

# Meeting News

## **Munich International Summer Academy of Practical Dermatology July 22-27, 2007 in Munich, Germany**

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For more than five decades, the Department of Dermatology of the Ludwig-Maximilians University of Munich has invited German-speaking dermatologists for an intensive one week practical update in dermatology ("Fortbildungswoche für praktische Dermatologie und Venerologie"). For most German-speaking dermatologists, the "Munich Week" at the end of July every other year has developed into one of their most important biannual updates in practical dermatology. This year the course was expanded to the international English-speaking dermatological community with 500 participants from 5 continents and held in cooperation with the International Society of Dermatology. Besides plenary lectures covering important areas of clinical dermatology, major emphasis was put on the acquisition of practical knowledge and skills through courses and small group discussions of clinical cases. Fifty clinical dermatologists were invited speakers, some of which are presented in this brief summary.



**James Krueger, New York**  
**Psoriasis vulgaris**

*Clinical manifestations and therapeutic options.* The clinical and histological features were presented, along with an explanation of the immunological circuits expressed in the disease and causing the clinical pathology. **The Ying/Yang** activation of innate and acquired immune cell types, produced by epidermal keratinocytes, directly affect T cells and various dendritic cells (DCs) and vice versa. Two new immune concepts have recently been published (Nature, Vol 445, February 2007) with focus on IL-23 produced by CD11-DCs analogue to TIP-DC (TNF- and iNOS-producing DCs) amplify Th17 cells with hyperproliferative IL-22 effect on keratinocytes. The key players in psoriasis are thus CD11-DCs, plasmacytoid-DCs and macrophages involved in the innate immunity; antigen- or auto-antigen-

specific T cells involved in acquired immunity; a dysbalance of cytokines and chemokines; and an altered keratinocyte response.

Treatment with targeted therapeutics e.g. biologicals aimed at TNF: Infliximab (Remicade), Etanercept (Enbrel), Adalimumab (Humira) or more generally at T cells: Efalizumab (Raptiva) was argued to be used early in the treatment strategy before or at least not in conjunction with potential tumour promoting agents such as UV-light or cyclosporine.

**Annegret Kuhn, Heidelberg/Düsseldorf**

**Cutaneous lupus erythematosus (CLE)**

*Clinical spectrum and therapeutic options.* The classification of cutaneous lupus erythematosus (CLE) has been the subject of intensive research (see

[www.euscle.org](http://www.euscle.org)) during the past few years and now includes acute CLE (ACLE), subacute CLE (SCLE), chronic CLE (CCLE, including lupus profundus) and intermittent CLE (ICLE, including LET, LE-tumidus) according to the Düsseldorf classification list. Basic treatment options include UV-protection and elimination of photosensitizing drugs. Use of topical calcineurin inhibitors may be effective in cases of ACLE, SCLE and LET

Recent advances in biotechnology have made progress in the treatment strategy with the use of Mycophenolate sodium or efalizumab against SCLE while other biologicals might worsen the clinical course.

**Sakari Reitamo, Helsinki**  
**Topical immunomodulators**

Commonly termed topical calcineurin inhibitors (TCI) represent a major advance in the treatment of atopic dermatitis (AD). A variety of studies have been conducted between TCI's and active comparators and demonstrated the benefit of use of TCIs in children and their long-term safety profile. Tacrolimus 0.1% is shown superior to Fluticason propionate as well as Hydrocortison buturate.

The use of TCIs has not been shown to increase the incidence of herpes simplex infections and has no effect on the routine childhood vaccination program. In contrast to topical steroids TCIs increase collagen synthesis which explains the non-atrophy effect. As proactive



Delegates on a boattrip (author to the right).

maintenance therapy, Tacrolimus 0.03% twice weekly to stabilize the skin barrier is suggested as a new treatment strategy for childhood AD. Caution should however be taken in cases of AD with large skin barrier defects where systemic absorption is possible. Application of TCIs is therefore contraindicated in Netherton's syndrome. To avoid compliance problems due to stinging and burning in the early treatment phase, use of ASA 500 mg 1 h before application is advocated.

#### **Melthem Önder, Ankara**

##### **Behçet's disease**

Characterized by recurrent orogenital ulcerations and skin eruptions, ocular, articular, gastrointestinal, neurological, and vascular involvements. Most prevalent in the Mediterranean countries, the Middle East and the Far East.

The etiopathogenesis is very complex with genetic, environmental, bacterial, viral and immunological factors. An update of Behçet has recently been published in JEADV (2007; 21 :1-10) stating that the possible aetiological factors form a broad spectrum, with infectious agents being the most probable ones. Whatever the stimulus is, the target tissue seems to be the small blood vessels, with various consequences of either vasculitis and/or thrombosis in many organ systems. The endothelium seems to be the primary target in this disease. The inflammation may be promoted and potentiated, if not initiated, by innate mechanisms induced by heat-shock-protein action on Toll-like-receptors

involved in steps preceding tissue damage by immune cells. Confusion exists about the thrombotic tendency in Behçet's disease, in terms of whether a primary hypercoagulability is present or whether it is secondary to inflammation.

Treatment options includes combination of topical corticosteroids, antimicrobial agents, sucralfate, amlexanox, silver nitrate and systemic treatment with corticosteroids, colchicine (0.5-2 mg/day), benztidine penicillin, Dapsone (100-150 mg/day), Thalidomid (100-300 mg/day), Azathioprine (2.5 mg/kg/day), Cyclophosphamide (2-3 mg/kg/day or 750 mg i.v. pulse), cyclosporine (5 mg/kg/day), Interferon, anti-TNF therapy and sulfapyridine. A treatment algorithm based on controlled studies was presented.

#### **Robert Schwartz, New Jersey**

##### **Kaposi's sarcoma (KS)**

Classical Kaposi's sarcoma (KS) represents 0.002% of all cancers with a M:F ratio of 10:1. Iatrogenic KS counts 3% of all cancers in immunosuppressed patients with a M:F ratio of 23:1. African KS is found endemic to represent 10% of all cancers and epidemic in 50% of all cancers with a M:F ratio of 2:1.

The oncogenic Human Herpes Virus (HHV-8) is necessary but not sufficient for KS development. Different diseases associated to KS includes SLE, Hodgkin, Myeloma, IDDM, Asthma, Biliary cirrhosis, Nephrotic syndrome, and Polymyalgia rheumatica.

KS therapies include Rapamycin (Sirolimus) which inhibits Mammalian Target of Rapamycin (mTOR) and has potential to prevent KS development.

#### **Christos Zouboulis, Dessau**

##### **Cryotherapy in dermatology**

The physical properties of the application of cold temperatures on tissues and the effect on different components are widely used for different types of dermatoses and neoplasms. Successful cryosurgery has recently been used to keloids using intralesional application with a patented needle connected to the conventional spray canister.

#### **Hans-Christian Wulf, Copenhagen**

##### **Management of photodynamic therapy induced pain**

Methyl aminolevulinate hydrochloride (MAL) is found to be less painful than aminolevulinic acid (ALA) in normal skin - but not in lesional skin. PDT treatment is general more painful in AK than in BCC cases. First treatment is more painful than second treatment; probably dependent on porphyrin IX concentration.

Treatment of the face is most painful. Laser is less painful than non-coherent light. Topical anaesthesia and painkillers do not decrease pain during illumination

#### **Christos Zouboulis, Dessau**

##### **Acne**

Topical treatment with retinoids, benzyol-peroxid, and antibiotics are

all more or less equal in efficacy, but the success of treatment depends mostly on patient compliance, which is highly dependent on the quality of the vehicle. Thus, choice of drug-base is essential (ointments, hydrogels, ambiphilic creme, lotions, creme o/w, or creme w/o).

### **Rosacea**

Incidence decreases with latitude (10-2%) in Europe. 40% of cases has relatives involved with a history of migraine.

Pathogenesis involves increased facial blood flow and skin temperature. The role of the *Demodex folliculorum* mite is disputed, but recent studies have shown that antigenic proteins related to a bacterium (*B. oleronius*), isolated from *D. folliculorum* mite, have the potential to stimulate an inflammatory response in patients with papulopustular rosacea. Different clinical rosacea variants are ocular rosacea (independent of skin involvement), lupoid (granulomatous) rosacea, rosacea conglobata, rosacea fulminans (pyoderma faciale) and Mb. Morbihan (facial lymphedema).

Treatment options include Pimecrolimus, Tetracyclin or low dose anti-inflammatory Doxycycline 2x40 mg/day, which may not have any antibiotic effect (JAAD 2007; 56:791-802).

In selected cases Isotretinoin might have effect on erythema and oedema but not on telangiectasia.

### **Ulrich Hengge, Düsseldorf** **Infectious and non infectious diseases of the genital area**

HPV induced tumours are cancer of the cervix (HPV 6, 11, 16, 18), vulvar, vaginal and penile cancer (HPV 59, 66, 68), Perianal (HPV 6, 11, 16, 18), Busche-Löwenstein (HPV 6, 11), SCC Epidermodysplasia (HPV 5, 8, 14, 17), Cutaneous SCC (HPV 1, 2, 4-9), Mb. Bowen (HPV 38, 41-49), Laryngeal, oral, pharyngeal (HPV 1, 2, 11, 16), Tonsillar cancer (HPV 6, 11, 16, 18), and Esophageal cancer (HPV 2, 3, 11, 16, 18).

While 80% of HPV infections undergo spontaneous remission, 20% develops into CIN with 3% occurrence of cervical cancer. Lichen sclerosus is classified as an autoimmune disease with auto-antibodies to the extracellular matrix protein (ECM-1) (Oyama et al. 2003). The use of Tacrolimus daily for 10-20 weeks is recorded as effective.

Tinea in immunosuppressed patients is an increasing problem especially in organ-transplanted patients where 25% of renal transplant patients are at risk for development of disseminated tinea in skin and nails. Likewise invasive *Trichophyton rubrum* in Infliximab-treated patients has been demonstrated. The oral antifungal to be used are allylamin (Terbinafin) with only weak interaction with CyA (cytochrom 450) or Triazoles (Fluconazol, Itraconazol) which however process strong interaction with cytochrom 450 and found contraindicated with antihistamine,

cholesterol-lowering agents, and benzodiazepine.

New anti-fungals such as Triazols (Posaconazol, Voriconazol) and Echinocandins (Caspofungin, Micafungin, Anidulafungin) are approved for invasive and esophageal candidiasis and aspergilosis. Both groups have better fungistatic effect for candida spp. but are very expensive.

### **Herbert Hönigsmann, Wien** **Sun beds: Risk and benefits**

A historical review of sun habit lifestyles points out that sun tanning has been in fashion for the last century. This is believed to be mostly due to the cosmetic and movie industry showing healthy looking tanned front page models.

New high-power sunlamps emit doses 15 times higher than the sun with a known potential skin cancer risk. The argument that sun bed induced pigmentation is a protection factor against subsequent UV-induced damage from natural sunlight is incorrect, as the protection factor for sun beds is only 1.3 against UV-induced erythema. Likewise vitamin D deficiency can not be used as an argument for the use of artificial sunlight equipments.

People obsessed with having a permanent deep tan are also known as tanorexics - a majority of these individuals also suffer from anorexia. This picture might be explained by an UV-induced endorphine addiction

since exposure to opioid-antagonists is associated with withdrawal symptoms.

#### **Percy Lehmann, Wuppertal**

#### **Work-up of the photosensitive patient**

Generally accepted guidelines for phototesting are lacking. Laboratory data may help to exclude differential diagnoses, for example, porphyries; however, in most cases of photodermatoses they are of no help. Since skin lesions often subside rapidly after sun exposure, it is desirable to induce the dermatosis in a given test area with appropriate test protocols.

Progress has been made to induce a variety of photodermatoses in loco, for example, polymorphous light eruption (PLE), hydroa vacciniforme, chronic actinic dermatitis (CAD) (including persistent light eruption), solar urticaria (SU) and lupus erythematosus (LE). Photo-patch testing: JEADV 2004; 18: 672–682

#### **Chronic actinic dermatoses**

Usually caused by UVB.

However, also low threshold for UVA.

#### **Persistent light eruption**

1. Test on previous involved skin.
2. UVA 3×60 J/cm<sup>2</sup> (most cases).

3. UVB 1.5 MED-UVB.
4. Reading 24–72 h post-radiation.

#### **Solar urticaria**

1. Test on non-involved skin.
2. UVA, UVB, UVC + visible light.
3. Reading immediately post-radiation.

#### **Lupus erythematosus**

1. UVA 30 J/cm<sup>2</sup>.
2. Back and forearms.
3. Reading + biopsy 24–48 h post-radiation.

#### **Leading photoallergens**

1. NSAID.
2. Phenothiazides (chlorpromazin, promethazine).
3. Desinfections (tetrachlor-salicylanilin).
4. UV-filters (hydroxy-4-methoxybenzophenon).

Treatment dose based on MED has low significance, since MED determination shows extreme differences (× 5) in different body areas.

Special lectures were given by **John MacGrath (London)** on inherited skin diseases, **Michael Meyrer (Dresden)** on sexually transmitted diseases

and **Rudolf Happle (Marburg)** with new aspects and recent advances in paediatric dermatology – besides several others highly competent clinical lectures coordinated by **Thomas Ruzicka** and **Matthias Volkenandt (Münich)** who hosted this nicely organized congress.

Astellas Pharma are cordially thanked for sponsoring this meeting.

#### **Idiopathic photodermatoses**

1. PLE
2. Hydroa vacciniformia
3. Solar urticaria

#### **Chronic actinic dermatoses**

1. Persistent light eruption
2. Actinic reticuloid
3. Photosensitive eczema
4. Photoagg. AD

#### **Chemical photodermatoses**

1. Phototoxic
2. Photoallergical

#### **UV-inducible photodermatoses**

1. LED
2. Porphyria
3. Hailey-Hailey
4. Darier
5. HIV-infections