunistic infections such as listeriosis, legionellosis and pneumocystis have been reported in patients receiving Humira. Other serious infections seen in clinical trials include pneumonia, pyelonephritis, septic arthritis and septicaemia. Hospitalisation or fatal outcomes associated with infections have been reported. *Tuberculosis:* Tuberculosis, including reactivation and new onset of tuberculosis, has been reported in patients receiving Humira. Reports included cases of pulmonary and extra-pulmonary i.e. disseminated. Before initiation of therapy with Humira, all patients must be evaluated for both active or inactive ('latent') tuberculosis infection. Appropriate screening tests, should be performed in all patients, local recommendations may apply. If active tuberculosis is diagnosed, Humira therapy must not be initiated. If latent tuberculosis is suspected, a physician with expertise in the treatment of tuberculosis should be consulted and the benefit/risk balance of therapy with Humira should be considered. If inactive ('latent') tuberculosis is diagnosed, appropriate treatment must be started with anti-tuberculosis prophylaxis treatment before the initiation of Humira, and in accordance with local recommendations. In patients who have several or significant risk factors for tuberculosis despite a negative test for tuberculosis, anti-tuberculosis prophylaxis treatment should also be considered before the initiation of Humira and in patients with a past history of latent or active tuberculosis in whom an adequate course of treatment cannot be confirmed. Some patients who have previously received treatment for latent or active tuberculosis have redeveloped tuberculosis while being treated with Humira. *Other opportunistic infections:* Opportunistic infections, including invasive fungal infections have been observed in patients receiving Humira. These infections have not consistently been recognised in patients taking TNF-antagonists and this resulted in delays in appropriate treatment, sometimes resulting in fatal outcomes. For patients who develop the signs and symptoms such as fever, malaise, weight loss, sweats, cough, dyspnoea, and/or pulmonary infiltrates or other serious systemic illness with or without concomitant shock, an invasive fungal infection should be suspected and administration

HUMIRA
adalimumab
THINK DEEP, STRIKE DEEP [1]

ow the skin with HUMIRA[1]

ts with moderate-to-severe
psoriatic arthritis[2,3]

of Humira should be promptly discontinued. Diagnosis and administration of empiric antifungal therapy in these patients should be made in consultation with a physician with appropriate expertise. *Hepatitis B Reactivation:* Reactivation of hepatitis B (HBV) has occurred in patients receiving a TNF-antagonist including Humira, who are chronic carriers of this virus (i.e. surface antigen positive), with some fatal outcomes. Patients should be tested for HBV infection before initiating treatment. Patients that test positive should have a consultation with a physician. In patients who develop HBV reactivation, Humira should be stopped and effective anti-viral therapy with appropriate supportive treatment should be initiated. Carriers of HBV should be closely monitored for signs and symptoms of active HBV infection throughout therapy and for several months following termination of Humira. *Neurological events:* Humira has been associated, in rare cases, with new onset or exacerbation of clinical symptoms and/or radiographic evidence of central nervous system demyelinating disease including multiple sclerosis and optic neuritis, and peripheral demyelinating disease, including Guillain-Barré syndrome. Caution should be exercised when considering Humira in patients with pre-existing or recent-onset central or peripheral nervous system demyelinating disorders. *Allergic reactions:* Postmarketing serious allergic reactions including anaphylaxis have been received following Humira administration. If an anaphylactic reaction or other serious allergic reaction occurs, administration of Humira should be discontinued immediately and appropriate therapy initiated. *Malignancies and lymphoproliferative disorders:* In clinical trials, more cases of malignancies including lymphoma and leukaemia have been observed among patients receiving a TNF-antagonist compared with control patients. These data cannot exclude a possible risk of malignancy in patients including children and adolescents treated with TNF antagonists. Furthermore, there is an increased background lymphoma risk in RA patients. Rare postmarketing cases of hepatosplenic T-cell lymphoma have been identified in patients treated with adalimumab. Some of these cases have occurred in young adult patients on concomitant treatment with azathioprine or 6-mercaptopurine used for inflammatory bowel disease. The potential risk with the combination of azathioprine or 6-mercaptopurine and Humira should be carefully considered. A risk for the development of hepatosplenic T-cell lymphoma in patients treated with Humira cannot be excluded. Caution should be exercised in considering Humira treatment of patients with a history of malignancy. All patients, and in particular patients with a medical history of extensive immunosuppressant therapy or psoriasis patients with a history of PUVA treatment should be examined for the presence of non-melanoma skin cancer prior to and during treatment with Humira. Caution should also be exercised when using TNF-antagonists in COPD patients, as well as in patients with increased risk of malignancies due to heavy smoking. With current data it is not known if adalimumab treatment influences the risk for developing dysplasia or colon cancer. All patients with UC who are at increased risk for dysplasia or colon carcinoma (e.g. patients with long-standing UC or primary sclerosing cholangitis), or who had a prior history of dysplasia or colon carcinoma should be screened for dysplasia at regular intervals before therapy and throughout their disease course. This evaluation should include colonscopy and biopsies per local recommendations. *Haematologic reactions:* Pancytopaenia including aplastic anaemia has rarely been reported with TNF blocking agents. Adverse events of the haematologic system, including cytopaenia (eg thrombocytopaenia, leucopaenia) have been reported with Humira. Patients should be advised to seek immediate medical attention if they develop signs and symptoms suggestive of blood dyscrasias. *Vaccinations:* Patients on Humira may receive concurrent vaccinations, except for live vaccines. It is recommended that JIA patients, if possible, be brought up to date with all immunisations in agreement with current immunisation guidelines prior to initiating Humira therapy. *Congestive heart failure:* Humira should be used with caution in patients with mild heart failure (NYHA class I/II) and discontinued in patients who develop new or worsening symptoms of congestive heart failure. *Autoimmune process:* Humira may result in the formation of autoimmune antibodies. If a patient develops symptoms suggestive of a lupus-like syndrome following treatment with Humira and is positive for antibodies against double-stranded DNA, further treatment with Humira should not be given. *Surgery:* There is limited safety experience of surgical procedures in patients treated with Humira. The long half life of Humira should be taken into consideration when a surgical procedure is planned, and the patient should be monitored for infections. *Small bowel obstruction:* Failure to respond to treatment for CD may indicate the presence of fixed fibrotic stricture that may require surgical treatment. Available data suggest that Humira does not worsen or cause strictures. *Elderly population:* The frequency of serious infections among Humira treated subjects over 65 years of age was higher than those under 65 years of age. Some of those had a fatal outcome. Particular attention regarding the risk for infection should be paid when treating the elderly. **Interactions:** Combination of adalimumab with other biologic DMARDS (e.g. anakinra and abatacept) or other TNF-antagonists is not recommended. **Pregnancy and lactation:** Administration of adalimumab is 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. Administration of live vaccines to infants exposed to adalimumab in utero is not recommended for 5 months following the mother's last adalimumab injection during pregnancy.Women must not breast-feed for at least five months after the last Humira treatment. **Driving and machinery:** Humira may have a minor influence on the ability to drive and use machines. **Side Effects:** From clinical trials unless marked '1' which indicates spontaneous reporting data.Very common ≥ 1/10: Respiratory tract infections (including lower and upper respiratory tract infection, pneumonia, sinusitis, pharyngitis, nasopharyngitis and pneumonia herpes viral), leucopaenia (including neutropaenia and agranulocytosis), anaemia, lipids increased, headache, abdominal pain, nausea and vomiting, elevated liver enzymes, rash (including exfoliative rash), musculoskeletal pain, injection site reaction (including injection site erythema). Common ≥ 1/100 to < 1/10: Systemic infections (including sepsis, candidiasis and influenza), intestinal infections (including gastroenteritis viral), skin and soft tissue infections (including paronychia, cellulitis, impetigo, necrotising fasciitis and herpes zoster), ear infections, oral infections (including herpes simplex, oral herpes and tooth infections), reproductive tract infections (including vulvovaginal mycotic infection), urinary tract infections (including pyelonephritis), fungal infections, joint infections, benign neoplasm, skin cancer excluding melanoma (including basal cell carcinoma and squamous cell carcinoma), thrombocytopaenia, leucocytosis, hypersensitivity, allergies (including seasonal allergy), hypokalaemia, uric acid increased, blood sodium abnormal, hypocalcaemia, hyperglycaemia, hypophosphotaemia, dehydration, mood alterations (including depression), anxiety, insomnia, paraesthesias (including hypoaesthesia), migraine, nerve root compression, visual impairment, conjunctivitis, blepharitis, eye swelling, vertigo, tachycardia, hypertension, flushing, haematoma, cough, asthma, dyspnoea, GI haemorrhage, dyspepsia, gastroesophageal reflux disease, sicca syndrome, worsening and new onset of psoriasis (including palmplanter pustular psoriasis) [1], alopecia[1], pruritis, urticaria, bruising (including purpura), dermatitis (including eczema), onychoclasis, hyperhydrosis, muscle spasms (including blood creatine phosphokinase increased), haematuria, renal impairment, chest pain, oedema, pyrexia[1], coagulation and bleeding disorders (including activated partial thromboplastin time prolonged), autoantibody test positive (including double stranded DNA antibody), blood lactate dehydrogenase increased, impaired healing. Uncommon ≥ 1/1000 to < 1/100: Opportunistic infections and tuberculosis (including coccidioidomycosis, histoplasmosis and mycobacterium avum complex infection), neurological infections (including viral meningitis), eye infections, bacterial infections, diverticulitis[1], lymphoma, solid organ neoplasm (including breast cancer, lung neoplasm and thyroid neoplasm), melanoma, idiopathic thrombocytopaenic purpura, sarcoidosis[1], cerebrovascular accident[1], tremor, neuropathy, diplopia, deafness, tinnitus, myocardial infarction[1], arrhythmia, congestive heart failure, pulmonary embolism[1], chronic obstructive pulmonary disease, interstitial lung disease, pneumonitis, pleural effusion[1], pancreatitis, dysphagia, face oedema, cholecystitis and cholelithiasis, bilirubin increased, hepatic steatosis, night sweats, scar, rhabdomyolysis, systemic lupus erythematosus, nocturia, erectile dysfunction, inflammation, vascular arterial occlusion, aortic aneurysm, thrombophlebitis. Rare ≥ 1/10,000 to < 1/1,000: Leukaemia[1], anaphylaxis[1], demyelinating disorders (e.g. optic neuritis, Guillain-Barré syndrome) [1], pancytopaenia, multiple sclerosis, cardiac arrest, pulmonary fibrosis[1], intestinal perforation[1], reactivation of hepatitis B[1], autoimmune hepatitis[1], erythema multiforme[1], cutaneous vasculitis[1], Stevens-Johnson syndrome[1], angioedema[1], lupus-like syndrome[1]. Very rare < 1/10,000: Liver failure[1].Not known: Hepatosplenic T-cell lymphoma[1], Merkel Cell Carcinoma[1] **Prescribers should consult the summary of product characteristics for further information on side effects. Overdose:** No dose-limiting toxicity was observed during clinical trials. The highest dose level evaluated has been multiple intravenous doses of 10 mg/kg (approximately 15 times the recommended dose). **Legal Category:** POM Marketing Authorisation Numbers/Presentations: Vial : EU/1/03/256/001 ; 1 pack contains 2 cartons each containing 1 single use vial and empty sterile injection syringe, needle and vial adapter, Pre-filled Syringe : EU/1/03/256/003; Each carton contains 2 single use pre-filled syringes in a blister, Pre-filled Pen: EU/1/03/256/008; Each carton contains 2 single use pre-filled pens in a blister. Further information is available from AbbVie Limited, Block B, Liffey Valley Office Campus, Quarryvale, Co. Dublin. Date of revision of PI: February 2013 PI/256/008

References: 1. Humira Summary of Product Characteristics available at www.medicines.ie **2.** Menter A, Tyring SK, Gordon K, et al. Adalimumab therapy for moderate to severe psoriasis: a randomized controlled phase III trial. J Am Acad Dermatol. 2008; 58(1):106-115. **3.** Gordon K, Papp K, and Poulin Y et al. Long-term efficacy and safety of Adalimumab in patients with moderate to severe psoriasis treated continuously over 3 years: Results from an open-label extension study for patients from REVEAL. J Am Acad Dermatol, 2012; 2: 241-251.

Date of preparation: September 2013 IREHUM120149(1)a

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Poster Presentations

01 Stevens Johnson Syndrome secondary to lamotrigine – A rare and severe adverse drug reaction.
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St. James's Hospital, Dublin

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Department of Dermatology, University Hospital Galway, Ireland

03 Childhood Rashes Presenting to Primary Care.
Dr Mairi Ferguson, Dr Jennifer Crawley
Royal Victoria Hospital, Belfast

04 Atypical sarcoidosis presenting with a breast mass
Keating C1,Hughes R1, Molloy E2 Lally A1
1:Department of Dermatology 2:Department of Rheumatology
St Vincents University Hospital, Dublin 4

Poster Abstracts

POSTER ABSTRACT 01
Stevens Johnson Syndrome secondary to lamotrigine –
A rare and severe adverse drug reaction.
Maguire, C, Higgins, E, Barnes, L
St. James's Hospital, Dublin

Case History
A 31-year-old woman presented with a one day history of a rapidly progressing skin and mucosal rash. She had a background history of intellectual disability and had commenced lamotrigine 16 days previously as a mood stabiliser. This had been discontinued the day prior to presentation.

Clinical examination revealed scattered targetoid plaques on her face, neck, upper torso and back, some of which were bullous. She had bilateral conjunctival hyperaemia with exudate and haemorrhagic crusting and erosions of her lips and oral mucosa. She was unwell with tachycardia and pyrexia.

Clinical impression was of Stevens-Johnson syndrome (SJS) secondary to lamotrigine.

Investigations revealed elevated white cell count with neutrophilia and elevated inflammatory markers. Skin biopsy showed focal superficial epidermal necrosis, abundant apoptotic keratinocytes, lymphocytic exocytosis and a subepidermal split.

Treatment was commenced with IV hydrocortisone, intensive skin care with emollients and oral care and topical ophthalmic treatments including regular steroid drops and lubricants. Her condition deteriorated over the next 48 hours with extension of her skin involvement and new vulval erosions. Systemic steroids were discontinued after 3 days. With continued multidisciplinary team care, she stabilised and slowly improved over the following two weeks.

This case represents a rare but serious adverse effect of lamotrigine, an increasingly used anti-epileptic and mood stabilising drug. As a result, the incidence of severe cutaneous adverse reactions attributed to this medication is rising . Early recognition and cessation of the culprit drug is important in managing SJS and so we highlight lamotrigine as a potential cause. Prompt ophthalmological, oral and gynaecological review in patients with Stevens-Johnson syndrome is imperative to minimise debilitating long term sequelae.

Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis: Assessment of Medication Risks with Emphasis on Recently Marketed Drugs. The EuroSCAR-Study, Mockenhaupt M, Viboud C, Dunant A, et al, J Invest Dermatol. 2008 Jan;128(1):35-44

POSTER ABSTRACT 02
Generalised pruritic eruption in an atopic 8 year old boy:
Lichen planus
Eilis Nic Dhonncha, Aizuri Murad, Catherine Foley, Pauline Marren
Department of Dermatology, University Hospital Galway, Ireland

An 8-year-old Nigerian boy was referred to Dermatology with a six week history of an itchy rash that started on his legs and spread to his trunk and arms. He was known to have mild eczema and asthma. There was a family history of atopy. Examination showed excoriated papules on his trunk and extensor surfaces of his limbs, psoriasiform and lichenified plaques on the back. There were no abnormalities of his nails, scalp or mucous membranes. Apart from mild eosinophilia, his ANA, ENA, complement levels, HIV serology,

Poster Abstracts

viral hepatitis serology, liver and renal function were normal. Skin histology showed hyperkeratosis, patchy hypergranulosis, rete peg elongation, interface inflammation and upper dermal lichenoid inflammation. Clinical and histological features were consistent with lichen planus.

Lichen planus is an inflammatory disease of unknown aetiology typically occurring in adults, with a rare occurrence of only 2-3% in patients less than fourteen years of age1. In children, the clinical appearance is usually atypical. Although majority of cases are idiopathic, there have been reports of childhood lichen planus following Hepatitis B vaccination. Oral and nail involvement are rare. Corticosteroids, topical and systemic, are the mainstay of treatment2. Other systemic treatments that have been used in children include dapsone, acitretin, and griseofulvin. Our patient was treated with oral prednisolone (0.5mg/kg/day), topical betamethasone valerate 0.1% and emollients with good response. Majority of cases reported to have resolved within one year, although post-inflammatory hyperpigmentation may persist for longer in dark skinned children. Clinical variation in childhood lichen planus and therapeutic options are presented.

References:

1) Brice SL, Barr RJ, Rattet JP. Childhood lichen planus -A question of therapy. J Am Acad Dermatol 1980; 3: 370-376.
2) Pandhi D, Singal A, Bhattacharya SN. Lichen Planus in Children. A series of 316 patients. Paediatric Dermatology April 29 2013. doi: 10.1111/pde.12155

POSTER ABSTRACT 03
Childhood Rashes Presenting to Primary Care.
Dr Mairi Ferguson, Dr Jennifer Crawley
Royal Victoria Hospital, Belfast

Children frequently present to their GP with a fever and a rash, which often has an infectious cause. The vast majority of these patients are diagnosed and managed in primary care. However, as waiting times for GP appointments increase, more patients are likely to attend A&E, where dermatologists may be called upon to offer an opinion on the diagnosis. It is therefore important to be familiar with the morphology of these exanthems and enanthems. This article will describe some of the most commonly encountered infections and their associated skin morphology.

Scarlet fever is a rash caused by group A beta-haemolytic streptococcus. There has been a recent increase in sporadic outbreaks. Skin signs include a strawberry tongue, followed by the characteristic exanthema. This consists of punctum on a diffuse erythematous base, and Pastia's lines which may be seen in the flexures. Prior to the era of antibiotics, this infection often resulted in death from complications such as rheumatic fever and glomerulonephritis. Early detection and treatment with antibiotics is therefore imperative.

Measles has had a recent resurgence, which may be a result of a reduced uptake of the childhood MMR vaccination. This paromyxovirus causes an unpleasant infection, with the potential to cause serious complications and even death. Koplik spots can be a helpful in detecting early infection. The characteristic rash which follows consists of a blanching maculopapular rash, with cephalocaudal spread. Early diagnosis can help reduce the rate of transmission of this highly infectious virus.

Hand foot and mouth disease is an infection usually caused by coxsakie A16 virus. The illness is mild and short-lived in most children. It often presents with vesicular acral lesions and mouth ulcers. It can therefore be mistaken for erythema multiforme or primary HSV.

Another commonly encountered rash in primary care is slapped cheek syndrome caused by parvovirus B19. The morphology includes erythematous cheeks and a lacy erythema on the limbs. The associated illness is usually mild however, certain high risk groups such as pregnant patients or those with hereditary anaemias can have far more serious outcomes. This makes correct diagnosis essential.

POSTER ABSTRACT 04
Atypical sarcoidosis presenting with a breast mass
Keating C1,Hughes R1, Molloy E2 Lally A1
1:Department of Dermatology 2:Department of Rheumatology
St Vincents University Hospital, Dublin 4

Sarcoidosis is a granulomatous disease of unknown origin. The breast is involved in less than 1% of cases. Breast can be a primary of secondary site of presentation.

A 33 year old woman presented to the breast clinic with a tender discharging right breast mass. Malignancy was ruled out by ultrasound-guided biopsy. Histology showed non-caseating granuloma formation. Multiple courses of oral antibiotics failed to result in improvement and she was commenced on low dose oral steroids and a diagnosis of granulomatous mastitis was made by her breast surgeon.
She subsequently developed tender subcutaneous nodules on her lower limbs clinically consistent with erythema nodosum and polyarthralgia involving her ankles, knees, arms and legs. She also complained of fatigue and malaise but no fevers or weight loss. Referral to rheumatology and dermatology was arranged.
A deep incisional biopsy was performed and histology again showed non-caseating giant cell granulomas in the breast tissue. There was no evidence of vasculitis. Tissue was sent for fungal and atypical mycobacterial culture and PCR..., all of which were negative .

A diagnosis of atypical sarcoidosis was made and she initially responded to oral corticosteroids but her symptoms flared when the dose was tapered below 15mg. She commenced hydroxychloroquine 200mg twice daily and tapered her dose of prednisolone with excellent clinical response.

Sarcoid involving the breast alone is rare and should be a diagnosis of exclusion, ruling out infective, vasculitic and malignant differential diagnoses. Oral steroids are a good first-line therapeutic option but restricted by their long term side effects and steroid sparing agents such as methotrexate, hydroxychloroquine or azathioprine may be considered.

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References: 1. Ting PT, et al. *J Cutan Med Surg.* 2005;9:10–15.

Date of preparation: May 2012.
p15/04/12

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Registrars' Symposium

01. 9.30am **A rare adverse event in TL – 01 phototherapy:**
A case series of 4 patients. Fahy, C.M.R., McDonald I, O'Donnell B, Gaynor L, Murphy G.M, Lenane P, Moloney F. National Photobiology Unit, Mater Misericordiae University Hospital, Dublin.

02. 9.40am **A rare case of Adiposis Dolorosa presenting in an acute setting in young male.**
Q. Razali, C. Kiely, M. Connolly, R. Hughes, AM. Tobin.
Adelaide and Meath Hospital, Tallaght.

03. 9.50am **Treatment resistant Nodular purigo which resolved during coincidental treatment with a glucagon-like peptide-1 agonist.**
R Hughes1, C Kiely1, M Connolly1, J Gibney2, AM Tobin1
Departments of Dermatology1 and Endocrinology2,
AMNCH, Tallaght.

04. 10.00am **Eccrine hidrocystomas of the nose treated with intralesional botulinim toxin.**
R Hughes1, C Keating1, R Barry2, A Lally1.
1.Department of dermatology, St Vincent's University hospital
2. Department of dermatology , St/ James' Hospital.

05. 10.10am **Subacute Cutaneous Lupus Erythematosus:**
A Paraneoplastic Phenomenon in Oropharyngeal Squamous Cell Carcinoma.
A. Murad1, H. Rowley2, N. Mulligan3, B. O Donnell1
Department of Dermatology1,
Ear Nose and Throat2 and Histopathology3,
Mater Misericordiae University Hospital, Dublin, Ireland.

06. 10.20am **Biologics induced bullous pemphigoid.**
C. Kiely, M. Connolly, Q. Razali, R. Hughes, AM. Tobin.
Adelaide and Meath Hospital, Tallaght

07. 10.30am **Cutaneous T-cell lymphoma in a 13-year-old boy**
S.J. O'Shea, B. O'Donnell
Department of Dermatology, Children's University Hospital, Temple Street, Dublin.

08. 10.40am **An unusual purpuric eruption in a young girl**
S.J. O'Shea, B. O'Donnell
Department of Dermatology,
Children's University Hospital, Temple Street, Dublin.

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Adverse events should be reported to 1800-244 255 or Ireland.drugsurveillance@gsk.com.

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References: 1. Langner A *et al. BJD* 2008;**158**,122-129 2. Summary of Product Characteristics for Duac®, available from www.medicines.ie. Accessed November 2011.
3. Lookingbill *et al. JAAD* 1997;**37**:590-5.

Date of preparation: November 2011. IE/CBP/0021/11

Case Presentation abstracts

ORAL ABSTRACT 01
Psychological distress and quality of life in hidradenitis suppurativa.
G Kelly1, CM Sweeney1, A Lally1, AM Tobin2, B Kirby1.
1Dermatology Research Group, St. Vincent's Hospital, Dublin 4.
2Adelaide and Meath Hospital, Tallaght, Dublin 24.

Background:
Hidradenitis suppurativa [HS] is a relapsing, inflammatory disease characterised by recurrent abscesses and suppuration primarily affecting the axillary, inguinal and inframammary folds. A small number of studies have suggested that HS causes a significant emotional burden and impairment in quality of life.1

Objectives:
To determine the prevalence of anxiety, depression, worry and quality of life impairment in patients with HS using standard screening questionnaires.

Methods:
Thirty five patients [age 26-64 years, M:F 11:24, Hurley stage I/II/III] completed 3 questionnaires – the Hospital Anxiety and Depression Scale [HADS], the Penn State Worry Questionnaire [PSWQ] and the Dermatology Life Quality Index [DLQI]. The severity of HS was measured using the Hurley classification.

Results:
Of the 35 patients with HS, 17 patients [48.6%] had probable pathological anxiety as defined by a HADS depression score of >11. This appears higher than the prevalence of anxiety in psoriasis where anxiety has been reported in 20-43% of psoriatic patients.
Four HS patients [11%] had probable pathological depression based on HADS depression scores of >11. This is comparable to the prevalence of depression in a psoriatic population, where estimates in the literature range from 10-60%.

Thirty one percent of HS patients had pathological worry (PSWQ >60) in keeping with prevalence rates of worry in psoriatic patients.
Impairment in quality of life was reported in a significant proportion of HS patients with 19 patients [54.2%] having a DLQI of >10. Nine of these patients [26%] had a DLQI >21.

There was no correlation between stage of disease, based on Hurley classification, and levels of anxiety, depression, worry or quality of life impairment.

Conclusion:
Hidradenitis suppurativa is associated with a high prevalence of pathological anxiety, depression, worry and a marked impairment in quality of life. Screening HS patients to identify patients requiring psychological intervention should be undertaken in order to optimize patient care.

1.Onderdijk AJ et al.Depression in patients with hidradenitis suppurativa. J Eur Acad Dermatol Venereol. 2013;27:473-8.

ORAL ABSTRACT 02
What is the relevance of contact allergy to sodium metabisulfite and which concentration of the allergen should we use?
Nicola Ralph, Saroj Verma, Subha Merry, Brian Kirby, Aoife Lally, Paul Collins. St Vincent's University Hospital, Dublin.
St Vincent's University Hospital, Dublin

The prevalence of contact allergy to sodium metabisulfite (SMB) has increased from 1.4-1.7% to 3.4-6.8% in published series over the past 20 years. However, the relevance of SMB positive patch test reactions is still debated (Madan V, Walker SL, Beck MH. Contact Dermatitis 200757:173-176, Garcia-Gavin J, Parente J, Goossens A Contact Dermatitis 2012;67:260-269). We added SMB 1% in petrolatum to the British standard series in February 2009. Well defined erythema occurs at test sites but whether this is true contact allergy is uncertain in some of our patients and the source of sensitisation is sometimes difficult to determine. The aim of this study was to review contact allergy to SMB in our cohort and to investigate different concentrations of the allergen in order to define the most appropriate concentration for patch testing. Methods: Patients' medical records were reviewed between February 2009 and December 2011 in order to obtain information on patient demographics, clinical presentation and source of sensitisation. Patients attending for patch testing between January 2012 and June 2013 were tested to three strengths of SMB (1%, 0.1%, 0.01%) (Chemotechnique Diagnostics, Vellinge, Sweden), following ethics committee approval. Allergens were applied to the upper back using IQ ultra chambers (Chemotechnique Diagnostics, Vellinge, Sweden). The allergens were removed after 48 hours and were interpreted according to the International Contact Dermatitis Research Group (ICDRG) criteria.

Results: Fifty-six of 616 patients (9%) were positive to SMB 1% between February 2009 and 2011. Three hundred and eighty patients were patch tested between January 2012 - June 2013 and 14 (8 female, 6 male) were positive to SMB 1%, eight to SMB 0.1%, and three to 0.01% SMB. Mean positive patch test results differed significantly between each concentration of SMB (ANOVA p=0.05), (Turkey's Multiple Comparison p<0.05). The reactions were considered relevant in 9/14 (64%), of which two were occupational (photographer and cleaner). The other seven patients gave a history of previous exposure to products containing SMB including cosmetics, bleaching agents and hair dyes. There were no irritant reactions. Our study confirms recent reports of the increasing prevalence of SMB allergy, which is relevant in the majority of patients (Goosens et al 2012). SMB 1% in petrolatum is superior to lower concentrations of the allergen.

ORAL ABSTRACT 03
Case mix and referral patterns of general practitioner graduates of the UCD Diploma in Dermatology.
McDonald I, Fahy C, Powell F, Moloney F.
Dermatology Dept, Mater Misericordiae University Hospital, Dublin.

Dermatological conditions are estimated to be involved in 25% of general practice consultations. Formal education and training in dermatology within general practice has however, been disproportionally low. The UCD Graduate Diploma in Dermatology is targeted at general practitioners (GPs) and aims to equip graduates with the knowledge and expertise required to recognise and treat skin disorders commonly encountered in general practice. Participants keep a log of their dermatology consultations while completing the diploma, documenting diagnosis, treatment and follow-up of patients including referral to secondary care. Our aim was to examine the case mix of dermatoses presenting to general practice, referral patterns between primary and secondary care and to assess changes in referral patterns over two sequentially run semesters of the diploma. The logbooks of 30 graduates from both semesters over three years were anonymised and retrospectively reviewed. Diagnoses were categorised into infective, inflammatory, benign lesion, premalignant lesion, malignant lesion, vascular, disorders of mucous membrane, disorders of skin appendages, cosmetic dermatology and genodermatoses. Referrals were categorised in the same way. Results from each semester were also compared. Sixty log books were assessed amounting to 1525 dermatological consultations and 118 different clinical diagnoses. Inflammatory conditions were the most commonly diagnosed (719/1525, 47.24%), followed by infective (472/1525, 30.95%), malignant (108/1525, 7.08%) and benign lesions

(105/1525, 6.88%). A total of 310 cases (20.32%) were referred to secondary care and of these, 204 (13.37%) were referred to a dermatology service. Inflammatory cases represented the largest referral group (115/310, 37.09%) followed by malignant lesions (95/310, 30.64%) and infective cases (38/310, 12.26%). While 87.96% (95/108) of presumed malignant lesions were referred to secondary care, only 68.42% (65/95) of referrals were made to a dermatology service. There was a statistically significant increase in the overall referral rate between the 1st semester (134/741, 18%) and the 2nd semester (176/781, 22.5%), p=0.04 and also in the number of presumed malignant lesions that were referred to secondary care between the two semesters (41/741 Vs 67/781), p=0.02.

In conclusion, almost 80% of dermatological conditions seen were managed within general practice with 13.37% referred to a dermatology service. Approximately one third of presumed malignant lesions referred were referred to a service other than dermatology. The significant increase in the number of referrals in the second semester of the diploma, particularly of presumed malignant lesions may suggest an increased clinical awareness following an educational intervention.

ORAL ABSTRACT 04
First experiences using in vivo reflectance confocal microscopy on melanocytic lesions in Ireland – the bridge between dermoscopy and histopathology.
M Sadlier, A Alani, K Ahmad, B Ramsay. Department of Dermatology University Hospital Limerick

Refectance confocal microscopy (RCM) is a non-invasive method of imaging human skin at a cellular level in vivo. Using optical imaging with an 830nm diode laser it creates high-resolution horizontal sections of the skin from the level of the stratum corneum to the papillary dermis. RCM has been shown to significantly improve the diagnostic accuracy of melanocytic lesions when compared to examination with dermoscopy alone. The dermatology department in University Hospital Limerick is the first Irish hospital to use RCM in a clinical setting. We describe the impact RCM has had on the diagnosis and management of melanocytic lesions seen in our daily practice.

The RCM is located in our outpatients department. Patients with suspicious or equivocal pigmented lesions attending dermatology clinics have their lesions imaged with RCM during their consultation. The microscope (Vivascope 1500, Lucid, Rochester, N.Y.) is attached to the skin with a disposable plastic window attached to a metal ring. This is magnetically connected to the lens-housing to stabilise the imaging site. Before confocal imaging a dermatoscopic image is taken. A consultant dermatologist (BR) performs microscopy and reviews the images taken.

We will describe the first malignant melanomas (MM) (n=8; 7 patients; 5 female; mean age 51 years) and spitz naevi (n=2) imaged with RCM in our department. All final diagnoses were based on histopathological analysis. We used the RCM method, an algorithm based on the identification of six morphological features, to differentiate between benign and malignant melanocytic lesions.2 They were seen in all 8 cases of MM and will be presented. The criteria were also present in both patients with spitz naevi. RCM helped confirm the diagnosis of MM in these patients and was supportive in the diagnoses of spitz naevi.
RCM helps bridge the gap between dermatoscopy and histologic analysis of melanocytic lesions. Reviewing the cellular in-vivo images along with the histology of melanocytic lesions has enhanced our interaction with our pathology colleagues at dermpath meetings. RCM has improved our diagnostic confidence and in time we hope it will increase our diagnostic specificity and reduce unnecessary excisions of melanocytic lesions

References:
1. Pellacani G, Cesinaro AM, Seidenari S. Reflectance-mode confocal microscopy of pigmented skin lesions-improvement in melanoma diagnostic-specificity. J Am Acad Dermatol. 2005;53:979-85.
2. Guitera P, Pellacani G, Longo C et al. In vivo reflectance confocal microscopy enhances secondary evaluation of melanocytic lesions. J Invest Dermatol. 2009;129:131-138

ORAL ABSTRACT 05
A study of the Dermatology Consult Service at a Dublin Teaching Hospital. Verma S, Reid C, Eustace K, Hughes R, Keating C, Lally A, Kirby B, Collins P. St Vincent's University Hospital, Dublin 4.

Skin disease affects 15-20% of the population. In one study, 35.9% of hospitalised patients had cutaneous signs and was the reason for admission in 13.4% of cases. However, there was no mention of skin disease in the admission notes in half of these cases (Nahass 1995). Studies of dermatology inpatient consults from Europe, India and America have highlighted poor diagnostic accuracy of skin disease even for common dermatoses. A prospective study of inpatient consults in our institution between 2003 and 2004 documented 149 cutaneous diagnoses in 615 patients.

The aim of the current study was to assess the diagnostic accuracy of the referring doctor and to establish the range of cutaneous diagnoses and the investigations required in order to compare our experience with our previous study and the international experience. 130 inpatient dermatology consults were received between August and November 2012. The median age of in-patients seen was 62 years (16-95 years) and 61.5% were male. Consults were requested most frequently by internal medicine (66.2%), surgery (19.2%), and psychiatry (6.2%). The most common reason for requesting a dermatology consult (77.7%) was to establish a diagnosis. The most frequent preliminary diagnoses considered by the referring team were; infection (28.5%), drug eruption (17.7%), dermatitis (7.7%), and skin cancer (6.2%). No diagnosis was suggested in 21 consults (16.2%). There was reference to a dermatology condition in the admission note in 48 consults (36.9%). 78.5% of consultations were seen the day requested and reviewed by consultant dermatologists in 107/130 cases (82%). The majority of consultations did not require diagnostic investigations (62.3%). The main diagnosis was dermatitis in 22.3%, infection 20.7%, drug eruption 14.6%, and benign lesions in 8.6% of patients. The accuracy of the preliminary diagnosis was 67% for psoriasis, 65% for infection, 62.5% for skin cancer, 61% for drug eruptions and 22% for vasculitis. Topical therapy was prescribed in 50% and systemic therapy in 27.7% of patients. The majority of patients had not seen or planned to seen their own general practitioner about their skin condition (82.0%).

These findings are similar to our previous study and the literature on hospital inpatient dermatology consultations in terms of type of dermatoses, diagnostic accuracy and investigations. Hospital based dermatology services have a central role in the care of the acute hospital inpatients. All inpatient admission notes should refer to cutaneous examination. Ensuring that dermatology is on the undergraduate medical student curriculum and providing dermatology education at postgraduate level should improve skills in the management of cutaneous disease.

ORAL ABSTRACT 06
Drug survival of fumaric acid esters for psoriasis:
A retrospective study.
Nuriah Ismail MB, Paul Collins MD, Sarah Rogers MD, Brian Kirby MD & Aoife Lally MD. Department of Dermatology, St. Vincent's University Hospital, Elm Park, Dublin 4, Ireland.

Background:
Fumaric acid esters (FAEs) have been used for over 30 years in the management of psoriasis

Objectives:
To determine drug survival of FAEs in patients with psoriasis, treatment limiting adverse drug events and range of effective drug doses.
Methods A retrospective single-centre study assessing all patients commenced on FAEs between October 2003 and July 2012 was carried out. Demographic data, length of treatment, reasons for discontinuation of FAEs, side effects and range of doses were recorded.

Results 249 patients (160, 64% male) were included. The mean age at which FAEs were commenced was 44.5 years (range 17-82 years). The mean length of treatment was 28 months (range 1 week – 150 months). In patients commenced on FAEs ≥4 years prior to inclusion in this study, the four year drug survival was 60% (64/107). Fumaric acid esters were discontinued in 146/249 (59%), with reasons including lack of efficacy in 59/146(40%) and gastrointestinal upset in 39/146 (27%). Lymphopenia (‹1.0 x 109 L-1) was observed in 133/249 (53%), necessitating cessation in 16/249 (6%) due to persistent counts of ‹0.50 x 109 L-1. Proteinuria was detected on urinary dipstick in 170/249 (68%) with worsening creatinine in 4/170 (2%). Fumaric acid esters were subsequently discontinued in 1 patient. A very low dose of FAEs (‹240mg daily) was successful in maintaining control of psoriasis in 26 (10%) patients. Their mean treatment duration was 64 months (range 32-150 months).

Conclusion This study indicates that FAEs have a four year drug survival rate of 60% which compares favourably with reported four year survival rates of 40% observed with etanercept and adalimumab and 70% observed with infliximab1. Longer drug survival is more likely in the significant subgroup of patients in whom a very low dose of FAEs is sufficient to control disease. Further work is required to elucidate factors that may predict which patients will respond to low dose therapy and therefore have a greater likelihood of successful prolonged therapy with FAEs.

1. Gniadecki R, Kragballe K, Dam TN, Skov L. Comparison of drug survival rates for adalimumab, etanercept and infliximab in patients with psoriasis vulgaris. Br J Dermatol 2011 164:1091-6.

ORAL ABSTRACT 07
Audit of serum vitamin D3 levels in patients with cutaneous lupus erythematosus.
Eoin Storan, Bairbre Wynne.
Department of dermatology, St. James's Hospital, Dublin 8.

Vitamin D is synthesized in the skin following exposure to ultraviolet (UV) light. Patients with cutaneous lupus are advised to avoid sun exposure and to wear high-factor sunscreen due to photoaggravation and are subsequently at risk of developing vitamin D deficiency. Low levels of vitamin D have been shown to correlate with disease activity. It is recommended that all patients with cutaneous lupus be screened for vitamin D deficiency.

Our primary aim was to assess our level of screening for vitamin D deficiency in patients with cutaneous lupus. Our secondary aim is to assess the level of sun protection used by patients and the level of patient education regarding lupus and photoaggravation.

Data was extracted from the electronic laboratory record. Between October 2012 and April 2013, we checked a serum 25(OH) vitamin D level on all patients with cutaneous lupus attending the lupus clinic. We also created a questionnaire to ascertain if these patients were taking calcium, vitamin D or bisphosphonate therapy. In addition, we asked patients about their practice of wearing sunscreen, specifically regarding SPF factor used, frequency of application, and sun exposure habits. Per our local laboratory assays for 25 (OH) vitamin D 25, a level of 25-80 nmol/L signified vitamin D insufficiency and ‹ 25 nmol/L signified vitamin D deficiency.

In total, data was collected on 24 patients. This included 21 (87.5%) females and 3 (12.5%) males. Upon initial observation of practice, no patients had a baseline vitamin D3 level.

Screening and use of the questionnaire revealed that 12 patients (50%) were vitamin D insufficient and 7 (29.1%) were vitamin D deficient. 14 (58.3%) patients reported wearing sunscreen every day, 5 (20.8%) in summer only, 3 (12.5%) if they perceived the day to be sunny and 2 (8.3%) never wore sunscreen. All 24 patients were aware that sunshine aggravates cutaneous lupus. 7 (29.2%) patients did not know to check if their sunscreen blocked UVA radiation.

Only 8 (33.3%) patients were taking vitamin D supplements. Of these, 4 patients were vitamin D insufficient (range 35-79).

All patients treated at the lupus clinic are given information on sun protection following diagnosis. It is now standard that we check 25 (OH) vitamin D levels on all patients with cutaneous lupus. If they are found to be insufficient or deficient they are supplemented with 1000 international units of vitamin D3. Levels are rechecked 6 monthly.

ORAL ABSTRACT 08
Methylisothiazinoline sensitization: recent patch test results from North Leinster.
R. Hellen, L. Jennings, CA. Egan.
Our Lady of Lourdes Hospital, Drogheda, Co Louth.

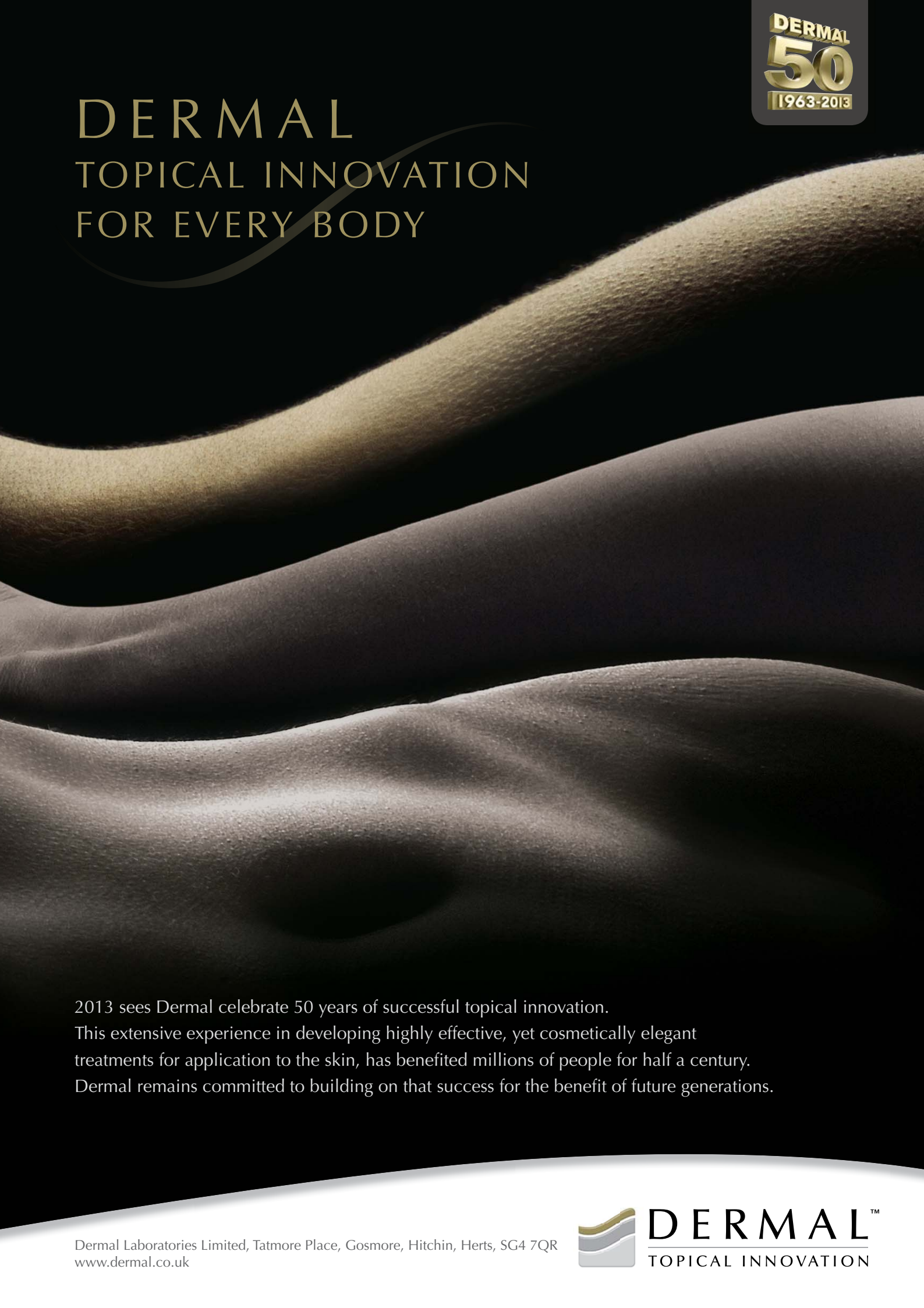
Background: European MCI and MI sensitization rates have been on the rise in recent years. The permitted concentration of MCI/MI for both 'rinse off' and 'leave on' products is 15ppm. Since June 2005, MI alone has a permitted concentration in cosmetics of 100ppm. MI (0.2% aq) was added to the standard series in 2012 for closer monitoring of this trend.

Aim: To investigate the prevalence of MCI and MI sensitization in an Irish population.

Methods: Patch test results of 203 patients from 2011 to 2012 were retrospectively reviewed. Of these 198 patients were tested against the standard series. In 2011 patients were tested against MCI/MI (0.02% aq) and 2012 patients were also tested against MI (0.2% aq).

Results: In 2011, the prevalence rate of MCI/MI sensitization was 4.21% (4/95). In 2012, the prevalence rate of MCI/MI and MI sensitization was 5.82% (6/103) and 8.73% (9/103) respectively. 55.56% (5/9) of positive reactions for MI had negative reactions to MCI/MI. 1.94% (2/103) were positive to the MCI component of MCI/MI.

Conclusion: The addition of MI (0.2% aq) to the standard series has revealed a high prevalence of MI sensitization in our population. This recent trend in MI sensitization suggests that the current permitted concentration of MI in cosmetics is too high.



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2 8 week regimen study, clinical trial of 25 patients aged 12 to 60 years **3** 4 week regimen study, clinical trial of 24 patients aged 2 months to 6 years
4 Kurtz ES & Wallo W.J Drugs Dermatol 2007;6(2): 167-170 **5** Fowler J & Silverberg N. Semin Med Surg 2008;27(2 Suppl):8-10

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*IMS, AC Nielsen Jan 2012

1. Cork MJ et al. Getting results from emollient therapy on atopic eczema. Derma Prac 2004. Vol.12 No. 3; 16-20

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Optimal therapeutic effect can be assessed approximately 8 weeks after treatment. If the treated area shows an incomplete response at the follow-up examination, the treatment should be carefully re-evaluated and management reconsidered. Clinical data on treatment for more than one treatment course of 2 or 3 consecutive days is not available. Clinical data on treatment of more than one area is not available. Clinical data on treatment in immunocompromised patients is not available, but systemic risks are not expected since ingenol mebutate is not absorbed systemically. **Contraindications:** Hypersensitivity to the active substance or to any of the excipients. See SmPC for full list of excipients. **Special warnings and precautions for use:** *Eye exposure:* Contact with the eyes should be avoided. If accidental exposure occurs, the eyes should be flushed immediately with large amounts of water, and the patient should seek medical care as soon as possible. Eye disorders such as eye pain, eyelid oedema and periorbital oedema should be expected to occur after accidental eye exposure of Picato®. *Ingestion:* Picato® must not be ingested. If accidental ingestion occurs the patient should drink plenty of water and seek medical care. *General:* Administration of Picato® is not recommended until the skin is healed from treatment with any previous medicinal product or surgical treatment and should not be applied to open wounds or damaged skin where the skin barrier is compromised. Picato® should not be used near the eyes, on the inside of the nostrils, on the inside of the ears or on the lips. *Local skin responses:* Local skin responses such as erythema, flaking/scaling, and crusting should be expected to occur after cutaneous application of Picato® (see SmPC for further information). Localised skin responses are transient and typically occur within 1 day of treatment initiation and peak in intensity up to 1 week following completion of treatment. Localised skin responses typically resolve within 2 weeks of treatment initiation when treating areas on the face and scalp and within 4 weeks of treatment initiation when treating areas on the trunk and extremities. Treatment effect may not be adequately assessed until resolution of local skin responses. *Sun exposure:* Studies have been conducted to assess the effects of UV irradiation of the skin following single and multiple applications of ingenol mebutate gel, 100 mcg/g. Ingenol mebutate gel did not demonstrate any potential for photo irritation or photo allergic effects. However, due to the nature of the disease, excessive exposure to sunlight (including sunlamps and tanning beds) should be avoided or minimised. *Management of actinic keratosis:* Lesions clinically atypical for actinic keratosis or suspicious for malignancy should be biopsied to determine appropriate treatment. **Fertility, pregnancy and lactation:** *Pregnancy:* There are no data from the use of ingenol mebutate in pregnant women. Animal studies showed slight embryo-fetal toxicity. Risks to humans receiving cutaneous treatment with ingenol mebutate are considered unlikely as Picato® is not absorbed systemically. As a precautionary measure, it is preferable to avoid the use of Picato® during pregnancy. *Breast-feeding:* No effects on the breastfed newborn/infant are anticipated as Picato® is not absorbed systemically. The nursing mother should be instructed that the newborn/infant avoid physical contact with the treated area for a period of 6 hours after application of Picato®. *Fertility:* No fertility studies have been performed with ingenol mebutate. **Undesirable effects:** *Summary of the safety profile:* The most frequently reported adverse reactions are local skin responses including erythema, flaking/scaling, crusting, swelling, vesiculation/pustulation and erosion/ulceration at the application site of ingenol mebutate gel. Following the application of ingenol mebutate, most patients (>95%) experienced one or more local skin response(s). Infection at the application site has been reported when treating face and scalp. 499 patients with actinic keratosis with exposure to Picato® 150 mcg/g or 500 mcg/g were treated in four vehicle controlled phase 3 studies enrolling a total of 1,002 patients. Patients received field treatment (area of 25 cm^2) with Picato® at concentrations of 150 mcg/g or 500 mcg/g or vehicle once daily for 3 or 2 consecutive days respectively. Frequencies are as follows: 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) and not known (cannot be estimated from the available data). *Adverse reactions observed for face and scalp (Picato® 150 mcg/g gel):* Very common: Application site: pustules, erosion, vesicles, swelling, exfoliation, scab, erythema, pain. Common: Headache, eyelid/periorbital oedema, application site: infection, pruritus, irritation. Uncommon: Eye pain, application site: discharge, paraesthesia, ulcer. *Adverse reactions observed for trunk and extremities (Picato® 500 mcg/g gel):* Very common: Application site: pustules, erosion, vesicles, swelling, exfoliation, scab, erythema. Common: Application site: pain, pruritus, irritation. Uncommon: Application site: paraesthesia, ulcer, warmth. Description of selected adverse reactions: The incidence of local skin responses that occurred at an incidence >1% in both the 'face/scalp' and the 'trunk/extremities', respectively are: application site erythema (94% and 92%), application site exfoliation (85% and 90%), application site scab (80% and 74%), application site swelling (79% and 64%), application site vesicles (13% and 20%), application site pustules (43% and 23%) and application site erosion (31% and 25%). Severe local skin responses occurred with an incidence of 29% on the face and scalp and with an incidence of 17% on the trunk and extremities. The incidence of severe local skin responses that occurred at an incidence >1% in both the 'face/scalp' and the 'trunk/extremities', respectively are: application site erythema (24% and 15%), application site exfoliation (9% and 8%), application site scab (6% and 4%), application site swelling (5% and 3%) and application site pustules (5% and 1%). *Long-term follow up:* A total of 198 patients with complete clearance at day 57 (184 treated with Picato® and 14 treated with vehicle) were followed for additionally 12 months. The results did not change the safety profile of Picato®. **Marketing Authorisation Holder:** LEO Pharma A/S, Industriparken 55, DK-2750 Ballerup, Denmark. **Marketing Authorisation Numbers:** Picato® 150 micrograms/gram (mcg/g) gel: EU/1/12/796/001, Picato® 500 micrograms/gram (mcg/g) gel: EU/1/12/796/002. **Available only on prescription. Date of First Authorisation:** November 2012. **Additional information is available on request from LEO Pharma, Cashel Rd., Dublin 12. Telephone: 01 4908924 or email: medical-info.ie@leo-pharma.com. Date of preparation of API:** December 6th 2012.

REFERENCES 1. LEO Pharma. Picato® 500 mcg/g (ingenol mebutate) gel Summary of Product Characteristics. Date of revision of SmPC: November 2012. **2.** LEO Pharma. Picato® 150 mcg/g (ingenol mebutate) gel Summary of Product Characteristics. Date of revision of SmPC: November 2012. **3.** Meda. Fluorouracil 5% cream Summary of Product Characteristics. Date of revision of SmPC: August 2011. **4.** Almirall. Diclofenac sodium 3% gel Summary of Product Characteristics. Date of revision of SmPC: February 2012. **5.** Meda. Imiquimod 5% cream Summary of Product Characteristics. Date of revision of SmPC: April 2010. (www.medicines.ie)

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