SUPPLEMENTUM NO. 2

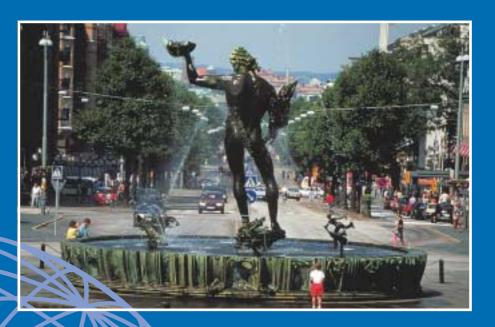
no. 2a, vol. 6, May 2001 ISSN 1402-2915

29th Nordic Congress of Dermatology and Venereology

forum for

dermato-venereology

June 7-10, 2001 in Göteborg, Sweden



WELCOME TO GÖTEBORG



Abstracts in the 29th Nordic Congress of Dermatology and Venereology

and Information from the Nordic Societies

Organizing and Scientific Committee

Olle Larkö, MD, Professor Ewa Voog, MD, Senor consultant Håkan Mobacken, MD, Assoc Professor Jan Faergemann, MD, Assoc Professor Inger Forsell, Administrator

Supplement No 2, 2001, Forum for Nordic Dermato-Venereology

Contents

Overview: Abstract titles and chairmen	3
Program at Glance	11
Abstracts in the 29th Nordic Congress of Dermatology and Venereology	12
Abstract author index	53
Stadgar för Nordisk Dermatologisk Förening	55
Möten i Nordisk Dermatologisk Förening 1910–2001	56
Protokoll från Nordisk Dermatologisk Förening	57
Ekonomisk redogörelse för 1998, 1999 och 2000	59
Necrologies	60
Members in the national societies	61
Denmark	61
Finland	65
Iceland	
Norway	
Sweden	

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

<u>Session and Chairmen</u> PLENARY LECTURE 1 Chair: Yaar, M., U.S.A.	<u>Titles</u> <i>The Role of Telomere Damage in Melanogenesis</i> +Yaar, Mina; Eller, Mark S.; Gilchrest, Barbara A.	<u>Abstract No</u> PL1:1
PLENARY LECTURE 2 Chair: Meheus, A., BELGIUM	<i>Epidemiology of sexually transmitted infections in Europe</i> +Meheus, André	PL2:1
PLENARY LECTURE 3 Chair: Ruzicka, Th., GERMANY	Immunomodulating therapy in dermatology: Today and into the next millennium +Ruzicka, Thomas	PL3:1
COURSE 1 - IMMUNOLOGY FOR THE DERMATOLOGIST	<i>Immunology for the dermatologist</i> +Ranki, Annamari; Nordlind, Klas	C1:1
Chair: Ranki, A., FINLAND Co-Chair: Nordlind, K., SWEDEN	Combining histamine with tumour necrosis factor-a leads to greatly enhanced inhibition in keratinocyte growth +Harvima, Ilkka; Kivinen, Petri K.; Horsmanheimo, Maija; Hyttinen, Mika	C1:2
COURSE 2 - MYCOLOGY RELEVANT FOR THE DERMATOLOGIST	<i>Mycology relevant for the dermatologist</i> +Faergemann, Jan; Svejgaard, Else	C2:1
Chair: Faergemann, J., SWEDEN Co-Chair: Svejgaard, E., DENMARK	<i>Onychomycosis - Diagnosis and treatment</i> +Sigurgeirsson, Bárdur	C2:2
COURSE 3 - LASER COURSE Chair: Bjerring, P., DENMARK Co-Chair: Troilius, A., SWEDEN	<i>Laser course</i> +Troilius, Agneta	C3:1
	<i>Treatment of Hailey-Hailey disease and Mb. Bowen with ultrapulsed CO₂-laser.</i> +Lauritzen, Edgar; Troilius, Agneta	C3:2
COURSE 4 - COURSE ON BASIC SKIN SURGERY Chair: Jemec, G., DENMARK Co-Chair: Stenquist, B., SWEDEN	Course on basic skin surgery +Jemec, G.; Stenquist, B.	C4:1
COURSE 5 - SELF-ASSESSMENT DERMATOPATHOLOGY COURSE Chair: Barr, R.J, U.S.A.	Self-assessment dermatopathology course +Barr, Ronald J.	C5:1
SYMPOSIUM 1 - PSORIASIS Chair: Badsgaard, O., DENMARK	Psoriasis genetics +Samuelsson, Lena	S1:1
Co-Chair: Reitamo, S., FINLAND	<i>New therapies</i> +Skov, Lone	S1:2
SYMPOSIUM 2 - LEG ULCERS Chair: Hansson, C., SWEDEN	<i>Conservative treatment</i> +Hansson, Carita	S2:1
Co-Chair: Gottrup, F., DENMARK	<i>Organisation of the wound healing area</i> +Gottrup, Finn	S2:2
	Diagnostics in venous leg ulcer +Karlsmark, Tonny	S2:3
	3 Forum for Nord Derm Ven Vo	l. 6, 2001 - Suppl. 2

3

Session and Chairmen	<u>Titles</u>	Abstract No
	Conservative treatment – compression +Bjellerup, Mats	\$2:4
	A surgical approach for the treatment of leg ulcer +Jørgensen, Bo	S2:5
SYMPOSIUM 3 - SEXOLOGY Chair: Moi, H., NORWAY Co-Chair: Voog, E., SWEDEN	Lubrikasjonsproblemer hos kvinner - vestibulitt – psykologiske faktorer. +Langfeldt, Thore	\$3:1
SYMPOSIUM 4 - ATOPIC DERMATITIS Chair: Langeland, T., NORWAY	<i>What regulates T lymphocyte migration?</i> +Thestrup-Pedersen, Kristian	S4:1
Co-Chair: Thestrup-Pedersen, K., DENMARK	Discrepancies in prevalence of atopic dermatitis +Broberg, Ann	S4:2
	Environmental factors influencing the expression of atopic dermatitis - With focus on the possible influence of measles mumps and rubella-vaccination, measles infection, hormonal contraception use and insulin-dependent diabetes mellitus. +Braae Olesen, Anne	S4:3
	Treatment options in atopic dermatitis +Reitamo, Sakari	S4:4
SYMPOSIUM 5 - HIV-INFECTION Chair: Pehrsson, PO., SWEDEN Co-Chair: Sandström, E., SWEDEN	Adverse effects of HIV-treatment +Sandström, Eric	S5:1
	HIV treatment - from death to survival +Gisslén, Magnus	\$5:2
SYMPOSIUM 6 - PEDIATRIC DERMATOLOGY Chair: Kalimo, K., FINLAND	Fungal infections in children +Faergemann, Jan	\$6:1
Co-Chair: Broberg, A., SWEDEN	Papular eruption secondary to molluscum contagiosum +Olafsson, Jon Hjaltalin; Davidsson, Steingrimur	\$6:2
	Treatment of small children with portwine stains What sort of anaesthesia do we use? +Mørk, Nils-Jørgen	\$6:3
	Child with chronic skin disease: How is the family getting along? +Koulu, Leena	S6:4
	Fraud and non-medicinal treatments in pediatric dermatology +Serup, Jørgen	S6:5
SYMPOSIUM 7 - PHOTODYNAMIC THERAPY Chair: Wennberg, A-M., SWEDEN	Photodynamic therapy. Mechanisms and procedures +Moan, Johan; Juzenas, Petras; Juzeniene, Asta; Ma, Li-Wei; Iani	S7:1 , Vladimir
Co-Chair: Wulf, H.C., DENMARK	Photodynamic therapy with 5-aminolevulinic acid of recalcitrant foot and hand warts. +Stender, Ida Marie; Renhua, N.; Fogh, H.; Gluud, C.; Wulf, HC	S7:2
	<i>Imaging fluorescence of basal cell carcinomas</i> +Ericson, Marica; Sandberg, Carin; Wennberg, Ann-Marie; Gudmundsson, Fredrik; Rosén, Arne; Larkö, Olle	\$7:3

4

Session and Chairmen		<u>Abstract No</u> S7:4
	Skin autofluorescence in demarcation of basal cell carcinoma +Renhua, Na; Rossen, Kristian; Stender, Ida-Maria; Wulf, Hans Ch	-
	Photodynamic therapy for psoriasis and extramammary Paget´s disease +Ros, Anne-Marie	\$7:5
SYMPOSIUM 8 - OCCUPATIONAL	Epoxy dermatitis – what is new?	
DERMATOLOGY Chair: Björkner, B., SWEDEN	+Bruze, Magnus	S8:1
Co-Chair: Bruze, Magnus, SWEDEN	<i>Occupational Plant Dermatoses.</i> +Paulsen, Evy	S8:2
	Irritant contact dermatitis - clinical and experimental aspects. +Lindberg, Magnus	S8:3
	<i>Trends in occupational dermatology</i> +Björkner, Bert	S8:4
SYMPOSIUM 9 - URETHRITIS Chair: Skov-Jensen, J., DENMARK Co-Chair: Lidbrink, P., SWEDEN	<i>Treatment of mycoplasma genitalium infections</i> +Falk, L.; Skov Jensen, J.	S9:1
SYMPOSIUM 10 - GENODERMATOSES Chair: Vahlquist, A., SWEDEN Co-Chair: Gedde-Dahl, T., NORWAY	Genodermatoses - An Introduction +Vahlquist, Anders	S10:1
	Epidermal transglutaminase (TGM1) mutations in lamellar and non-lamellar ichthyoses – a larger spectrum than anticipated +Pigg, Maritta; Vahlquist, Anders; Gedde-Dahl, Tobias; Gånemo, A Virtanen, Marie; Westermark, Per; Haußer, Ingrid; Dahl, Niklas	S10:2 Agneta;
	Life Quality Assessment in Ichthyosis Patients. +Gånemo, A.; Vahlquist, A.; Sjödén, P-O; Lindberg, M.	S10:3
	"Scandinavian" keratin mutations in epidermolytic hyperkeratosi. (bullous ichthyosis). +Virtanen, M.; Gedde-Dahl, T.; Mørk, N-J; Bowden, P.; Vahlquist, A	S10:4
	<i>Epidermolysis bullosa simplex: Molecular characterization of the mutational spectrum in Danish patients</i> +Sørensen, C.B.; Ladekjær-Mikkelsen, AS.; Andresen, B.S.; Brand Veien, N.K.; Buus, S.K.; Anton-Lamprecht, I.; Kruse, T.A.; Jensen, P.K.A.; Eiberg, H.; Bolund, L.; Gregersen, N.	S10:5 rup, F.;
	<i>The role of plectin for the integrity of human skin</i> +Koss-Harnes, Dörte; Høyheim, B.; Gedde-Dahl, T.	S10:6
	Hereditary hypotrichosis simplex of the scalp. Clinical and molecu investigations in a Danish family. +Bygum, Anette; Betz, RC; Nöthen, MM; Ibsen, HHW; Rasmussen, Brandrup, F	S10:7
	Ichthyosis-Prematurity Syndrome – an unknown, frequent and an Mid-Scandinavian disease +Kampman, Petra T	cient " S10:8
SYMPOSIUM 11 - SKIN INFECTIONS Chair: Olafsson, J., ICELAND	<i>Resistance to antibiotics in dermatology</i> +Gaustad, Peter	S11:1
Co-Chair: Christensen, O., NORWAY	5 Forum for Nord Derm Ven Vol. 6	, 2001 - Suppl. 2

5

_

Session and Chairmen	Titles	ract No
	<i>Skin manifestations of streptococcal infections</i> +Broberg, Ann	S11:2
	Dermatomycoses of the feet - more than meets the eye at first sight?	S11:3
	+Sigurgeirsson, Bárdur <i>Tropical skin infections/infestations in travellers</i> +Brandrup, Flemming	S11:4
SYMPOSIUM 12 - NORDIC DERMATOLOGY IN EUROPE Chair: Jemec, G., DENMARK Co-Chair: Bergbrant, Ing-Marie, SWEDEN	The European Specialist Section (U.E.M.S) - Responsibilities and rights. +Bergbrant, Ing-Marie	S12:1
SYMPOSIUM 13 - SKIN TUMOURS Chair: Helland, S., NORWAY	Solar keratosis, Bowen's disease and keratoacanthoma - are they all squamous cell carcinoma? +Sviland, Lisbet	\$13:1
	PUVA and skin tumours +Karvonen, Jaakko	\$13:2
	Incidence of skin cancer in patients following organ transplantation. +Lindelöf, Bernt	\$13:3
Chair: Mörk, N-J., NORWAY	Y Narrowband UVB phototherapy for psoriasis +Ferguson, James	<i>S14:1</i>
Co-Chair: Jansén, C., FINLAND	<i>UVA1 Phototherapy.</i> +Podda, Maurizio; Grundmann-Kollmann, Marcella; Kaufmann, Roland	\$14:2 1
	UVA exposure and the risk of cutaneous malignant melanoma +Moan, Johan	S14:3
	UVA radiation enhances metastatic properties of melanoma cells +Pastila, Riikka; Leszczynski, Dariusz	S14:4
	UVA and photoprotection +Larkö, Olle	\$14:5
SATELLITE SYMPOSIUM 1 - ROCHE	Inflammatory acne: Can resistance reveal how antibiotics work? +Cove, Jonathan H.	SAT1:1
SATELLITE SYMPOSIUM 2 - GALDERMA	<i>Do We Need Combination Therapy?</i> +Faergemann, Jan	SAT2:1
	Amorolfine + Terbinafine Combination: Results of a Clinical Trial in France +Baran, Robert L.	SAT2:2
	Rationale for Combination Therapy +Evans, E.G.V.	SAT2:3
	Amorolfine + Itraconazole Combination: Results of a Clinical Trial in Spain +Lecha, Mario	SAT2:4
SATELLITE SYMPOSIUM 3 - SCHERING-PLOUGH	Treatment of urtcaria with antihistamines – new aspects	

6

<u>Session and Chairmen</u> SATELLITE SYMPOSIA 4 – PHOTOCURE	<u>Titles</u> Photodynamic theray – an effective treatment in AK and BCC. An over practice and benefits	<mark>stract No</mark> erview of its
FREE COMMUNICATIONS, FRIDAY a.m. Chair: Helland, S., NORWAY	Arthritis and quality of life among members of the Nordic Psoriasis Associations. Data from the Nordic quality of life study +Molin, Lars; Zachariae, Hugh; Zachariae, Robert; Blomqvist, Kirsti; Davidsson, Steingrimur; Mørk, Cato; Sigurgeirsson, Bardyr	0-1
	Prevalence of fibromyalgia in patients with psoriasis +Thune, Per	0-2
	Use of alternative therapy in psoriatics from the nordic countries: A survey from 5739 members of the Nordic Psoriasis Associations +Mørk, Cato; Zachariae, Hugh; Zachariae, Robert; Blomqvist, Kirsti; Davidsson, Steingrimur; Molin, Lars; Sigurgeirsson, Bardyr	O-3
	<i>Erythromelalgia: A syndrome of dysfunctional vascular dynamics</i> +Mørk, Cato; Asker, C.; Salerud, G.; Kvernebo, K.	0-4
	Treatment of Psoriasis in the Nordic Countries: A Survey from 5739 Members of the Nordic Psoriasis Associations. +Zachariae, Hugh; Zachariae, Robert; Blomqvist, Kirsti; Davidsson, Steingrimur; Molin, Lars; Mørk, Cato; Sigurgeirsson, Bardyr	O-5
	<i>Psoriasis-related quality of life in 6497 Nordic patients</i> +Zachariae, Robert; Zachariae, Hugh; Blomqvist, Kirsti; Davidsson, Steingrimur; Molin, Lars; Mørk, Cato; Sigurgeirsson, Bardyr	O-6
	Palmoplantar pustulosis, smoking and autoimmunity +Michaëlsson, Gerd; Hagforsen, E; Nordlind, K	0-7
	Botulinum toxin A improves life quality in severe primary focal hyperhidrosis +Swartling, Carl; Naver, Hans; Lindberg, Magnus	O-8
	An 8-year experience with routine SL mix patch testing supplemented with compsitae mix +Andersen, Klaus E.; Paulsen, E.; Hausen, B.M	O-18
FREE COMMUNICATIONS, FRIDAY p.m. Chair: Andersen, K.E., DENMARK	<i>Measurements of colour in port wine stains using a quantitative method</i> +Helsing, Per; Lyngsnes Randaberg, L.; Mørk, NJ	O-9
	<i>Treatment of chronic hand dermatoses with UVB/TL01</i> +Nordal, Eli J.	O-10
	Ichthyosis-Prematurity Syndrome - an unknown, frequent and ancie "Mid-Scandinvian" recessive disease Kampman, Petra	nt 0-11
	A randomized double-blind study comparing photodynamic therapy (PDT) with Metvix® to PDT with placebo cream in actinic keratosis +Bjerring, Peter; Funk, J.; Roed-Petersen, J.; Söderberg, U.	0-12
	A pivotal study of photodynamic therapy (PDT) with Metvix [®] 160 mg cream in patients with basal cell carcinoma (BCC) with a risk of com <i>cations and poor Cosmetic outcome using conventional therapy.</i> +Wennberg, AM; Horn, M; Wulf, HC; Warloe, T; Rhodes, L; Fritsch, C; Kaufmann, R; de Rie, M; Wolf, P; Stender, I; Solér, A; Wong, G; Lang, T Legat, K; Pavel, S; Larkö, Olle	pli- O-13

Session and Chairmen	TitlesAbstDifferences in sun exposure doses in SED and sun burning episodes when sunbathing at the beach on holidays in Southern versus Norther Europe +Thieden, Elisabeth; Philipsen, P.A.; Heydenreich, J.; Sandby-Møller, J.; Wulf, H.C. Dermatan sulphate is released by proteinases of common pathogenic bacteria and inactivates antibacterial a-defensin +Schmidtchen, Arthur; Frick, Inga-Maria; Björck, Lars	0-14
FREE COMMUNICATIONS, SATURDAY a.m. Chair: Jansén, C., FINLAND	Quality of life and hand eczema +Lindberg, Magnus; Wallenhammar, Lena-Marie; Meding, Birgitta Allergic contact dermatitis from 2,2-bis[4-(2-hydroxy-3-	0-17
	methacryloxypropoxy)phenyl]-propane (BIS-GMA) +Kanerva, Lasse; Jolanki, Riitta; Estlander, Tuula NOSQ - The Nordic Occupational Skin Questionnaire - a tool for	0-19
	surveying work-related skin diseases +Lindberg, Magnus; Susitaival, Päivikki; Meding, Birgitta; Svensson, Åk Kanerva, Lasse; Flyvholm, Mari-Ann	O-20 te;
	Allergic contact dermatitis to budesonide reactivated by inhalation of the allergen +Isaksson, Marléne; Bruze, Magnus	0-21
	Cross-reactivity between nickel and cobalt demonstrated by systemic administration of nickel and cobalt? +Hindsén, Monica; Spirén, A.; Bruze, M.	0-22
	The ASP84 glu variant of the MC1R gene in Norwegian melanoma patients +Helsing, Per; Høyheim, Bjørn	0-23
	Dermatological DNA Laboratory in Oslo: Diagnostic services +Søyland, Elisabeth	O-24
FREE COMMUNICATIONS, SATURDAY p.m. Chair: Bäck, Ove, SWEDEN	Radiotherapy increases skin collagen synthesis in breast cancer patients +Riekki, Riitta; Parikka, M.; Jukkola, A.; Salo, T.; Oikarinen, A.	0-25
	Twisted collagen fibrils. Significance for hypermobile patients +Kobayasi, Takasi; Ullman, Susanne	O-26
	Congenital onset ichthyosis in Norway: Are our patients satisfied with their treatment? +Mørk, Nils-Jørgen; Gedde-Dahl, Tobias	0-27
	STD in Latvia in the year 2000 +Rubins, Andris; Jakabsone, I.; Rubins, S.; Chigorevska, L.	O-29
	HSV-2 antibodies in STD-patients, healthy pregnant females, blood donors and medical students in Bergen. +Nilsen, Arvid; Marsden, HS; Langeland, N; Matre, R; Haarr, L	O-30
	Higher number of leukocytes in urethral male smear obtained with a blunt metal curette in comparison with a calcium alginate swab	0-31

8

Session and Chairmen	<u>Titles</u> <u>Abst</u>	<u>ract No</u>
	+Stang, Henning; Moi, H.; Loeb, M.; Barlinn, C.; Gjertsen, I.; Halsos, A-M Kramer, P.; Thorvaldsen, J.	1.;
	Amelanotic malignant melanoma – a report of 5 cases +Odegard, Brit; Larsen, Tove Eeg	O-32
POSTER PESENTATION	Antibodies against nicotinic acetylcholine receptors in sera from patients with palmo plantar pustulosis +Michaëlsson, Gerd; Hagforsen, E; Nordlind, K; Lefvert, A-K; Mustafa, .	Р-33 А
	Methotrexate and Psoriasis - Can we reduce the need of Liver Biopsies? An evaluation of aminoterminal propeptide of type III procollagen (PIIINP) in routine screening for methotrexate induced liver fibrosis. +Søgaard, Helmer; Zachariae, Hugh; Heickendorff, Lene	P-34
	<i>Sensitization to inhalant and food allergens in childhood</i> +Jøhnke, Hanne; Norberg, L.; Andersen, KE; Bindslev-Jensen, C; Høst, A	P-35 4
	Syndrome of endogenous intoxication in patients with mycrobial eczema +Prokhorov, Dimitry	P-36
	Study of expression of fas-receptor on the lymphocytes of peripheral blood in patients with pemphigus +Pritulo, Olga	P-37
	<i>MED/MPD in thin and thick skin</i> +Nordal, Eli J.	P-38
	<i>The potential role of oxidative stress in elicitation of contact allergy</i> +Kaur, Sirje; Zilmer, Mihkel; Eisen, Maigi; Kullisaar, Tiiu; Vihalemm, Ti Rehema, Aune	P-39 iiu;
	Patch test reactions with dental screening series +Kanerva, Lasse; Aalto-Korte, K; Estlander, T; Hannuksela, M; Harvima RJ; Hasan, T; Horsmanheimo, M; Jolanki, R; Kalimo, K; Lahti, A; Lammintausta, K; Lauerma, A; Niinimäki, A; Rantanen, T; Turjanmaa, T Vuorela, A-M	
	The Importance of Understanding Exposure in Risk Assessment +McNamee, Pauline	P-41
	<i>Does imiquimod normalise hair growth in alopecia areata?</i> +Sommerfeld, Beatrice; Popova, I.	P-42
	Successful treatment for multiple superficial basal cell carcinoma using imiquimod 5% cream - A case report +Eklind, Jan; Lidbrink, Peter	P-43
	Photodynamic therapy (PDT) with Metvix® cream versus topical treatment with Efudix® cream in patients with multiple actinic keratosis on sundamaged skin. +Kampman, Petra; Lützow-Holm, Claus; Christensen, Ole	P-44
	Misoprostol improves symptoms in patients with erythromelalgia +Mørk, Cato; Kvernebo, K	P-45
	Improved quality of life and disease severity in Norwegian patients with psoriasis after climatotherapy at the Canary Island +Mørk, Cato; Wahl, A	P-46
	A need for pregnancy carrier test for junctional epidermolysis bullosa Herlitz in Sweden?	P-47

9

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

<u>Titles</u> <u>Abstr</u>	act No
+Gedde-Dahl, Tobias; Holmberg, Eva; Kristoffersson, Ulf	
Generalized basaloid follicular hamartoma treated with X-ray - A case report +Broberg, Ann; Landys, Karl; Ternesten-Bratel, Annika	P-48
Pili torti et canaliculi and agenesis of teeth. Report of a new "pure" hair-teeth ectodermal dysplasia +Selvaag, Edgar	P-49
Phototoxicity to diuretics and antidiabetics in the cultured keratinocyte cell line HaCaT. Evaluation by clonogenic assay and single cell gel electrophoresis (Comet assay) +Selvaag, Edgar; Petersen, Anita B.; Gniadecki, Robert; Thorn, Tine; Wulf, Hans Christian	P-50
Clinical findings and environmental factors related to urod gene and HFE gene mutations in Danish patients with porphyria cutanea tarda +Bygum, Anette; Christiansen, Lene; Thomsen, Kristian; Brandrup, Flemming	P-51
<i>Dermatovenereological service in Estonia</i> +Kukk, Terje; Poder, A.; Kangur, A.; Silm, H.	P-52
Gonorrhea in a new millennium +Thune, Turid; Rustad, Lisbeth	P-53
Microscopic view of methylene blue (MB) stained urethral smear of the male attending STD outpatient clinic and its relation to C. Trachomatis infection	P-54
+Vagoras, Andrius; Sumila, A.; Lapinskaite, G.; Marciukaitiene, I.	
Infectious skin diseases in recently returned travellers +Gasior-Chrzan, Barbara; Falk, Edvard S.	P-55
Long-term effectiveness of terbinafine vs. itraconazole in onychomycosis: A 5-year blinded prospective follow-up study +Sigurgeirsson, Bárdur; Olafsson, Jón H.; Steinsson, Jón; Paul, Carle; Billstein, Stephan; Evans, E. Glyn V.	P-56

10

Session and Chairmen

	THURSDAY 7/6	FRIDAY 8/6	S.	SÅTURDAY 9/6	SUND	SUNDAY 10/6	
ø		S: Psoriasis	Case Reports	S: Photodynamic	Case Reports		00
6		S: Leg ulcers		therapy S: Occupational dermatology		S: Skin Tumours S: Photodermatology	0
10		COFFEE - Exhibition/poster visit	er visit	COFFEE - Exhibition/poster visit	er visit		10
		S. Sevoloriv	Free communications			COFFEE - Exhibition/poster visit	
11		S: Atopic dermatitis		S: Urethritis S: Genodermatoser	Free communications Poster discussion	Plenary lecture Th. Ruzicka: Immuno-Modulating therapy in dermatology: today & into the next millenium	11
12			-		LUNCH	AWARDS CEREMONY	12
		FUNC	E			CLOSING REMARKS	
13				Plenary lecture	lecture		(* *
		Plenary lecture	scture	A. Meneus: Epidemiology or sexually transmitted infections in Europe: A changing pattern	y or sexually transmitted anging pattern		
14	REGISTRATION	M. Yaar: What controls melanogenesis?	slanogenesis?	Nordic Dermatology Association	ogy Association		14
	Satellite Symposiums:	COFFEE - Exhibition/poster visit	visit	General Assembly			
15	15.00 PhotoCure Photodynamic Therapy - an effective treatment in AK and RCC An overview of it's	S: HIV-infection	Free communications	COFFEE - Exhibition/poster visit	er visit		15
	practice and clinical benefits.	S: Pediatric dermatology		S: Skin infections	Free communications		
16	76 27			S: Nordic dermatology in Europe	Poster discussion		16
	17.00 Galderma Onychomycosis.						
17	17.30 Schering-Plough Treatment of urtcaria with antihistamines – new aspects						17
18		17.45 Bus to the Opera House: Musical Evita	louse:				10
19	Opening Ceremony & Welcome Reception	Buffet before performanc	υ	18.45 Bus to Banquet dinner	e Je		19
]

** Courses Dermatopathology Immunology Laser Surgery

Courses: Mycology Skin Surgery

*

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

Supplementum til kongress.p65

11

08-05-01, 08:13

PL1:1

THE ROLE OF TELOMERE DAMAGE IN MELANOGEN-ESIS

+Yaar, Mina* (U.S.A.); Eller, Mark S. (U.S.A.); Gilchrest, Barbara A. (U.S.A.) *Reston University School of Medicine

*Boston University School of Medicine

Delayed tanning, a photoprotective cutaneous response to UV irradiation, appears within 3-4 days after a single UV exposure and clinically parallels increased tyrosinase activity in melanocytes. The action spectrum for the tanning repsonse closely parallels the action spectrum for DNA damage as determined by pyrimidine dimer formation, suggesting that UV-induced DNA damage may be a melannogenic signal. In prokaryotes, photoprotective SOS responses are initiated during the repair of DNA damage through single-stranded DNA fragments (ssDNA) excised by nucleotide excision repair enzymes. To determine if in eukaryotes melanogensis is part of an SOS-like response, and because almost all UV-induced DNA damage as thymine dimers or (6-4) photoproducts involves adjacent thymidines, we stimulated cells with the substrate for these photoproducts, thymidine dinucleodites (pTPT). pTpT entered the nucleus and lead to a substantial increase in melanin content, compared to deoxyadenine dinucleotide (pdApdA), a dinucleotide rarely involved in photoproduct formation, used as control. Furthermore, pTpT increased tyrosinase mRNA and protein in cells, establishing that pTpT affects gene expression. Interestingly, as with UV irradiation, pTpT responses were mediated, at least in part through induction of p53 and p53-regulated genes. pTpT applied topically to guinea pig skin also induced pigmentation. Moreover, the pTpTinduced tanning was protective against UV irradiation as determined by histologic evaluation of UV-irradiated skin. Some but not all other ssDNA could induce melanogenesis, and effective sequences were noted to have hormology to telomeres, the terminal portions of eukaryotic chromosomes that consist of tandem repeats of TTAGGG. Prior studies have suggested that telomeres form a loop structure at the end of chromosomes. Telomere loop disruption results in degradation of the 3' single-stranded overhand and, similar to the active ssDNA, induces p53 and p53-regulated genes. These findings suggest that telomere damage during UV irradiation might disrupt the loop structure and thereby serve as one signal for melanogenesis. To test this hypothesis, three 11-base oligonucleotides were designed: one homologous to the telomere, one complementary and one unrelated. In cultured pigment cells, pTpT increased melanin content 3-fold above control, but the telomere homologue increased it by 10-fold. No effect on melanogenesis was observed with the complementary or unrelated ssDNA. To determine the role of p53 in ssDNA stimulated melanogenesis in vivo, we examined their effect on pigmentation of p53 (+/+) and p53 (-/-) mice ears. After 15 daily topical applications over 3 weeks, Fontana-Masson staining revealed substantial increase of epidermal melanin in the ears of p53 (+/+) mice related with telomere homologues but not control sequences. In contrast, p53 (-/-) ears did not pigment in response to any of the ssDNA. We propose that, at least in part, tanning is a response to UV-induced telomere damage resulting in exposure of the TTAGGG repeat, activation of p53 and upregulation of tyrosinase. Topical applications of telomere homologues may stimulate photoprotective responses in human skin.

PL2:1

EPIDEMIOLOGY OF SEXUALLY TRANSMITTED INFEC-TIONS IN EUROPE

+Meheus, André* (BELGIUM) *University of Antwerp

HIV/AIDS, genital herpes (HSV-2 and HSV-1), genital human papillomavirus (HPV) infection and C. trachomatis infection are the sexually transmitted infections (STI) of major public health importance in Europe during the last decade.

For HIV/AIDS the highest rates are seen in Spain, Portugal and Italy where the main exposure category is through sharing equipment for injecting drug use; the lowest rates are seen in the United Kingdom and the Scandinavian countries where the main exposure category is through homosexual transmission. Incidence of HIV infection is now relatively stable in most countries while the incidence of AIDS decreased significantly due to effective and earlier treatment.

The number of clinical cases of genital herpes has levelled off in recent years, but HIV 2 antibody rates in the general population (5% in teenagers, 10% in 20–29 years and 20% above 35 years) indicate the importance of asymptomatic infection.

HPV infection is also largely asymptomatic with 3 to 10% of adult women being infected with high-risk (oncogenic) HPV genotypes and 10 to 35% of women being infected with any type. Algorithms for cervical cancer screening combining testing for oncogenic HPV and Pap smear are actually evaluated.

Chlamydial infections have decreased considerably in countries that established comprehensive control programmes (e.g. Sweden), but in most countries infection rates remained high. In the UK, median prevalence in women was 4,5% in general practice, 4,6% in antenatal units, 4,8% and 5,1% in gynaecology clinic and family planning clinic attenders respectively, 8% in women seeking abortion and 16,4% in STD clinic attenders.

Gonorrhoea and syphilis have reached very low levels in Western Europe, but a major epidemic of syphilis and of the other STI occurred in Russia and the other Newly Independent States (NIS) of the former Soviet Union since 1992. This epidemic peaked in 1997/98. While incidence of syphilis is now below 5 per 100.000 in Western European countries, rates are between 100 and 300 per 100.000 in NIS countries.

Hepatitis B (HB) infection is the only STI for which an effective vaccine is available. Universal HB vaccination of infants and/or adolescents in Central and Southern European countries shall nearly eliminate HB infection as an STI there. Northern European countries still adhere to a vaccination policy of high-risk groups, an ineffective and inefficient approach.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

PL3:1

IMMUNOMODULATING THERAPY IN DERMATOLOGY: TODAY AND INTO THE NEXT MILLENIUM

+Ruzicka Thomas* (GERMANY)

*Heinrich-Heine-University

Immunomodulating drugs are developing to become the most important class of compounds for dermatology for the next years. Among these, immunosuppressive macrolides seem particularly promising The prototype drug cyclosporine is highly efficient in a number of inflammatory skin diseases, but its use is restricted due to side effects and is recommended only in severe cases. For the topical treatment of mild to intermediate severity diseases, topical immunomodulating agents are preferable. Among these, Tacrolimus and Ascormycin seem particularly promising. Tacrolimus has been shown to be effective in atopic eczema, but its efficacy in psoriasis is limited to occlusive treatment only. Other possible applications for Tacrolimus are emerging such as Pyoderma gangraenosum and rheumatoid ulcers. The mechanism of action of Tacrolimus includes the modulation of a number of cytokines in epidermis and inflammatory cells.

Besides immunosuppressive macrolides, other classes of immunomodulating agents are emerging, such as TNF-antagonists and Imiquimod.

The rapidly expanding field of topical and systemical immunomodulators hold particularly promise for improved therapeutic possibilities in dermatology at the beginning of the 3rd millennium.

C1:1

IMMUNOLOGY FOR THE DERMATOLOGIST

+Ranki, Annamari* (FINLAND); Nordlind, Klas** (SWEDEN) *Helsinki University Hospital,**Karolinska Hospital

Immunological mechanisms play a pivotal role in a vast majority of skin disorders. Skin and mucous membranes first meet environmental noxious compounds, and many reactions we see are the results of either innate immunity defence mechanisms or of immunocompetent cells residing in the skin. The complement system bridges innate and adaptive immunity. For an inflammatory reaction to develop, inflammatory cells must be recruited from the blood stream to the skin. This is mediated by an orderly expression of several types of adhesion molecules both on the inflammatory cells and on e.g. endothelial cells and keratinocytes. The role of innate immunity in skin diseases will be addressed by Hanna Jarva and Kristian Thestrup-Pedersen will discuss what regulates T lymphocyte migration into the skin.

Antigen take up, whether of microbial or autoimmune origin, by dendritic cells and presentation to T lymphocytes is a central initial event in many skin diseases. The network of cytokines regulating the outcome of the T cell response is complex. The use of new immunosuppressive drugs targeting specific phases of T cell-mediated reactions are in the frontline of treatment of atopic dermatitis and psoriasis. This area will be discussed by Sakari Reitamo. Also, new in vitro diagnostic methods for delayed hypersensitivity reactions of the skin will be described by Lena Lundeberg.

Mast cells, abundant in the dermis, are a group of pluripotent inflammatory cells that play a role not only in atopy and urticaria but also in psoriasis. The latest knowledge about the role of mast cells in skin immune and allergic reaction will be highlighted by Ilkka Harvima.

In the group of blistering skin diseases, either an autoimmune reaction towards or a hereditary mutation of a specific structure of the epidermis-dermis adhesion junction may be present. Kaisa Tasanen-Määttä will enlighten the specificity of the autoantibodies in bullous skin diseases.

C1:2

COMBINING HISTAMINE WITH TUMOR NECROSIS FACTOR-α LEADS TO GREATLY ENHANCED INHIBI-TION IN KERATINOCYTE GROWTH

+Harvima, Ilkka* (FINLAND); Kivinen, Petri K.* (FINLAND); Horsmanheimo, Maija* (FINLAND); Hyttinen, Mika* (FINLAND)

*Kuopio University Hospital

Increased mast cell numbers can be found in the upper dermis and in contact with the epidermis of the psoriatic lesion and chronic leg ulcers. However, the role of mast cells in epidermal pathology is not known. In this work, we have studied the effect of mast cell mediators, histamine and TNF-a, on the growth and viability of cultured human keratinocytes by using 3H-thymidine incorporation and MTT assays of proliferating keratinocytes in the presence of Keratinocyte-SFM medium (Gibco). In addition, an in vitro epithelialization model was developed to study the effect of histamine and TNF-a on the growth area of keratinocyte epithelium in the presence of 10% fetal calf serum and Dulbecco's modification of Eagle's medium. Histamine at 0.5 mM and TNF-a at 500 U/ml alone inhibited maximally by about 40% and 20%, respectively, the 3H-thymidine incorporation. However, the combination of 1 µM or 0.5 mM histamine with increasing concentration of TNF-a resulted in clear potentiation in this inhibition. The growth of keratinocyte epithelium was inhibited dose-dependently by 17-46% at 0.01-1 mM histamine, respectively, but was inhibited by only up to 25% at 500 U/ml TNF-a. The combination of 0.05 mM or 0.5 mM histamine with 100 U/ml or 500U/ml TNF-a led to potentiation in inhibition (up to 87% inhibition) of epithelium growth. MTT assay revealed that the potentiation in growth inhibition by simultaneous effect of histamine and TNF-a is due to greatly enhanced cytotoxicity. However, histamine and TNF-a alone were not cytotoxic under experimental conditions. In conclusion, the concentration of histamine in mast cell granules is about 100 mM and in the dermal skin about 0.05-0.1 mM. Thus, mast cells can be inhibitory, and even cytotoxic, to keratinocytes in the microenvironment between mast cells and keratinocytes.

13

Forum for Nord Derm Ven Vol. 6, 2001 – Suppl. 2

MYCOLOGY RELEVANT FOR THE DERMATOLOGIST

+Faergemann, Jan* (SWEDEN); Svejgaard, Else** (DEN-MARK)

*Sahlgrenska University Hospital,**Bispebjerg Hospital

This course will try to cover the important aspects of dermatomycology. Basal knowledge in the taxonomy and laboratory diagnostic methods of fungi is essential for all dermatologists. Dermatophyte infections of skin and hair are increasing in several Nordic countries due to an increase in immigrants from countries where tinea capitis is more common. The problem of correct diagnosis and treatment of onychomycosis will be discussed. Cutaneous *Candida* infections are still a problem and to be able to diagnose and treat the various clinical manifestations correctly is important. The various predisposing factors, diagnostic procedures and treatments of *Malassezia (Pityrosporum)* related skin diseases will be discussed.

C2:2

C2:1

ONYCHOMYCOSIS - DIAGNOSIS AND TREATMENT

+Sigurgeirsson, Bárdur* (ICELAND)

*University Hospital

When considering onychomycosis, sample technology is of paramount importance. A recent study showed that positive cultures in patients with onychomycosis ranged from 56% to 88% depending on the sampling technology used. Sampling technology also depends on the type of onychomycosis and different technologies will be discussed. Sampling of patients with tinea pedis will also be discussed.

Regarding treatment of onychomycosis a recent study where the long-term efficacy of terbinafine and itraconazole are compared will be reviewed. In this study the objective was to examine long-term cure and relapse rates, after treatment with continuous terbinafine and intermittent itraconazole in onychomycosis. This is a long-term prospective follow-up study in three centers in Iceland. Included were 151 patients aged 18 to 75 years with a clinical and mycological diagnosis of dermatophyte toenail onychomycosis. In a previous double-blind, double-dummy study, patients were randomized to receive either terbinafine (250 mg/day) for 12 or 16 weeks or itraconazole (400 mg/day) for 1 week in every 4 weeks for 12 weeks or 16 weeks (first intervention). Patients who did not achieve clinical cure at month 18, or experienced relapse/re-infection were offered an additional treatment with terbinafine (second intervention).

The primary efficacy criterion was mycological cure, defined as negative results on microscopy and culture at the end of follow-up without requiring second intervention treatment. Secondary efficacy criteria included clinical cure without second intervention treatment, and mycological and clinical relapse rates.

Median duration of follow-up was 54 months. At end of study mycological cure without second intervention treatment was found in 34/74 (46%) of terbinafine-treated subjects and 10/77 (13%) of itraconazole-treated subjects

(p<0.001). Mycological and clinical relapse rates were significantly higher in itraconazole vs. terbinafine-treated patients (53% vs. 23% and 40% vs. 17%, respectively). Of the 72 patients who received subsequent terbinafine treatment, 82% achieved mycological cure, and 92% clinical cure.

Continuous terbinafine provided superior long-term mycological and clinical efficacy and lower rates of mycological and clinical relapse, when compared to intermittent itraconazole, in the treatment of onychomycosis.

C3:1

LASER COURSE

+Troilius, Agneta* (SWEDEN) *Malmö University Hospital

This laser course will give you an idea of what is possible to do today with different kind of lasers and intense pulsed light sources within the field of dermatology. The technology is improving and it has given us many more possibilities to help our patients.

We will discuss:

Treatment of vascular lesions including vascular tumours and malformations.

Pigment lesions-possibilities and drawbacks.

Tattoos - cultural and traumatic.

Different colours e.g. green can sometimes be resistant for treatment and also hard materials e.g. metal car paint. There is also been some reports of allergies after treatment.

Vaporisation with CO_2 or Erbium – possibilities and problems. Abstract of a dissertation regarding CO_2 -treatment of hidroadenitis will be given and also treatment results after CO_2 treatment of Morbus Hailey-Hailey and Morbus Bowen.

Hair removal – good results with dark hair on pale skin however multiple treatments are needed.

PDT and it's possibilities will also be discussed.

C3:2

TREATMENT OF HAILEY-HAILEY DISEASE AND MB. BOWEN WITH ULTRAPULSED CO₂-LASER.

+Lauritzen, Edgar* (SWEDEN); Troilius, Agneta* (SWEDEN) *Malmö University Hospital

A 50-year-old man displayed Hailey-Hailey disease in the axillae, the groin and scrotum. The disease had earlier been treated with local steroids, disinfectants, and antibiotics without any significant improvement. The patient had many flares intense local treatment. We decided to resume CO_2 -laser evaporation with the ultrapulsed laser, and he was followed during a three-year period, which showed that the vegetative erosions could be removed. The patient had little adverse effect from the laser and was treated several times during the period.

The case is presented and a short survey of the subject is presented.

An 80-year-old man had a squamous cell carcinoma in situ on an ear, which spread superficially involving the conca and part of the helix. It was removed by surgery and skintransplantation but recurred at the same site. It was decided to apply CO_2 -laser evaporation for tumour removal and avoid skin grafting in the area. We used ultrapulsed CO_2 -laser for the tumour area and took biopsies to determine the tumour. The patient has had a 4 months period where the tumour has not revived. He is submitted to follow-up during an extended period of time to define efficacy of tumour removal.

The case is presented and a short survey of cases from the literature treated for Mb.Bowen with the $\rm CO_2$ -laser is discussed.

C4:1

COURSE ON BASIC SKIN SURGERY

+Jemec, G.* (DENMARK); Stenquist, B. (SWEDEN) *Roskilde Hospital

This 3 h-course will cover the essentials in skin surgery for those who plan to start dermatologic surgery or have some experience of the procedures in beforehand. The facilities, equipment and instruments necessary for surgery will be presented in detail. The outlines of the main danger zones, especially of the face, will be demonstrated and how to avoid lesions to nerves and arteries. Most important is a careful planning of all excisions and incisions. The relaxed skin tension lines (RSTL) or wrinkle lines should be used if possible. Skin marking is frequently helpful particularly when dealing with malignant lesions. Local infiltration anesthesia with adrenaline is normally used but regional blocks could be an alternative. An adequate undermining using a skin hook and blunt-ended scissors helps eversion of the wound edges and eases closure. The fusiform elliptical excision with primary suture should be the first option for repair but small flaps or a skin graft could be an alternative. Haemostatic equipment should always be available in the operating theatre. The postoperative care is essential and written instructions to the patients are a good routine. Complications can be avoided by careful planning of the procedure and discussion with your patient before surgery.

C5:1

SELF-ASSESSMENT DERMATOPATHOLOGY COURSE

+*Barr, Ronald J.* (U.S.A.)* *University of California

This is a self-assessment course with an emphasis on clinicopathological correlations utilizing actual microscopic slides with a multiple choice examination. Twenty (20) cases will be selected which will include a variety of interesting and important inflammatory and neoplastic lesions of the skin. Most cases will be basic but important lesions, but some will be newly described or unusual ones. The first portion of the course will be devoted to studying the slides. This will be followed by a discussion of the cases emphasizing important diagnostic clues and clinical correlations when relevant. The Course objective will be for the participant to evaluate his or her knowledge of dermatopathology and also learn additional diagnostic cirteria and an understanding of some unusual but significant disorders.

S1:1 PSORIASIS GENETICS

+Samuelsson, Lena* (SWEDEN) *Sahlgrenska University Hospital

Psoriasis is a chronic skin disorder affecting 2% of the population in northern Europe. The disease is characterised by hyperproliferation of keratinocytes and inflammatory infiltration. Several clinical forms exist although chronic plaque psoriasis is the most common variant. No major cause of psoriasis have been identified but current evidence suggest a central role for T lymphocytes.

Evidence for a strong genetic component in psoriasis susceptibility comes from familial clustering of the disease as well as a high concordance rate in monozygotic twins. The disease is today regarded to be a multifactorial disease with a complex genetic background, although in some large pedigrees a simple Mendelian inheritance pattern can be identified.

In order to identify genetic alterations rendering predisposition to psoriasis several genome scans have been performed by different groups using family set of configuration. This has led to the identification of several candidate loci but as of today, no single gene have been identified as disease-causing. One locus, PSORS1, has been identified and replicated in all genome scans. This locus resides within the HLA-region on chromosome 6p but seems to be different from the psoriasis-associated HLA-antigen Cw6. In this talk the present understanding of genetic causes to psoriasis will be reviewed. In addition, work on the PSORS5 locus identified in a family set from Southwest Sweden will be described in more detail.

S1:2

NEW THERAPIES

+Skov, Lone* (DENMARK) *Gentofte Hospital

The immune system and especially the T cells play an essential role in the pathogenesis of inflammatory skin diseases as psoriasis. During the resent years our detailed understanding of the pathogenesis of psoriasis has increased. This together with the development in biotechnology has made it possible to design specific response modifiers with a potential for greater effectiveness and fewer side effects than the known systemic therapies currently used for treatment of severe psoriasis. Several of the biological response modifiers such as monoclonal antibodies, recombinant cytokines and fusion proteins are already on the way in clinical trials.

S2:1 CONSERVATIVE TREATMENT

+Hansson, Carita* (SWEDEN) *Sahlgrenska University Hospital

There is consensus concerning some, but not all, of the conservative measures used to induce or speed venous ulcer healing, as for example the use of compression bandaging to counteract venous hypertension and to control oedema. Clinical infections and necrotic tissue are factors known to inflict negatively upon ulcer healing, and there is consensus about treating clinical infections with systemic antibiotics. Local ulcer treatment is also known to be of importance. Fibrin and necrotic tissue can be removed surgically, by larval therapy or by autolytic debridement. A moist environment is considered to enhance ulcer healing, which most modern topical wound dressings or wound preparations provides.

Still under debate for the treatment of venous ulcers are some topical therapies (like growth factors, hyperbaric oxygen, ultrasound, lasers, and electrical stimulation), as well as some systemic medical treatments (like zinc, fibrinolytics, hydroxyrutosides, prostaglandins, and methylxanthines).

S2:2

ORGANISATION OF THE WOUND HEALING AREA

+Gottrup, Finn* (DENMARK) *Copenhagen Wound Healing Center

Purpose: Improvements in prophylaxis and treatment of patients with all types of problem wounds could be achieved by focusing on the following topics: centralised treatment and care; standardised treatment plans; education for health care personnel; new drugs and materials; economical systems; societies, associations and publications.

Methods: The main organisatory effort should be the establishment of centralised treatment systems like wound centres integrated in the normal health care system. These systems should generate standardised treatment plans and generate the scientific basis for these. This development has to go along with the new economical systems, which presently are established in many health care systems. Education and information on wound treatment and care will be provided through societies, associations, journals and review systems.

Results: This development has been started some years ago in Denmark by the establishment of Copenhagen Wound Healing Centre in 1996. The Centre is a full-integrated hospital unit in the socialised government health care system of Denmark and consists of an outpatient clinic (8.000 consultations/year) and an inpatient ward (20 beds). All types of wound problems can be referred. The multi-disciplinary staff consists of 52 clinical and 8 research related persons employed full time for treatment of problem wounds. The structure of a national system for wound treatment and

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

16

care has been prepared, and presently negotiations for a full acceptance and integration in the health care system are going on.

Conclusion: The future goal for the organisation of wound healing and care is to be a full integrated part of the National Health Care Systems. With the establishment of the European Union this development should be realisable in this part of the World.

S2:3

DIAGNOSTICS IN VENOUS LEG ULCERS

+Karlsmark, Tonny* (DENMARK) *Copenhagen Wound Healing Center

Leg ulcers can be caused by many diseases. Venous leg ulcers are the most common cause, but an increasing amount of ischaemic and diabetic ulcers are seen. In order to choose the right treatment to the ulcer it is urgent to have established the correct diagnosis.

Clinical differential diagnosis as well as diagnostic tools including ankle/brachial index and examination for venous insufficiency using continuous wave ultrasound Doppler will be demonstrated.

S2:4

CONSERVATIVE TREATMENT - COMPRESSION

+Bjellerup, Mats* (SWEDEN) *Helsingborg Hopsital

The prerequisite for venous ulceration is venous hypertension. Venous hypertension is almost always secondary to incompetent venous valves or in rare cases to venous outflow obstruction. Venous hypertension results in elongated, tortuous capillaries producing microedema and cellular hypoxia. Venous hypertension may be counteracted by external compression delivered by compression bandages or pneumatic pumps or a combination.

Treatment with compression bandages has been shown to heal up to 85% of patients within three months and almost all patients are healed within 12 months.

Bandages may be classified according to three parameters, namely; interval between changes, elasticity and adhesive properties.

Interval between changes: In young and mobile patients it is preferred to remove bandages during night time. This may be achieved with compression stockings and non-adhesive bandages applied by the patient. Stay-on bandages replaced with weekly intervals by health personnel are preferred in older patients unable to apply bandages on their own. *Elasticity:* Bandages are classified as short-stretch, longstretch and intermediates. Short-stretch bandages deliver no sub-bandage pressure in rest but high pressure during work. Long-stretch bandages deliver high pressure during rest and intermediate during work.

Adhesive properties: Bandages are classified as unprepared, adhesive and cohesive. The two latter have better stay-on properties.

Hints about choice of compression bandages:

Mobile patients: Any type of bandage may be used.

Immobile patients: Long-stretch or medium-stretch bandages should be used since muscle pump function is absent. Compression pump may be supplemented.

Patients with arterial insufficiency: Low-stretch bandages should be used. Compression pump may be supplemented.

S2:5

A SURGICAL APPROACH FOR THE TREATMENT OF

LEG ULCER

+Jørgensen, Bo* (DENMARK) *Bispebjerg University Hospital

The policy of the Wound Healing Centre of Copenhagen is to perform excision of the ulcer followed by transplantation with an auto-split-skin-graft when conservative treatment does not give satisfactory results.

Local surgery for venous insufficiency may be carried out at the same time. The indications and contra indications for the procedure are displayed and the surgical methods are presented. The results of a one year follow up are displayed.

We stress that the venous leg ulcer is a chronic disease eventually demanding repetitive surgery.

S3:1

LUBRIKASJONSPROBLEMER HOS KVINNER -

VESTIBULITT - PSYKOLOGISKE FAKTORER.

+Langfeldt, Thore* (NORWAY) *Institutt for Klinisk Sexologi og Terapi

Selv om lubrikasjons variasjoner hos kvinner tilsvarer ereksjonsvariasjoner hos menn, har ikke begrepene vært anvendt identisk. I litteraturen har man anvendt frigiditet, kjønnskulde, om manglende evne til å lubrikere, mens ereksjonssvikt aldri har vært beskrevet som kjønnskulde. Jeg skal i dette foredraget ta opp ulike kulturelle og psykologiske aspekter ved lubrikasjon hos kvinner og sette dette inn i en utviklingsmessig sammenheng. Samt belyse de psykoterapeutiske implikasjonene.

S4:1 WHAT REGULATES T LYMPHOCYTE MIGRATION?

+Thestrup-Pedersen, Kristian* (DENMARK) *University of Aarhus

Inflammatory skin diseases represent accumulations of activated leukocytes in the skin. Besides pustular psoriasis, bullous pemphigoid or Sweet's syndrome, where neutrophils or eosinophils form the majority of cells, most other inflammatory diseases show a predominance of activated T lymphocytes. This is remarkable as both neutrophil granulocytes and monocytes are able to move both quicker and through more narrow pores in vitro than lymphocytes.

We have used an in vitro technique to measure T lymphocytes chemotaxis. Approx. 10% of blood lymphocytes are capable of showing in vitro chemotaxis towards various cytokines, but there are specific patterns of reactivity. Thus, pro-inflammatory cytokines like IL-8 induce chemotaxis of both CD4+ and CD8+ T lymphocytes via the upregulation of IL-8 receptors on the T cells. IL-10 only attracts CD8+ cells, but not CD4+ cells; psoriasin is vice versa. The T cell derived cytokines, IL-2 and IL-4, do not exhibit any chemotactic activity on T lymphocytes themselves, but will block a continued chemotactic response by down-regulating chemokine receptors on the T lymphocytes.

We have put foreward a hypothesis which includes the dynamics of T lymphocyte chemotaxis by showing how proinflammatory cytokines released from damaged cells in the skin will attract T lymphocytes into the area ("migration"). Once the T cells meet IL-2 and IL-4 i.e. they recognized other activated T lymphocytes, migration is stopped ("focussing"). The capacity to migrate is likely regulated via chemokine receptor expression.

S4:2

DISCREPANCIES IN PREVALENCE OF ATOPIC DERMA-TITIS

+Broberg, Ann* (SWEDEN) *Sahlgrenska University Hospital

Epidemiologic studies including the prevalence and severity of Atopic Dermatitis (AD) are important to discover specific environmental risk factors in AD (1). In order to compare prevalence rates in different studies, a common disease definition is essential. We performed a study in Göteborg and Kristianstad during October 1997-March 1998 with the aim to evaluate the cumulative incidence (Schultz Larsen's questionnaire), point prevalence (clinical examination) and severity of AD (SCORAD) among 5.5 year old children (2). The UK working party's criteria were used for the clinical diagnosis of AD. There was a significantly higher point prevalence of eczema in Kristianstad (12%) than Göteborg (8%). This may be a true difference, but we cannot exclude the possibility that differences in the treatment, and in the dermatologists' assessments may be the reason for the discrepancy.

S4:3

ENVIRONMENTAL FACTORS INFLUENCING THE EXPRESSION OF ATOPIC DERMATITIS.

- With focus on the possible influence of measles mumps and rubella-vaccination, measles infection, hormonal

contraception use and insulin-dependent diabetes mellitus.

+Braae Olesen, Anne* (DENMARK) *University Hospital of Aarhus

The incidence of AD seems to have increased substantially over the past 40 years. This has led to an intense search for environmental causes of AD. Up to 2/3 of children with AD have IgE-mediated allergic reactions. Studies suggest that these children have a Th-2 immune reactivity pattern with low interferon- γ and increased interleukin-4 production. Measles virus (MV) seems to induce a prolonged Th-2 type immune response. In contrast, IDDM seems to be associated with a Th-1 immune reactivity pattern.

A historical follow-up was performed among a random population sample of 10,000

3-15-year-old children drawn from the Danish Medical Birth Register. Data were collected by a mailed questionnaire, from the Danish National Population Register and the Children's database at Statistics Denmark to study the association between MMR-vaccination, measles infection, hormonal contraception and AD. In a case-control study of the association between AD and IDDM, 920 diabetic children were identified in the Danish Registry for Childhood Diabetes while the population sample served as a control group.

The incidence ratio of AD increased after MMR-vaccination and after measles infection compared with children who were not exposed to MMR-vaccination and measles infection.

The cumulated AD incidence up to age 15 was one third lower among diabetic cases than among non-diabetic controls. The AD incidence was decreased before but not after onset of IDDM. Atopic dermatitis was not associated with pre-pregnancy use of hormonal contraception.

The main results have established an association between MV exposure (measles, mumps and rubella-vaccination and measles infection) and increased AD incidence, which suggest that MV may play a causative role in the immune response modulation and hence initiation of AD expression. The inverse association between AD and later development of IDDM indicates that an inherited or acquired propensity to develop Th-2 immune responses protects against IDDM development.

The main findings are in agreement with the Th1/Th2 hypothesis, which provide a strong argument for continued research into association between atopic diseases and possible short and long term changes of the immune system that may initiate disease expression.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

18

S4:4

TREATMENT OPTIONS IN ATOPIC DERMATITIS

+Reitamo, Sakari* (FINLAND) *Helsinki University Hospital

All current treatment options of atopic dermatitis depend on corticosteroids, as the secondary treatments (UV and immunosuppressive therapies) act usually only as steroidsparing therapies. In contrast, numerous studies with the new topical immunomodulatory agents have shown promising results for both adults and children as corticosteroidreplacing agents. Currently two such compounds, tacrolimus (FK 506) and the ascomycin derivative pimecrolimus (SDZ-ASM 981) are available for clinical studies. These compounds show a similar structure with differences at two sites of the molecule, that are responsible for differences in affinity to the binding-protein and lipophilicity. Treatment with these agents has been tolerated well, with adverse events reported mainly at site of treatment. The most common adverse events have been a burning and/or stinging sensation, and itch at site of application. Other reported adverse events include folliculitis and alcohol intolerance. The main difference between corticosteroids and tacrolimus/pimecrolimus is the lack of atrophogenicity of the new treatments. As there has been no signs of local or systemic immune suppression it seems that the new topical immune modulatory agents could have a potential to replace corticosteroids as the primary treatment of atopic dermatitis.

S5:1

ADVERSE EFFECTS OF HIV-TREATMENT

+Sandström, Eric* (SWEDEN) *Söder Hospital

Introduction of HAART has made a dramatic change in the life expectancy of HIV-infected patients. Since its introduction in 1996 it has been drastic decrease in HIV-related conditions and deaths due to AIDS. There are now 13 drugs on the market and the standard of care is to use three or more in combination. Since many of these drugs, notably the protease anhibitors, affect the cytochrome P450 system a number of drug interactions can influence the efficacy and adverse effects of these and other drugs. Most patients experience adverse effects after starting HAART. The most common are nausea, headache, fatigue and loose stools. However, some adverse effects seem to be class specific. Mitachondrial toxicity seems to be a property of nucleoside analogues while metabolic toxicity seems to be most prominent with protease inhibitors and skin rashes seem to be primarily caused by non-nucleoside analogues. Furthermore a number of adverse effects seem to be drugspecific. Zidovudine causes anaemia, and myopathy. Stavudine has been associated with pancreatitis and probably lipodystrofi. Zalatabine has been shown to be a potent cause of neuropathy and oral ulcers, while lavimudine seems to be the least toxic among this class of drugs. Didanosine is mostly known by the patients because of its unpleasant formulation and diarrhoea. It can also cause pancreatitis and neuropathy. Finally abacavir has been free from common sideeffects causes though it causes a hypersensitivity syndrome in about 3 percent of the patients. Among the nonnucleoside analogues nevirapine frequently causes rashes which can sometimes be fatal and classified as Steven Johnson syndrome. Efavirenz causes rashes and is mostly plagued by night mares and other symptoms from the nervous system. The protease inhibitors as a class metabolic distarbancies lead to increased insulin resistance and elevated blood lipid levels with concerns as to the future effect on coronay heart disease. Among the specific adverse effects indinavir can cause renal stones and renal toxicity in addition to dry skin and toe nail changes. Rashes have been documented in a number of studies. Ritonavir frequently causes perioral parestesial and gastrointestinal upset, particularly if not phased in. Saquinavir seems to be the drug with least recorded adverse effects, however, most of its use has been with suboptimal doses. Nelfinavir is a frequent cause of gastrointestinal upset but otherwise seems well tolerated. The latest addition lopinavir, also causes gastrointestinal upset.

Thus the use of these drugs have many subjective adverse effect that are important to the quality of life, as well as an increasing number of medical concerns mainly would regard to change in the habitus and future risk of coronary heart disease. These difficulties should however not overshadow the dramatic change in life expectancy in HAART treated patients.

S5:2

HIV-TREATMENT - from death to survival

+Gisslén, Magnus* (SWEDEN) *Sahlgrenska University Hospital

The introduction of highly active antiretroviral therapy (HAART) against HIV must be considered a significant milestone in medical history. Since 1996, when protease inhibitors were made available and combination therapy became the treatment of choice against HIV, the morbidity and mortality have significantly declined in the western world. To date, 13 different drugs with three different target mechanisms are available for HIV treatment (Nucleoside analogues Reverse Transcriptase Inhibitors (NRTI), Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTI) and Protease Inhibitors (PI)). Treatment with a combination of at least 3-4 drugs from at least 2 different groups of drugs dramatically decrease the viral replication and leads often to a significant increase in the CD4-cell count. In parallel, a considerably increase in quality of life is seen in most patients but drug intolerance and high pill burden are major drawbacks that limit the use of HAART in some patients. With a lot of patients harboring drug-resistant virus the treatment regimens are getting more complicated and more individualized, to fit each patient and each virus. Several new drugs, some with new mechanisms to fight the virus. are under development and hopefully could some of these problems be solved within the soon future.

S6:1 FUNGAL INFECTIONS IN CHILDREN

+Faergemann, Jan* (SWEDEN) *Sahlgrenska University Hospital

The panorama of fungal infections in children is different from that of adults. Tinea unguium and tinea pedis are rare in children. However, tinea capitis and tinea corporis due to both zoophilic and antropophilic dermatophytes are more common in children. Expecially tinea capitis due to *Trichophyton viotaceum,T. tonsurans* or *T. sudanense* is now common in several of the Nordic countries. Skin infections due to yeasts are generally more common in adults. However, chronic mycocutaneous candidiasis is a disease that usually starts in childhood. Fungal infections in immunosuppressed patients are seen both in children and adults.

S6:2

PAPULAR ERUPTION SECONDARY TO MOLLUSCUM CONTAGIOSUM

+Olafsson, Jon Hjaltalin* (ICELAND); Davidsson, Steingrimur (ICELAND) *Landspitali University Hospital

"Papular acrodermatitis of childhood" also named Gianotti-Crosti syndrom (GCS) is a selflimiting eruption originally believed to be connected to hepatitis-B infection but now considered an unspecifik reaction related to several viral diseases. In 1998 one case of CGS and poxvirus was reported (Cutis 1998, vol 61, 265–267).

In a 3-year period we have observed 29 patients with molluscum contagiosum in our practice, who have also had a papular eruption similar to GCS. Their mean age was 4.4 years (range 2–8 years) 14 boys and 15 girls. Atopic eczema was observed in 13 patients (6 boys, 7 girls). The eruption healed within 4 weeks in all instances but it seems that there is not a connection between the disappearance of molluscum and the eruption. The eruption was treated with a medium strength steroid cream in 24 cases but no treatment was given in 5 cases. Healing time of the eruption was 1–4 weeks. Skin biopsies from CGS lesions are often nonspecific.

S6:3

TREATMENT OF SMALL CHILDREN WITH PORTWINE STAINS. WHAT SORT OF ANAESTHESIA DO WE USE?

+Mørk, Nils-Jørgen* (NORWAY) *Rikshospitalet

The golden standard for the treatment of portwine stains in children is the flashlamp-pumped dye laser with a wavelength of 585 mm and a pulse duration of 450 microseconds. Marked improvement is experienced by 70–80 % of the patients after several treatments. The younger patients require fewer treatment sessions indicating the benefit of starting the treatment during the first year of life.

Optimal analgesia and deep sedation are required for laser treatment of portwine stains in children. This can be performed with little anaesthetic equipment but skilled anaesthetic personnel is a prerequisite.

To relieve pain the patients have topical local anaesthetic (prilocain-lidocain EMLA) at the site of venous cannulation. Sedation is induced either by thiopental 5 mg/kg or propofol 2-2.5 mg/kg. The use of opioids will provide sufficient general analgesia for the laser treatment. Alfentanil has an optimal kinetic profile for short procedures. The doses should be titrated by giving 10 μ g/kg iv each time deepening of analgesia is needed. If the laser treatment is scheduled to be longer than 5–10 minutes, it is beneficial to use fentanyl at a dose of 2 μ g/kg. Additional doses of sedative or analgetic drugs are given when needed.

The anaesthetic challenge during these procedures is to balance the need for analgesia and sedation to the risk of respiratory depression. Additional oxygen reduces the need for assisting the patients with mask ventilation. Post procedure pain is relieved by rectal administration of paracetamol or paracetamol in combination with codeine.

In conclusion these procedures can be performed without complications in an open ward/day care unit. A sufficient level of sedation and analgesia is achieved by drugs with a short duration. A close collaboration between the dermatologist and the anaesthesiologist is necessary during laser treatment.

S6:4

CHILD WITH CHRONIC SKIN DISEASE: HOW IS THE

FAMILY GETTING ALONG ?

+Koulu, Leena* (FINLAND) *Turku University Central Hospital

The general experience among dermatological patients, as well adults as adolescents, is that they are not taken seriously. Chronic illness in the family easily provokes depression. Based on a material of written personal stories by adolescents emotional experiences of the families are presented.

A psychoeducational, cognitive-behavioral coping program for families is described in the lecture. The method is closely modeled after the Coping with Depression Course by Lewinsohn et al. The course is designed for use with groups of six to eight participants and consists of ten 2-hour sessions. The areas covered are relaxation, pleasant events, irrational and negative thoughts, social skills, communication, and problem solving. The aim of the course is to prevent major depression and to improve quality of life in families having children with chronic dermatitis. The parents and adolescents have their own sessions.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

Symposia

S6:5 FRAUD AND NONMEDICINAL TREATMENTS IN

PEDIATRIC DERMATOLOGY

+Serup, Jørgen* (SWEDEN) *Linköping University Hospital

Dermatologists often recommend nonmedicinal treatments as exemplified by moisturisers used for ichthyosis and atopic dermatitis. Patients themselves frequently use cosmetic products, special branded products and alternative medicine parallel to or substituting topical medicines.

Registered medcine is the only category where the claimed content of active chemical(s) is strictly assured including stability during the shelter life period of the product. The established manufacturers of cosmetic products of course share a similar interest that the content of their products is honest and assured.

In recent years nonmedicinal products especially branded or launched for use in skin diseases were noted in Scandinavia exemplified by the product Skin-Cap produced in Spain. This product contained clobetasole propionate. Another product named Psorial contained halcionide and triamcinolone. A British study found dexamethasone in 8 of 11 Chineese herbal creams analysed with gas chromatography. Fradulent manufacturers operate internationally and easily replace problem markets with convenient markets and internet trade.

Complementary treatments which may interfere with regular treatment also include acupuncture, homeopathy and others.

Dermatologists should actively detect fraud related to nonmedicinal products and treatments and notify authorities about suspected cases. Unauthorized enrichment of inefficient products with high potency steroids seems to be especially frequent. The product is typically produced by some unknown manufacturer and promoted directly to the patient who, shopping the dermatologist´s office, may enthusiastically report on some "wonderful" alternative remedy miraculous in his case, and even ask the dermatologist to recommend it to other sufforers of skin diseases.

S7:1

PHOTODYNAMIC THERAPY. MECHANISMS AND PROCEDURES

+Moan, Johan* (NORWAY); Juzenas, Petras* (NORWAY); Juzeniene, Asta* (NORWAY); Ma, Li-Wei* (NORWAY); Iani, Vladimir* (NORWAY)

*The Norwegian Radium Hospital

Photodynamic therapy (PDT) of cancer is based on the tumour selective uptake and retention of certain photosensitizers, and on light exposure of the tumour. The selective uptake/retention is related to a number of physiological factors in tumours: A low pH, a large vascular volume, a large number of macrophages, a leaky vasculature, and a poor lymphatic drainage. PDT acts through vascular damage (water-soluble sensitizers, short incubation times), through direct tumour cell inactivation (lipophilic sensitizers, long incubation times) and through immunological effects. Tumour tissue appears to be more sensitive to PDT than normal tissue. The most recent version of PDT, socalled ALA-PDT, is based on the tumour selective production of the photosensitizer protoporphyrin IX from 5-aminolevulinic acid (ALA). Determinants for tumour selectivity of ALA-PDT are: Differences in enzyme activities between tumour tissue and normal tissue, a high permeability of the stratum corneum of skin overlaying tumours, a high tumour blood flow and a high tumour temperature. PDT is efficient only in the presence of O2. Since O2 is consumed during PDT, since vascular damage develops, whereby the oxygen supply is reduced, and since the optical penetration depth changes during PDT (oxy-hemoglobin has a lower absorbance at 630 nm than hemoglobin), the mode of light delivery (fluence rate, fractionation of the exposure) is of crucial significance for the outcome of PDT. The use of ALA derivatives with different lipophilicities and the choice of other wavelengths than 630 nm may be advantageous under certain conditions.

S7:2

PHOTODYNAMIC THERAPY WITH 5-AMINOLEVULINIC ACID OF RECALCITRANT FOOT AND HAND WARTS

+Stender, Ida Marie* (DENMARK); Renhua, N.** (DEN-MARK); Fogh, H.** (DENMARK); Gluud, C.** (DENMARK); Wulf, HC** (DENMARK) *Hudklinikken,**Bispebjerg Hospital

Photodynamic therapy (PDT) with topical 5-aminolevulinic acid (ALA) followed by irradiation with incoherent light (ALA-PDT) for warts have had beneficial results.

In 45 patients, 232 recalcitrant foot and hand warts were in a parallel, double-blind clinical trial randomly assigned to six repetitive ALA-PDT or placebo-PDT interventions combined with standard treatment encompassing paring followed by a keratolytic.

Both the number of vanishing warts and the difference in relative wart area of persisting warts one and 2 months after last treatment were significant (p< 0.05) in favor of ALA-PDT.

ALA-PDT is superior to placebo-PDT both when wart area and number of vanishing warts are considered.

S7:3

IMAGING FLUORESCENCE OF BASAL CELL CARCINOMAS

+Ericson, Marica* (SWEDEN); Sandberg, Carin** (SWEDEN); Wennberg, Ann-Marie** (SWEDEN); Gudmundsson, Fredrik* (SWEDEN); Rosén, Arne* (SWEDEN); Larkö, Olle** (SWEDEN)

*Chalmers University of Technology,**Sahlgrenska University Hospital

It has been shown that the photodynamic technique for treatment of skin cancer also can be applied for diagnostic purposes. By imaging the fluorescence from the photosensitiser in the skin, the tumor extension can be determined.

21

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

In a study carried out at the Sahlgrenska University Hospital in Göteborg, the fluorescence from protoporphyrin IX, Pp IX, in ALA treated basal cell carcinoma, BCC, was investigated as a function of ALA application time.

The Pp IX fluorescence was recorded by using a CCD camera equipped with filter during irradiation of light in the wavelength region 360-405 nm. The contrast, defined as the ratio between the fluorescence intensity in tumor and normal skin, was evaluated as a function of ALA application time. 40 patients were included in the study. The patients were randomly allocated to 4 groups, each group with a certain ALA application time of 1, 2, 3 and 4 hours respectively.

A significant difference in contrast between the groups with 1 and 3 hours of ALA application was obtained. Between the other groups no significant difference could be detected due to the large variance within the groups. However, the trend indicated that the 3 hour ALA application yields a better contrast in general. The fluorescence variance in normal skin is up to 40%, which means that a contrast value above the normal variance level could indicate abnormalitv.

The study showed a correlation between the fluorescence images and histological pattern however the individual variations were large. Further studies are planned in order to further improve and optimise the technique.

S7:4

SKIN AUTOFLUORESCENCE IN DEMARCATION OF

BASAL CELL CARCINOMA

+Renhua, Na* (DENMARK); Rossen, Kristian* (DENMARK); Stender, Ida-Maria* (DENMARK); Wulf, Hans Christian* (DENMARK)

*Bispebjerg Hospital

In skin cancer treatment, it may be difficult to find the borders (e.g. demarcation) of the tumors, since part of it may not be visible to the naked eyes. This may result in incomplete removal of tumor tissue, and consequent recurrence and repeated treatments, which may cause tissue destruction and cosmetic morbidity.

Employed a fluorescence spectrometer system, we measured in vivo the autofluorescence of BCCs and normal surrounding skin for its potential in tumor demarcation. Protoporphyrin IX (PpIX) fluorescence and histopathology examination were used as control methods.

The 370:452 nm fluorescence was 53% (18-84%) (median (range)) lower in the BCCs than in normal skin (p<0.001). This low intensity fluorescence extended beyond the visible tumor border for at least 3 mm in 56% of the tumors. The extension was comparable to that of the protoporphyrin IX fluorescence. Gross detection of the autofluorescence provoked by 370 nm radiation enabled finding the borderlines of the BCCs, of which 58% were verified by histopathology examination.

The autofluorescence detection may be useful in skin cancer demarcation. Gross detection of autofluorescence is sim-

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

ple, fast and non-invasive. Yet its effectiveness is limited in deeply located tumors. Its clinical utility remains to be tested in practice.

S7:5

PHOTODYNAMIC THERAPY FOR PSORIASIS AND EXTRAMAMMARY PAGET 'S DISEASE

+Ros, Anne-Marie* (SWEDEN) *Karolinska Institute

Photodynamic therapy (PDT) for psoriasis using 5-aminolevulinic acid (ALA) topically has been tried in recent years, since long-term side effects are supposed to be less than after PUVA. A few studies have been published and shown moderate effectiveness in the treatment of psoriasis. Our study confirm this. The moderate clinical effect and the intense patient discomfort during therapy makes PDT with ALA not suitable for psoriasis according to our experience.

Extramammary Paget's disease is a neoplasm that is usually treated with surgery or radiation. It is common with recurrences after therapy. PDT with ALA for extramammary Paget's disease may be effective according to a few case reports. Our experience confirm the effectiveness although several treatments are often necessary. Pain during and after therapy is a problem.

S8:1

22

EPOXY DERMATITIS - WHAT IS NEW?

+Magnus Bruze

Department of Occupational and Environmental Dermatology University Hospital, 205 02 Malmö, Sweden

Epoxy resin systems always consist of a resin and a hardener and may also contain a reactive diluent. The most commonly used epoxy resin is based on the monomer diglycidylether of bisphenol A (DGEBA), which is the major contact sensitizer in resins of this type. Commercial epoxy resins are mixtures of oligomers of different molecular weights and it is the concentration of DGEBA which determines their sensitizing capacity. Other types of epoxy resin used are based on diglycidylether of tetrabromobisphenol A, tetraglycidyl-4,4'methylenedianiline, triglycidyl derivative of p-aminophenol and o-diglycidyl phtalate.

There are many hardeners which may act at either room temperature (cold curing) or elevated temperature (thermal curing). The cold curing hardeners are mostly polyamines, polyamides or isocyanates. The hardeners used for thermal curing are acids and anhydrides or aldehyde condensation products, e.g. phenol-formaldehyde resins, melaminformaldehyde resins and urea-formaldehyde resins.

Reactive diluents contain one or more epoxide groups and they are used primarily to reduce the viscosity of the epoxy resin system. The epoxy reactive diluents are either aromatic, like phenyl and cresyl glycidylether or aliphatic, e.g. butyl and allyl glycidylether, all being potent sensitizers.

In the standard patch test series an epoxy resin based on DGEBA is present. To trace contact allergy to other resins,

hardeners and reactive diluents, these must be tested separately. Test series with epoxy substances are available from the major suppliers of patch test preparations. In a finished product there will always be residual monomers, higher concentrations when cold curing has been used and it is therefore often advisable to patch test finished products to which the patients are exposed. In the lecture attention will be paid to news within the epoxy dermatits area.

S8:2

OCCUPATIONAL PLANT DERMATOSES.

+Paulsen, Evy* (DENMARK) *Odense University Hospital

The development of the sesquiterpene lactone (SL) mix and the Compositae mix has made routine screening for Compositae allergy possible and has substantiated the leading role of this plant family in plant dermatitis in Europe today.

The prevalence of occupational sensitization to Compositae ranges between 11% (SL mix) (1) and 28% (Cornpositae mix) (2) in consecutively tested Compositae-allergic patients. A Danish study in gardeners and greenhouse workers detected a sensitization rate almost twice as high as in consecutively tested persons (3).

This presentation deals with the results of routine screening with the SL mix supplemented with various Compositae extracts and allergens in consecutive eczema patients in the Danish county of Funen where a large greenhouse industry is located.

In the first 8 years the prevalence of Compositae sensitization was 190/4386 (4.3%) and 42 (22%) of these were suspected to be occupationally sensitized. This group was characterized by a lower mean age and a larger proportion of persons without any other contact allergies compared to the non-occupationally sensitized. Chrysanthemum (*Dendranthema*), marguerite daisies (*Argyranthemum frutescens*) and lettuce (*Lactuca sativa*) were the most important sensitizers. Most of the patients were/had been employed in some kind of greenhouse job or floristry, but home helps (attending clients' plants) and a zone therapist (sensitized by among other things arnica in a massage oil) were also diagnosed as occupationally sensitized.

Finally, some rare causes of occupational plant dermatoses will be presented.

References

- J.S. Ross H. du Peloux Manage, J.L.M. Hawk I.R. White. Sesguiterpene lactone contact sensitivity: clinical patterns of Compositas dermatitis and relationship to chronic actinic dermatitis. Contact Dermatitis 1993; 29: 84–87.
- B.M. Hausen. A 6-year experience with Compositae mix. Am J Contact Dermatitis 1996; 7: 94-99.
- E. Paulsen, J. Søgaard, K.E. Andersen. Occupational dermatitis in Danish gardeners and greenhouse workers (III). Compositas-related symptoms. Contact Dermatitis 1998; 38: 140-146.

S8:3

IRRITANT CONTACT DERMATITIS - CLINICAL AND EXPERIMENTAL ASPECTS.

+Lindberg, Magnus* (SWEDEN) *Karolinska Institutet

During the past decade there has been an increasing interest in irritant contact dermatitis. It has been demonstrated that chemically different irritants will produce different responses at the cellular and sub-cellular level following application on the skin surface. From a clinical point of view, irritant contact dermatis can also be presented in different clinical forms and can also be a negative worsening factor in other skin diseases.

The presentation will cover aspects on the mechanisms of irrritancy and the implications for the clinical appearence of irritant contact dermatitis.

S8:4

TRENDS IN OCCUPATIONAL DERMATOLOGY

+Björkner, Bert* (SWEDEN) *Malmö University Hospital

Since the father of occupational dermatology, Bernardino Ramazzini, published his book Diseases of Tradesmen, 1700, an increasing number of chemicals, that can cause hazards in the workplace has been introduced during the years. On the other hand there is a greater knowledge of the offending chemicals in the environment and their effects on humans. During the years, there has been a greater awareness among dermatologists and others of the importance of occupation in the causation of dermatologic diseases. New offending substances are repeatedly introduced on the market, while other chemicals are expelled, not because of the possible hazard they can cause on exposed workers, but mostly for technical and economical reasons. The industrial world is complex and ever changing. For instance, service industries have slowly enlarged in the last century to become the largest employers, rather than manufacturing. The knowledge of a few key industrial processes is in order to understand the exposure that may have produced a patient's dermatosis and to establish an occupational association. Without such basic information, it is difficult to intelligently approach the problem of work-related skin disease or, often, to manage it effectively. A knowledge of industrial processes is also vital if primary preventive measures are to be taken to protect other workers who may be at risk.

TREATMENT OF MYCOPLASMA GENITALIUM INFEC-TIONS.

S9:1

+Falk, L.* (SWEDEN); Skov Jensen, J.** (DENMARK) *Örebro Medical Centre Hospital,**Statens Serum Institut

Aim: To compare signs, symptoms and efficacy of treatment among STD-clinic attendees with ure-thritis and cervicitis and to estimate the prevalence of Mycoplasma genitalium (Mg) and Chlamydia trachomatis (Ct)

Methods: 464 women and 532 men attending an STD outpatient clinic were tested for Mg and Ct by PCR on first voided urine and for women also by PCR (Mg) and cell culture on endocervical swab (Ct). Patients with cervicitis and or urethritis were treated with doxycycline for 8 days or lymecycline ten days. All Mg and Ct positive patients were requested for a check up visit. Asymptomatic untreated Mg positive patients or patients attending due to Mg contact tracing or Mg positive at check up were treated with azithromycin 500 mg first day and 250 mg the following 4 davs.

Results: 26 Mg positive women (prevalence 5,6%) aged 16-39 years (median 22) and 41 Mg positive men (7.7%) aged 18-55 years (median 27) were found. Corresponding figures for Ct were 44 women (prevalence 9.5%) aged 16-32 years (median 22) and 60 men (11.3%) aged 16-56 years (median 23.5). There was no significant difference in symptoms between Mg positive and Ct positive women (OR 1.4, 95% CI 0.48 to 4.17), but fewer Mg positive women had signs of cervicitis and or urethritis (OR 0.49, 95% CI 0.14 to 0.60). Mg positive men more often had symptoms of urethritis than Ct positive men (OR 2.9, 95% CI 1.82 to 5.12) whereas signs were more common among Ct positive men (OR 0.35, 95% CI 0.09 to 0.79). 4/41 Mg positive men and 4/26 Mg positive women had a concurrent Ct infection, 20 of 31 (64,5%) of the Mg positive patients initially treated with tetracyclines were still Mg positive at check up. None of the 38 patients with a 5-day course of azithromycin were Mg positive at follow up.

Conclusion: Only slight differences in the distribution of signs and symptoms between Mg and Ct positive patients were found. Whereas the standard treatment for uncomplicated urethritis and cervicitis with tetracyclines appeared to have a good clinical efficiency at follow-up, it did not eradicate Mg. A five day-course of azithromycin eradicated Mg efficiently, however, further studies are needed since patients treated with azithromycin were not directly comparable to those treated with tetracyclines.

S10:1

GENODERMATOSES - AN INTRODUCTION

+Vahlquist, Anders* (SWEDEN) *University Hospital, Uppsala

No aspect of Dermatology has gained so rapidly in knowledge over the past 10 years as the genodermatoses. To date the etiologies have been unravelled for more than 30 mono-

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

genetic skin disorders, including epidermolysis bullosa, lamellar ichthyosis, Netherton's syndrom, erythrokeratodermia variabilis, ectodermal dysplasia, Darier's and Hailey-Hailey diseases. It is now often possible not only to make a correct diagnosis in ambiguous cases, but also to offer prenatal diagnosis and carrier analysis to parents in need of that. This symposium will exemplify ongoing research in the field of epidermal genodermatoses where Nordic scientists have made considerable contributions both to the advance of molecular genetics and to the understanding of skin biology. Hopefully, this knowledge will soon lead to more sophisticated treatments and eventually also to gene therapy for the most disabling diseases. In the meantime it is important to try to improve current therapies and to learn more about how we can help our patients to improve their quality of life.

S10:2

EPIDERMAL TRANSGLUTAMINASE (TGM1) MUTA-

TIONS IN LAMELLAR AND NON-LAMELLAR ICHTHY-

OSES - A LARGER SPECTRUM THAN ANTICIPATED

+Pigg, Maritta* (SWEDEN): Vahlquist, Anders** (SWEDEN): Gedde-Dahl, Tobias*** (NORWAY); Gånemo, Agneta*' (SWEDEN); Virtanen, Marie** (SWEDEN); Westermark, Per* (SWEDEN); Haußer, Ingrid**** (GERMANY); Dahl, Niklas* (SWFDFN)

*Rudbeck Laboratory and **University Hospital, Uppsala, ***Rikshospitalet and University of Oslo, ****University of Heidelberg

Autosomal recessive congenital ichthyosis (ARCI) is a clinically and genetically heterogeneous disorder of keratinization. Mutations in the transglutaminase 1 (TGM1) gene may be associated with the clinical subtypes lamellar ichthyosis (LI) and non-bullous congenital ichthyosiform erythroderma (CIE). LI and CIE usually represent severe phenotypes of ARCI with general scaling of the skin. We investigated the TGM1 gene in ARCI patients of various phenotypes. In patients from 29 families, clinically classified as LI or CI, TGM1 gene 16 different mutations were found in 86% of the alleles. In four patients from three unrelated families, clinically presenting a milder phenotype (non-LI, non-CIE) five different missense mutations in the TGM1 gene were found (Val209Phe; Arg687His; Tyr365Cys; Arg396Leu; Asp306Glu). Electron microscopy (EM) of skin biopsies showed a picture consistent with

IC type I or IC type II in patients showing TGM 1 gene mutations despite of the clinical expression. The findings indicate that mutations in the TGM1 gene may cause ARCI clinically classified as neither LI nor CIE but with the EM diagnosis IC type I or IC type II. The five missense mutations found in the non-LI, non-CIE patients might result in less severe phenotypic consequences than in LI/CIE, due to conservative amino acid substitutions or the specific heterozygous combination of mutations.

S10:3 LIFE QUALITY ASSESSMENT IN ICHTHYOSIS PA-TIENTS.

+Gånemo, A.* (SWEDEN), Vahlquist, A.* (SWEDEN); Sjödén, P-O* (SWEDEN); Lindberg, M.* (SWEDEN) *University Hospital, Uppsala

Non-bullous ichthyosis is either present at birth (Lamellar ichthyosis 'LI') or develops in early childhood (Ichthyosis vulgaris 'IV' or X-linked recessive ichthyosis 'XRI'). Today there is no cure for ichthyosis. Skin diseases, such as psoriasis and atopic eczema, have been shown to have a significant adverse impact on the Health-Related Quality Of Life (HRQOL). The overall aim of the present study was to investigate the HRQOL in patients with LI, XRI and IV. To this end we used the Dermatology Life Quality Index (DLQI) and SF-36, and a subjective measure of disease activity, using a visual analogue scale (VAS). One aim was to study the correlation between VAS and the Quality of Life instrument DLQI and SF 36. All participants received the following questionnaires: DLQI, SF-36, VAS and sociodemographic questions. A total of 121 persons aged 17-78 years completed the questionnaires (LI 37, XRI 36, IV 48). The proportion of males was 55% in the group as a whole. The mean total score for DLQI was 6.1, significantly higher for LI than for XRI (7.70 vs 4.17). SF-36 showed significantly lower (worse) scores for the study group compared to the Swedish norm scores, in 6 of the 8 dimensions. No difference in SF-36 was found between men and women or between the groups LI, XRI or IV. The estimated correlation between the instruments were in the expected direction and mostly statistically significant. The results confirm the general impression that ichthyosis has an adverse impact on the HRQOL. However, the clinical symptoms may not always give a good guidance as to how the patient experiences her quality of life.

S10:4

"SCANDINAVIAN" KERATIN MUTATIONS IN EPIDERMOLYTIC HYPERKERATOSIS (BULLOUS ICH-

THYOSIS).

+Virtanen, M.* (SWEDEN); Gedde-Dahl, T. (NORWAY); Mørk, N-J, (NORWAY); Bowden, P. (UK); Vahlquist, A. (SWEDEN)

*University Hospital, Uppsala

Epidermolytic hyperkeratosis is a rare inherited disease of the skin caused by a dominant-negative mutation in keratin 1 (K1) or 10 (K10). Keratins are the major structural proteins in epidermis and mutations cause instability of the intermediate filament and keratinocyte fragility. No curative treatment is available, but some patients benefit from retinoid therapy. More knowledge is needed about the genotype/phenotype correlation in epidermolytic hyperkeratosis and the mechanism of action of retinoids including the regulation of keratin expression. Fifteen patients were identified in Scandinavia, 13 with a generalised disease and 2 with localised lesions. Different types of mutation were identified such as point, splice site, deletion, and deletion-insertion mutations. An association was found between mutations in K1 and the appearance of palmoplantar keratoderma. Only patients with K10 mutation benefited from retinoid treatment, although no differences in the effects on mRNA levels for K1 and K10 were detected. However, retinoids caused a pronounce down-regulation of K2e in upper epidermis and upregulation of K4 not normally present in the skin.

In conclusion, several novel keratin mutations have been shown to cause epidermolytic hyperkeratosis, and a few examples of genotype/phenotype correlations have been found. Treatment with retinoids is only useful for patients carrying a K10 mutation, possibly because they are less vulnerable to the pronounced down-regulation of K2e also seen in normal skin.

S10:5

EPIDERMOLYSIS BULLOSA SIMPLEX: MOLECULAR CHARACTERIZATION OF THE MUTATIONAL SPEC-TRUM IN DANISH PATIENTS

+Sørensen, C.B.* (DENMARK); Ladekjær-Mikkelsen, A.-S.* (DENMARK); Andresen, B.S.* (DENMARK); Brandrup, F.** (DENMARK); Veien, N.K.*** (DENMARK); Buus, S.K.*** (DENMARK); Anton-Lamprecht, I.**** (GERMANY); Kruse, T.A.* (DENMARK); Jensen, P.K.A.* (DENMARK); Eiberg, H.***** (DENMARK); Bolund, L.* (DENMARK); Gregersen, N.* (DENMARK)

*Aarhus University Hospital,**Odense University Hospital,***The Dermatology Clinic,****Ruprecht-Karls University,*****Genome Group/RC Link

Epidermolysis Bullosa Simplex (EBS) is a group of autosomal dominant inherited skin disorders caused by mutations in the keratin 5 or 14 genes. Three major subtypes of EBS has been classified clinically, Weber-Cockayne (WC), Koebner (K), and Dowling-Meara (DM), of which the DM form is the most severe. The severity of the disease seems to correlate with the position of the mutation in the genes.

We have investigated three Danish families with EBS-WC, two families with EBS-K, and two sporadic cases with the DM form of EBS in order to analyse the mutational spectrum in Danish EBS patients. PCR amplification was performed of all exons and flanking intron regions in the two genes using genomic DNA purified from the patients, relatives, and unrelated normal individuals. Automatic sequencing revealed three novel EBS-associated mutations in K14, as well as a novel and a known mutation in K5. None of these mutations were found in 100 normal alleles. An identical mutation in K14 was found in the three seemingly unrelated EBS-WC families indicating that these families were related by a common ancestor. This was supported by molecular haplotyping of the mutant chromosome in the three families.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

S10:6 THE ROLE OF PLECTIN FOR THE INTEGRITY OF

HUMAN SKIN

+Koss-Harnes, Dörte* (NORWAY); Høyheim, B. (NORWAY); Gedde-Dahl, T. (NORWAY) *The National Hospital, Oslo

Plectin (from greek: plectin, web or network), one of the plakin proteins, is a plaque protein and acts as a general crosslinking element of intermediate filaments and is expressed in a wide variety of tissues.

The human plectin gene PLEC1 consists of 32 exons . Various isoforms exist, produced by alternative spicing of different first coding exons into exon 2 including a rodless isoform. Plectin ablation in mice by gene targeting lead to a severe phenotype. Plectin has also been shown to serve as a autoantigen in paraneoplastic pemphigus.

Mutations in the human plectin gene, first reported in 1996, cause a recessive type of Epidermolysis bullosa with muscular dystrophy. Recently a dominant mutation in the plectin gene was shown to cause Epidermolysis bullosa Simplex-Ogna (EBS-O). The clinical signs of plectin skin diseases aswell as their ultrastructural and genetical background will be discussed.

S10:7

HEREDITARY HYPOTRICHOSIS SIMPLEX OF THE

SCALP. CLINICAL AND MOLECULAR INVESTIGATIONS

IN A DANISH FAMILY

+Bygum, Anette* (DENMARK): Betz, RC (GERMANY): Nöthen, MM (GERMANY); Ibsen, HHW (DENMARK); Rasmussen, HB (DENMARK); Brandrup, F (DENMARK) *Odense University Hospital

A nine generation Danish family with Hereditary Hypotrichosis Simplex of the Scalp, Toribio-Quinones type (HHS) is presented with clinical data and the results of molecular genetic studies.

50 family members were examined and 21 were affected. A genomewide linkage scan (using highly polymorphic microsatellite markers) was performed on DNA from the Danish family. Spanish descends from the originally described family (reported by Toribio and Quinones) were included in the linkage study.

The patients start to loose scalp hair in childhood or early puberty and it progresses to an almost total alopecia by the early twenties. There is no sign of cicatricial alopecia or other associated ectodermal defects. There is no sex difference. Morphological examination of hairs by light and scanning electron microscopy shows a normal hair shaft morphology

The study identified the locus for HHS on the short arm of chromosome 6 (6p21.3). A search for candidate genes in the region has identified several candidate genes for HHS. The locus is distinct from the locus for other primary hair disorders, namely monilethrix, on chromosome 12q13 and alopecia universalis congenitalis on chromosome 8p21. Further research based on a candidate gene approach is going on. The identification of the HHS gene may shed light on the molecular mechanisms of human hair development.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

26

S10:8

ICHTHYOSIS-PREMATURITY SYNDROME - AN UN-KNOWN, FREQUENT AND ANCIENT

"MID-SCANDINAVIAN" RECESSIVE DISEASE

+Petra T. Kampman (NORWAY) Department of Dermatology, Rikshospitalet, Oslo

In Norway a new case of the lchthyosis-Prematurity Syndrome (IPS) is born annually. IPS is a previously unrecognized syndrorne of obstetric, pediatric and dermatological significance (1). It was first observed as unique in the early 1980ies by its skin ultrastructural features and published as "Ichthyosis congenita type IV" (2). IPS is an autosomal recessive disease. The mutation, carried by 2% of the population of Middle Norway, must be of prehistorical origin. A few cases are reported from Finland and Italy. In IPS the pregnancy is complicated by polyhydramnion and an opaque amnion fluid due to shedding of large amounts of epidermally derived cells. Premature birth occurs in the 32nd week of pregnancy. Due to aspiration of the amnion debris the child may become severely asphyctic after delivery, and in unrecognized cases these children might not survive. At birth the skin is covered by thick caseous desquarnating epidermis which surprisingly improves to a benign dryness of the skin within the first 1-2 weeks. The dry skin may later be misdiagnosed as atopic skin, partly due to the accompanying dermographism and atopic manifestations during infancy. A typical case-history will be presented.

- 1. Gedde-Dahl T Jr. The lchthyosis-Prematurity Syndrome (IPS) (abstr.) Case presentation at Syndromdiagnostikk, Dept.s of Medical Genetics and Pediatrics, Ullevål Hospital, Oslo (K.H. Ørstavik) on Aug. 28th, 1996.
- 2. Anton-Lamprecht l. The Skin. In: Papadimitriou JM, Henderson DW, Spagnolo DV (eds.): Diagnostic ultrastructure of non-neoplastic diseases. Churchill Livingstone, Edinburgh, 1992 b, pp. 459-550.

S11:1

RESISTANCE TO ANTIBIOTICS IN DERMATOLOGY

+Gaustad, Peter* (NORWAY) *National Hospital

Bacterial resistance has compromised the effectiveness of antibiotics and resistance is one of the major problems of medicine today. Resistance can be defined as the situation where the minimal inhibitory concentration is higher than the concentration in the focus of infection. The evolution of resistance in Gram positive coci, a major group of pathogens in dermatology, started in the 60ties with penicillinresistant Staphylococcus aureus. In the 80ties emerged the MRSA (methicillin resistant S. aureus), in the 90ties the vancomycin-resistant enterococci and at present we have to handle with vancomycin-intermediate resistant S. aureus. Resistant bacteria can occur by mutation. More commonly resistance emerge from the ability of bacteria to pick up resistance genes by three mechanisms: transformation, transduction or conjugation. The genes might code for 1)

efflux pumps ejecting antibiotics from bacteria, 2) decreased permeability of the bacteria 3) enzymes that degrade the antibiotics 4) decreased binding of antibiotics to the target. Resistance genes can be transferred from Gram positives to Gram negatives and vice versa. Resistance genes can reside on the bacterial chromosome or on plasmids. In dermatological patients the most important resistance problems are found in the Gram positive pathogens S. aureus, Propionibacterium acnes and, to some extent, streptococci. More than 90% of S. aureus are resistant to penicillin, and more than 50% of the strains are methicillin resistant. Multiresistance including macrolides and tetracyclines is common and resistance to quinolones is developing rapidly. Streptococcus pyogenes is still susceptible to penicillin, but increasing problems with macrolides and tetracyclines have been reported. After oral or systemic treatments, P. acnes develops resistance in more than 50% of the cases. To limit the development of antibiotic resistance, it is necessary to establish an antibiotic policy.

S11:2

SKIN MANIFESTATIONS OF STREPTOCOCCAL INFEC-TIONS

+Broberg, Ann* (SWEDEN) *Sahlgrenska University Hospital

The major streptococcal pathogens in humans belong to group A, commonly referred to as Streptococcus pyogenes (S. pyogenes). Group B, C and G streptococci can also act as pathogens in humans. Involvement of streptococci in cutaneous diseases can be by 1) direct infections of skin or subcutaneous tissue,2) secondary infection, 3) tissue damage from circulating toxin,4) skin lesions attributed to allergic hypersensitivity to streptococcal antigens. Streptococci can also provoke other skin disease. Red infiltrate in the face was seen in four patients (3 females and one male, aged 8, 9, 10 and 24 years) with sore throat. S. pyogenes was isolated from throat swabs and the skin lesions disappeared during treatment with antibiotics in all patients. The skin lesions are suggested to be a reaction to the streptococcal infection.

S11:3

DERMATOMYCOSES OF THE FEET - MORE THAN MEETS THE EYE AT FIRST SIGHT?

+Sigurgeirsson, Bár_ur* (ICELAND) *University Hospital

Dermatomycoses of the feet are common. Recent studies have shown a prevalence of 2–10% for onychomycosis and 10–20% for tinea pedis. Both ailments are sometimes considered trivial, but this is not the case. Onychomycosis is often accompanied with dermatomycoses of the adjacent skin such as interdigital or plantar ("moccasin type") tinea pedis. Several studies have shown that these diseases can have severe impact on quality of life and should not be trivialized. Fissuring, inflammation, itching and pain are signs and symptoms, and patients may also have reduced self-esteem. Recently dermatomycoses of the feet and toeweb intertrigo have been linked to erysipelas, a severe and potentially life-threatening disease. Individual case reports in the literature and personal experience has shown that dermatomycoses can aggravate the symptoms of atopic dermatitis, can cause hand eczema, erythema nodosum and other reactive dermatoses.

Several cases from the literature, individual studies and the author's cases will be presented.

S11:4

TROPICAL SKIN INFECTIONS/INFESTATIONS IN TRAVELLERS

+Brandrup, Flemming* (DENMARK) *Odense University Hospital

In recent decades we have witnessed a remarkable growth in international travel, resulting in considerable variation in exposure to – not only cultural and climatic conditions – but also to infectious agents and toxic stings and bites; e.g. when young backpackers are touring tropical areas for a few months. An increasing occurrence of rare tropical skin diseases has been recorded among travellers, some of which will be presented in a short clinical review, covering dermatoses caused by protozoan (cutaneous leishmaniasis), helminths (cutaneous larva migrans, strongyloidiasis, onchocerciasis, swimmers itch), arthropods (tungiasis, myiasis, bed bugs, tick bites) and some bacterial infections (swimming pool-granuloma, ecthyma, buruli ulcer). Treatment with the anti-parasitic drugs Ivermectin, Albendazole and Sodium stibo-gluconate will be discussed.

S12:1

27

THE EUROPEAN SPECIALIST SECTION (U.E.M.S) -

RESPONSIBILITIES AND RIGHTS.

+Bergbrant, Ing-Marie* (SWEDEN) *Sahlgrenska University Hospital

European Union of Medical Specialists was created in 1958 when representatives delegated by the professional organisations of medical specialists of the six member countries of the very new European Community (EEC) met in Brussels. Membership was further expanded as additional countries joined the EEC. In addition three European Free Trade Association (EFTA) countries – Iceland, Norway and Switzerland – are full members of U.E.M.S.

U.E.M.S. is the European representative organisation of the various National Associations of medical specialists in the member countries, working through 36 Specialists Sections. The objectives of U.E.M.S. includes the promotion of quality patient care through the harmonisation and improvement of quality of specialist medical care in the member countries and the encouragement and facilitation of Continuing Medical Education for European specialists.

U.E.M.S. has produced charters on Specialists Training, Visitation of Training Institutions and Continuing Medical Education (CME) and in January 2000 formally established the European Accreditation Council for CME.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

S13:1 SOLAR KERATOSIS, BOWEN'S DISEASE AND KERATO-

ACANTHOMA - are they all squamous cell carcinoma?

+Sviland, Lisbet* (NORWAY) *Haukeland Sykehus

There can be no doubt that solar UVR is a potent carcinogenic agent and the entities to be discussed are all associated with sun exposure There appears to be a general consensus that solar keratosis and Bowen's disease are "precursor" lesions of squamous cell carcinoma, but there is considerable dispute about prevalence and rate and incidence of transformation to invasive malignancy. Regression has been reported by several authors but disputed by others who maintain that both solar keratosis and Bowen's disease are superficial squamous cell carcinoma that keep growing albeit slowly.

A number of viral agents have been implicated in the aetiology of both Bowen's disease and keratoacanthoma (KA). Especially human papillomavirus (HPV) has been studied, but the role of this virus in the aetiology arid pathogenesis remains to be elucidated. KA continues to cause diagnostic problems and there are many examples which have been reclassified as squamous cell carcinoma on subsequent clinical grounds. Explanations include malignant transformation of a KA and the possibility that KA is a type of squamous cell carcinoma that usually regress because it is caused by HPV that has disappeared.

S13:2

PUVA AND SKIN TUMOURS

+Karvonen, Jaakko* (FINLAND) *Oulu University Hospital

PUVA is a potentially carcinogenic treatment. Both 8-methoxypsoralen (8-MOP) and trioxsalen plus UVA are mutagenic in bacteria. Topical 8-MOP-PUVA induced multiple squamous cell carcinomas (SCC) in a lifelong study in mice whereas topical trioxsalen-PUVA did not.

In the American PUVA study 618 SCCs were observed in 144 out of 1,380 patients treated with systemic 8-MOP-PUVA and followed-up for13 years. The relative risk (RR) for contracting SCC in the whole group was 12 and for those patients with more than 300 treatments it was 33. In Sweden, 67 SCCs were observed in 2,447 patients treated with systemic 8-MOP-PUVA (RR 7.7). The risk for melanoma after systemic 8-MOP-PUVA was moderately increased in the American PUVA study but not in European studies.

No increase in SCC has been reported after topical trioxsalen-PUVA. In a Finnish-Swedish 15-year follow-up study on 944 psoriatics only 3 SCCs were observed; the expected value being 2.8 (RR 1.1). Only one small study including 158 patients has been published on the SCC risk of topical 8-MOP-PUVA; no SCCs were found.

In conclusion: Systemic 8-MOP-PUVA causes SCCs in a dosedependent manner. Topical trioxsalen-PUVA is clearly less carcinogenic. The data on topical 8-MOP-PUVA are too limited for conclusions. The data for the risk of melanoma after B-MOP-PUVA are contradictory.

S13:3

INCIDENCE OF SKIN CANCER IN PATIENTS FOLLOW-

ING ORGAN TRANSPLANTATION. +Lindelöf, Bernt* (SWEDEN) *Karolinska Hospital and Institute

More than 400 000 organ transplantations have been performed in the world since 1960s. The quality of life of these patients is generally good, but along with the new organ follows chronic immonosuppression in order to prevent organ rejection. It is well established that these patients run a high risk of developing cancer and most often the skin is affected.

We linked a population-based cohort of 5356 patients who had received organ transplants in Sweden (1970–1994) with the Swedish Cancer Registry, to identify all cancer cases. After a mean follow-up of 5.6 years post-transplantation, 172 patients developed 325 non-melanoma skin cancers and six malignant melanomas. The relative risk of nonmelanoma skin cancer was 108.6 (95% CI = 94.6–123.1) for men and 92.8 (95% CI = 73.2–116.0) for women. The highest risk were noted for upper limbs, and the risk increased with time. No significant increase in malignant melanomas was noted. Except for the lip, which is also sun-exposed, other epithelial sites did not show comparable increases in cancer risk.

The Swedish cohort currently comprising of 6457 patients constitute the basis for ongoing studies on cancer risks of other sites than skin and nested case-control studies of factors possibly influencing carcinogenesis, such as immunosuppression and sun tanning habits. The histopathological characteristics of the non-melanoma skin cancers are presently reviewed and 450 questionnaires have been sent to patients in order to analyse the importance of sun exposure both before and after the transplantation.

S14:1

NARROWBAND UVB PHOTOTHERAPY FOR PSORIASIS

+Ferguson, James* (UNITED KINGDOM) *Ninewells Hospital and Medical School

Narrowband UVB (TL-01) phototherapy plays a significant role in the management of psoriasis and an increasingly reported number of other skin conditions, including atopic dermatitis, vitiligo, mycosis fungoides (patch stage) and a range of photodermatoses.

Although the majority of studies conducted which compare TL-01 efficacy with traditional broadband UVB sources are of a non-controlled type, the weight of evidence points towards greater efficacy with a trend towards faster clearance with less minimal erythema doses required. Broadband UVB has been shown to be less effective than PUVA with a tendency for use in guttate rather than the more problematic plaque psoriasis. Recent studies comparing PUVA with TL-01 suggest a variety of outcomes, presumably dependent on methodology and patient selection. Metanalysis of the clearance data suggests a slight preference for PUVA over TL-01 with, as yet, no significant difference in relapse rate.

28

08-05-01, 08:13

The long-term cancer risk of TL-01 in humans is unknown and is likely to remain so for many years. Using mouse cancer studies, which compared TL-01 with broadband, it appears that the TL-01 has a similar carcinogenic effect per course of effective treatment as the broadband source. Limited human data with broadband UVB suggests it to be significantly less carcinogenic than PUVA (1). The current trend of switching from PUVA to narrowband appears justified on current data. There is a need to establish a cohort follow-up skin cancer study of TL-01 treated subjects.

S14:2

UVA1 PHOTOTHERAPY.

+Podda, Maurizio* (GERMANY); Grundmann-Kollmann, Marcella* (GERMANY); Kaufmann, Roland* (GERMANY) *J.W. Goethe-University of Frankfurt

Ultraviolet A1 (UVA1) phototherapy is a phototherapeutic modality employing long- wavelength ultraviolet A radiation of 340-400 nm. Currently, three different irradiation protocols are in use with a UVA1 single dose above 100 J/ cm² in the high-dose regimen, 50-60 J/cm² in the mediumdose or 20-30 J/cm² in the low-dose regimen. High-dose UVA1 phototherapy had been originally described for the treatment of patients with acute, severe exacerbation of atopic dermatitis. These results have been corroborated by several studies indicating that high-dose UVA1 therapy is superior to conventional UVA-UVB phototherapy and at least as effective as topical fluocortolone therapy in atopic dermatitis. Good results have also been reported using a medium or even low -dose protocol, possibly at a price of faster recurrence of symptoms.

In the last years, therapeutic effects of high-, medium- or low-dose UVA1 have also been described for various other dermatoses, like urticaria pigmentosa, localized scleroderma, systemic sclerosis, lichen sclerosus et atrophicus, keloids, cutaneous T-cell lymphoma and graft versus host disease. T-cells are either directly or indirectly responsible for the etiopathology of most of these UVA1 sensitive diseases and, indeed, recent studies indicate that the mechanism of action of UVA1 is through selective cytotoxic effects via induction of apoptosis in infiltrating T-cells. The known induction of collagenase-1 (MMP-1) expression by UVA could, furthermore, account for the effects observed in sclerosing diseases.

UVA1 has broadened and improved phototherapy, however, despite these documented benefits due to the novelty of the therapeutic approach very little is known on potential long-term side effects, e.g. carcinogenic risk. Thus, indications for UVA1 should be considered carefully, the cumulative dose well documented, and patients followed up at regular intervals.

S14:3

UVA EXPOSURE AND THE RISK OF CUTANEOUS MALIGNANT MELANOMA

+Moan_Johan* (NORWAY)

*The Norwegian Radium Hospital

Ultraviolet radiation A (UVA) is localised in the wavelength region 320-400 nm. Since cancer induction is believed to be caused by DNA damage, and since DNA practically does not absorb UVA, it was for decades anticipated that UVA exposure had nothing to do with skin cancer. The action spectra of non-melanomas in mice supported this view. However, the first experimental action spectrum for cutaneous malignant melanoma (CMM) indicated that UVA might be of significance. Also epidemiological observations are in argument with this indication. Thus, the latitude gradient for UVA is much smaller than that of UVB. This may explain the fact that the latitude gradient of CMM incidence among Caucasians is smaller than that of non-melanoma skin cancer incidence. The incidence rate of CMM has increased for decades and the introduction of sunscreens absorbing UVB has not reduced the rate of increase. Comparisons of skin cancer incidence rates among Caucasians and Africans and among albinos and non-albinos indicate that melanin may be a chromophore - although not the only one - for CMM induction.

S14:4

UVA RADIATION ENHANCES METASTATIC PROPER-

TIES OF MELANOMA CELLS

+Pastila, Riikka* (FINLAND); Leszczynski, Dariusz* (FINLAND)

*STUK

29

Presently available sunscreens protect relatively well against UVB radiation but are poor absorbers of UVA radiation. Considering that people are spending more and more time in the sun, because of the misconception of the purpose of sunscreen, there is a growing urgency to develop more efficient UVA-protective lotions. UVA, although not as potent as UVB radiation in induction of DNA damage, can also exert significant effect on body's physiology. Previously, we demonstrated (Photochem. Photobiol. 64, 1996, 936) that UVA radiation-induced activation of protein kinase C signaling pathway leads to increase in expression of major histocompatibility antigens (MHC class I and II). Here, we hypothesize that the same mechanism may lead to UVAinduced alteration in expression of adhesion molecules in melanoma cells. If proven, it could suggest that melanoma cells in primary tumors, located in epidermis/dermis, might become prone to metastasis following UVA exposure. As an in vitro experimental model we used C57BL/6 mouse melanoma cell lines B16-F1 and B16-F10 and syngeneic endothelial cells. UVA irradiation induced decline in the surface expression of E-cadherin and increase in the expression of N-cadherin in B16-F1 and B16-F10 cell lines. This change is a well-known marker of metastatic melanoma phenotype. The decline in cadherin E expression was accompanied by a significant decline in homotypic melanoma-

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

melanoma adhesion (clustering) that is cadherin E-dependent. This suggests, that following UVA irradiation, the bonds between melanoma cells in the primary tumor might weaken/loosen what might facilitate detachment of single cells from the solid tumor mass and their migration into capillary blood vessels. Also, we observed that the singledose UVA irradiation of melanoma cells (8-12 J/cm²) causes an increase in melanoma cell adhesiveness to non-irradiated endothelium with the peak-response 24 h after irradiation. The use of multiple-dose irradiation protocol of melanoma cells $(4 \times 2 \text{ I/cm}^2)$ increased melanoma adhesion already at 1-h time-point, what suggests that a fractionated dose of UVA-radiation is more efficient than the bolus irradiation. In conclusion, it appears that UVA irradiation might enhance metastatic potential of melanoma cells by weakening melanoma-melanoma binding within solid tumor mass and by enhancing adhesiveness of melanoma cells to endothelium what, in turn, may facilitate extravasation of tumor cells in internal organs. This observation, together with the previously reported UVA-induced expression of MHC antigens, further supports the notion of the urgent need for the development of more effective UVA-absorbing sunscreen lotions.

S14:5

UVA AND PHOTOPROTECTION

+Larkö, Olle* (SWEDEN) *Sahlgrenska University Hospital

Recent epidemiological data suggest that UVA plays an important role in the development of malignant melanoma. Human experimental data is largely lacking although the increased melanoma incidence among sunbed users indicates that UVA may be important in the induction of malignant melanoma.

The sunscreen layer applied is far less than used in the tests for determining the sun protection factor (SPF).

Sunscreens have been advocated as protection against sun damage. However, many sunscreens still protect poorly against UVA. One of the problems is that UVA protective sunscreens with an absorption spectrum in the long wavelength UVA tend to get coloured. Also, some UVA protective ingredients can be photodegraded during irradiation. Titanium dioxide and zinc oxide may be a solution but these agents can act as photocatalysts. Coating of the particles has been used to overcome this problem. A UVA protection factor has still to be decided upon.

Meanwhile, the basic UVA protection should consist of clothes and sunscreening products with a high SPF.

SAT1:1

INFLAMMATORY ACNE: CAN RESISTANCE REVEAL HOW ANTIBIOTICS WORK?

+Cove, Jonathan H.* (UNITED KINGDOM) *School of Biochemistry and Molecular Biology

It is well known that acne is a multifactorial disease of the pilosebaceous follicles. However, even in cases of severe disease only a small proportion of ducts is affected at any one time. Long standing evidence points to the over-production of sebum, and the involvement of bacteria in acne pathogenesis. Paradoxically, seborrhoea persists after resolution of the disease and it is evident that Koch's postulates cannot be demonstrated in acne. A model in which inflammatory acne can be considered as an infection of functionally blocked pilosebaceous follicles by Propionibacterium acnes is proposed which may help explain outstanding issues relating to, initiation, progression and perhaps resolution of the disease. Propionibacteria are not found in microcomedones (non-inflamed lesions) which are first visible during the adrenarche in acne-prone individuals. Thus comedogenesis appears to be independent of bacterial infection and may be driven by high levels of bioactive interleukin-1a derived from ductal keratinocytes. Microcomedones may progress to inflamed lesions but the factors that trigger this are unknown. Evidence for the involvement of propionibacteria is inconclusive although antibiotic treatment can be less effective for patients with predominantly antibiotic-resistant P. acnes. Analysis of immunological events in early lesions indicates a type IV hypersensitivity response to one or more persistent lesional antigens. These may or may not be bacterial. However it is possible that the potent adjuvant activity of P. acnes could up-regulate this immune response. Antibiotics are widely used in the treatment of acne. Although they reduce the numbers of propionibacteria on the skin, other modes of action may contribute to or explain their therapeutic efficacy. These not only include anti-inflammatory effects but also activities such as antioxidant and inhibition of ketratinocyte proliferation. With current global concern over the rising prevalence of antibiotic resistance among pathogens and commensal bacteria, including propionibacteria, it is timely to reappraise the role of antibiotics in the treatment of acne.

SAT2:1

DO WE NEED COMBINATION THERAPY?

+Faergemann, Jan* (SWEDEN) *Sahlgrenska University Hospital

Jan Faergemann, doctor of medicine 1975, specialist in dermatology and venereology 1980, PhD 1979. Ass.prof. in dermatology and venereology. He has been visiting professor at the Department of Dermatology, University of California. He is the author of 174 papers, published, in press or accepted for publication in various international journals. His main area of interest is in dermato-mycology. He is the referee for several well-known international journals. He is at the advicery board for three international journals He has been the chairman, co-chairman and speaker at 110

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

international meetings with 134 presentations. He has been the organiser for joint meetings in mycology. He is the president for the Swedish Society for Clinical Mycology.

Onychomycosis is one of the most common skin conditions. While estimates for prevalence in the general population range from 3% to more than 20%, fungal nail infections are more prevalent in the elderly and in those with peripheral vascular disease or diabetes mellitus.

The current oral treatments for fungal nail diseases, including terbinafine, itraconazole and fluconazole, are superior to griseofulvin. These newer agents achieved mycological cure rates of about 70% in clinical trials of toe nail infections. However, the published efficacy rates vary, particularly with itraconazole. There are some disadvantages associated with these treatments. First, there is a significant failure rate (up to 30%) under clinical trial conditions. Second, drug penetration may be low. In some cases, the location of the infection (for example, in the lateral margins of the nail) is associated with low drug levels, suggesting uneven distribution of the antifungal agent in the nail plate. Third, while adverse events are not common, they occur in about 10% of patients. Serious adverse events are very rare. Finally, one potentially serious limitation is the risk for drug interaction, which is greatest with the azoles. This limits the concomitant use of medications such as terfenadine, digoxin, cyclosporin and tacrolimus. While, individually, these features do not necessarily limit the use of oral antifungals, taken together they constitute a disadvantage.

One potential future management strategy is to use combinations of drugs or treatments to improve success rates and reduce the duration of therapy. Such an approach would potentially include combined antifungal drug and nail removal or antifungal/antifungal drug combinations. The purpose of this symposium is to examine the value of using two or more antifungal drugs - combination therapy in the management of fungal nail disease.

SAT2:2

AMOROLFINE + TERBINAFINE COMBINATION: RE-SULTS OF A CLINICAL TRIAL IN FRANCE

+Baran, Robert L.* (FRANCE) *Nail Disease Centre

Dr. Baran is Head of the Nail Disease Centre in Cannes, France and is the Official Investigator of Drugs at the Cannes General Hospital. He received his medical training at the Hôspital Saint-Louis in Paris, France. Dr. Baran has authored and edited several books focused on diseases of nails and their management. He is a member of many professional societies, and helped to found the International Society for Dermatological Surgery and the European Nail Society. Dr. Baran currently serves on the editorial boards of Cutis, The Journal of Dermatological Treatment, and Mikologia Lekarska (Poland). In addition, he is author or co-author of more than 400 publications in major peer-reviewed journals.

Onychomycosis is a difficult infection to treat, and current interest has focused on combination therapies. A multicenter, randomized, prospective study was conducted to compare the combination of amorolfine and terbinafine versus terbinafine alone for the treatment of dermatophytic toenail onychomycosis with matrix involvement. Patients were randomly assigned to one of the following treatment groups: 1) 5% amorolfine nail lacquer once weekly for 15 months plus terbinafine 250 mg daily for 6 weeks (AT-6); 2) 5% amorolfine nail lacquer once weekly for 15 months plus terbinafine 250 mg daily for 12 weeks (AT-12); or 3) terbinafine 250 mg daily for 12 weeks (T-12). The primary efficacy criterion was negative culture and microscopy at 3 months. Secondary efficacy criteria included negative culture and microscopy at visits up to 18 months, clinical cure (defined as <10% of disease remaining), and combined mycologic-clinical clearance.

A total of 147 patients were included in the study (50 patients in the AT-6 group; 48 patients in the AT-12 group; and 49 patients in the T-12 group). At week 12, mycologic evaluation revealed a negative examination and culture in 35% of the AT-6 group, 28% of the AT-12 group, and 17% of the T-12 group. Clinical responses at 18 months were significantly improved in the AT-12 group versus the T-12 group (85% vs 59% cure or improvement, P<.05); the clinical response in the AT-6 group was numerically superior (66%). The combined clinical and mycologic response at 18 months was also significantly superior in the AT-12 group (P<.05), with a 72% cure rate versus a 37% cure rate in the T-12 group and a 44% cure rate in the AT-6 group. An economic evaluation found that the cost per patient cured was in favor of group AT-12, followed by AT-6 and T-12.

In summary, the results of this study show that there is an improvement in the treatment of severe onychomycosis when a combination of amorolfine topical nail lacquer and oral terbinafine therapy is used. Combination therapy is cost effective and the dosing schedule (short systemic therapy plus once-weekly topical treatment) may favor patient compliance.

SAT2:3

31

RATIONALE FOR COMBINATION THERAPY

+Evans, E.G.V.* (UNITED KINGDOM) *University of Leeds

Professor Glyn Evans is Professor of Medical Mycology in the Department of Microbiology, the University of Leeds and also Director of the Mycology Reference Laboratory, an internationally recognized reference laboratory for the diagnosis of fungal infections. Professor Evans is a graduate of both the University of Wales College at Cardiffe and the University of Glasgow, where he earned a PhD in Medical Mycology. In 1992 he was admitted as a Fellow of the Royal College of Pathologists (UK) and also as a Fellow of the Institute of Biology (UK). Professor Evans' research interests concern molecular epidemiology, diagnosis and treatment of fungal infections. He has authored or co-authored seven books, contributed a number of chapters to textbooks and published more than 100 original papers and review articles in learned journals. He was until May 2000 the President of the International Society for Human and Animal Mycology (ISHAM). He was also the President of the British Society for Medical Mycology (BSMM) from 1995-1998 and a Member of the Council of the European Confederation of

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

Medical Mycology from 1997-1998. Professor Evans is frequently invited to give lectures and to chair sessions at national and international meetings.

There are now much improved oral therapies for onychomycosis. This follows the introduction of terbinafine, itraconazole and fluconazole. Amorolfine is also used topically to treat onychomycosis but it is reserved for treating 'mild' distal disease. The in vitro activity of these newer drugs is around 10–200 times higher than griseofulvin against dermatophytes; the most active is terbinafine, which is approximately 20 times more potent than itraconazole.

All these drugs inhibit the synthesis of ergosterol, an important component of the fungal cell membrane, and they interfere at different points in the synthetic pathway. The azoles inhibit the enzyme 14-a-demethylase, terbinafine acts earlier on squalene epoxidase, and amorolfine inhibits g-14-reductase and g-7-8 isomerisation. These minor differences in mode of action have subtly varying effects on the fungal cells, and potentially the drugs could be synergistic.

Despite the improvements in therapy, approximately 20% of patients with onychomycosis fail on antifungal therapy. This is not likely to be due to development of resistance by the dermatophyte to the antifungal used to treat the patient. Failure is more likely to be due to kinetic problems or because the fungal forms are not sensitive to the concentrations of antifungal agents that can be achieved in the nail.

One approach to improving cure rates would be to exploit any synergy between drugs by using combination therapy. For example, topical amorolfine could be used in conjunction with oral therapies. Indeed, there is good evidence of synergy in vitro between terbinafine and the triazoles and also between amorolfine and griseofulvin, and terbinafine and ketoconazole. Further studies of potential synergy between amorolfine and other antifungals in vitro are needed to examine a wider range of fungi than studied to date, and to include all potential nail pathogens.

Synergy between combinations of amorolfine and other antifungals has also been demonstrated in animal models of dermatophytosis. Topical amorolfine has been used concurrently with oral antifungals in patients with onychomycosis. Most of this work was done with amorolfine in combination with griseofulvin, although preliminary studies have also been carried out with terbinafine and itraconazole. The results have been encouraging but further studies are needed. The rationale for this approach is that the amorolfine applied topically will penetrate the nail plate, diffusing to the nail bed, while the orally administered drug penetrates the matrix and nail bed. Any synergy between the two drugs will mean an increased antifungal activity at lower concentrations of both drugs.

In summary, combinations of antifungals, specifically a topical and oral agent, may be able to achieve higher cure rates and in a shorter time than is currently possible.

SAT2:4

AMOROLFINE + ITRACONAZOLE COMBINATION:

RESULTS OF A CLINICAL TRIAL IN SPAIN

+Lecha, Mario* (SPAIN) *University of Barcelona

Professor Glyn Evans is Professor of Medical Mycology in the Department of Microbiology, the University of Leeds and also Director of the Mycology Reference Laboratory, an internationally recognized reference laboratory for the diagnosis of fungal infections. Professor Evans is a graduate of both the University of Wales College at Cardiffe and the University of Glasgow, where he earned a PhD in Medical Mycology. In 1992 he was admitted as a Fellow of the Royal College of Pathologists (UK) and also as a Fellow of the Institute of Biology (UK). Professor Evans' research interests concern molecular epidemiology, diagnosis and treatment of fungal infections. He has authored or co-authored seven books, contributed a number of chapters to textbooks and published more than 100 original papers and review articles in learned journals. He was until May 2000 the President of the International Society for Human and Animal Mycology (ISHAM). He was also the President of the British Society for Medical Mycology (BSMM) from 1995-1998 and a Member of the Council of the European Confederation of Medical Mycology from 1997-1998. Professor Evans is frequently invited to give lectures and to chair sessions at national and international meetings.

There are now much improved oral therapies for onychomycosis. This follows the introduction of terbinafine, itraconazole and fluconazole. Amorolfine is also used topically to treat onychomycosis but it is reserved for treating 'mild' distal disease. The in vitro activity of these newer drugs is around 10–200 times higher than griseofulvin against dermatophytes; the most active is terbinafine, which is approximately 20 times more potent than itraconazole.

All these drugs inhibit the synthesis of ergosterol, an important component of the fungal cell membrane, and they interfere at different points in the synthetic pathway. The azoles inhibit the enzyme 14-a-demethylase, terbinafine acts earlier on squalene epoxidase, and amorolfine inhibits g-14-reductase and g-7–8 isomerisation. These minor differences in mode of action have subtly varying effects on the fungal cells, and potentially the drugs could be synergistic.

Despite the improvements in therapy, approximately 20% of patients with onychomycosis fail on antifungal therapy. This is not likely to be due to development of resistance by

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

the dermatophyte to the antifungal used to treat the patient. Failure is more likely to be due to kinetic problems or because the fungal forms are not sensitive to the concentrations of antifungal agents that can be achieved in the nail.

One approach to improving cure rates would be to exploit any synergy between drugs by using combination therapy. For example, topical amorolfine could be used in conjunction with oral therapies. Indeed, there is good evidence of synergy in vitro between terbinafine and the triazoles and also between amorolfine and griseofulvin, and terbinafine and ketoconazole. Further studies of potential synergy between amorolfine and other antifungals in vitro are needed to examine a wider range of fungi than studied to date, and to include all potential nail pathogens.

Synergy between combinations of amorolfine and other antifungals has also been demonstrated in animal models of dermatophytosis. Topical amorolfine has been used concurrently with oral antifungals in patients with onychomycosis. Most of this work was done with amorolfine in combination with griseofulvin, although preliminary studies have also been carried out with terbinafine and itraconazole. The results have been encouraging but further studies are needed. The rationale for this approach is that the amorolfine applied topically will penetrate the nail plate, diffusing to the nail bed, while the orally administered drug penetrates the matrix and nail bed. Any synergy between the two drugs will mean an increased antifungal activity at lower concentrations of both drugs.

In summary, combinations of antifungals, specifically a topical and oral agent, may be able to achieve higher cure rates and in a shorter time than is currently possible.

0-1

ARTHRITIS AND QUALITY OF LIFE AMONG MEMBERS OF THE NORDIC PSORIASIS ASSOCIATIONS. DATA

FROM THE NORDIC QUALITY OF LIFE STUDY

+Molin, Lars* (SWEDEN); Zachariae, Hugh (DENMARK); Zachariae, Robert (DENMARK); Blomqvist, Kirsti (FIN-LAND); Davidsson, Steingrimur (ICELAND); Mørk, Cato (NORWAY); Sigurgeirsson, Bardyr (ICELAND) *Örebro Medical Centre Hospital

The aim of the study was to estimate the prevalence of arthritis and evaluate the quality of life, related to psoriasis and arthritis, in a large sample of members of the psoriasis associations from the Nordic countries, and to compare the results with psoriasis patients recruited from Nordic dermatologists and Nordic university clinics.

Material. Answer from 6.849 members of the psoriasis associations were accepted for the analysis and were compared with the answers from 702 patients treated by dermatologists and at university clinics.

Methods. The members and the patients answered a questionnaire including a great number of questions regarding severity and extent of psoriasis lesions and joint involvement together with questions regarding quality of life, stress and health in relation to psoriasis as well as to arthritis.

Results. Arthritis had been diagnosed in 30% of the psoriatics by a rheumatologist or a dermatologist. The prevalence of arthritis was the same among members and patients. Arthritis was more common in Finland, Norway and Sweden than in the other countries. Individuals with arthritis exhibited greater impairment of quality of life, longer psoriasis duration, and greater self-reported psoriasis severity than those without joint complaints. Patients recruited from the clinics reported greater impairment of quality of life than the other groups.

Conclusions. The study indicates that arthritis is more prevalent than previously accepted and is an important factor to include in the evaluation of the quality of life in individuals suffering from psoriasis.

0-2

PREVALENCE OF FIBROMYALGIA IN PATIENTS WITH PSORIASIS

+Thune, Per* (NORWAY) *OMNIA

The frequency of fibromyalgia (FM) varies in different population studies and is usually estimated to be about 1-3%. A low prevalence of 0.75% has been reported in Finland while one study in Norway showed a very high prevalence of 10.5%. The reason for this discrepancy can be different diagnostic criteria but may also be the result of overdiagnosis. Although the symptoms of FM overlap considerably with those of spondyloarthropathy (SP) the frequency of FM in psoriasis (PS) and particularly among those with musculoskeletal pain, has apparently never been investigated. This was the aim of the present study which comprised 1269 patients with PS who were consequtively investigated during a three years period from 1997 till 2000. There were 704 f. (55.5%) and 565 m. (44.5%), mean age 48 yrs. Among these were 335 patients (26.4%), 217 f. and 118 m. mean age 54 yrs. who complained of musculoskeletal pain. The mean duration of skin symptoms and of joint and muscle pain were 7-yrs and 6-yrs respectively. All patients were seronegative. In 105 patients (8.4%), 95 f. and 12 m. (mean age 50 yrs) the symptoms were compatible with the ACR-90 criteria for FM. The diagnosis of FM was made by a dermatologist in 51, by a rheumatologist in 29 and by a general practioner and confirmed by a dermatologist in 25 patients.In 70/105 patients with FM was the diagnosis of PS unknown despite the fact that the skin symptoms had lasted for 3½ year at a mean. Their muscle and joint complaints had lasted for about 12 years.

It appeared that 21/105 patients with FM had first-degree relatives with PS while 5/105 had second-degree relatives with PS. The other patients did not know. – Radiological sacroiliitis occurred in 2/66 patients with FM, while arthritis in fingers and/or wrists was observed in 17/66 patients, osteochondrosis and spondylosis occurred in 22/66 and arthritis of the toes in 3/66. – QUESTION: what should the diagnosis be: FM/PS? PS/SP? or PS/SP/FM?

A closer cooperation between dermatologists and rheumatologists seems urgent.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

O-3

USE OF ALTERNATIVE THERAPY IN PSORIATICS FROM THE NORDIC COUNTRIES: A SURVEY FROM 5739

MEMBERS OF THE NORDIC PSORIASIS ASSOCIATIONS

+Mørk, Cato* (NORWAY); Zachariae, Hugh (DENMARK); Zachariae, Robert (DENMARK); Blomqvist, Kirsti (FIN-LAND); Davidsson, Steingrimur (ICELAND); Molin, Lars (SWEDEN); Sigurgeirsson, Bardyr (ICELAND) *The National Hospital

Alternative therapy is widely used in patients with chronic disorders, and psoriasis is no exception. The aim of this study was to find the present and previous use of "alternative medicine" and "alternative treatment" among psoriasis patients within the Nordic countries. The data were obtained from a questionnaire-based Quality of Life study of 5739 members of the psoriasis associations of Denmark, Finland, Iceland, Norway, Sweden and the Faeroe Islands. 15,9% of psoriatics had tried to be on a diet, more than 40% from Iceland against only 7,2% from Sweden. Dietary measures were more frequent among female and young patients. 17,9% (26,6%) of the patients with psoriasis reported use of alternative medicine; the same figures for alternative treatment were 11,4% (17,1%). The popularity of these therapies were highest in Iceland, where 26,6% (42,1%) took or had taken alternative medicine or 17,2% (26,0%) other alternative treatment. The figures for the same two categories on Faeroe Islands were 8,7% (25,6%) and 8,7% (23,5%), respectively. Women, younger patients and patients with a longer disease duration had a higher previous use of alternative therapies. A relation was seen between present use of alternative therapies and lower Quality of Life, a higher degree of subjective stress and with a greater self-reported disease severity. The patient satisfaction with their contact with the physician was in general high. Different traditions and trends could possibly explain some of the variations in the use of alternative therapies.

0-4

ERYHTROMELALGIA: A SYNDROME OF DYSFUNC-

TIONAL VASCULAR DYNAMICS

+Mørk, Cato* (NORWAY); Asker, C. (SWEDEN); Salerud, G. (SWEDEN); Kvernebo, K. (NORWAY) *The National Hospital

*The National Hospital Erythromelalgia (EM) is a condition defined by red, hot and burning extremities with exacerbation of symtpoms by warming and relief by cooling. Microvascular arteriovenous shunting has been hypothesised to be the common pathogenetic mechanism in patients with EM. During EM attacks the skin blood flow is maldistributed from nutritive, towards increased thermoregulatory perfusion. The anatomical arteriovenous anastomoses (AVA) are located in acral

cal arteriovenous anastomoses (AVA) are located in acral areas, mainly plantar and palmar aspects of hands and feet. By relating the perfusion changes, using Laser Doppler Perfusion Imaging (LDPI) and capillary video microscopy, in plantar and dorsal aspects of the feet in patients with primary EM (n=14) and controls during central body heating with the occurrence of EM symptoms, we wanted to test the shunt-hypothesis.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

Symptoms were induced in 8 patients after heat provocation. In the plantar region of the foot, the location of numerous AVA, the symptomatic EM patients significantly increased the LDPI flux as compared to asymptomatic patients with EM and controls. In the dorsal region with few AVA no significant differences between the groups were demonstrated. On the other hand, the number of visible or active capillaries in symptomatic EM patients decreased significantly, as compared to asymptomatic patients and controls.

In conclusion, the combination of increases global perfusion and fewer visible capillaries during EM symptoms indicates a steal phenomenon from the capillaries to the AVA. The blood flow is maldistributed away from the nutritive, towards the increased thermoregulatory perfusion. These findings give further support to the shunt-hypothesis.

O-5

TREATMENT OF PSORIASIS IN THE NORDIC COUN-

TRIES: A SURVEY FROM 5739 MEMBERS OF THE

NORDIC PSORIASIS ASSOCIATIONS.

+Zachariae, Hugh* (DENMARK); Zachariae, Robert (DEN-MARK); Blomqvist, Kirsti (FINLAND); Davidsson, Steingrimur (ICELAND); Molin, Lars (SWEDEN); Mørk, Cato (NORWAY); Sigurgeirsson, Bardyr (ICELAND) *Aarhus University Hospital

The aim of the study was to show the present spectrum of psoriasis treatment within the Nordic countries. The data were obtained from a questionnaire-based quality of life study of 5739 members of the psoriasis associations of Denmark, Finland, Iceland, Norway, Sweden and the Faeroe Islands. They showed that the two most commonly used active agents were topical steroids (89.7% total use and 49.4% present use) and calcipotriol (73.1% total use and 35.8% present use) with only small variations in use between the countries. Marked differences between the countries were, however, found within all other types of psoriasis therapy including the patients' use of alternative treatments. The different countries had each their significant priorities. The use of dithranol in Finland was almost the double of the average. While 14.2% of Danish members had received grenzrays within the last week only 0.1% of the Finns went through the same treatment. 13.1% of the Finnish psoriatics were on PUVA against 3.8% Danes, and PUVA was almost nonexistent for patients from the Faeroe Islands. The use of non-PUVA phototherapy was highest in Norway and Sweden. Almost 10% of the Danes were presently on methotrexate, which was used far more than etretinate or cyclosporin. In contrast, Finnish patients more often received etretinate than other systemic agents, and in Iceland there was a higher present use of cyclosporine than of etretinate. The popularity of alternative therapies was highest in Iceland, where 26.6% had taken "alternative medication" during the last week. For the Faeroe Islands the figure for the same category was 8.7%. The results of the study suggest that different treatment patterns should be taken into consideration, whenever discussing outcome of psoriasis in different countries.

O-6 PSORIASIS-RELATED QUALITY OF LIFE IN 6497

NORDIC PATIENTS

+Zachariae, Robert* (DENMARK); Zachariae, Hugh (DEN-MARK); Blomqvist, Kirsti (FINLAND); Davidsson, Steingrimur (ICELAND); Molin, Lars (SWEDEN); Mørk, Cato (NORWAY); Sigurgeirsson, Bardyr (ICELAND) *Aarhus University Hospital

Aim: The aim of the study was to investigate psoriasis-related QOL in a large sample of members of the psoriasis associations from the Nordic countries, and to compare the results with results from psoriasis patients recruited from Nordic dermatologists or Nordic University clinics.

Methods: A total of 5795 members and 702 patients rated their psoriasis severity and completed Nordic versions of the Psoriasis Disability Index (PDI) and the Psoriasis Life Stress Index (PLSI). The respondents also completed a number of questions concerning demographic and life style factors.

Results: Several factors, including age, marital status, smoking, and wine consumption were significantly associ-ated with severity and QOL. When controlling for demographic factors, self-reported severity emerged as the overall most significant predictor, explaining between 24 and 29% of the variation in psoriasis-related QOL, with the remaining factors only accounting for five to seven percent of the variation. Although correlated with self-reported severity, PASI scores did not emerge as a significant predictor of QOL. Norwegian psoriatics generally reported greater disease severity and greater impairments of QOL than psoriatics from the remaining Nordic countries. Whether Norwegian psoriatics have a higher threshold for seeking medical assistance and if this is of importance is not known.

Conclusion: Although self-reported severity may be the most important predictor, further research is needed to determine factors explaining the remaining variance in psoriasis-related QOL.

0-7

PALMOPLANTAR PUSTULOSIS, SMOKING AND AUTOIMMUNITY.

+Michaëlsson, Gerd* (SWEDEN); Hagforsen, E (SWEDEN); Nordlind, K (SWEDEN) *University Hospital

Aim of the study. 1. To investigate if patients with palmoplantar pustulosis (PPP) have antibodies to nicotinic acetylcholine receptors (nAChR) in addition to antibodies to thyroglobulin, thyroperoxidase and gliadin. 2. To investigate if PPP sera with/without antibodies to nAChR give a positive immunofluorescense (IF) in palmar skin from healthy non-smokers/smokers.

Methods. Sera were obtained from 45 patients with PPP (43 were smokers), from 23 patients with longstanding palmar hand eczema (15 had been or were smokers). Twenty-five per cent of the PPP patients had antibodies to thyroglobu-

lin, thyroperoxidase and 25% had IgA antibodies to gliadin, some had both types. Palmar skin was obtained from healthy non-smokers/smokers. Antibodies to nAChR were quantitaed by RIA. Double staining was used for identification of positive structures.

Results. Forty-two percent of the PPP sera had moderately elevated levels of nAChR antibodies in contrast to none of the hand eczema sera. Sixty-eight percent of the positive PPP sera induced a typical IF pattern in the papillary dermis in palmar skin from a non-smoker, with double staining identified as associated with endothelium. Five percent of the hand eczema patients displayed a similar pattern. When palmar skin from a smoker was used there was-in addition to the endothelial staining-also a staining of the acrosyringium, indicating that there is an upregulation of the autoantigen by smoking. The most intense IF was observed in sera with antibodies both to nAChR and thyroid antigens or gliadin. Furthermore 38% of sera without antibodies to nAChR but with antibodies to thyroglobulin, thyroperoxidase and/or o gliadin, displayed the same IF pattern although with lower intensity.

Conclusions. PPP may be an autoimmune disease precipitated by smoking with the autoantigen localized to the papillary endothelium and the acrosyringium. There may be an overlap between this antigen and those in autoimmune thy roid disease and gluten sensitivity explaining the high prevalence of these disorders in PPP patients.

O-8

BOTULINUM TOXIN A IMPROVES LIFE QUALITY IN SEVERE PRIMARY FOCAL HYPERHIDROSIS.

+Swartling, Carl* (SWEDEN); Naver, Hans (SWEDEN); Lindberg, Magnus (SWEDEN) *Uppsala University

The aim of this study was to assess quality of life with the Dermatology Life Quality Index (DLQI) before and after treatment with intradermal injections of botulinum toxin in a group of patients with severe focal hyperhidrosis. DLQI was administered to 58 randomly chosen patients before and after treatment. All patients answered the DLQI questionnaire prior to treatment and 53/58 at mean 5.2 months after treatment. The mean DLQI score in the 58 patients before treatment was 10.3 (2-23). In the group of 16/53 patients who had a relapse of sweating when answering the DLQI a second time, no significant improvement was seen (score 10.6 before and 8.8 after treatment (p=0.21)). In patients without relapse a 76% improvement was obtained (DLQI was reduced from 9.9 to 2.4 - p<0.0001). The study showed that focal hyperhidrosis may considerably reduce life quality and the disability ex-perienced by the patients can be largely reversed by botulinum toxin injections.

O-9 MEASUREMENTS OF COLOR IN PORT WINE STAINS USING A QUANTITATIVE METHOD

+Helsing, Per* (NORWAY); Lyngsnes Randaberg, L. (NORWAY); Mørk, NJ (NORWAY) *National Hospital

Port wine stains are congenital vascular malformations characterised by ectatic blood vessels. Lesions are treated with lasers in early childhood. Few port wine stains clears completely, most have residual changes after treatment.

Evaluation of treatment have so far been based on rather subjective methods, and there has been a substantial need for more objective methods to evaluate treatment success. Skin reflectance measurements produces curves that still have to analysed, and are difficult to use in a clinical setting.

Treatment success must be when treated area are perceived indistinguishable from normal skin. By using the CIE 1976(L*a*b*) system for colour perception, where change in colour is calculated as DE, we have followed 10 children with port wine stains during treatment.

DE was measured between treatments, and the hypothesis was that this value will reach a limit. We will also try to relate DE to the more subjective evaluations we use today.

O-10

TREATMENT OF CHRONIC HAND DERMATOSES WITH UVB/TL01

+Nordal, Eli J.* (NORWAY) *Ullevål Sykehus

UVB/Tl01 irradiation has proven to be efficient in whole body treatment for several chronic inflammatory dermatoses. Chronic hand and foot dermatoses of different genesis represent a great problem in clinical dermatological practice.

The last months we have had available TL01 equipment for hand and foot treatment with plates of 20W tubes \times 6, effect 4.3 mW/cm² (Esshå elagentur, Värnamo, Sweden).

So far 20 patients have entered the study with three weekly treatments up to 9 weeks. They have been assessed according to a modified scoring system of Vocks/Plötz/Ring1.

The preliminary results indicate that psoriatics are the best responders. There is little or none effect in PPP, and varying results in eczema/pompholyx where some respond well and some experience increased disease activity.

Detailed results from a relatively large material will be presented.

0-11

ICHTHYOSIS-PREMATURITY SYNDROME - AN UN-KNOWN, FREQUENT AND ANCIENT "MID-SCANDINVIAN" RECESSIVE DISEASE

+Kampman, Petra (NORWAY) Rikshospitalet

In Norway one new case of the Ichthyosis-Prematurity Syndrome (IPS) is born annually. IPS is a previously unrecognized syndrome of obstetric, pediatric and dermatological significance [1]. It was first observed as unique in the early 1980ies by its skin ultrastructural features and published as "Ichthyosis congenita type IV" [2]. IPS is an autosomal recessive disease. The mutation, carried by 2% of the population of Middle Norway, must be of prehistorical origin.

In IPS the pregnancy is complicated by polyhydramnion and an opaque amnion fluid due to shedding of large amounts of epidermally derived cells. Premature birth occurs in the 32nd week of pregnancy. Due to aspiration of the amnion debris the child may become severely asphyctic after delivery, and in unrecognized cases these children might not survive. At birth the skin is covered by thick caseous desquamating epidermis which surprisingly improves to a benign dryness of the skin within the first 1–2 weeks. The dry skin may later be misdiagnosed as atopic skin, partly due to the accompanying dermographism and atopic manifestations during infancy.

A typical case-history will be presented.

0-12

A RANDOMIZED DOUBLE BLIND STUDY COMPARING PHOTODYNAMIC THERAPY (PDT) WITH METVIX® TO PDT WITH PLACEBO CREAM IN ACTINIC KERATOSIS

+Bjerring, Peter* (DENMARK); Funk, J. (NORWAY); Roed-Petersen, J. (DENMARK); Söderberg, U. (DENMARK) *Marselisborg Hospital

Metvix[®] (methyl 5-aminolevulinate) is a new topical photosensitiser with very high lesion selectively. In this phase III trial, Metvix[®] PDT was compared to placebo PDT in patients with actinic keratosis (AK).

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

36

Methods: 39 patients with clinically diagnosed AK were randomised blindly to either placebo or Metvix[®] PDT. Scales and crusts were removed from the lesions before cream application. After 3 h application time a light dose of 75 J/ cm² was given using red light (570–670 nm). Lesion response was assessed after 3 months.

Results: 33 patients with 75 lesions were included n the efficacy analysis. 97% of the lesions were located in face/ scalp and 93% were of thin or moderate thickness. The lesion complete response rates were:

Lesion type n	Metvix® %	PDT n	Placebo %	PDT
Thin	7/9	78	4/16	25
Moderate	25/32	78	2/16	12
Thick	0/0	0	0/2	0
TOTAL	32/41	78	6/34	18

The expected local phototoxic reactions were transient and mainly of mild or moderate severity.

Conclusions: Metvix[®] PDT is an efficacious treatment for patients with AK. In the present study, lesion were only treated once. Re-treatment of residual lesions has previously been shown to result in higher response rates (>90%). Lesion preparation, placebo cream application and illumination alone do not have any significant treatment effect on AK lesions.

O-13

A PIVOTAL STUDY OF PHOTODYNAMIC THERAPY (PDT) WITH METVIX® 160 MG/G CREAM IN PATIENTS WITH BASAL CELL CARCINOMA (BCC) WITH A RISK OF COMPLICATIONS AND POOR COSMETIC OUTCOME USING CONVENTIONAL THERAPY

+Wennberg, AM* (SWEDEN); Horn, M (AUSTRIA); Wulf, HC (DENMARK); Warloe, T (NORWAY); Rhodes, L (UNITED KINGDOM); Fritsch, C (GERMANY); Kaufmann, R (GER-MANY); de Rie, M (THE NETHERLANDS); Wolf, P (AUS-TRIA); Stender, I (DENMARK); Solér, A (NORWAY); Wong, G (UNITED KINGDOM); Lang, K (GERMANY); Legat, K (AUSTRIA); Pavel, S (GERMANY); Larkö, Olle (SWEDEN) *Sahlgrenska University Hospital

Objective: Patients (pts) with "high-risk" BCC in need of advanced surgery or radiation therapy with a risk of complications and poor cosmetic outcome, received a new selective photosensitizer, methyl 5-aminolevulinate (Metvix®) to determine response rate, cosmetic outcome and side effects.

Methods: Ninety-four pts with clinical and histological diagnosis of BCC (mid-face, large, recurrent) excluding morpheic and highly infiltrating lesions, received one treatment cycle with Metvix® PDT (two treatments one week apart). After lesion preparation and three hrs of occulusion with Metvix® cream, the lesion was illuminated with 75 J/cm² of red light (570–670 nm). If there was non-complete response after three months as assessed clinically and by histology, the lesion was retreated.

Results: Ninety-four pts with 123 lesions were treated and included in safety analysis, 60% of the lesions were located in face/scalp and 40% of the patients received two treatment cycles. Eighty-five pts with 108 lesions were included in primary efficacy analysis, nine pts were excluded by external reviewer because they did not fulfil the definition of having a "high-risk" BCC lesion. Clinical lesion evaluation resulted in complete response rate of 87%, which dropped to 74% when excluding lesions with a positive histology. 75% of pts had good or excellent cosmetic outcome at three months which increased to 85% by 12 months. 67% of pts reported adverse events, mostly expected local phototoxic reactions like erythema and burning sensation/pain. The symptoms were transient, and mostly of mild severity. Twelve months follow-up of 68 pts with 80 lesions histologically confirmed CR at three months, showed a lesion recurrence rate of 9%. No lesions in face/scalp or on extremities did recur and in the group of pts with complete response after one treatment cycle, only one lesion recurred.

Lesion location	Clinical CR 3 months (all lesions, n=123)	Clinical CR 3 months (high-risk, n=108)	Histology CR 3 months (high-risk, n=108)	Recurrence 12 month (of hist. CR 3 months)
Face/scalp	64/74,86%	57/65, 88%	45/65,69%	0%
Extremities	16/17, 94%	14/15, 93%	13/15, 87%	0%
Truncus/neck	27/32, 84%	23/28, 82%	22/28, 79%	7/21, 32%

Conclusion: Metvix[®] PDT was effective in pts with "highrisk" BCC, and the cosmetic result was good and improved by time. Metvix[®] PDT was well tolerated and may be a good alternative to conventional modalities which have the risks of disfiguration and inferior cosmetic outcome. Five years follow-up is underway to determine long-term recurrence rate.

0-14

37

DIFFERENCES IN SUN EXPOSURE DOSES IN SED AND SUN BURNING EPISODES WHEN SUNBATHING AT THE BEACH ON HOLIDAYS IN SOUTHERN VERSUS NORTH-ERN EUROPE

ERN EUROPE

+Thieden, Elisabeth* (DENMARK); Philipsen, P.A. (DEN-MARK); Heydenreich, J. (DENMARK); Sandby-Møller, J. (DENMARK); Wulf, H.C. (DENMARK) *Bispebjerg Hospital

Aim: To investigate differences in solar UVR doses in SED and number and severity of sun burning episodes when sunbathing at the beach in either Southern or Northern Europe.

Methods: From June to October 1999, 298 subjects (4-67 years old, mean 25 years old) participated in an investigation of sun habits. They wore a personal, electronic, UV dosimeter in a wristwatch, Sun-Saver, and filled a sun diary in total 21,119 days hereof 54% days holiday/days off work/ school.

Results: 61 persons had holidays in Southern Europe in total 897 days. They spent 54.3% of the days sunbathing at the beach (mean 8 days, range (1–30 days)) while 157 subjects were sunbathing at the beach on holidays/weekends in Denmark and Northern Europe in only 10.3% out of 10,358 possible days (mean 7 days, range (1–26 days)). The mean sun exposure dose per day measured on the UV do-simeter on the wrist when sunbathing at the beach was 5.3 SED in Denmark/Northern Europe and 9 SED in Southern Europe. From a former study, we know, that doses received on the wrist should be doubled to get the total sun exposure doses. The subjects got sunburned 22% of the days in Southern Europe but only 15% of the days in Northern Europe. Severity and the extension of the sunburns were almost the same.

Conclusion: People going to Southern Europe for holidays spent 54% of the days sunbathing at the beach and got almost the double UVR dose per day than in Denmark/Northern Europe. Sun burning episodes are also more frequent when going south.

0-15

DERMATAN SULPHATE IS RELEASED BY PROTEINASES OF COMMON PATHOGENIC BACTERIA AND INACTI-

VATES ANTIBACTERIAL α-DEFENSIN

+Schmidtchen, Arthur* (SWEDEN); Frick, Inga-Maria (SWEDEN); Björck, Lars (SWEDEN) *Lund University Hospital

Defensins represent an evolutionarily conserved group of small peptides with potent antibacterial activities. We here report that extracellular proteinases secreted by the human pathogens Pseudomonas aeruginosa, Enterococcus faecalis, Proteus mirabilis and Streptococcus pyogenes, release dermatan sulphate by degrading dermatan sulphate-containing proteoglycans, such as decorin. Dermatan sulphate was found to bind to neutrophil-derived a-defensin and this binding completely neutralized its bactericidal activity. During infection, proteoglycan degradation and release of dermatan sulphate may therefore represent a previously unknown virulence mechanism, which could serve as a target for novel antibacterial strategies.

O-17

QUALITY OF LIFE AND HAND ECZEMA

+Lindberg, Magnus* (SWEDEN); Wallenhammar, Lena-Marie (SWEDEN); Meding, Birgitta (SWEDEN) *Norrbacka

Hand eczema accounts for an estimated 90 per cent of occupational skin diseases. In Sweden, about 10 per cent of the population of working ages, have hand eczema during a year. Twice as many women as men, most of them young and otherwise healthy, suffer from hand eczema. Reports from the Occupational Injury Information System, at the National Board of Occupational Safety and Health in Sweden, show that work-related skin disease is common in hairdressers, metal-workers, cooks, kitchen maids, dental nurses, cleaners, housekeeping service workers, nurses and

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

nurse's assistants. About half of the work-related hand eczema cases are related to wet work. Our hypothesis is, that quality of life (QOL) is affected by hand eczema, and that women may report reduced QOL, compared to men, e.g. due to the influence of more frequent wet exposure, at work and at home.

Objectives. The aims of the study are to investigate if the questionnaires Short Form 36 (SF-36) and Dermatology Life Quality Index (DLQI) are suitable instruments for the investigation of QOL in patients with hand eczema. If so, the plan is to use them in a larger regional study in Sweden.

Methods. The two different instruments, SF-36 and DLQI, for assessment of QOL, are used in 100 consecutive patients with hand eczema diagnosis, at the Department of Occupational Dermatology, Stockholm. The questionnaires have earlier been used and evaluated in other diagnoses. SF-36 is one of the most used and reliable instruments for measuring health-related QOL, e.g. the relative influence of different diseases on function and well being. DLQI was designed by Dr AY Finlay, Cardiff, Wales, UK, in the beginning of the 1990s. This questionnaire has since then been used to measure QOL in different skin diseases. These instruments have, as far as we know, not previously been used to study QOL in hand eczema.

Results and conclusion. The data has been collected and results and conclusions will be presented at the congress.

0-18

38

AN 8-YEAR EXPERIENCE WITH ROUTINE SL MIX

PATCH TESTING SUPPLEMENTED WITH COMPOSITAE MIX.

+Andersen, Klaus E.* (DENMARK); Paulsen, E. (DENMARK); Hausen, B.M: (GERMANY) *Odense University Hospital

Odense University Hospital

Routine patch testing with sesquiterpene lactone (SL) mix, supplemented with Compositae mix and other Compositae extracts and allergens where appropriate, was evaluated over an 8-year period. 190 of 4,386 patients tested (4.3%) were Compositae-sensitive, 143 females (mean age 51.5 years) and 47 males (mean age 55 years), and 83% of reactions considered clinically relevant. 62% had 2 or more other contact allergies, most often to nickel, fragrance and colophony. SL mix detected 65%, Compositae mix 87% of Compositac-allergic patients, and the overall detection rate with both mixes was 93%. Few irritant reactions and no cases of clear-cut active sensitization were recorded with the mixes, but our results emphasize the importance of differentiating late-appearing reactivation reactions from patch test sensitization. The weakly positive Compositae mix reactions could reflect some irritancy, but as they were associated with fragrance and/or colophony allergy to a higher degree than weakly positive SL mix reactions, they probably represented cross-reactions. In conclusion, the detection rate with SL mix was high enough to support its continued use as a screening mix and it was very well and rather safely supplemented by aimed testing with Compositae mix.

O-19 ALLERGIC CONTACT DERMATITIS FROM 2,2-BIS[4-(2-

HYDROXY-3-METHACRYLOXYPROPOXY)PHENYL]-PROPANE (BIS-GMA)

+Kanerva, Lasse* (FINLAND); Jolanki, Riitta (FINLAND); Estlander, Tuula (FINLAND) *Finnish Institute of Occupational Health

Background. Allergenic epoxy di(meth)acrylates (EPODMA) such as 2,2-bis[4-(2-hydroxy-3-methacryloxypropoxy)phe-nyl]-propane (BIS-GMA) are widely used e.g. in dentistry and in ultraviolet (UV)-curable printing processes.

Objective. We studied concomitant and cross-sensitivity of patients with allergic patch test reactions to BIS-GMA, to other EPODMA and to diglycidyl ether of bisphenol A (DGEBA).

Patients and Methods. Patient records from September 1985 to December 1999 from our patch test clinic were reviewed. Patch tests were performed according to the recommendations of ICDRG.

Results. During 1985–1999 13 patients had an allergic patch test reaction to BIS-GMA. All these patients also reacted to DGEBA. 7 out of 10 patients with an allergic patch test reaction to DGEBA reacted to BIS-GMA. 4/13 patients developed BIS-GMA allergy from dental composite resins. 6/13 patients had apparently been sensitized from DGEBA and no exposure to BIS-GMA was known. One patient (1/13) had been sensitized from 2,2-bis[4-(2-hydroxy-3 acryloxypropoxy)phenyl]-propane (BIS-GA). 3/13 patients had first been sensitized to DGEBA, and then concomitantly to BIS-GMA or BIS-GA.

Conclusion. To find out the causative agent of allergic contact dermatitis in patients with allergic patch test reactions to BIS-GMA and/or DGEBA, patients with an allergic patch test reaction to DGEBA should also be tested to BIS-GMA and other EPODMA, and patients with an allergic patch test reaction to EPODMA should be tested to DGEBA.

O-20

NOSQ - THE NORDIC OCCUPATIONAL SKIN QUES-TIONNAIRE- A TOOL FOR SURVEYING WORK-RE-

LATED SKIN DISEASES

+Lindberg, Magnus* (SWEDEN); Susitaival, Päivikki (FINLAND); Meding, Birgitta (SWEDEN); Svensson, Åke (SWEDEN); Kanerva, Lasse (FINLAND); Flyvholm, Mari-Ann (DENMARK) *Norrbacka

Objectives: Work-related skin diseases are common in many occupations. For surveying occupational skin diseases and exposure, questionnaire-tools are needed. Questionnaires have to be standardized to facilitate comparable epidemiological research, workplace assessments, and evaluation of workplace interventions.

Methods: A Nordic group supported by the Nordic Council

of Ministers has developed a questionnaire-tool for work-related skin problems.

Results: Nordic Occupational Skin Questionnaire (NOSQ) includes two questionnaires for separate purposes. NOSQlong is a in-depth survey tool for research purposes. NOSQshort is a 4-page questionnaire for screening work-related skin problems at workplaces (e.g. by occupational health services). It can also be used for monitoring the frequency of skin conditions in workplaces with known dermatitis risks. NOSQ-short is an exerpt from NOSQ-long. The NOSQ-INFO version of the questionnaire also includes information and instructions to the researcher and can be seen as a manual. The questions included covers e.g. occupational history, atopic symptoms, self-reported hand and forearm eczema, exacerbating factors, self-reported contact urticaria on hands and forearms, consequences and life impact of dermatoses, skin symptoms, skin tests, exposures, and protective glove use.

Conclusions: The NOSQ-short and NOSQ-long questionnaires will be available first in English, Danish, Swedish and Finnish. NOSQ-INFO will be available only in English. NOSQ-short, NOSQ-long, and INFO questionnaires will be placed in a special internet site. The site will also include literature background, translation guidelines, and information to researchers on skin disease questionnaire use.

0-21

ALLERGIC CONTACT DERMATITIS TO BUDESONIDE REACTIVATED BY INHALATION OF THE ALLERGEN

+Isaksson, Marléne* (SWEDEN); Bruze, Magnus (SWEDEN) *Malmö University Hospital

Aim of the study: To study if inhalation of budesonide would result in reactivation of patch tests caused by budesonide and potentially cross-reacting substances.

Method: A randomized, double-blind, placebo-controlled study was initiated, in which 15 non-asthmatics hypersensitive to budesonide were provoked with budesonide or placebo by inhalation 6 weeks after having been patch tested with budesonide, its R and S diastereomers and potentially cross-reacting substances. Lung function was monitored using spirometry and repeated peak expiratory flow rate measurements.

Results: In 4/7 subjects inhaling budesonide reactivation of previously positive patch tests and other skin lesions occurred in contrast to no one of the 8 who inhaled placebo (P=.026).

Reactivation of a potentially cross-reactive substance was also noted.

Conclusion: A patient hypersensitive to budesonide should not be given budesonide as an inhalant. The study design described may be used in studies on cross-reactivity.

O-22

CROSS-REACTIVITY BETWEEN NICKEL AND COBALT DEMONSTRATED BY SYSTEMIC ADMINISTRATION OF

NICKEL AND COBALT?

cross-sensitization.

+Hindsén, Monica* (SWEDEN); Spirén, A. (SWEDEN); Bruze, M. (SWEDEN) *Universitetssjukhuset MAS

Objectives: Concomitant patch test reactions to nickel and cobalt have been frequently reported. The present study was designed to demonstrate if these reactions represent

Materials and methods: Females hypersensitive to nickel and cobalt were patch tested with serial dilutions of nickel sulphate and cobalt chloride. If they also were hypersensitive to another allergen they were patch tested also with a serial dilution of this allergen or a serial dilution of the irritant sodium lauryl sulphate. The females were patch tested on the upper back. One month later when the test reactions were healed the patients were randomized into 3 groups which were challenged orally with 3 mg nickel sulphate, 1 mg cobalt chloride or placebo. Flare-up reactions of previous patch test reactions were read in a blind way.

Results: Several flare-up reactions were observed on sites previously tested with nickel and cobalt. No flare-up reactions were however seen in patients given placebo or on sites tested with other allergens or sodium lauryl sulphate.

Conclusion: Flare-up reactions in healed patch tests to both nickel and cobalt after oral administration with either nickel or cobalt, may speak in favour of a cross-reactivity mechanism.

O-23

THE ASP84 GLU VARIANT OF THE MC1R GENE IN NORWEGIAN MELANOMA PATIENTS

+Helsing, Per* (NORWAY); Høyheim, Bjørn (NORWAY) *National Hospital

Individuals with red hair and fair skin are at risk of melanoma development. These phenotypic traits are regulated by the melanocyte stimulating hormone receptor, MC1R. Variants of the MC1R gene have been associated with red hair and fair skin in humans, one of these, the Asp84Glu variant with melanoma.

69 melanomas, 9 atypical naevi and 20 benign naevi were analyzed for the Asp84Glu mutation by nested PCR and RFLP, followed by sequencing.

The Asp84Glu allele was found in one melanoma. This finding indicates that the Asp84Glu variant allele is rare in melanoma patients in Norway.

O-24

DERMATOLOGICAL DNA LABORATORY IN OSLO: DIAGNOSTIC SERVICES

+Søyland, Elisabeth* (NORWAY) *Rikshospitalet

The laboratory at the Department of Dermatology, Rikshospitalet, has since June 1995 focused on DNA-technology for the purpose of basic research and development of a diagnostic service. Its name is DDL for short.

Situated since March 2000 in the "New" Rikshospital, the facilities has been located partly at the Dermatological Outpatient section, partly integrated in the Research Laboratory for Internal Medicine, and in close cooperation with the Institute of Forensic Medicine. Economically it has until now (2001) been run exclusively on external funding raised by volontary activities by professionals at the Department of Dermatology, but with one research fellow under the Norwegian Council of Science.

This communication restricts focus on it current diagnostic capability which is within monogenic diseases, whereas the long-term goal is also to include genetic predisposition to atopy and to psoriasis, and to perform quick skin fungus diagnostics.

Due to 40 years research activity within epidermolysis bullosa and 30 years within congenital ichthyosis, DNA diagnosis within these groups have been given preference. Due to the high frequency of Herlitz disease in Eastern Scandinavia (i.e.Sweden) and of lamellar ichthyosis in Western Scandinavia (i.e.Norway) the high frequency mutations in the LAMB3-locus and the TGM1-locus are currently tested along with haplotype diagnosis both for neonatal, prenatal and gene carrier purposes. For the many other gene loci which may be involved in junctional EB, haplotyping of minisatelittes around these loci are performed to pinpoint the most probable locus involved in individual families.

DDL is additionally using triplex minisatellite-haplotyping around the keratin II cluster on chromosome 12, and is preparing a similar set of haplotype-triplets around the keratin I cluster on chromosome 17 and around the epidermal differentiation cluster (EDC) on chromosome 1, to be able to help pinpointing the correct gene cluster for familial cases of epidermolysis bullosa simplex, dyskeratoses and hair anomalies.

Future diagnostics may derive from our basic research projects: plectin on 8q24 and the "melanoma" gene on chromosome 9p.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

40

40

08-05-01, 08:13

O-25

RADIOTHERAPY INCREASES SKIN COLLAGEN SYNTHE-SIS IN BREAST CANCER PATIENTS

+Riekki, Riitta* (FINLAND); Parikka, M. (FINLAND); Jukkola, A. (FINLAND); Salo, T. (FINLAND); Oikarinen, A. (FINLAND) *University of Oulu

To study the mechanisms of irradiation-induced fibrosis, collagen synthesis was analyzed in radiotherapy-treated human skin. The subjects were ten randomly chosen women, who had been treated for breast cancer with operation and radiation therapy. The mean age of the subjects was 53 years and the interval from radiotherapy was 7–94 months. The irradiated skin area was compared to the corresponding healthy skin area of the subject.

Skin biopsies were obtained from both the irradiated skin area and the corresponding control skin area. Suction blisters were also induced on both skin areas of the subjects. The skin biopsies were analyzed for type I and type III collagen synthesis by in situ-hybridization technique. An immunohistochemical staining for type I collagen producing fibroblasts was also performed. Suction blister fluid was analyzed for procollagen propeptides of types I and III collagens (PINP and PIIINP) with radioimmunoassay.

The amount of fibroblasts positive for type I collagen synthesis was found to be increased in radiotherapy-treated skin. A slight increase of fibroblasts positive for type III collagen synthesis was also found in irradiated skin. In suction blister fluid, the levels of both PINP and PIIINP were markedly increased in irradiated skin compared to nontreated skin.

We conclude that these results confirm the hypothesis that skin collagen synthesis is increased as a result of irradiation. The results provide information on the molecular basis of connective tissue fibrosis induced by radiotherapy.

O-26

TWISTED COLLAGEN FIBRILS. SIGNIFICANCE FOR DIFFERENTIATION OF HYPERMOBILE PATIENTS

+Kobayasi, Takasi* (DENMARK); Ullman, Susanne (DEN-MARK)

*Bispebjerg Hospital

Twisted collagen fibrils (TCF) show remarkable shapes in dermis. TCF are formed by the enzyme systems with inherited defects. TCF have been found in normal skin of Ehlers Danlos syndrome (EDS) and in the lesions of some other inherited dermatoses. EDS presents the cardinal symptoms of joints, skin and vessel in wide variation at varied degrees. The severe cases show typical clinical symptoms of EDS and the milder are often dubious for the exact diagnosis. The latter, hypermobility syndrome (HS), have been separated from EDS by clinical symptoms. HS patients spread widely among the inhabitants. This study intends to evaluate clinical diagnosis of EDS and HS by the ultrastructural points of view. Skin biopsy specimens, 450 EDS and HS, 5 Marfan syndrome, 1 Osteogenesis imperfecta, 1 Homocysteinuria, 1 Prolidase deficiency, 6 Tuberous sclerosis and ca 100 normal and other acquired dermatoses were studied by routine electron microskopy. Skin biopsy specimens from 20 hypermobile and 7 normal persons were prepared for immune electron microscopy for collagen types I and III. The results were corresponded with Beighton's score index (BI) as the marker of the clinical symptoms.

TCF in normal skin were characteristic for EDS and HS. TCF appeared in forms of flower-like, zigzag bordered and polygonal cut-surfaces of collagen fibrils. Disarrays and various thickness of the collagen fibrils were the accompanying changes. The patients with BI higher than 5 showed distinct changes of TCF and lower ratio of collagen types I/III than 1.0. Seemingly, BI 5 was the border between EDS and HS. Non-hypermobile and TCF-positive persons were also found. They were considered as hypermobile gene-carriers. Four percent of the normal persons had TCF in the dermis. TCF in the other hypermobile disorder were dubious. Involvement of the internal organs was found in the patients of EDS, HS and gene-carriers at a rate of about 10%. They showed manifold disorders, for instance chronic pains, riskful attacks for life and birth complication.

HS is considered to be formes frustes of EDS. TCF indicate disposition for once of the inherited abnormalities of collagen fibrils.

0-27

41

CONGENITAL ONSET ICHTHYOSIS IN NORWAY: ARE OUR PATIENTS SATISFIED WITH THEIR TREATMENT?

+Mørk, Nils-Jørgen* (NORWAY); Gedde-Dahl, Tobias (NORWAY)

*Rikshospitalet

To evaluate treatment modalities in ichthyosis a questionnaire was during year 2000 sent to 98 patients. Up to now 72 (73,4%) have responded. These had been clinically evaluated by one or both of the authors. 34 of the patients previously had an electronmicroscopic examination showing the type of congenital ichthyosis (1) and their mutations were known (2).

Of the 72 questionnaires received 34 patients (47,2%) used acitretin continuously.

In these subjective evaluations by the patients 27 of 34 (79,4%) were very satisfied with their treatment where as 7 (20,6%) reported a moderate effect of the treatment.

The side effects of the retinoids are well known. 4 (11%) of the patients using acitretin had to use a wig because of the hairloss. X-rays of the skeleton was done on a regular basis in 25 patients (73,5%) to detect possible hyperostosis due to the retinoid therapy. These findings will be discussed.

- Anton-Lamprecht 1. The skin. In: Papadimitriov JM, Henderson DW, Spangnolo DV (ed.): Diagnostic ultrastructure of nonneoplastic diseases. Churchill Livingstone, Edinburgh, 1992, 459–550.
- 2. Pigg M et al Eur J. Hum Genet 1998; 6: 589–596/Pigg M, Uppsala Thesis 2000.

O-29

STD IN LATVIA IN THE YEAR 2000

+Rubins, Andris* (LATVIA); Jakabsone, I. (LATVIA); Rubins, S. (LATVIA); Chigorevska, L. (LATVIA) *Medical Akademy of Latvia

In the Latvia morbidity of STD, particularly of syphilis and gonorrhoea, is one of the highest in Europe. The aim of the study was to characterize and analyse the increase in and epidemiology of STDs and normalizing the situation.

In Latvia, the registration of syphilis and gonorrhoea cases is carried out by the state Centre of Sexually Transmitted and Skin Diseases keeping the file of STDs and contagious skin diseases and summarizing data on the morbidity rate of STDs in the country. Each monthly registered morbidity rate is reported to the State Environment and Health Centre that sends summarized information on all infectious diseases to the State Statistics Committee.

In the past decade the highest level of syphilis morbidity in Latvia was in 1996 when 125 cases were recorded per 100,000 inhabitants.

The highest level of morbidity of gonorrhoea morbidity has been observed in 1993 with 162 cases per 100,000 inhibitants.

Currently a decrease in syphilis and gonorrhoea morbidity has been observed, while the incidence of HIV/AIDS has become most alarming. In 1999, the indices of syphilis and gonorrhoea morbidity in Latvia were 62 and 45 per 100,000 inhibitants, but in 2000 there were 1,021 syphilis new cases (9 of them congenital) and 745 with gonorrhoea, however HIV (466 incidences in 2000) infection has appeared in Latvia.

Conclusion: Syphilis and gonorrhoea are the most widespread STDs in the Latvia showing a tendency torwards decrease. The flare-up of HIV presents an alarming threat to the society.

To restrict the spread of STDs and HIV/AIDS in Latvia it is of utmost importance to direct attention prophylactic measures and information among the population, particularly among young people.

O-30

HSV-2 ANTIBODIES IN STD-PATIENTS, HEALTHY PREGNANT FEMALES, BLOOD DONORS AND MEDICAL

STUDENTS IN BERGEN.

+Nilsen, Arvid* (NORWAY); Marsden, HS (UNITED KING-DOM); Langeland, N; Matre,R; Haarr, L *University of Bergen

We have examined the prevalence of HSV-2 antibodies in 600 patients attending our outpatient clinic for sexually transmitted diseases (STD), using three different assays. For comparison we have also examined healthy pregnant females, blood donors and medical students (100 in each group).

The three assays are one ELISA gG2 assay (1), one peptid-55 based ELISA assay (2) and HSV-2 specific IgG ELISA (GULL laboratories).

Among STD-patients we found 9,7–14,1% to be HSV-2 seropositive, in healthy pregnant women the corresponding figures were 10–11,9%, in blood donors between 5–7%, whereas only 2–3% of the medical students were HSV-2 seropositive. Increasing HSV-2 seropositivity was statistically associated with increasing age, whereas we did not find a correlation to gender, number of sexual partners, age at sexual debut or the presence/absence of previous STDs.

The prevalences are somewhat lower than reported in comparable groups elsewhere. The results will be presented and factors that may relate to the relatively low seroprevalence among our STD-patients will be discussed. The sensitivity, specificity and predictive values will be discussed as possible limitations concerning future use of these assays.

- 1. Ho DWT, Field PR, Sjøgren-Jansson E, Jeansson S, Cunningham AL. Indirect ELISA for the detection of HSV-2 specific IgG and IgM antibodies with glycoprotein G (gG-2). J Virol Methods 1992;36:249-64.
- 2. Marsden HS, MacAulay K, Murray J et al. Identification of an immunodominant sequential epitope in glycoprotein G of herpes simplex virus type 2 that is useful for serotype-specific diagnosis. J Med Virol 1998;56:79–84.

0-31

HIGHER NUMBER OF LEUCOCYTES IN URETHRAL MALE SMEAR OBTAINED WITH A BLUNT METAL CURETTE IN COMPARISON WITH A CALCIUM ALGI-

NATE SWAB.

+Stang, Henning* (NORWAY); Moi, H. (NORWAY); Loeb, M. (NORWAY); Barlinn, C. (NORWAY); Gjertsen, I. (NORWAY); Halsos, A-M. (NORWAY); Kramer, P. (NORWAY); Thorvaldsen, J. (NORWAY) *Ullevål University Hospital

Objectives: In the studies which have determined the cutoff value of polymorphonuclear leukocytes (PMNL) needed for the diagnosis of urethritis in males in high power field microscopy of urethral smear, cotton tipped swabs have been used for urethral sampling. Comparison of a positive DNA amplification tests for *Chlamydia trachomatis* (CT) with microscopy diagnosis of urethritis, have found a highly variable sensitivity of the microscopy ranging from 29% to 88%. In the Nordic countries, metal curette is widely used instead of cotton swab for urethral sampling. Comparison between these two sampling methods has not been published.

Methods: 190 male patients consulting the municipal outpatient department for STD in Oslo were enrolled in a study comparing the two methods of urethral sampling. 130 patients met the inclusion criteria. The patients were rando-

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

mised into two groups based on which method was used first. The urethral smears were stained with methylenblue. The mean number of PMNL of 5 consecutive fields from the area with highest concentration of leukocytes was recorded at high power field (HPF-1000x) microscopy.

Result: Swab was used as first smear in 52 patients with a mean of 6.9 PMNL/HPF and metal curette was used first in 78 patients with a mean of 21.6 PMNL/HPF. The mean difference between the two methods was 14.7 PMNL/HPF (95% CI 8.1–21.4) giving a p-value <0.0001. Swab was used as the second smear in 78 patients with a mean on 7.8 PMNL/HPF and metal curette as second smear in 52 patients with a mean of 5.3 PMNL/HPF giving a non-significant difference. 61 (48%) of the patients had more than 4 PMNL/HPF in the first smear, indicating a urethritis. Sixteen (12.3%) of the patients had positive chlamydia test (LCR), all but one had >4 PMNL/HPF in the first smear with both sampling methods.

Conclusion: Microscopy of urethral smear taken with a blunt metal curette revealed a higher number of PMNL in comparison with urethral smear taken with a calcium alginate swab. Urethral samples for microscopy taken with a metal curette for pre-screening of chlamydia may have a higher sensitivity for a diagnosis of chlamydia urethritis than swab samples. However, the cut-off value for the diagnosis of urethritis for samples taken with a metal curette should be considered.

O-32

AMELANOTIC MALIGNANT MELANOMA - A REPORT OF 5 CASES

+Odegard, Brit* (NORWAY); Larsen, Tove Eeg (NORWAY) *Ullevaal Hospital

Approximately 2 per cent of all melanomas are amelanotic. These lesions are difficult to diagnose due to their lack of the pigment changes generally associated with melanomas. They are often mistaken for other, more benign, skin lesions, which may lead to a diagnostic delay as well as inadequate treatment and a worsening of the prognosis. In the following we will describe five cases of amelanotic melanoma seen in our department from 1995 to 2000. There was a diagnostic delay in 4 out of five patients, still all except one are doing well with no signs of recurrence or metastases three years or more after removal of their tumour. The fifth patient died from metastases. Three of the patients had had previous melanomas. We conclude that in order to diagnose amelanotic melanoma, awareness of this clinical entity as well as a high index of suspicion is necessary, maybe especially in patients who has had previous melanomas. In addition, early biopsy should be taken from solitary skin lesions that do not respond to seemingly adequate treatment.

P-33

ANTIBODIES AGAINST NICOTINIC ACETYLCHOLINE RECEPTORS IN SERA FROM PATIENTS WITH PALMO PLANTAR PUSTULOSIS

+Michaëlsson, Gerd* (SWEDEN); Hagforsen, E (SWEDEN); Nordlind, K (SWEDEN); Lefvert, A-K (SWEDEN); Mustafa, A (SWEDEN) *University Hospital

*University Hospital

Aim of the study: 1.To investigate if patients with palmoplantar pustulosis (PPP) have antibodies to nicotinic acetylcholine receptors (nAChR) in addition to antibodies to thyroglobulin, thyroperoxidase and gliadin. 2. To investigate if PPP sera with/without antibodies to nAChR give a positive immunofluorescense (IF) in palmar skin from healthy subjects.

Methods: Sera were obtained from 45 patients with PPP (43 were smokers), from 23 patients with longstanding palmar hand eczema (15 had been or were smokers). Twenty-five per cent of the PPP patients had antibodies to thyroglobulin, thyroperoxidase and 25% had IgA antibodies to gliadin, some had both types. Palmar skin was obtained from healthy non-smokers. Antibodies to nAChR were quantitated by RIA. Double staining was used for identification of positive structures.

Results: Forty-two percent of the PPP sera had moderately elevated levels of nAChR antibodies in contrast to none of the hand eczema sera. Sixty-eight percent of the positive PPP sera induced a typical IF pattern in the papillary dermis in palmar skin from a non-smoker, with double staining identified as associated with endothelium. Eight percent of the hand eczema patients displayed a similar pattern. The most intense IF was observed in sera with antibodies both to nAChR and thyroid antigens or gliadin. Furthermore one third of sera without antibodies to nAChR but with antibodies to thyroglobulin, thyroperoxidase and/ or o gliadin, displayed the same IF pattern although with lower intensity.

Conclusions: PPP may be an autoimmune disease precipitated by smoking with the autoantigen localized to the papillary endothelium. There may be an overlap between this antigen and those in autoimmune thyroid disease and gluten sensitivity explaining the high prevalence of these disorders in PPP patients.

P-34

METHOTREXATE AND PSORIASIS - CAN WE REDUCE THE NEED OF LIVER BIOPSIES? AN EVALUATION OF AMINOTERMINAL PROPEPTIDE OF TYPE III PROCOLLAGEN (PIIINP) IN ROUTINE SCREENING FOR METHOTREXATE INDUCED LIVER FIBROSIS.

+Søgaard, Helmer* (DENMARK); Zachariae, Hugh (DEN-MARK); Heickendorff, Lene (DENMARK) *University Institute of Pathology

In hepatic fibrosis there is increased synthesis of predominantly type III collagen, and the radioimmunoassay of aminoterminal propeptide of type III procollagen (PIIINP) measures these propeptides, which are cleaved off during collagen synthesis and released into circulation. A number of studies have shown that although the test is not organ specific, it can be utilised as a valuable non-invasive marker of liver fibrogenesis. Several groups have studied PIIINP as an indicator for development of fibrosis in methotrexate (MTX)-treated psoriatic patients. All these studies demonstrated significantly higher levels in patients with hepatic fibrosis than in patients with normal histological features or steatosis alone. The aim of the study was to evaluate if serial normal serum levels of aminoterminal propeptide of type III procollagen (PIIINP) might indicate, that no significant fibrosis is taking place in the liver, and thereby reduce the need for repeated liver biopsies in psoriatics treated with methotrexate (MTX). The clinical records of seventy psoriatics, who in the years 1989/90 were on MTX and had both a liver biopsy without fibrosis and a normal PIIINP, were examined and followed until the patient stopped taking the drug. The follow-up time was from one to eleven years. A total of 189 liver biopsies and 329 analyses of PIIINP were recorded. Twenty-one patients had only one and no further biopsies, but their data included at least two to three PIIINP samples obtained within a year around the time of the biopsy, and at least two were taken either prior to or at the time of the biopsy. The remaining patients had from two to seven liver biopsies each and a total of 267 analyses of PIIINP. In the study period only four patients developed fibrosis of the liver as shown by liver biopsies, and all these four patients developed elevated serum PIIINP levels. In addition two further patients, one of them with psoriatic arthritis, had elevated PIIINP, but normal liver biopsy. No liver fibrosis was missed in the 63 patients with consistently normal PIIINP levels. Thus the present data support the view, that as long as PIIINP is consistently normal in serial investigations, there is minimal risk of development of substantial liver fibrosis.

P-35

SENSITIZATION TO INHALANT AND FOOD ALLER-GENS IN CHILDHOOD

+Jøhnke, Hanne* (DENMARK); Norberg, L. (DENMARK); Andersen, KE (DENMARK); Bindslev-Jensen, C (DENMARK); Høst, A (DENMARK) *Odense University Hospital

Aim: To investigate prospectively the prevalence of sensitization to grass, birch, house dust mite, egg and milk in children at birth and at 3, 6, 12 and 18 months of age.

Method: A random sample of 562 newborn infants, born in a one-year period at the University Hospital of Odense between Oct.1998 and Oct.1999, is followed prospectively. At birth and at three months intervals parents are interviewed and infants examined clinically. Environmental factors such as exposure to food, infections, vaccinations, pets, tobacco and socio economic factors are registered. Blood samples are drawn for analysis of total IgE, specific IgE and histamine release. Patch testing with milk and egg is performed synchronously with skin prick test in order to evaluate the possibility of type I and IV allergy. Blood is stored for later DNA analysis and cytokine profile. Data are recorded on the newborns' parents and siblings regarding atopy, and skin prick test are offered.

Results: This presentation reports on the occurrence of sensitization to five common allergens. Sensitization to grass, birch, house dust mite, egg and milk have preliminary been examined in cord blood and in blood from children 3 and 6 months by total IgE, RAST-analysis and basophil histamine release. Measurements showed a sensitization rate of 16%, 25% and 27% to one or more allergens using the basophil histamine release response (HR), and a sensitization rate of 11%, 9% and 13% using the RAST method. Total IgE in cord blood and in blood samples was increased in 21%, 4% and 13% of the children. The most common reactions were to egg and milk. Using the HR response, sensitization to egg showed a rate of 11,3%, 17,2% and 17,5% and to milk a rate of 3,6%, 11% and 11,7%. The RAST method showed a sensitization to egg in 0,3%, 2,8% and 6,7% and to milk in 4,8%, 2,0% and 2,8%.

Conclusion: Newborn children show sign of in utero sensitization or sensitization in early childhood. In cord blood and in blood samples from 3 and 6 months old children positive specific IgE and basophil reactivity was found to allergens such as egg and milk, and to a lesser degree to inhalant allergens as grass, birch and mite.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

44

44

08-05-01.08:13

P-36 SYNDROME OF ENDOGENOUS INTOXICATION IN

PATIENTS WITH MYCROBIAL ECZEMA.

+Prokhorov, Dimitry

Nowadays the syndrome of endogenous intoxication is presented by complex pathologic polyetiologic process which attends majority of somatic diseases and dermatosis. I markers of endogenous intoxication were studied in 63 patients with mycrobial eczema. Moleculs middle mass (MMM) was defined by scrinning method, for definition of antibodies method of hard-phase immunoenzymic analysis. Wasneed the increase of level of moleculs middle mass was determined in blood serum and the lowering of MMM in urine (blood – $0,350\pm0,09$ conditional units; urine – $35,5\pm0,1$ conditional units; normal indexes: blood – $0,242\pm0,01$ conditional units; urine – $39,2\pm0,3$ conditional units). It was also registered the high level of antibody to lipopolysaccharide Esherichia coli K30 in blood serum in 1,5 times higher, than in healthy people.

Received data show the presence of a considerable extent of endogenous intoxication in patients with mycrobial eczema. Clinical manifistation of endotoxicosis can corulated with the heaviness of flow dermatosis, the extensiveness of skin affection and disturbance of the common state of organism.

P-37

STUDY OF EXPRESSION OF FAS-RECEPTOR ON THE LYMPHOCYTES OF PERIPHERAL BLOOD IN PATIENTS WITH PEMPHIGUS

+Pritulo, Olga

Two-parametric immunophenotypic analysis of expression of antigen CD95 (FAS/APO-1) on superficial membrane of lymphocytes of peripheral blood in patients with pemphigus on the background of hormonotherapy was done by means of running laser cytofluorometry (cytofluorometer FACScan, Becton Dickinson). It was determined, that in the examined patients the number of CD4+CD95+ - lymphocytes were 4,2±1,6%. Within subpopulation of CD4+ - lymphocytes 10,8±2,4%, cells carried marker of activation antigen HLA-DR. CD95+ - lymphocytes of patients with pemphigus membrano-associated FAS-receptor didn't express.The Number of common population of CD95+ - lymphocytes and immunoregulatory CD4+ - subpopulation of lymphocytes in these patients was within the limits of normative meaning. Received data indicate the presence of inverse interaction in the number of lymphocytes with phenotype CD4+HLA-DR+ and number of CD4+CD95+ - lymphocytes in patients with pemphigus at hormonotherapy.

P-38

MED/MPD IN THIN AND THICK SKIN

+Nordal, Eli J.* (NORWAY) *Ullevål Sykehus

There is no available knowledge of tolerance to UV irradiation by different sources and in different body areas. In UV treatment of hand and foot dermatoses dose enhancement has been done according to experience, and not led by MED/ MPD measurements.

In six healthy volunteers we have measured MED after TL01 irradiation in thin skin (dorsal aspect of hands, buttocks) and in thick skin (palms). In the same localizations we have measured MPD after P-TL01 and PUVA irradiation.

As TL01 source we have used a plate of 20W tubes \times 6, effect 4.3 mW/cm² (Esshå elagentur, Värnamo, Sweden) and as UVA source a PUVA 180, effect 6.4 mW/cm² (Waldmann, Germany).

With TL01 irradiation the MED of palms was 20–43 times MED of buttocks, with MED of dorsal aspect of hands lying close above buttocks. Addition of psoralen did not change this ratio, and MPD was only slightly lower than MED. Though the numbers are few the findings are consistent.

PUVA MPD measurements revealed inconsistent results with MPD of palms to be 1.7–6.7 times MPD of buttocks, and the values for dorsal aspects of hands lying in between.

The study should be repeated in a larger number with standardized conditions including measurement of plasma psoralen.

P-39

45

THE POTENTIAL ROLE OF OXIDATIVE STRESS IN ELICITATION OF CONTACT ALLERGY

+Kaur, Sirje* (ESTONIA); Zilmer, Mihkel (ESTONIA); Eisen, Maigi (ESTONIA); Kullisaar, Tiiu (ESTONIA); Vihalemm, Tiiu (ESTONIA); Rehema, Aune (ESTONIA) *Tartu University

Reactive oxygen species (ROS) are involved in the pathogenesis of inflammatory skin diseases. To evaluate the extent of oxidative stress (OS) in reactive patch test sites, we compared the iron status and the glutathione redox status in the positive to 5% NiSO4 patch test areas and in the normal skin in eight female volunteers.

We found a 4-fold increase of the iron level in the positive patch test areas in comparison with the healthy skin (p<0.001 by Wilcoxon's signed range test). Iron derives mostly from storage proteins and it is released under the influence of ROS generated by inflammatory cells. The unbound iron binding capacity (UIBC) and possible total binding capacity of iron sequesters (TIBC) were significantly (p<0.005) increased in the positive patch test sites. There was only a slight difference in the diene conjugate amounts between the inflamed and the healthy skin, which indicated that lipid peroxidation did not take place although the iron level was high. The most important low-molecular weight

antioxidant in skin cells is glutathione that removes ROS by conjugation. The oxidized glutathione (GSSG) level was markedly increased in the positive patch test sites in comparison with the normal skin (p<0.003). Because of a concomitant GSH increase in the reactive patch test sites, the difference in the GSSG/GSH ratios was below statistical significance. There was a positive correlation between the iron level and the GSSG amount.

Recently, mechanisms underlying the contact sensitization during patch testing have been investigated. Our results suggest that the positive patch test reaction is accompanied by potent OS. ROS released during inflammation can oxidize metals to higher oxidation states and favour the contact sensitization.

P-40

PATCH TEST REACTIONS WITH DENTAL SCREENING SERIES

+Kanerva, Lasse* (FINLAND); Aalto-Korte, K (FINLAND); Estlander, T (FINLAND); Hannuksela, M (FINLAND); Harvima, RJ (FINLAND); Hasan, T (FINLAND); Horsmanheimo, M (FINLAND); Jolanki, R (FINLAND); Kalimo, K (FINLAND); Lahti, A (FINLAND); Lammintausta, K (FINLAND); Lauerma, A (FINLAND); Niinimäki, A (FINLAND); Rantanen, T (FINLAND); Turjanmaa, K (FIN-LAND); Vuorela, A-M (FINLAND) *Finnish Institute of Occupational Health

Background. Dental products contain many allergens and may cause problems both to patients undergoing dental treatment and to dental personnel from occupational exposure. Single patch test clinics may not study sufficient numbers of patients to get reliable data on uncommon allergens.

Objective. To get information on dental allergens based on a multicenter study.

Material and Methods. The Finnish Contact Dermatitis Group tested more than 4000 patients (for most allergens 2300–2600 patients) with dental screening series. Conventional patch testing was performed. The total number and percentage of irritant [scored as irritant (IR) or doubtful (?)] and allergic (scored as +, ++ or +++) patch test reactions, respectively, were calculated, as well as the highest and lowest percentage of allergic patch test reactions recorded by the different patch test clinics. A reaction index (RI) was calculated giving information on the irritancy of the patch test substances.

Results. The most frequent allergic patch test reactions were caused by nickel (14.6%), ammoniated mercury (13%), mercury (10.3%), gold (7.7%), benzoic acid (4.3%), palladium (4.2%) and cobalt (4.1%). 2-hydroxyethyl methacrylate (2.8%) provoked most of the reactions caused by (meth)acrylates. Menthol, peppermint oil, ammonium tetrachloroplatinate and amalgam alloying metals provoked no (neither allergic nor irritant) patch test reactions.

Conclusion: Patch testings with allergens in the dental screening series, including (meth)acrylates and mercury need to be performed to detect contact allergy from dental products.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

P-41

THE IMPORTANCE OF UNDERSTANDING EXPOSURE IN RISK ASSESSMENT

+McNamee, Pauline* (UNITED KINGDOM) *Procter & Gamble Technical Center Ltd.

The development of ingredients and products for the consumer market requires, as part of the pre-marketing safety assessment, a thorough evaluation of their potential to induce contact allergy and/or elicit clinically relevant Allergic Contact Dermatitis (ACD). An essential element of the skin sensitisation risk assessment process is the evaluation and understanding of the relationship between skin sensitisation hazard (the inherent potential of an ingredient to cause allergic skin sensitisation) and actual skin sensitisation risk where the latter relates to the induction of contact allergy and/or elicitation of ACD under exposure conditions typical of all intended and foreseeable uses of the product by consumers.

Many of the chemicals in common use today possess, to some degree, the potential to cause contact allergy. However the fact that a chemical is a contact allergen does not mean that it cannot be formulated into a consumer product at safe levels. For example, it is well known that ingredients, such as certain preservatives, which have known contact allergy potential can be formulated into consumer products at levels that are safe and do not result in an unacceptable incidence of skin reactions so long as the in use exposures are below the recognised thresholds for induction and elicitation of sensitisation. It is equally well known that these same ingredients can trigger significant ACD when formulated into products inappropriately, e.g. at too high a level. This is based on the knowledge that all allergens demonstrate dose-response and threshold characteristics and that exposure is a key parameter to take into account. The dose response for induction of skin allergy and elicitation of ACD can be directly influenced by a number of factors including, for example, the vehicle system/product matrix in which the allergen is presented to the skin, the frequency and duration of exposure, underlying skin irritation and whether the ingredient/product is occluded (e.g. deodorant application versus shampoo use).

It has been known for over a decade that an understanding of the concentration (dose/unit area) of allergen applied to skin, rather than the absolute amount (volume) applied is more important to the understanding of skin sensitisation risk. It is only relatively recently, however, that such exposure scenarios have been used to understand whether consumer exposure in use to an allergen is acceptable relative to an established "safety" or "uncertainty" factor. Such a safety/uncertainty factor is calculated taking into account the differences that might exist (e.g. matrix/vehicle effects, occlusion versus non-occlusion, frequency and duration of exposure) between the potential exposure for the consumer versus those used to establish known safe benchmarks such as the No Effect Level (NOEL) determined from pre-clinical studies and confirmed in such human studies as the Human Repeat Insult Patch Test (HRIPT). This poster details the principles of exposure-based risk assessment for consumer products using MCI/MI as a case study.

P-42 DOES IMIQUIMOD NORMALISE HAIRGROWTH IN

ALOPECIA AREATA?

+Sommerfeld, Beatrice* (SWEDEN); Popova, I. (SWEDEN) *Lidingö Hudläkarmottagning

Imiquimod is a synthetic molecule with potent immune modifying activities. The induction of among others, interferon-g (IFN-g) and interleukin-12 (IL-12), favour an immune TH1 reaction and an immune deviation away from a TH2 response, which may dominate in alopecia. Some anecdotal reports have indicated beneficial effects in alopecia.

Background: We report 5 cases of alopecia areata which all had regrowth of hair after treatment with imiquimod 5% cream (Aldara^M). Three women, age 41 to 52 years, one girl, 11 years, and one man 52 years, with a recurrent alopecia areata. None had family history of alopecia, but the girl suffered atopic eczema (AE) since the age of one, and the man had periods of AE. Except for the man, none had previously had any abnormal hairloss. The hairless areas varied from 2 cm in diameter to 16*3.5 cm .Two patients had two spots involved. Two of the women and the man indicated stress as a possible provoking factor, while the others had no obvious reason. The duration of the present hairloss varied between 2.5 month and one year.

Procedures: The patients applied the cream locally once daily or each second day. Most patients were seen after 6 weeks and than followed up for different time periods. Except for the girl, photos were taken before treatment and at each follow up visit. In the girl treatment was initiated with clobetasol propionat solution for a total of 4 weeks during a two month period with no effect on hairgrowth.

Follow-up: In the girl, after 6 weeks of treatment with imiquimod 5% cream 2/3 of the area was regrown. Except for one woman all had regrowth after 6 weeks. The patients were followed for different periods ranging from 18 weeks to 9 months. In all patients regrowth was observed. The treatment was well tolerated without any serious reaction. The man experienced light ulceration and erythema on a once daily application, which disappeared when the dose was reduced to each second day.

Conclusion: In 5 patients with alopecia areata, partly or total regrowth of hair were observed after local application of imiquimod 5% cream. As spontaneous regrowth may occur in these patients, this can not be excluded. However, in our opinion it is highly unlikely that, by chance, all should start regrowing spontaneously at the time of treatment. Further controlled studies are warranted.

P-43

SUCCESSFUL TREATMENT FOR MULTIPLE SUPERFI-CIAL BASAL CELL CARCINOMA USING IMIQUIMOD 5% CREAM-A CASE REPORT

+Eklind, Jan* (SWEDEN); Lidbrink, Peter (SWEDEN) *Huddinge University Hospital

Imiquimod stimulates interferon-a and other cytokines and is shown to have a broad anti-tumor effect in animal studies. A double blind pilot study has shown total / partly clearance of BCC in man.

Background: We report an otherwise healthy 72 year-old Caucasian man who had had multiple superficial basal cell carcinomas (BCC) since 1965. His sister and uncle also have multiple superficial BCC, but he had no known immune defect or immunosuppressive treatment. He has had approximately 2-3 various surgical procedures each year, mostly ordinary excisions and he subsequently developed a syringes- and operation-phobia. Since the patient refused any surgical procedures, he was referred from the Department of Plastic Surgery at Karolinska Hospital, where they had done three excisions and taken one punch biopsy. One excision showed residual of BCC in the left eyebrow. The punch biopsy lateral to the left eyebrow showed multifocal growth of superficial BCC. He also had several lesions on the trunk. When we first saw him in December 1999 he had a total of 29 clinically visible superficial BCC, the majority on the trunk. The earlier pathological findings were matching. A non-invasive treatment was desirable and possible with imiquimod 5% cream (Aldara®).

Procedure: To start with, the patients 29 lesions were treated three times a week by a nurse. After four applications he reacted with itch, redness and even shallow ulcerations. The treatment was paused for one week. After 8 weeks most of the lesions were significantly reduced and some still ulcerated. He was treated for a total of 16 weeks.

Follow-up: We saw him again after four weeks. All treated areas were slightly erytematous. He accepted four punch biopsies of which one revealed rests of BCC. Clinical follow-up at 4, 6 and 8 months showed no signs of recurrences of the treated lesions.

Conclusions: A man with a history of multiple BCC was successfully treated with local imiquimod, which seems to be a good alternative treatment modality.

P-44

Photodynamic therapy (PDT) with Metvix[®] cream

versus topical treatment with Efudix[®] cream in patients

with multiple actinic keratosis on sun-damaged skin. +Kampman, Petra* (NORWAY); Lützow-Holm, Claus (NORWAY); Christensen, Ole (NORWAY)

*Rikshospitalet

This clinical trial compares response and sideeffects of traditional topical 5-FU-treatment (Efudix® cream) and photodynamic therapy with a new fotosensitizer containing 5aminolevulinate (Metvix® cream 160 mg/g) on multiple and thick actinic keratosis (AK) on sundamaged skin.

Methods: 12 patients (10 male; 2 female; mean age 72 (59 - 82 yrs)) with multiple and symmetrical distribuated AK-lesions, including 6 referance-lesions > 4mm were – in a defined bodyarea – treated with Efudix[®] on the right half of the body, and Metvix[®]-PDT on the left half of the body in the same patient.

Metvix[®] cream was applied 3 hours before irradiation with blue light 420 nm 5 J/cm². Efudix[®] cream was applied b.i.d . for 3 weeks. The response of the treated lesions was evaluated after 3 months when all lesjons on the left body side wher retreated with Metvix[®]-PDT now using red light 570-670 nm 75 J/cm².

Results: 12 patients with a total of 546 AK-lesions (mean 46), mainly located in sunexposed sites of the head were included in this explorative trial. 11 patients where included in the effect analysis, as one patient dropped out. Evaluation was performed after 6 and 12 months.

Metvix® PDT was tolerated well with mild transcient local sideeffects as erythema and oedema short time after treatment. In the Efudix®-treated sites the expected sideefekts (erythema, oedema, erosions) were seen. PDT-treatment was therefore preferred subjectively, but most of the patients favoured the treatment with the best clinical outcome.

Conclusion: In this study 5-FU was significantly more effective than Metvix[®]-PDT in clearing/ reducing AK. Metvix[®]-PDT was without major side effects compaired to the wellknown side effects of 5-FU. Metvix[®]-PDT was only effective in rather thin AK, indicating that thick AK lesions need to be manipulated with curretage before PDT to enhance penetration of Metvix[®] cream.

P-45

MISOPROSTOL IMPROVES SYMPTOMS IN PATIENTS WITH ERYTHROMELALGIA

+Mørk, Cato* (NORWAY); Kvernebo, K (NORWAY) *The National Hospital

Erythromelalgia (EM) is a condition defined by red, hot and painful extremities. Warmth induce and intensifies the discomfort while cold provides relief. We have previously postulated microvascular arteriovenous shunting as a pathogenetic mechanism. According to this hypothesis there is maldistribution of skin perfusion, with increased thermoregulatory flow and a relative deficit of nutritive perfusion with tissue hypoxia. Vasodilatation may enhance nutritional blood flow, improve tissue oxygenation and remission of symptoms have been reported after infusions with vasodilators (alprostadil/prostacycline/sodium nitroprusside). No single medication or treatment has been universally helpful and treatment reports in the literature are limited to single or few cases, not placebo controlled.

The objective of the present study was to determine whether misoprostol, an oral, synthetic prostaglandin E1 analogue and vasodilator, leads to clinical improvement in patients with EM in a non-randomised, placebo compared study. 21 patients aged 44,7+12,5 years (mean+SD) were treated with placebo for six weeks followed by misoprostol for the next six weeks.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

Pain and cooling score, ability to induce pain after central body heating and global self-assessment of EM suffering was significantly reduced after the intervention. The symptoms deteriorated in most patients after discontinuation of the study medication.

In conclusion, we have performed the first placebo controlled clinical trial in patients with EM. Misoprostol significantly reduces EM symptoms. The long term effect seems to be limited. A redistribution of microvascular perfusion is a possible mechanism for the beneficial effect.

P-46

IMPROVED QUALITY OF LIFE AND DISEASE SEVERITY IN NORWEGIAN PATIENTS WITH PSORIASIS AFTER CLIMATOTHERAPY AT THE CANARY ISLAND

+Mørk, Cato* (NORWAY); Wahl, A (NORWAY) *The National Hospital

Little attention has been paid to the effect of different treatment regimes or treatment settings on quality of life in patients suffering from psoriasis. The aim of the present study was to explore the effect of climatotherapy on quality of life and disease severity in Norwegian patients with psoriasis. Supervised climatotherapy at the Norwegian Health Center consists of a 3-week health care programme including sun exposure, sea bathing, psychosocial and physical stimulation in a relaxing atmosphere, and education with emphasis on improving the coping abilities in relation to the disease, its treatment and its consequences. Over a two year period (1994–1996), patients were invited to complete the Dermatology Life Quality Index (DLQI) on arrival and at the end of the treatment period. Psoriasis severity (PASI / PSI) was also assessed by the dermatologist pre and post treatment. The sample consists of 229 patients from the season 1994/1995 and 230 patients from the season 1995/ 1996. Descriptive analyses were performed to assess the characteristics of the sample. Paired t tests were performed to assess the differences in the mean pre - and post treatment values for quality of life and psoriasis severity. In the sample from 1994/1995, 59.8% was men and in the sample from 1995/1996, 59.1% was men. With regard to age, the mean age of the sample from 1994/1995 was 47.7 (13.4) 23–82 (range), and for the sample from 1995 / 1996, the mean age was 49.8 (13.4) 22-86 (range). Results from the paired t tests showed a significantly decrease on a 0.01 level in impairment of quality of life and psoriasis severity in both seasons. In the absence of a permanent cure for psoriasis, the goal is to minimise the extent and severity of the condition to the point where it no longer substantially disrupts the patient's quality of life.

P-47

A NEED FOR PREGNANCY CARRIER TEST FOR JUNCTIONAL EPIDERMOLYSIS BULLOSA HERLITZ IN

SWEDEN?

+Gedde-Dahl, Tobias* (NORWAY); Holmberg, Eva (SWE-DEN); Kristoffersson, Ulf (SWEDEN) *Rikshospitalet

Over 2% of the population in Västerbotten and Norrbotten and about 1% of the total Swedish population are heterozygous carriers for this disasterous disease with an average life span of 7 months and with profound disturbances and tragedies in 2-4 Swedish families annually (1,2). Herlitz disease is caused by nonsense mutations in either of the 3 genes LAMC2, LAMB3 and LAMA3 coding for the 3 polypeptides laminin g2, b3, a3. These polypeptides participate in the heterotrimeric laminin-5 molecule essential for the anchorage of the hemidesmosomal anchoring fibrils traversing lamina rara into the lamina densa of the dermoepidermal junction (3,4). Strong association to dinucleotide markers on chromosome 1q32 have pinpointed one predominant haplotype involved in almost all Swedish JEB-H families. Six families spread over Northern Scandinavia are compound for this and another uniform haplotype, suggesting 2 prehistoric mutations. The common haplotype appears to carry the LAMB3 mutation R635X, first discovered in 1994 (5) and later found in 45% of all JEB-H alleles examined in Caucasians (6). From the 8 new Herlitz cases born 1999-2000 in Northern Sweden, the proportion of R635X to the other prehistoric mutation is 14:2.If in Sweden, with 90.000 annual births, all pregnant woman was offered and chose to have a blood sample examined for these two mutations, 900 pregnant women will be shown to be mutation carriers. Of their husbands 9 will be carriers, hence 9 couples will annually be offered chorion villus sample (CVS) and 1-3 fetuses shown to develop Herlitz disease, preventable by interruption of pregnancy. These figures assume an overall carrier frequency of 1%, which may be an underestimate. Relative to the health expenses of keeping Herlitz babies alive at an optimal life quality untill death, a pregnancy screening may be beneficial also in terms of health economy.

P-48

GENERALIZED BASALOID FOLLICULAR HAMARTOMA TREATED WITH X-RAY - A CASE REPORT

+Broberg, Ann (SWEDEN); Landys, Karl (SWEDEN); Ternesten-Bratel, Annika (SWEDEN)

A 16-year-old woman noted a thinning of scalp hair and eyebrows and during the same period a papular skin lesion appeared in the face. The skin lesion slowly progressed and she was first seen at the Department of Dermatology in February 1989. The histopathological picture of the facial papules was consistent with generalized basaloid follicular hamartoma (1). The skin lesions progressed during the following years and during this period isotretinoin and CO_2 -laser treatment were used without effect. A plastic surgeon was consulted but surgery was declined. In 1998 the hamartoma had progressed severely. We could not find any re-

port of treatment in the literature and because the progress was so severe in our patient, treatment with Caelyx[®], a liposomal anthracycline, of an absolute dose of 50 mg i.v.×1/ week and then Interferon Alfanative[®] 3.0 MIU s.c.×3/week for a period of 24 weeks was started. The treatment did not have any effect on the skin lesion.

In December 1998, a treatment with superficial x-ray therapy was started using 70 kV energy in fractionation of 3.0 Gy per day up to the tumor dose of 45.0 Gy. During the last 3 years 4 different areas in the face have been treated. The effect of the x-ray treatment has been very good which is clearly seen both clinically and in skin biopsies from different facial areas.

P-49

PILI TORTI ET CANALICULI AND AGENESIS OF TEETH. REPORT OF A NEW "PURE " HAIR-TEETH ECTODER-MAL DYSPLASIA

+Selvaag, Edgar (GERMANY)

Ectodermal dysplasias comprise a heterogeneous group of inherited developmental disorders affecting tissue and organs of ectodermal origin. Their classification is based on malformations in hair, teeth, nails, and sweat glands as the major criteria. Ectodermal dysplasias are thus divided into 11 subgroups based on a minimum of two ectodermal signs with or without other developmental defects. A four generation norwegian family with structural hair shaft abnormalities and agenesis of teeth is presented. The index person had suffered from stiff and rough hair since childhood. His paternal relatives also showed stiff and rough hair. Hair samples from him and his relatives were fixed onto probes with double-sided scotch tape, coated with 30 nm layer of gold/palladium alloy in a Polaron E 5100 Sputter Coater (Polaron Equipment Ltd., Watford, U.K.), and the specimens were then examined and photographed in a Philips SEM 515 microscope (Philips, Eindhoven, The Netherlands). The scanning electron microscopical investigation of hairs showed pili torti et canaliculi. Odontological investigations revealed agenesis of certain teeth, primarily the upper and/or lower incisors. No associated defects of nails, disturbances of sweat gland function, nor other defects of ectodermal or other origin were found. The dental abnormalities became obvious with the eruption of the permanent teeth. The ectodermal dysplasia seems to be inherited autosomal-dominantly, and has not been described previously.

P-50

PHOTOTOXICITY TO DIURETICS AND ANTIDIABETICS IN THE CULTURED KERATINOCYTE CELL LINE HaCaT. EVALUATION BY CLONOGENIC ASSAY AND SINGLE CELL GEL ELECTROPHORESIS (COMET ASSAY)

+Selvaag, Edgar (GERMANY); Petersen, Anita B. (DEN-MARK); Gniadecki, Robert (DENMARK); Thorn, Tine (DENMARK); Wulf, Hans Christian (DENMARK)

The oral antidiabetics tolbutamide, glibenclamide, and glipizide, and the diuretics bendroflumethiazide, butizide, furosemide, hydrochlorothiazide, and trichlormethiazide were investigated for their potential to cause phototoxicity in the HaCaT cell line. The cells were incubated with different concentrations of the drugs and then exposed to UVA1 irradiation. Cell survival was evaluated in a clonogenic assay and phototoxic DNA damage was investigated by the single cell gel elctrophoresis (comet assay). The effects of the antioxidants L-ascorbic acid, and α -tocopherol on oxidative DNA damage were also assessed. Bendroflumethiazide, furosemide, hydrochlorothiazide, trichlormethiazide, or tolbutamide induced dose-dependent phototoxicity in the clonogenic assay. Cells incubated with bendroflumethiazide, tolbutamide, and glibenclamide, and irradiated with UVA1 demonstrated an increased oxidative DNA damage revealed as alkali-labile sites in the comet assay. Pretreatment with L-ascorbic acid, or a-tocopherol, suppressed the UVA-induced DNA damage in cells incubated with 1 mM of bendroflumethiazide, furosemide, glibenclamide, glipizide, tolbutamide, and trichloromethiazide, further implying the involvement of reactive oxygen species in the phototoxic DNA damage. These results indicate a link between phototoxic and photocancerogenic potential of the sulfonamide-derived oral antidiabetic and diuretic drugs, as it has previously been recognized for psoralen, chlorpromazine, and fluoroquinolones. Excessive exposure to UV light may be deleterious for patients treated with oral antidiabetic and diuretic drugs.

P-51

CLINICAL FINDINGS AND ENVIRONMENTAL FACTORS RELATED TO UROD GENE AND HFE GENE MUTATIONS IN DANISH PATIENTS WITH PORPHYRIA CUTANEA TARDA.

+Bygum, Anette* (DENMARK); Christiansen, Lene (DEN-MARK); Thomsen, Kristian (DENMARK); Brandrup, Flemming (DENMARK) *Odense University Hospital

The manifestation of porphyria cutanea tarda (PCT) is based on genetical and environmental factors. The cytoplasmic enzyme activity of uroporphyrinogen decarboxylase (UROD), which catalyzes the fifth step in the haemsynthesis, is decreased by 60–90% in patients with active skin lesions. Demonstration of mutations in the UROD gene, located at chromosome 1p34, discriminates familial PCT (fPCT) from sporadic cases (sPCT). Furthermore, mutations in the haemochromatosis gene (HFE) may be implicated in the aetiology of PCT.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

The aim of the present study was to classify a series of Danish patients according to UROD and HFE mutations and by clinical investigation to identify environmental risk factors of importance for the clinical expression in predisposed patients. The study includes 53 Danish PCT patients with clinically overt disease in 36 and clinical remission in 17 patients at the time of inclusion. 25% of our patients had fPCT as seven different, probably disease related mutations were identified in 13 patients. 15% were homozygotic for the HFE C282Y mutation and 6% (3 patients) met current clinical and biochemical criteria for expressed haemochromatosis. No statistically significant difference was found between sPCT and fPCT regarding age of onset, clinical severity, sex, liver function tests and iron storage parameters. However, daily alcohol intake and use of oestrogens were reported more frequently in the sPCT group compared to the fPCT group. The present study shows that genetic, biochemical and clinical data should be taken into account when diagnosing PCT. Mutations in UROD as well as HFE can predispose to PCT. Examination for mutations in UROD and HFE genes help to establish the diagnosis and in cases with HFE mutations more intensive phlebotomy may be necessary to prevent liver disease.

P-52

DERMATOVENEROLOGICAL SERVICE IN ESTONIA

+Kukk, Terje* (ESTONIA); Poder, A. (ESTONIA); Kangur, A. (ESTONIA); Silm, H. (ESTONIA) *University of Tartu, Clinic of Dermatology

Changes are expected in Estonian health policy over the next few years. At present the situation in dermatovenerology in Estonia is the following.

There are 83 dermatovenerologists (aged up to 67) active in the field as well as 11 postgraduate students/residents. Altogether, this makes 6 specialists per 100,000 inhabitants (data as of first of January, 2000), which is twice as high as the figure for the Nordic countries.

Most of the dermatovenerologists are concentrated in Tallinn and Tartu (35 and 17, respectively). With the exception of the Hiiumaa county, there is at least one dermatologist in every county (Figure). In 1999 Estonia had 841 reported cases of syphilis (with 7 of the patients under age 5), 1103 cases of gonorrhoea, 3413 cases of chlamydiosis, and 3508 cases of trichomonosis. There are four specialized dermatovenerological hospitals, situated in Tartu, Tallinn, Pärnu, and Narva, with a total of 160 beds plus 46 beds in daydepartments. In 1999, 3316 patients were hospitalized and 1026 received treatment in the day-departments. All four hospitals also have outpatient departments. A visit to a dermatovenerologist does not require a reference from the family doctor. The changes expected will involve a radical decrease in the number of beds with a corresponding increase in outpatient treatment.

The training center for medical students in Estonia is the University of Tartu, whose Clinic of Dermatovenerology (50 beds) makes up the base for training future dermatovenerologists. Residency takes place in the same clinic as well as in Tallinn Dermatological Hospital (70 beds) and lasts for three years.

We suggest a close collaboration between the Nordic countries and Estonia in the area of postgraduate training of dermatovenerologists.

P-53

GONORRHEA IN A NEW MILLENNIUM.

+Thune, Turid* (NORWAY); Rustad, Lisbeth (NORWAY) *Haukeland University Hospital

During the last two decades Neisseria gonorrhoeae has become a rare cause of sexually transmitted disease in Norway (1). We have looked at all the gonococcal infections registered at the STD clinic, department of Dermatology and Venereology, Haukeland University Hospital, Bergen, Norway, from Jan. 1st 1997 until Dec. 31st 2000. 45 infections caused by N. gonorrhoeae were verified by cultivation. The aim of the study was to look at the gonococcal susceptibility to antibiotics. During the study we also made some other interesting observations:

- Most of the patients were males.
- More than 50% caught the infection abroad.
- Most of the males caught the infection from a casual partner.
- Less than 10% had a co-infection with chlamydia trachomatis.
- Resistance to the commonly used antibiotics was high.

Because of the high resistance against penicillin, antibiotics in the quinolone group have been recommended as the drug of choice against infections caused by N. gonorrhoeae (2). Quinolone resistance is increasing. About 35% of the gonococci cultured were either resistant or showed reduced sensitivity to fluorinated quinolone. When the infection was imported from the southeastern part of Asia the percentage was nearly 60. This is coherent with the gonococcal susceptibility to antibiotics found in this area (3). The knowledge of the increasing resistance against quinolone should be taken into account in the treatment uncomplicated gonorrhea.

P-54

MICROSCOPIC VIEW OF METHYLENE BLUE (MB) STAINED URETHRAL SMEAR OF THE MALE ATTEND-ING STD OUTPATIENT CLINIC AND ITS RELATION TO

C. TRACHOMATIS INFECTION

+Vagoras, Andrius* (LITHUANIA); Sumila, A. (LITHUANIA); Lapinskaite, G. (LITHUANIA); Marciukaitiene, I. (LITHUA-NIA)

*University Hospital

Objectives: Study aimed to evaluate empiric difference of microscopic view of MB stained urethral smear and to look for a possible relation of these differences to C. trachomatis infection.

Methods: Urethral smears of 215 men attending outpatient clinics of veneral diseases were taken using 1 µl bacteriological plastic loop. Direct microscopy of the methylene blue stained smear was performed at "bed-side". Finding of >4 of polymorphonuclears (PMNL) in more than 5 "eye" field on high power magnification, was considered as diagnosis of urethritis. Depending on distribution pattern of PMNL's in the smear urethritis was categorized: 1-PMNL's are detected only in the stands of mucous; 2-PMNL's only between the epithelial cells (EC); 3-PMNL's are detected both in mucous and in between the EC; 4-PMNL's and EC are within a large amount of mucous. First voided urine of every patient was tested by in-horse PCR (Uppsala University) for presence of C. Trachomatis.

Results: Prevalence of chlamydial infection 11.2%. Urethritis was diagnosed in all cases (n=24) of C. trachomatis infection and was prevalent in 57% (123 of 215). Distribution of C. trachomatis urethritis cases depending on morphology view category presented in the table.

Number of category	1.	2.	3.	4.
Number of total				
cases CT infection	1	12	5	6
Total number of urethritis	31	26	19	47

Conclusion: It could be that not all differences in morphological view of smear depend on the stage of urethritis, sample taking etc. This study find OR=0.1 (CI; 0.005-0.750) and p=0.0295 for category 1; OR=6 (CI; 2-18) and p=0.0003 for category 2. Bigger size of study population and determining of other possible urethral pathogens could uncover statistical significant relations, which could be explained by specificity of urethritis caused by different infection agent or non-infectious origin.

P-55

INFECTIOUS SKIN DISEASES IN RECENTLY RETURNED TRAVELLERS

+Gasior-Chrzan, Barbara* (NORWAY); Falk, Edvard S. (NORWAY)

*University of Tromsø

For many infections acquired during travel, skin lesions may be the only visible clinical findings and often present a diagnostic dilemma. Some skin diseases may develop weeks or months after the patient has returned from a trip. The approach to the recently returned traveller must start with detailed travel and dermatological histories and complete physical examination. Not all skin lesions that appear during and after travel to exotic locations are caused by unusual infections. This presentation focuses on the usual manifestation of infections in immunocompetent hosts. Six cases will be presented and discussed.

51

Supplementum til kongress.p65

P-56

LONG-TERM EFFECTIVENESS OF TERBINAFINE vs.

ITRACONAZOLE IN ONYCHOMYCOSIS: A 5-year

blinded prospective follow-up study

+Sigurgeirsson, Bárdur* (ICELAND); Olafsson, Jón H. (ICELAND); Steinsson, Jón (ICELAND); Paul, Carle (ICE-LAND); Billstein, Stephan (ICELAND); Evans, E. Glyn V. (ICELAND)

*University Hospital

Objective: To examine long-term cure and relapse rates, after treatment with continuous terbinafine and intermittent itraconazole in onychomycosis.

Design: Long-term prospective follow-up study.

Setting: Three centers in Iceland.

Subjects: 151 patients aged 18 to 75 years with a clinical and mycological diagnosis of dermatophyte toenail ony-chomycosis.

Interventions: In a double-blind, double-dummy study, patients were randomized to receive either terbinafine (250 mg/day) for 12 or 16 weeks or itraconazole (400 mg/day) for 1 week in every 4 weeks for 12 weeks or 16 weeks (first intervention). Patients who did not achieve clinical cure at month 18, or experienced relapse/re-infection were offered an additional treatment with terbinafine (second intervention).

Main outcome measures: The primary efficacy criterion was mycological cure, defined as negative results on microscopy and culture at the end of follow-up without requiring second intervention treatment. Secondary efficacy criteria included clinical cure without second intervention treatment, and mycological and clinical relapse rates.

Results: Median duration of follow-up was 54 months. At end of study mycological cure without second intervention treatment was found in 34/74 (46%) of terbinafine-treated subjects and 10/77 (13%) of itraconazole-treated subjects (p<0.001). Mycological and clinical relapse rates were significantly higher in itraconazole vs. terbinafine-treated patients (53% vs. 23% and 40% vs. 17%, respectively). Of the 72 patients who received subsequent terbinafine treatment, 82% achieved mycological cure, and 92% clinical cure.

Conclusion: Continuous terbinafine provided superior longterm mycological and clinical efficacy and lower rates of mycological and clinical relapse, when compared to intermittent itraconazole, in the treatment of onychomycosis.

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

AUTHOR INDEX

Aalto-Korte, K, P-40 Andersen, KE, O-18, P-35 Andresen, B.S., S10:5 Anton-Lamprecht, I., S10:5 Asker, C., O-4 Baran, Robert L., SAT2:2 Barlinn, C., O-31 Barr, Ronald J., C5:1 Bergbrant, I.-M., S12:1 Betz, R.C., S10:7 Billstein, Stephan, P-56 Bindslev-Jensen, C, P-35 Bjellerup, Mats, S2:4 Bjerring, Peter, O-12 Björck, Lars, O-15 Björkner, Bert, S8:3 Blomqvist, K., O-1, O-3, O-5, O-6 Bolund, L., S10:5 Bowden, P., S10:4 Braae Olesen, Anne, S4:3 Brandrup, F., S10:5, S10:7, S11:4, P-51 Broberg, A., P-48, S11:2, S4:2 Bruze, M., S8:1, O-21, O-22 Buus, S.K., S10:5 Bygum, A., P-51, S10:7 Chigorevska, L., O-29 Christensen, Ole, P-44 Christiansen, Lene, P-51 Cove, Jonathan H., SAT1:1 Dahl, Niklas, S10:2 Davidsson, S., O-1, O-3, O-5, O-6, S6:2 de Rie, M, O-13 Eiberg, H., S10:5 Eisen, Maigi, P-39 Eklind, Jan, P-43 Eller, Mark S., PL1:1 Ericson, Marica, S7:3 Estlander, T, O-19, P-40 Evans, E. Glyn V., P-56, SAT2:3 Faergemann, Jan, C2:1, S6:1, SAT2:1 Falk, Edvard S., P-55 Falk, L., S9:1 Ferguson, James, S14:1 Flyvholm, M.-A., O-20 Fogh, H., S7:2 Frick, Inga-Maria, O-15 Fritsch, C, O-13 Funk, J., O-12 Gasior-Chrzan, B., P-55 Gaustad, Peter, S11:1 Gedde-Dahl, T., S10:2, S10:4, S10:6, O-27, P-47, Gilchrest, B.A., PL1:1 Gisslén, Magnus, S5:2 Gjertsen, I., O-31 Gluud, C., S7:2 Gniadecki, Robert, P-50 Gottrup, Finn, S2:2 Gregersen, N., S10:5 Grundmann-Kollmann, Marcella, S14:2 Gudmundsson, F., S7:3 Gånemo, A., S10:2, S10:3

Haarr, L, O-30 Hagforsen, E, O-7, P-33 Halsos, A-M., O-31 Hannuksela, M, P-40 Hansson, Carita, S2:1 Harvima, Ilkka, C1:2 Harvima, R.J., P-40 Hasan, T, P-40 Hausen, B.M., O-18 Haußer, Ingrid, S10:2 Heickendorff, L., P-34 Helsing, Per, O-23, O-9 Heydenreich, J., O-14 Hindsén, Monica, O-22 Holmberg, Eva, P-47 Horn, M, O-13 Horsmanheimo, M, C1:2, P-40 Hyttinen, Mika, C1:2 Høst, A, P-35 Høyheim, B., S10:6, O-23 Iani, Vladimir, S7:1 Ibsen, HHW, S10:7 Isaksson, Marléne, O-21 Jakabsone, I., O-29 Jemec, G., C4:1 Jensen, P.K.A., S10:5 Jolanki, Riitta, O-19, P-40 Jukkola, A., O-25 Juzenas, Petras, S7:1 Juzeniene, Asta, S7:1 Jøhnke, Hanne, P-35 Jørgensen, Bo, S2:5 Kalimo, K, P-40 Kampman, Petra, S10:8, P-44 Kanerva, L., O-19, O-20, P-40 Kangur, A., P-52 Karlsmark, Tonny, S2:3 Karvonen, Jaakko, S13:2 Kaufmann, R, S14:2, O-13 Kaur, Sirje, P-39 Kivinen, Petri K., C1:2 Kobayasi, Takasi, O-26 Koss-Harnes, D., S10:6 Koulu, Leena, S6:4 Kramer, P., O-31 Kristoffersson, P-47 Kruse, T.A., S10:5 Kukk, Terje, P-52 Kullisaar, Tiiu, P-39 Kvernebo, K., O-4, P-45 Ladekjær-Mikkelsen, A.-S., S10:5 Lahti, A, P-40 Lammintausta, K., P-40 Landys, Karl, P-48 Lang, K, O-13 Langeland, N, O-30 Langfeldt, Thore, S3:1 Lapinskaite, G., P-54 Larkö, Olle, O-13, S14:5, S7:3 Larsen, Tove Eeg, O-32 Lauerma, A, P-40 Lauritzen, Edgar, C3:2 Lecha, Mario, SAT2:4

53

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

Lefvert, A-K, P-33 Legat, K, O-13 Leszczynski, Dariusz, S14:4 Lidbrink, Peter, P-43 Lindberg, M., S10:3, O-17, O-20, O-8, S8:3 Lindelöf, Bernt, S13:3 Loeb, M., O-31 Lyngsnes Randaberg, L., O-9 Lützow-Holm, Claus, P-44 Ma, Li-Wei, S7:1 Marciukaitiene, I., P-54 Marsden, H.S., O-30 Matre, R., O-30 McNamee, Pauline, P-41 Meding, Birgitta, O-17, O-20 Meheus, André, PL2:1 Michaëlsson, Gerd, O-7, P-33 Moan, Johan, S14:3, S7:1 Moi, H., O-31 Molin, Lars, O-1, O-3, O-5, O-6 Mustafa, A, P-33 Mørk, Cato, O-1, O-3, O-4, O-5, O-6, P-45, P-46 Mørk, N.-J., S6:3, S10:4, O-9, O-27 Naver, Hans, O-8 Niinimäki, A., P-40 Nilsen, Arvid, O-30 Norberg, L., P-35 Nordal, Eli J., O-10, P-38 Nordlind, Klas, C1:1, O-7, P-33 Nöthen, MM, S10:7 Odegard, Brit, O-32 Oikarinen, A., O-25 Olafsson, Jon Hjaltalin, S6:2, P-56 Parikka, M., O-25 Pastila, Riikka, S14:4 Paul, Carle, P-56 Paulsen, Evy, S8:2, O-18 Pavel, S, O-13 Petersen, Anita B., P-50 Philipsen, P.A., O-14 Pigg, Maritta, S10:2 Podda, Maurizio, S14:2 Poder, A., P-52 Popova, I., P-42 Ranki, Annamari, C1:1 Rantanen, T, P-40 Rasmussen, H.B., S10:7 Rehema, AuneP-39 Reitamo, Sakari. S4:4 Renhua, N., S7:2 Renhua, Na, S7:4 Rhodes, L, O-13 Riekki, Riitta, O-25 Roed-Petersen, J., O-12 Ros, Anne-Marie, S7:5 Rosén, Arne, S7:3 Rossen, Kristian, S7:4 Rubins, Andris, O-29 Rubins, S., O-29 Rustad, Lisbeth, P-53 Ruzicka, Thomas, PL3:1 Salerud, G., O-4 Salo, T., O-25

Samuelsson, Lena, S1:1 Sandberg, Carin, S7:3 Sandby-Møller, J., O-14 Sandström, Eric, S5:1 Schmidtchen, Arthur, O-15 Selvaag, Edgar, P-49, P-50 Serup, Jørgen, S6:5 Sigurgeirsson, B., C2:2, P-56, S11:3, O-1, O-3, O-5, O-6 Silm, H., P-52 Sjödén, P-O, S10:3 Škov, Lone, S1:2 Skov Jensen, J., S9:1 Solér, A, O-13 Sommerfeld, Beatrice, P-42 Spirén, A., O-22 Stang, Henning, O-31 Steinsson, Jón, P-56 Stender, Ida Marie, S7:2, S7:4, O-13 Stenquist, B., C4:1 Sumila, A., P-54 Susitaival, Päivikki, O-20 Swartling, Carl, O-8 Svejgaard, Else, C2:1 Svensson, Åke, O-20 Sviland, Lisbet, S13:1 Söderberg, U., O-12 Søgaard, Helmer, P-34 Sørensen, C.B., S10:5 Søyland, Elisabeth, O-24 Ternesten-Bratel, Annika, P-48 Thestrup-Pedersen, Kristian, S4:1 Thieden, Elisabeth, O-14 Thomsen, Kristian, P-51 Thorn, Tine, P-50 Thorvaldsen, J., O-31 Thune, Per, O-2 Thune, Turid, P-53 Troilius, Agneta, C3:1, C3:2 Turjanmaa, K, P-40 Ullman, Susanne, O-26 Vagoras, Andrius, P-54 Wahl, A, P-46 Vahlquist, Anders, S10:1, S10:2, S10:3, S10:4 Wallenhammar, Lena-Marie, O-17 Warloe, T, O-13 Veien, N.K., S10:5 Wennberg, Ann-Marie, S7:3, O-13 Westermark, Per, S10:2 Vihalemm, Tiiu, P-39 Virtanen, Marie, S10:2, S10:4 Wolf, G, O-13 Wong, G, O-13 Wulf, H.C., S7:2, S7:4, O-13, O-14, P-50 Vuorela, A-M, P-40 Yaar, Mina, PL1:1 Zachariae, Hugh, O-1, O-3, O-5, O-6, P-34 Zachariae, Robert, O-1, O-3, O-5, O-6 Zilmer, Mihkel, P-39

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

54

STADGAR FÖR NORDISK DERMATOLOGISK FÖRENING

antagna vid föreningens första möte i Köpenhamn 1910, ändrade i Köpenhamn 1935, i Stockholm 1946, i Århus 1977 och senast vid föreningens 26:e möte i Reykjavik den 14 juni 1993

- §1 Föreningens syfte är att befrämja samarbete i vetenskap, undervisning och praktisk läkekonst mellan dermatovenereologer i de 5 nordiska länderna (Danmark, Finland, Island, Norge och Sverige.
- §2 Som nya medlemmar kan antas personer i de 5 länderna vilka är verksamma inom dermatologi och venereologi. För inträde fordras, att den som söker om medlemskap föreslås av dermatologisk förening av samma nation; beslut om inval fattas på allmänt möte vid varje kongress med enkel röstövervikt.
- §3 Till hedersledamot kan föreningens allmänna möte kalla den som gjort osedvanligt stora insatser för föreningen eller för nordisk dermatologi och/eller venereologi. För kallelse krävs 2/3 majoritet. Förslag till hedersledamot skall inges skriftligen till generalsekreteraren minst tre månader före allmänna mötet. Förslagen skall godkännas av föreningens styrelser för att kunna presenteras för allmänna mötet.
- §4 Årsavgiften bestämmes vid varje kongress. Medlem som uppnått 65 levnadsår är befriad från avgift.
- §5 Föreningen håller ett möte i regel vart tredje år i ett av de nordiska länderna. Tid och plats för nästa möte bestämmes på varje möte.
- §6 Vid mötet hålls ett sammanträde för föreningsangelägenheter varvid följande ärenden skall förekomma:
 - 1. Kassa förvaltarens berättelse.
 - 2. Revisorernas berättelse jämte frågan om ansvarsfrihet.
 - 3. Årsavgift för kommande 3-årsperiod.
 - 4. Val av styrelse samt 2 revisorer för kommande 3-årsperiod.
 - 5. Val av forskningskommitté
 - 6. Tid och plats för nästa möte fastställes.
 - 7. Antagning av nya medlemmar.
 - 8. Övriga ärenden
- §7 Styrelsen består av: generalsekreteraren samt 9 styrelsemedlemmar och 9 suppleanter (1 från Island och 2 från vart och ett av de övriga länderna). Som extraordinarie medlem ingår den vid mötet verksamme presidenten såvitt han ej i förväg är medlem i styrelsen. Styrelsen väljer inom sig ordförande och dessutom generalsekreterare, som samtidigt är föreningens kassaförvaltare. Generalsekreteraren väljes på obestämd tid, men bör ej fungera i mer än 12 år. De övrigas funktion sträcker sig från slutet av ett möte till slutet av nästa. Styrelsemedlemmarna kan återväljas för ytterligare två 3-årsperioder. De nationella föreningarna anmodas att senast 3 månader före mötet inkomma med förslag till sitt lands styrelsemedlemmar.
- §8 Det dermatologiska sällskapet i det land där mötet skall äga rum, lägger tillrätta kongressens vetenskapliga och övriga program och ombesörjer tryckningen av förhandlingarna i samråd med styrelsen. Varje föredragshållare och diskussionsdeltagare skall sända in ett referat till sekreteraren vid anmälan till kongressen. Föredraget hålles på danska, norska, svenska eller engelska.
- §9 För en förändring av dessa stadgar krävs 2/3 majoritet. Dylika ändringsförslag skall vara insända senast 3 månader före ett mötes avhållande.

55

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

		President	Sekreterare
1. Köpenhamn	1910	C Rasch	
2. Stockholm	1913	E Sederholm	K Marcus
3. Oslo	1916	C Boeck	K Grön
4. Köpenhamn	1919	C Rasch	A Kissmeyer
5. Stockholm	1922	A Afzelius	J Strandberg
6. Helsingfors	1924	J J Karvonen	B Grönroos
7. Oslo	1928	E Bruusgaard	K Grön
8. Stockholm	1932	A. Moberg	J Strandberg
9. Köpenhamn	1935	H Boas	S Emanuel
10. Helsingfors	1938	A Cedercreutz	T E Olin
11. Stockholm	1946	S Hellerström	M Tottie
12. Oslo	1949	N Danbolt	R Björnstad
13. Köpenhamn	1953	H Haxthausen	P-H Nexmand
14. Helsingfors	1956	T Putkonen	V Pirilä
15. Oslo	1959	N Danbolt	M H Foss
16. Göteborg	1962	G Seeberg	B Magnusson
17. Köpenhamn	1965	G Asboe-Hansen	H Schmidt
18. Åbo	1968	C E Sonck	E Lundell
19. Oslo	1971	N Danbolt	K Wereide
20. Stockholm	1974	N Thyresson	Ö Hägermark
21. Århus	1977	H Zachariae	J V Christiansen
22. Helsingfors	1980	K K Mustakallio	L Förström
23. Oslo	1983	G Rajka	L R Braathen
24. Uppsala	1986	L Juhlin	S Öhman
25. Köpenhamn	1989	N Hjorth	J Roed-Petersen
			G Lange Vejlsgaard
26. Reykjavik	1993	J H Olafsson	B Sigurgeirsson
27. Åbo	1995	V Havu	I Helander
28. Bergen	1998	S Helland	J Langeland
29. Göteborg	2001	O Larkö	H Mobacken, E Voog

Möten i Nordisk Dermatologisk Förening 1910–2001

56

NORDISK DERMATOLOGISK FÖRENING

Protokoll fört vid generalförsamling fredagen den 5 juni 1998 i Bergen

Inför generalförsamlingen hade ett föreberedande möte med NDF:s styrelse hållits 4 juni 1998. Närvarande: Anders Vahlquist, Gun-Britt Löwhagen, Jaakko Karvonen, Nils-Jörgen Mörk, Svein Helland, T Egelrud, Kristian Thestrup-Pedersen, Klaus E. Andersen, Lasse Kanerva, Jon Olafsson

1.	Val av ordförande för dagens möte	Till ordförande och protokolljusterare valdes kongresspresident Svein Helland.
2.	Kassaförvaltarens berättelse	Generalsekreteraren redogjorde för föreningens ekonomi, bilaga 1. Vid utgången av 1997 var utgående saldo SEK 545 260,47, d v s ökningen av tillgångarna som började 1989 har fortsatt. Föreningens kostnader i sam- band med det nordiska initiativet att söka arrangörsskap för världs- kongressen 2002 var SEK 43 828. Till årets kongress har SEK 108 345 utbetalats till de nationella föreningarna som resestipendier till yngre dermato-venereologer.
3.	Revisorernas berättelse jämte frågan om ansvarsfrihet	Revisionsberättelse, bilaga 2. Mötet beviljade ansvarsfrihet för åren 1995- 1997 för styrelsen och generalsekreteraren.
4.	Årsavgift för kommande 3-årsperiod.	Beslutades om oförändrad medlemsavgift SEK 30 per år.
5.	Val av styrelse och revisorer för kommande årsperiod	I enlighet med förslag från de nationella föreingarna valdes följande 3- styrelseledamöter:
		<u>Danmark</u> : Ordinarie: Else Svejgaard och Klaus E. Andersen. Suppleanter: Finn-Schultz-Larsen och Knud Kragballe.
		<u>Finland</u> : Ordinarie: Jaako Karvonen och Lasse Kanerva. Suppleanter: Annamari Ranki och Kristina Turjanmaa
		<u>Island:</u> Ordinarie: Bardur Sigurgeirsson. Suppleant: Jon Hjaltalin Olafsson.
		<u>Norge</u> : Ordinarie: Nils-Jörgen Mörk och Ole B. Christensen. Suppleanter: Svein Helland och Elisabeth Sjöland.
		<u>Sverige</u> : Ordinarie: Gun-Britt Löwhagen och Ove Bäck. Suppleanter: Inger Rosdahl och Mona Ståhle-Bäckdal.
		<u>Revisorer</u> : Tapio Rantanen och Kristian Thestrup-Pedersen.
6.	Antagning av nya medlemmar	Till nya medlemmar antogs samtliga personer som invalts i de nationella föreningarna sedan föregående möte 1995 i Åbo.
7.	Fastställande av tid och plats för nästa möte	Beslutades att hålla nästa möte i Göteborg, preliminärt 14-17 juni år 2001.



Till arrangörerna utgår ett garantibelopp SEK 110 000. Av eventuellt ekonomiskt överskott går hälften till föreningen.

Vad gäller språk vid nästa möte beslutades att engelska skall användas för all information och övrig korrespondens inför kongressen samt för föredragssammanfattningar (abstracts) och allt bildmaterial som används i samband med föredrag och posterutställningar. Vidare beslutades att under mötet skall hela tiden minst en parallellsession hållas på engelska samt att starkt rekommendera att en så stor andel som möjligt av övriga inslag sker på engelska.

Det beslutades att föreningen bör ha ett kapital inkluderande utestående garantibelopp om c:a SEK 300 000. Intäkterna under åren 1998–2001 beräknades till c:a SEK 200 000. Överskjutande kapital skall under 1998– 2001 användas enligt följande, under förutsättning att intäkterna blir de förväntade:

- I. Resestipendier för yngre dermatovenereologer till nästa kongress SEK 100 000. Stipendierna skall fördelas mellan länderna i förhållande till medlemsantalet.
- II. IFD Training Center i Moshi (se pkt 9) SEK 75 000 (USD 10 000).
- III. Utbytesstipendier för att göra det möjligt för nordiska dermato-venereologer att under 2–3 dagar studera förhållanden vid klinik inom specialiteten i annat nordiskt land. Ett stipendium à SEK 10 000 per land och år 1998–2001 utbetalas till respektive nationella förening som efter ansökan med bifogad inbjudan från mottagande klinik utser stipendiater. Stipendiater skall författa en reserapport som publiceras i Nordic Forum for Dermato-Venereology.
- IV. Bidrag till en nordisk forskarutbildningskurs under den kommande treårsperioden om SEK 50 000.
- V. Abonnemang på Acta Dermato-Venerelogica till kollegor i Baltikum till en kostnad av SEK 10 000 per år i tre år. Generalsekreteraren förhandlar med förlaget och utreder optimala betingelser för fördelning.
- VI. Ett garantibelopp om SEK 20 000 till Nordic Forum for Dermato-Venereology avsätts.
- VII. Generalsekreteraren får ett expenserkonto (se pkt 10) om maximalt SEK 20 000 per år. Medlen skall bland annat användas till besök vid de nationella föreningarnas årsmöten.

Föredragande: K. Thestrup-Pedersen . Se beslut 8 (II). Summan delas ut i av generalsekreteraren i samband med ILDS-möte i Florens, mars 1999.

Se beslut 8 (VII).

Beslutades att vid innevarande kongress utdela 2 st posterpris à SEK 5000. Till priskommittén utsågs Thor Langeland (smk), Gerd Michaelsson, Klaus E. Andersen, Lasse Kanerva och Jon Olafsson.

Torbjörn Egelrud Generalsekreterare

11. Övriga ärenden

Moshi, Tanzania

Svein Helland President

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

58

9. Ekonomiskt stöd till IFD Training Center

10. Expenserkonto för generalsekreteraren

8. Föreningens framtida verksamhet

EKONOMISK REDOGÖRELSE FÖR 1998, 1999 OCH 2000

Debet	
1998	SEK
Ingående saldo	545260,47
Medlemsavgifter	45730
Räntor	17479,61
Garantisumma	120278,92
Överskott, Bergen	99930
Övriga inbetalningar	40250
	868929

Kredit	
1998	SEK
Diverse utgifter	177420,52
Utgående saldo	691508,48

868929

Debet	
1999	SEK
Ingående saldo	691508,48
Medlemsavgifter	19993,64
Räntor	12776,36
Övriga inbetalningar	81900
	806178,48

Kredit	
1999	SEK
Diverse utgifter	177345
Utgående saldo	628833,48

806178,48

Debet	
2000	SEK
Ingående saldo	628833,48
Medlemsavgifter	31460
Räntor	14850,59
Övriga inbetalningar	1000
	676144,07

Kredit	
2000	
Diverse utgifter	

Diverse utgifter	68704
Utgående saldo	607440,07

676144,07

SEK

Necrologies

Gerda Frentz var født 25. juli 1942. Hun blev medicinsk kandidat i 1970 og speciallæge i dermato-venerologi 1979. Hun blev dr. med. i 1987 på en afhandling "Flowcytometric DNA-analysis of normal, premalignant and malignant epidermal tissues".

I 1994 modtog hun som den første praktiserende dermatolog et 5-årigt forskningsprofessorat under Statens Sundhedsvidenskabelige Forskningsråd. Hun blev ansat ved Københavns Kommunehospitals Institut for Sygdomsforebyggelse for at gennemføre et projekt omhandlende "nonmelanoma hudcancer i Danmark".

Hendes speciallægeuddannelse foregik ved københavnske hudafdelinger med den største del af tiden ved Finsen Instituttet. Fra 1989 var hun praktiserende speciallæge i Panoptikonbygningen i København. Gerda Frentz var uhyre aktiv indenfor det organisatoriske arbejde. Her skal blot nævnes hendes indsats som formand for Dansk Dermatologisk Selskab, indenfor bestyrelsen for Danske Dermatologers Organisation og indenfor bestyrelsen for EADV. Hun modtog i 1990 Schering-Plough Prisen.

Gerda Frentz var uhyre vellidt, hun var et ærligt, hårdt arbejdende menneske. Det var tragisk, at det ikke lykkedes for hende at få afsluttet sit store og vanskelige forskningsprojekt. Hun døde 9. november 1998.

Gadborg, Ejnar var født 16/6-1918. Han blev medicinsk kandidat i 1945 og modtog specialistanerkendelse i dermato-venerologi i 1957. Han blev dr. med. i 1956 på afhandlingen "Om metalallergi". Hans speciallægeuddannelse fandt sted på Finsen Instituttet, på Rigshospitalet og på Københavns Kommunehospital. Fra 1957 til 1987 var han praktiserende speciallæge i Randers. Han var formand for Danske Dermatologers Organisation fra 1970 til 1979. Fra 1971 til 1977 var han Lektor ved Århus Universitet. Han afgik ved døden 8. juli 1996.

Sven Ancher Kvorning var født 14. september 1916. Han blev medicinsk kandidat 1941 og speciallæge i dermatovenerologi 1953. I 1950 blev han dr. med. på en afhandling "Investigations into the pharmacology of skin fats and ointments". Han modtog sin dermato-venerologiske uddannelse på alle de daværende københavnske hudafdelinger. Fra 1962 og indtil 1982 var han overlæge og chef for Københavns Kommunehospitals dermato-venerologiske afdeling og fra 1967 til 1971 professor i hud- og kønssygdomme ved Københavns Universitet. Foruden sin interesse for begge dele af specialet havde han gennem årene bevaret interessen for farmakologi. Han har været formand for Dansk Dermatologisk Selskab foruden for de Københavnske Dermatovenerologers organisation. Sven Ancher Kvorning afgik ved døden 29. juli 1998.

Lennart Hellbe genomgick läkarutbildning på KI Stockholm, kom till Örebro i mitten av 60-talet, fick en BÖL-tjänst 1:a augusti 1970 och en ÖL-tjänst 13.e oktober 1975. Samtidigt med ÖL-tjänsten blev han förordnad som klinikchef, vilket uppdrag han behöll tills han gick i pension 31:a januari 1986 strax före sin 60-årsdag. Började då en ny karriär som aktiv politiker inom Miljöpartet, vilken han sedan fortsatte med fram till sin död 98-06-30.

Ludvigsen, Knud blev født 24. marts 1912. Han blev speciallæge i 1948 efter uddannelse på Marselisborg Hospital, Rigshospitalet og Finsen Instituttet. Han havde speciallægepraksis i Randers fra 1948 til 1988. Han havde været medlem af bestyrelsen for Dermato-Venerologernes Provinsorganisation og formand for Lægekredsforeningen i Randers. Knud Ludvigsen afgik ved døden i 2000.

Pedersen, Daniel var født 18. juli 1912. Han blev medicinsk kandidat i 1941 og modtog specialistanerkendelse i dermato-venerologi i 1949. Hans specialistuddannelse fandt sted på Rudolph Berghs Hospital og Marselisborg Hospital. Fra 1946 til 1948 var han fungerende kredslæge i Århus (Marselisborg). Han havde speciallægepraksis i Vejle i perioden fra 1949 til 1985. Daniel Pedersen afgik ved døden i 2000.

Veikko Pirilä, the father of the Finn Chamber, died suddenly in his own courtyard on 2 November 1998, at the age of 83. All who are interested in contact dermatitis knew



him pesonally or by name. His father Paavo Pirilä was Professor of Dermatology and Venereology at Helsinki University. Veikko followed in his father's footsteps and became specialist in dermatology and venereology in 1947. At that time, he was already interested in contact dermatitis and studied patch testing in Copenhagen. After returning home in 1947, he became Chief Doctor of the Department of Occupational Dermatology at the Finnish Institute of Occupational Health in Helsinki for 25 years. He also became professor of Dermatology and Venereology at Helsinki University in 1958.

Veikko Pirilä had already begun to develop his patch test chamber in the 1960s. The present form of the Finn Chamber was introduced in 1975. Veikko continued to develop the Finn Chamber test system and the manufacturing process in small steps until his death.

The Nordic Contact Dermatitis Research Group was founded in the 1960s, and Veikko Pirilä was one of its founders. The group expanded to the International Contact Dermatitis Research Group, in which Veikko was active until 1983. Veikko Pirilä remained active in the Finnish and Nordic Dermatological societies, as well as in the Finnish Society of Allergology and Clinical Immunology.

Reiter, Henry blev født 2. januar 1920. Han var kandidat fra 1946 og blev speciallæge i dermato-venerologi i 1957. Uddannelsen som speciallæge foregik på Rigshospitalet og Rudolph Berghs Hospital. Fra 1957 til 1985 drev han en stor speciallægepraksis i Hjørring. Henry Reiter døde i 2000.

Speiermann, Jørgen var født 9. juni 1932. Han fik medicinsk embedseksamen 1969 og blev speciallæge i dermatovenerologi i 1977. Speciallægeuddannelsen modtog han på Regionssjukhuset i Örebro og på Sahlgrenska Sjukhuset i Göteborg. Han havde speciallægepraksis i Greve Strand fra 1977 til 1997. Jørgen Speiermann døde 12. juni 1998.

Members in the national societies *denotes new members since last meeting

DENMARK

Aastrup, Bent Borgm.Jørgensens vej 6 DK-2930 Klampenborg

Ackermann, A. Bernard Med Center 530 First Avenue New York, NY 10016

Afzelius, Hans-Wilhelm Klostergade 20-22 DK-8000 Århus C

Agdell, Jan Slottsstadens Läkar-grupp Regementsgatan 50 S-217 48 Malmö

Agner, Tove Ibstrupvej 57 DK-2820 Gentofte

Albrectsen, Birgit Gentoftegade 28B DK-2820 Gentofte

Andersen, Bo Lasthein Strandhuse 45 Strandgården DK-5700 Svendborg

Andersen, Klaus Ejner Derm Avdelning I Odense Sygehus DK-5000 Odense C

Andersen, Peter Hundevadt Vester Strand Alle 55 DK-8240 Risskov

Andersen, Sven La Cour Jægersborg Allé 16, 3 DK-2920 Charlottenlund

Auken, Gunnar Bomhoffs Have 8 DK-1872 Frederiksberg

Avnstorp, Christian Sassvej 2 DK-2820 Gentofte

Avrach, Wolf Willy Banegårdsplatsen 1, 5 DK-1570 Köpenhamn

Baadsgaard, Ole Rahbeks Allé 32, ST.TV. DK-1801 Frederiksberg Balslev, Eva* Sassvej 2 DK-2820 Gentofte

Bang, Flemming da Cunha Mothsvej 66 DK-2840 Holte

Baran, Robert 42, Rue des Serbes F-06400 Cannes

Bech-Thomsen, Niels Linnésgade 16A, 2 DK-1361 Köpenhamn

Beck, Hans-Iver Pattburger Bogen 9A DE-24955 Harrislee

Bendsøe, Niels Anthony Vardavägen 249F S-223 71 Lund

Benefeldt, Eva Merete Marskensgade 2,5.TH DK-2100 Köpenhamn

Bergman, Bente Åbrinken 74 DK-2830 Virum

Bindslev-Jensen, Carsten Bispeengen 70, Anderup DK-5270 Odense N

Bjerring, Peter Stationsgade 4 DK-8240 Risskov

Björngren, Helene Box 4259 S-203 14 Malmö

Blichmann, Christa W. Svanevænget 13A DK-2100 Köpenhamn

Brandrup, Flemming Vestergade 30 DK-5600 Faaborg

Bro-Jørgensen, Anne Vibeke Skodsborgvej 179 DK-2850 Nærum

Brocks, Kim Mathias Lokesvej 4 DK-8800 Viborg

Brodersen, Ingelise Smedievej 23 DK-3400 Hillerød

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

61

Bryld, Lars Erik Dermatol. afd. K. 1502 Gentofte Hospital DK-2900 Hellerup

Bundgaard, Lise Kollemosevej 24B DK-2840 Holte

Buus, Sanne K* Tjalfesvej 22 DK-8230 Åbyhøj

Bygum, Anette* Mollegardet 31 DK-6000 Kolding

Carlsen, Karen Marie* Fasanvænget 286 DK-2980 Kokkedal

Castellani, Teresa Stengårds Allé 31C DK-2800 Lyngby

Christophers, Enno Hautklinik Schittenhelmstr 7 D-24105 Kiel

Christophersen, Jette Ved Højmosen 32 DK-2970 Hørsholm

Clemmensen, Ole Jacob Langelinie 78 DK-5230 Odense M

Cramers, Marie Kristine Hårbyvej 58 Vindskovg., Stjær DK-8660 Skanderborg

Dahl, Jens Christian Saltværksvej 168 DK-2770 Kastrup

Dam, Tomas Norman Barthsgade 7, 1th DK-8200 Aarhus N

Danielsen, Anne Grete* Hovmarksvej 85 DK-2920 Charlottenlund

Danielsen, Lis Skjoldagervej 22 DK-2820 Gentofte

Deleuran, Mette Åbyvej 47 DK-8230 Åbyhøj

Dybdahl, Helle* Kasted Byvej 6 DK-8200 Århus N

Egekvist, Henrik* Bjerget 5 DK-8382 Hinnerup Eriksen, Knud Svanevænget 2 3tv DK-2100 Köpenhamn

Esmann, Jørgen Moltkesvej 76E Bregentved DK-4690 HASLEV

Fischer, Annelise Drachmannsvej 17 DK-2930 Klampenborg

Flindt-Hansen, Henrik Jægersborgs Allé 35 DK-2920 Charlottenlund

Foged, Erik Klemens Nørlundvej 15 DK-7500 Holsterbro

Fogh, Hanne Ådalsvej 19A DK-2720 Vanløse

Fogh, Karsten Søtoften 36 DK-8250 Egå

Frankild, Søren Bo* Æblehaven 6 DK-8660 Skanderborg

Fregert, Sigfrid Mellanvängsvägen 5 S-223 55 Lund

From, Ellis Dr. Ingrids Hospital Box 1001, DK-3900 Nuuk

Gade, Margrethe Søbredden 5 DK-2820 Gentofte

Gammeltoft, Michala Dr Tværgade 41, 1. 4 DK-1302 Köpenhamn

Genner, Jørgen Retsmedicinsk Institut Fredrik den 5s Vej71 DK-2100 Köpenhamn

Gilg, Ingrid Virum Stationsvej 104 DK-2830 Virum

Gjede, Uffe Simons Bakke 66 DK-7700 Thisted

Gniadecka, Monica Christiansvej 19 DK-2920 Charlottenlund

Gniadicki, Robert* Christiansvej 19 DK-2920 Charlottenlund

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

Graudal, Bodil Charlotte Batzkes Bakke 11 DK-3400 Hillerød

Grunnet, Eva Ingersvej 8 DK-2920 Charlottenlund

Hagdrup, Hans Kenneth Skt. Anne Plads 2, 3 DK-5000 Odense C

Hagerman, Gösta Limhamnsvägen 22A S-217 59 Malmö

Halkier-Sørensen, Lars Højdedraget 25 DK-8660 Skanderborg

Hallinger, Lise Kokildehøjen 24 DK-8800 Viborg

Hammershøy, Ole Mariavænget 25 DK-5260 Odense S

Hansen, Bodil Stenvang Langdyssevej 15 DK-7700 Thisted

Hansen, Preben Mitchellstræde 5 Pleiehjemmet DK-2820 Gentofte

Hansen, Ulla Viggo Barfoeds Allé 2 DK-2750 Ballerup

Hansted, Birgitte Gyvelvej 3 DK-2942 Skodsborg

Hastrup, Nina Cicilie Ordrupvej 81B, 4TH DK-2920 Charlottenlund

Hattel, Thais Søndre Skovvej 60 DK-9000 Aalborg

Heidenheim, Michael Gentoftegade 45, 3 DK-2820 Gentofte

Held, Elisabeth* Norgesmindevej 24, 1 DK-2900 Hellerup

Hendel, Jørn Stumpedyssevej 30 Kettinge DK-2970 Hørsholm

Hendel, Lene Stumpedyssevej 30 Kettinge DK-2970 Hørsholm

62

Henningsen, Sten Juel Gartnersvinget 8 DK-2800 Lyngby

Henriksen, Lars Sanatorievej 14C DK-8680 Ry

Hentzer, Bent Clarasvej 10 DK-8700 Horsens

Heydenreich, Gerhard Box 124 DK-6100 Haderslev

Hjorther, Anne Birgitte C.F. Richs Vej 20 DK-2000 Frederiksberg

Hjortshøj, Anders Skovvej 29 Houstrup DK-6830 Nørre-Nebel

Hou-Jensen, Klaus Skodsborg Strandvej 218 DK-2942 Skodsborg

Hougaard, Frands Egernvej 33 DK-2000 Frederiksberg

Hædersdal, Merete* Valdemar Holmers G. 55 DK-2100 Köpenhamn Ø

Høyer, Henrik Algade 33, 3 DK-4000 Roskilde

Ibsen, Hans H. Windeløv Clausens Allé 45 DK-5250 Odense SV

Iversen, Lars Kirsebærhaven 7 DK-8660 Skanderborg

Iversen, Lise Marie Norborg Lindö S-611 93 Nyköping

Iversen, Normann Breum Brodalsgatan 7 S-711 34 Lindesberg

Jacobsen, Finn Kjær Egebjergvej 14 DK-8220 Brabrand

Jacobsen, Jette M. Urup Godthåbsvej 4 DK-8600 Silkeborg

Jacobsen, Keld Urup Godthåbsvej 4 DK-8600 Silkeborg Jansen, Elin Riedel Erik Dahlbergsgatan 35A SE-411 31 Göteborg

Jemec, Gregor Borut Ernst Prinsesse Alexanders Allé 18.3 DK-2920 Charlottenlund

Jensen, Axel Blegvad Fasanvej 16 Assentoft DK-8900 Randers

Jensen, Birgitte Løkke Hallandsparken 62 Høje Taastrup DK-2630 Taastrup

Jensen, Poul Erik Kordilgade 36, 2tv DK-4400 Kalundborg

Jeppsen, Lissi Voss Rugbjergvej 119 DK-8260 Viby J

Joensen, Høgni Debes Gamli Vegur 3 FR-188 Hoyvik

Johansen, Jeanne Duus Løvsangsvej 7, st DK-2900 Hellerup

Johansen, Urs Broby Søllerrød Park BL.9 Lejl 2 DK-2840 Holte

Jonsson, Lennart Lerdalavägen 8 S-541 41 Skövde

Juldorf, Finn Åsbogatan 16 S-262 41 Ängelholm

Justesen, Ole Solbyen 51 DK-9000 Aalborg

Jøhnke, Hanne Niels Uels alle 128DK-5250 Odense SV

Jørgensen, Birgit Borch Frederiksborgvej 5 DK-4000 Roskilde

Jørgensen, Gerda Reincke Ekenæsvej 34 DK-2850 Nærum

Jørgensen, Hans Lindeengen 153 DK-2740 Skovlunde

Jørgensen, Hans P Valnes Storgt. 15 N-1607 Fredrikstad Jørgensen, Hans Paulli H.P. Hanssens vej 5 DK-6400 Sønderborg

Jørgensen, Jørgen Skovvangen 3 DK-2920 Charlottenlund

Kaaber, Knud Bredgade 30 DK-7400 Herning

Kalsbøll, Mogens Strandvejen 174 DK-2920 Charlottenlund

Kamp, Peter Tornbyvej 10 DK-4600 Køge

Karlsmark, Tonny Ingersvej 22 DK-2920 Charlottenlund

Kassis, Vibeke Walther Jacob Erlandersens Gade 11 2tv.

DK-2100 Köpenhamn Ø

Kieffer, Marianne Elholm Frederiksdalsvej 171B DK-2830 Virum

Kirkegaard, Erik Syrenvænget 4 DK-3520 Farum

Kjær, Margrethe Gl. Køge Landevej 255B 1.tv DK-2650 Hvidovre

Kleiter, Ib Juulsvej 13 DK-4720 Præstø

Klemp, Per Helstedsvej 4C DK-3480 Fredensborg

Knudsen, Bodil Bygum Tibberup Allé 11 Hareskov DK-3500 Værløse

Knudsen, Erik A. Langs Hegnet 28 B DK-2800 Lyngby

Knudsen, Hans Ekkert Godthobsvej 113, St+h DK-2000 Fredriksberg

Knudsen, Lone Gammel Strand 40, 1. th DK-1202 Köpenhamn K

Kobayashi, Takasi Hvidkløvervej 1 DK-2400 Köpenhamn NV Kollander, Marianne Wingesvej 10 Over Hornbæk DK-8900 Randers

Kopp, Heinrich Skovlodden 28 DK-2840 Holte

Kragballe, Knud Klokkerfaldet 42 DK-8210 Århus V

Kristensen, Berit Hudklinikken Bredgade 50 DK-4400 Kalundborg

Kristensen, Johannes Kjeldstrup Skovvej 69A DK-2920 Charlottenlund

Kristensen, Mette Espe Vestergade 73 DK-6270 Tønder

Kristensen, Ove Hudklinikken Bredgade 50 DK-4400 Kalundborg

Kromann, Niels Ellekildehavevej 16 DK-3140 Ålsgårde

Kroon, Susanne Hostrups Have 24, 3th DK-1954 Frederiksberg C

Lange, Kamma Østerbrogade 53, 3 DK-2100 Köpenhamn Ø

Larsen, Allan Örnäsv 87 S-302 40 Halmstad

Larsen, Christian Grønhøj Rislundvej 7 DK-8240 Risskov

Larsen, Finn Schultz Fælledsvej 42 DK-7000 Fredericia

Larsen, Frederik Grønhøj Mikkelborg Allé 72 DK-2970 Hørsholm

Larsen, John Carl Grantoftevej 18 DK-3500 Værløse

Larsen, Poul Ølholm Jacob Adelsborgs Alle 30 DK-8240 Risskov

Laurberg, Grete Klostermarken 39 DK-9000 Aalborg

63

Lauritzen, Thomas Edgar Pile Alle 17A, 2th DK-2000 Fredriksberg

Laursen, Ruth Stoltze Dronninggårds Allé 83 DK-2840 Holte

Lindberg, Lena Lundåsvägen 12 B S-502 60 Borås

Lindskov, Rune Stockholmsvej 41B DK-3060 Espergærde

Lisby, Steen Solvej 2, 1 th DK-2000 Frederiksberg

Lissau, Bodil* Langagervej 3, 1 tv c/o Jonsson DK-2500 Valby

Lomholt, Hans* Skelbækvej 31 DK-8240 Risskov

Lorentzen, Henrik Frank* Herlufsvænge 17, St.th. DK-4700 Næstved

Løland, Einar Odd Brønderslevvej 28 DK-9900 Frederikshavn

Menné, Torkil Baunegårdsvej 57 DK-2900 Hellerup

Midelfart, Kjell Køpmannsgatan 31 N-7011 Trondheim

Mikkelsen, Flemming Les Jardins de Farnese-E2 470, Route de Cagnes FR-06140 Vence

Mikkelsen, Henrik Ingemann Ekenæsvej 34 DK-2850 Nærum

Molin, Lars Hudkliniken Regionssjukhuset S-701 85 Örebro

Mrowietz, Ulrich Hautklinik der Univ. Kiel Schittenhelmstr.7 D-24105 Kiel

Munkvad, Jan Mikael Ahlgade 30A DK-4300 Holbæk

Munkvad, Steffen Mundedammen 7B Allerslev DK-4320 Lejre

Möller, Halvor Hudkliniken Malmö Allmänna sjukhus S-214 01 Malmö

Møller, Rigmor Andersen Oppesundbyvej 1 Sundbylille DK-3600 Frederikssund

Nagyajta, Elisabeth Darko de Lundtoftevej 277

DK-2800 Lyngby

Nielsen, Aksel Otkjær Bredgade 30 DK-7400 Herning

Nielsen, Eivind Bøgvad Vestervænget 13 Hjerting DK-6710 Esbjerg V

Nielsen, Mads Frederik R. Bylaugsvænget 2 DK-2791 Dragør

Nielsen, Niels Henrik Rude Vang 59 DK-2840 Holte

Nielsen, Preben Løvgreen Frederiksborgvej 70 DK-4000 Roskilde

Nielsen, Regitze Henrik Svinget 3 DK-7100 Vejle

Nielsen, Ruth Hostrups have 42, 4tv DK-1954 Frederiksberg C

Nielsen, Torben Broe Mejdal Søvej 11A DK-7500 Holstebro

Niordson-Grysgaard, Ann-Marie Folehaveparken 16 DK-2970 Hørsholm

Nissen, Birge Knudsen Hovvejen 11 DK-7800 Skive

Nissen, Jeanette* P. Schmidtsvej 10 DK-8700 Horsens

Nyfors, Allan Yrkesmedisinsk avd. Haukeland sykehus N-5021 Bergen Nyfors, Birgit Forskjønnelsen 3 N-5018 Bergen

Nørholm, Asger Antoniusvej 2 Hasseris DK-9000 Aalborg

Obitz, Erik Rene Thorvaldsensgade 15 DK-8000 Århus C

Olivarius, Frederik De Fine Granholmen 32 DK-2840 Holte

Olsen, Lene Overgaard Ryvej 19 DK-2830 Virum

Osmundsen, Poul Erik Gedebakken 10 DK-3520 Farum

Ottevanger, Vibeke Lindevangsvej 14A DK-2950 Vedbæk

Oxholm, Anne Mette Granholmen 27 DK-2840 Holte

Paulsen, Evy Kløvervænget 6, 12 DK-5000 Odense C

Pedersen, Karsten Lund Solbækvej 44 DK-9300 Sæby

Pedersen, Kristian Jacob Knudsensvej 15 DK-9200 Aalborg SV

Pedersen, Niels B Carl Krooks gata 1A S-252 25 HELSINGBORG

Peters, Kurt Friedrich Lægehuset Paradisvej 4 DK-3700 Rønne

Petersen, Ane Marie Ahm* Classensgade 67, 1th DK-2100 Köpenhamn

Petersen, Carsten Sand Rymarksvej 66

DK-2900 Hellerup Petersen, Hans Overgaard

Sjøgrensgatan 11 S-593 34 Västervik

Petri, Michael Søbakkevej 2 DK-2840 Holte

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

Pock-Steen, Bodil Skovlybakken 4 DK-2840 Holte

Poulsen, Anne-Grethe Stestrupvej 109 Stestrup DK-4360 KRK-Eskilstrup

Poulsen, Jens Søgade 16 DK-4100 Ringsted

Poulsen, Poul Asmus Englandsgade 23, 1. DK-6700 Esbjerg

Rasmussen, Hanne H. Boje Langelinie 157 DK-5230 Odense M

Rasmussen, Kaj A. Svendborgsvej 4 DK-6000 Kolding

Rasmussen, Lars Pind Buen 3, 1, P.O. Box 434 DK-6000 Kolding

Rasmussen, Ole Gowertz Magnoliavej 15 DK-8260 Viby J

Ravnborg, Lisbeth Reymann Furesø Parkvej 27 DK-2830 Virum

Reymann, Flemming Parkovsvej 30B DK-2820 Gentofte

Ring, Johannes Derm Klin der Tech Univ Biedersteinerstr. 29 D-80802 München

Risum, Gunver Furesøvej 5 DK-2830 Virum

Roed-Petersen, Jytte Hvidegårdsparken 55 DK-2800 Lyngby

Roesdahl, Kresten Kraghsvej 1 DK-9800 Hjørring

Rorsman, Hans Hudkliniken Lasarettet S-221 85 Lund

Rosman, Niels Bukkardalen 5 Gadevang DK-3400 Hillerød

64

Rothenborg, Hans Walter Svanemøllevej 53 DK-2900 Hellerup

Rønnevig, Jørgen Richard Lersolveien 22 N-0875 Oslo 8

Schmidt, Lena Ejbye Bretagnevej 34B DK-3100 Hornbæk

Schou, Marie Notvägen 9 S-711 35 LINDESBERG

Schønning, Leif Piletoften 3 DK-2630 Taastrup

Secher, Lena Vibeke Amagerbrogade 18 DK-2300 Köpenhamn S

Seier, Kirsten Gothersgade 157, ST DK-1123 Köpenhamn K

Serup, Jørgen Ingeborgvej 42 DK-2900 Hellerup

Sindrup, Jens Hein Frederiksberg Allé 32, 4.th DK-1820 Frederiksberg C

Sjølin, Knud-Erik Fortunfortvej 4B DK-2800 Lyngby

Skov, Lone Frydenlund Park 30 DK-2950 Vedbæk

Skoven, Inger Grete Svenstrupvænget 5F DK-5260 Odense S

Skovgaard, Gunhild R. Lange Kathrinevej 14 DK-2900 Hellerup

Snitker, Gerda Florian Skovlybakken 25 DK-2840 Holte

Sommerlund, Mette* Jørgen Brønlundsvej 14 DK-8200 Aarhus N

Spaun, Eva Patologiska inst. Aalborg Sygehys DK-9100 Aalborg

Staberg, Bent Geelsvej 23 DK-2840 Holte Stahl, Dorrit Gammel Hovedgade 6 B DK-2970 Hørsholm

Stangerup, Maja Pouline Slettebjerget 83 DK-3400 Hillerød

Steinkjær, Bjarte Poliklin, Sentr. sj. Rogaland Armauer Hansensv. 20 N-4011 Stavanger

Stender, Ida-Marie Tonysvej 27 DK-2920 Charlottenlund

Stenderup, Jørgen Hjortholm Allé 17A DK-2400 Köpenhamn NV

Storrs, Frances* Univ Health, 3181 SW Jac Portland, OR 97201

Svejgaard, Else Lyngsøe Skovvang 67 DK-3450 Allerød

Svendsen, Inge Borup Lerbækvej 4 DK-2830 Virum

Svensson, Åke Norreg 17 S-289 00 Knislinge

Søderberg, Ulla Tegelbakken 17 Skåde DK-8270 Højbjerg

Søgaard, Helmer P. Heises vej 4 DK-8000 Århus C

Søltoft, Peter Haldor Laxness Vej 5 DK-9220 Aalborg

Sölvsten, Henrik Haraldsgade 15 DK-8260 Viby J

Søndergaard, Jørgen Dept of Dermatology Mafraq Hospital PO Box 2951, Abu Dhabi

Sørensen, Dennis John Kongensgade 30A, ST DK-4800 Nykøbing F

Tegner, Eva Hudkliniken Lasarettet S-221 85 Lund Ternowitz, Thomas Fuglemosevej 3A DK-8620 Kjellerup

Thestrup-Pedersen, Kristian Vestre Skovvej 3 DK-8240 Risskov

Thomsen, Henrik Klem Damgårdsvej 29 DK-2930 Klampenborg

Thomsen, Inger Birthe Mørck Rosenstandsvej 28 DK-2920 Charlottenlund

Thomsen, Kristian Borgm. Schneiders vej 76 DK-2840 Holte

Thormann, Jens Solvej 41 DK-7120 Vejle Ø

Thulin, Henning Danmarksgade 21 DK-6700 Esbjerg

Tissot, Jytte Sjølundsparken 28 DK-3150 Hellebæk

Traulsen, Jette Brandt Strandvejen 177 DK-2900 Hellerup

Ullman, Susanne Halsskovgade 2,5, Lejl. 504 DK-2100 Köpenhamn Ø

Unna, Paul Jacob Tækkerløkke 18 DK-6200 Aabenraa

Wad, Susanne* Ellesøpark 11 DK-2950 Vedbæk

Wadskov, Svend Chr. Winthers vej 18 DK-4700 Næstved

Walsøe, Ida Sylvia Mars Allé 98 DK-2860 Søborg

Wanscher, Birgitte c/o Kobberbøl-Jensen Kamstrupvej 105B DK-2610 Rødovre

Warming, Jørgen Løvenborgsvej 18 DK-4420 Regstrup

Veien, Niels Kren M.A. Schultz vej 17 DK-9000 Aalborg Weismann, Kaare Derm.-Ven Afd. Bispebjerg Hospital DK-2400 Köpenhamn NV Østerlind, Anne Lucja

Slotsgade 14A

DK-3400 Hillerød

G.W.

Wendelboe, Peter Johannes Ewalds vej 93 DK-8230 Åbyhøj

Verdich, Jesper Uldall Bogfinkevej 24 Kraghave DK-4800 Nykøbing F

Vesterager, Lene Kvædevej 99 DK-2830 Virum

Wildfang, Inger Louise Marselisvej 21 DK-8000 Århus C

Vissing, Susanne Fløistrup Hyrebakken 4 DK-3460 Birkerød

Witmeur, Olaf Frugtparken 21 DK-2820 Gentofte

Wolff-Snedorff, Annette* Nordre Strandvej 186 DK-3140 Ålsgårde

Worm, Anne Marie Grumstrupsvej 18 DK-2900 Hellerup

Wulf, Hans Chr. Olsen Bindesbøllsvej 23 DK-2920 Charlottenlund

Wærsted, Atle Höjbjerggårdsvej 22 DK-2840 Holte

Zachariae, Bobby Psykolog.inst., Århus Univ DK-8240 Risskov

Zachariae, Claus Otto Nyhavn 8, st DK-1051 Köpenhamn K

Zachariae, Hugh Ildervej 40 DK-8270 Højbjerg

Ågren, Magnus* Sørens Alle 4 B DK-3050 Humlebæk

Øhlenschlæger, Kirsten Vingårds Allé 43 DK-2900 Hellerup

Østerbye, Poul Overmarken 18 Haldrup DK-8700 Horsens



Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

FINLAND

Aalto-Korte, Kristiina Ulvilantie 11 a D 6 FIN-00350 HELSINKI Aaltonen, Tellervo Särkäntie 11 as 2 FIN-80150 JOENSUU

Aarnivuo, Teuvo Paimelantie 385 FIN-17110 KALLIOLA Abdulkareem, Hasan Hanneksenrinne RJ 144 FIN-60220 SEINÄJOKI

Ackermann, Leena Kalevankatu 54 C 31 FIN-00180 HELSINKI Ahokallio, Arja Terholantie FIN-04430 JÄRVENPÄÄ

Ahokas, Terttuliisa Munkinpolku 10 FIN-00330 HELSINKI Airola, Kristiina Soukanlahdentie 3A 7 FIN-02360 ESPOO*

Alanko, Kristiina 2, rue F, Baclesse L-1208 Alatalo, Elina Hauhianranta 18 FIN-54915 Saimaanharju

Antti, Jouko Peikkovuorentie 5 FIN-07940 LOVIISA

Aulamo, Sari* Valto Käkelän katu 8 as 3 FIN-53130 Lappeenranta

Autio, Pekka Keskussotilassairaala PL 50 FIN-00301 HELSINKI

Blomqvist, Kirsti Hopeasalmenranta 5 B FIN-00570 HELSINKI

Brandt, Heikki Liljatie 36 F 27 FIN-01300 VANTAA

Cajanus, Suvi Sofianlehdonkatu 9 A 7 FIN-00610 Helsinki

Dammert, Kai Talvitie 20 A FIN-90530 OULU

Erkko, Pekka Tontunmäentie 22E FIN-02200 ESPOO Eskelinen, Aarno FIN-56440 POHJALANKILA

Estlander, Tuula Mäntypaadentie 13 as 5 FIN-00830 HELSINKI

Fagerlund, Varpu-Liisa Putolankatu 12 FIN-24240 SALO

Forsten, Yrsa Aurakatu 20 A 5 FIN-20100 TURKU

Fräki, Jorma Kuntokuja 6 B 8 FIN-70200 KUOPIO

Förster, Johanna Hakolahdentie 5 A 1 FIN-00200 HELSINKI*

Förström, Lars Soukanlahdentie 3 B 1 FIN-02360 ESPOO

Geier, Ludwig Koivikkotie 19 B FIN-40250 JYVÄSKYLÄ

Granlund, Håkan Turjantie 14 B FIN-00610 HELSINKI

Grönroos, Mari Kapteeninkatu 7 B 34 FIN-00140 Helsinki

Haapasaari, Kirsi-Maria OYKS Ihotautiklinikka FIN-90220 OULU

Hagman, Johanna* V Citta di Castello 27/13 IT-00191 ROMA

Hannuksela, Matti Laihiankatu 6 FIN-53100 Lappeenranta

Hannuksela-Svahn, Anna Westye Egebergs gate 1C N-0177 OSLO

Happonen, Hannu-Pekka Koivusaarentie 8 A 1 FIN-00200 HELSINKI

Harvima, Ilkka Annikintie 2 B 32 FIN-70500 KUOPIO

Harvima, Rauno Saarijärventie 2 B 46 FIN-70460 KUOPIO

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

66

Hasan, Taina Silmäkkeenkatu 5 E 16 FIN-33560 TAMPERE

Havu, Väinö Käenpiiankuja 2 FIN-20600 TURKU

Heikkilä, Elina Yliopistonkatu 1 E 66 FIN-20100 TURKU

Heikkilä, Hannele Lepolantie 77 B FIN-00660 HELSINKI

Heino, Timo Keskuskatu 14 A 10 FIN-48100 KOTKA

Helander, Inkeri Vihtiläntie 4 FIN-21200 RAISIO

Helanen-Mikkola, Sirkka Unioninkatu 45 A 18 FIN-00170 HELSINKI

Helle, Juha Rantapolku 3 FIN-00330 HELSINKI

Hervonen, Kaisa Selininkatu 1 E 17 FIN-20100 TURKU

Hietanen, Anita Bredantie 6 F 31 FIN-02700 KAUNIAINEN

Hiltunen-Back, Eija Takametsäntie 17B FIN-00620 HELSINKI

Hjerppe, Mika* Ahkionkuja 18 E 10 FIN-33580 TAMPERE

Hollmén, Antero Kanervatie 14 FIN-70280 KUPIO

Horsmanheimo, Maija Helenankuja 7 A FIN-02700 KAUNIAINEN

Huotari-Orava, Riitta Vuorenmaanrinne 11D 11 FIN-60220 SEINÄJOKI

Huovinen, Saara Nikkarmäenkuja 1 FIN-20380 TURKU

Huttunen, Maria Sepontie 3 G 38 FIN-02130 ESPOO

66

Hyry, Heli Mannerheimintie 132B 44 FIN-00270 HELSINKI Hyvärinen, Maija Vanhatie 30 A 11 FIN-15240 LAHTI

Hyödynmaa, Ritva Lokinkuja 4 A 19 FIN-80100 JOENSUU

Hämäläinen, Tiina* Setterinkuja 5 FIN-33580 TAMPERE

Härö, Sakari Kallionlaita 3 FIN-02610 ESPOO

Höök-Nikanne, Johanna Lohjantie 54 B FIN-03100 Nummela

Immonen, Aila Nisulankatu 11 FIN-94100 KEMI

Isoherranen, Kirsi Kuusikallionkuja 3 A 3 FIN-02210 ESPOO

Itkonen-Vatjus, Raija Tapaninaho FIN-90900 KIIMINKI

Jackson, Päivi Uuvenperäntie 15 FIN-90810 KIVINIEMI

Jansén, Christer Yliopistonkatu 24 D 59 FIN-20100 TURKU

Jeskanen, Leila Viherniemenkatu 1 A 7 FIN-00530 HELSINKI

Jolanki, Riitta Työterveyslaitos Topeliuksenkatu 41 a A FIN-00250 HELSINKI

Juhela, Jukka Seponk. 12 FIN-29200 HARJAVALTA

Juvakoski, Timo Laurinlahdentie 15 D FIN-02320 ESPOO

Järveläinen, Reijo Ihotautipoliklinikka Etelä-Pohjanmaan keskussairaala FIN-60220 SEINÄJOKI

Järvikallio, Anitta Lakkapolku 2 C FIN-702801 KUOPIO

Järvinen, Kirsi-Marjut Dept of Dermatology Skin & Allergy Hospital FIN-00250 Helsinki Järvinen, Marketta Joonaksentie 4 C FIN-00370 HELSINKI

Järvinen, Timo Vikiöntie 34 FIN-90650 OULU

Kajanne, Heikki Pohj. Rautatienk. 17 C 20 FIN-00100 HELSINKI

Kalimo, Kirsti Honkatie 37 FIN-20540 TURKU

Kallioinen, Matti Ketokatu 24 FIN-90140 OULU

Kalliomäki, Pekka Hannulankatu 11 A 5 FIN-33580 TAMPERE

Kaminska, Renata A. Chydeniuksenkatu 7B FIN-67100 Kokkola

Kanerva, Lasse Työterveyslaitos Topeliuksenk. 41 aA FIN-00250 HELSINKI

Kanervo, Marja Alankotie 54 FIN-04400 JÄRVENPÄÄ

Kaprio, Leena Turuntie 132 FIN-02700 Kaunainen

Karenko, Leena Miilutie 4 FIN-00670 HELSINKI

Karesoja, Leena Rekikuja 8 FIN-00950 HELSINKI

Kariniemi, Arja-Leena Maisterintie 16 B FIN-02700 KAUNIAINEN

Karonen, Tiina Mannerheimintie 69 A 18 FIN-00250 Helsinki

Karppinen, Ari Höytämöntie 59 FIN-33880 Lempäälä

Karppinen, Liisa Kanneltie 21 FIN-00420 HELSINKI

Kartamaa, Matti Lahnatie 7 B 5 FIN-02170 ESPOO Karvonen, Jaakko Jaakonkuja 1 F 7 FIN-90230 OULU

Karvonen, Seija-Liisa Jaakonkuja 1 F 7 FIN-90230 OULU

Kauppinen, Kirsti Liljassaarentie 3 B 6 FIN-00340 HELSINKI

Kause, Laura* Munstenpellonkatu 4 A 4 FIN-20740 TURKU

Kekki, Outi-Maria Nallekarhuntie 34 FIN-36100 KANGSALA

Kero, Matti K-H KS Ihotautiosasto FIN-13530 HÄMEENLINNA

Keski-Oja, Jorma Osuuskunnantie 45 FIN-00660 HELSINKI

Kianto, Ursula Pietarinkatu 12 B 36 FIN-00140 HELSINKI

Kiistala, Raija Kulmakatu 1 B 4 FIN-00170 HELSINKI

Kiistala, Urpo Kulmakatu 1 B 4 FIN-00170 HELSINKI

Kilpiö, Olavi Niemenmäenkuja 1 B FIN-00350 HELSINKI

Kinnunen, Tuula Karinkannantie 51 FIN-90800 OULU

Kiraly, Csaba Julinintie 6 J FIN-53200 Lappenranta

Kivekäs, Kristiina* Juuruksentie 1 FIN-90240 OULU

Kivirikko, Sirpa* Tilkankatu 39 C 9 FIN-00300 HELSINKI

Koistinen, Anna-Maija Kaskilankuja 3 as 1 FIN-20540 TURKU

Komulainen, Mirja Oikokatu 2-4 as. 1 FIN-60100 SEINÄJOKI Kortekangas-Savolainen, Outi Huvilakatu 10 FIN-20720 TURKU

Kostiainen, Minna Nuotiokatu 3 FIN-15840 LAHTI

Kotovirta, Marja-Liisa Mäensyrjä 10 A FIN-02160 ESPOO

Koulu, Leena Sirkkalankatu 17 C FIN-20700 TURKU

Kousa, Merja Ihotautiosasto FIN-40930 KINKOMAA

Kuittinen, Kaarina Tontunmäentie 34 A FIN-02200 ESPOO

Kuokkanen, Kirsti Pastellintie 13 FIN-37630 VALKEAKOSKI

Kuoppamäki, Leena länsipuisto 18 B 31 FIN-28100 PORI

Kähäri, Veli-Matti Karjakuja 48 C 12 FIN-20540 TURKU Laakso, Liisa

Saaruantie 6 A 1 FIN-96440 ROVANIEMI

Lahti, Arto Hanhikaari 12 B FIN-90240 OULU

Lahtinen, Marjo-Riitta* Pajulahdentie 18 FIN-70260 KUOPIO

Laine, Arja Välitalontie 110 FIN-00660 HELSINKI

Lammintausta, Kaija Meltoistentie FIN-20900 TURKU

Lange, Terhikki Liisankatu 15 A 10 FIN-00170 HELSINKI

Langen, Marja Honkitie 2 FIN-91900 LIMINKA Lappalainen, Katriina

Larmi, Eva Jaakonpolku 7 B FIN-90230 OULU

67

Lassus, Allan Iso Roobertinkatu 8 A 7 FIN-00120 HELSINKI

Latvala, Kaija* Villilänkatu 6 E 20 FIN-33300 TAMPERE

Lauerma, Antti HYKS,Iho-ja Allergiasairaala Meilahdentie 2 FIN-00250 HELSINKI

Lauharanta, Jorma Kallvikintie 35 B FIN-00980 HELSINKI

Laukkanen, Arja Ahkiotie 2 A 27 FIN-70200 KUOPIO

Laukkanen, Kleta Friedrichstraße 12 DE-06667 WEISSENFELS

Laukkanen, Sakari Kirkkoveräjäntie 4 FIN-33950 PIRKKALA

Laulainen, Matti Lankkukatu 4 FIN-33400 TAMPERE

Launis, Juhani Holmanranta 21 FIN-01800KLAUKKALA

Laurikainen, Leena Ruskontie 118 FIN-21250 MASKU

Lavikainen, Reima* Punttelintie 7 FIN-48310 KOTKA

Lehmuskallio, Eero Kuusikkotie 18 A FIN-01380 VANTAA

Lehtinen, Katriina* Ranta-Kastellintie 14 F 3 FIN-90230 OULU

Leinonen, Marjatta Sivakkatie 11 FIN-90230 OULU

Leivo, Tomi* Melkonkatu 1 B 51 FIN-00210 HELSINKI

Linnavuori, Kimmo* Steniuksentie 25B 8 FIN-00320 HELSINKI

Lintu, Päivi* Mestarinkatu 7 as 26 FIN-20810 TURKU

Liutu, Mervi* TYKS Ihotautuklinikka Kiinanmyllynkatu 4-8 FIN-20520 TURKU

Lund, Sirkka Päkäräntie 14 FIN-28220 PORI

Lähteenmäki, Marja-Terttu Helsingin Lääkärikeskus Mannerheimintie 12 B FIN-00100 HELSINKI

Majamaa, Heli TAYS, Ihot. Pkl PL 2000 FIN-33521 Tampere

Malanin, Ken Tanhuantie 7 D FIN-53920 Lappeenranta

Mashkilleyson, Nikolai Fredrikinkatu 28 B 24 FIN-00120 Helsinki

Mattila, Jaana Pallaksentie 13 A FIN-65200 VAASA

Mattila, Leena Väinöläntie 31 FIN-21100 NAANTALI

Mattila, Rauni Ihotautiklinikka KYKS FIN-70210 KUOPIO

Mattila, Timo Kukkukuja 2 D FIN-90810 KIVINIEMI

Mattila, Ulla Hännikönk 57 FIN-20600 TURKU

Milán, Tiina* Uittamontie 16 as 3 FIN-20810 TURKU

Molander, Gerd Rönnträdsvägen 7 D8 FIN-02940 ESBO

Montonen, Outi Tahkokuja 7 A FIN-02760 Espoo

Muroma, Ali Huvilakatu 25 A 3 FIN-00150 HELSINKI

Mustakallio, Kimmo K. Välikatu 2 B 19 FIN-00170 HELSINKI Mustonen, Marja-Terttu Kyylintie 4 FIN-07230 MONNINKYLÄ

Mynttinen, Synnöve Koskenniskantie 5D39 FIN-48400 KOTKA

Mäkelä, Leeni* Bratislavankatu 1 F 72 FIN-20320 Turku

Mäki, Airi Kauppapvistikko 36 A 12 FIN-65100 VAASA

Mörtenhumer, Minna Matruusinkatu 11 B FIN-67100 Kokkola

Neittaanmäki, Heikki Savonlinnan keskussairaala Ihotautien poliklinikka FIN-57170 SAVONLINNA

Niemi, Kirsti-Maria Yrjö Liipolantie 5 FIN-02700 KAUNIAINEN

Niinimäki, Aila Ihotautiklinikka OYKS FIN-90220 OULU

Nissi, Tuula Vahdontie 489 FIN-21290 RUSKO

Nuutinen, Pauliina* Museokatu 37 A 18 FIN-00100 HELSINKI

Ohela, Kyllikki Kimpisenkatu 12 FIN-53100 Lappenranta

Oikarinen, Aarne Ihotautiklinikka OYKS FIN-90220 OULU

Oksman, Risto Rätiälänkatu 18 as 2 FIN-20810 TURKU

Paavilainen, Outi Tiilentekijänkatu 5 B 21 FIN-20810 TURKU

Pajarre, Reino Ihotautipoliklinikka Satalinnan sairaala FIN-29230 SATALINNA

Palatsi, Riitta OYKS, Ihotautiklinikka FIN-90220 OULU

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

Palosuo, Kati Koivuviita 14 E 22 FIN-02130 ESPOO

Panelius, Jaana Heikinniementie 6 as. 4 FIN-00250 HELSINKI

Partanen, Taina Rovastinkatu 16 B 16 FIN-70600 KUOPIO

Paukkonen, Kari Päivärinteentie 14 FIN-70940 JÄNNEVIRTA

Pekanmäki, Kalle Uudenmaankatu 31 F 25 FIN-00120 HELSINKI

Pekola, Pia Saunalahdentie 13 B 18 FIN-00330 HELSINKI

Peltola, Mari-Out Kaskipuunkaari 6 H 23 FIN-02340 ESPOO

Peltonen, Juha Rakuunatie 16 FIN-20720 TURKU

Peltonen, Leena Ohrakatu 20 FIN-20740 TURKU

Peltonen, Sirkku Rakuunatie 16 FIN-20720 TURKU

Perko, Ritva-Liisa Ruukinp. 5 FIN-70910 VOUORELA

Perttilä, Leena Ylä-Fallin kuja 4 FIN-690 Helsinki

Pesonen, Liisa Kuhatie 12-18 A 2 FIN-02170 ESPOO

Petäys, Tuula* PL 123 FIN-02401 KIRKKONUMMI

Pitkänen, Sari Kehrääjäntie 18A FIN-02660 ESPOO

Plosila, Mikko Runeberginkatu 29 B 60 FIN-00100 HELSINKI

Poikonen, Sanna* Sauvakatu 4-6 D 12 FIN-33580 TAMPERE

Porki, Irmeli Yhdyskatu 8 A 13 FIN-53100 Lappeenranta Puolakka, Tuula Kissanmaankatu 8 A 5 FIN-33520 TAMPERE

Puolijoki, Tuija Pohjalaistenraitti 7 FIN-60200 SEINÄJOKI

Puska, Pirkko Meritullinkatu 15 E 51 FIN-00170 HELSINKI

Raatikainen, Maria* Jyrkänne 3 FIN-01120 VÄSTERSKOG

Raekallio, Eeva Linnankatu 13 B 13 FIN-20100 TURKU

Raitala-Niemi, Riitta Tanhuankatu 6 B 4 FIN-20540 TURKU

Raitio, Anina* Lehtoranta 9 FIN-90500 OULU

Raitio, Hanna* Harjutie 12 A FIN-02730 ESPOO

Ranki, Annamari Sibeliuksenkatu 11B 28 FIN-00250 HELSINKI

Rantanen, Tapio Rautatienkatu 6 A 21 FIN-15100 LAHTI

Raudasoja, Riikka Muuttolantie 27 FIN-41340 Laukaa

Ravanti, Laura* Linnankatu 33 D 52 FIN 20100 TURKU

Rechardt, Leena Luoteisväylä 33 G FIN-00200 HELSINKI

Reitamo, Sakari HYKS, Iho-ja Allergiasairaala Meilahdentie 2 FIN-00250 HELSINKI

Remitz, Anita Pitkänsillanranta 5 B 32 FIN-00530 HELSINKI

Reunala, Timo Liutuntie 15 B 9 FIN-36240 KANGASALA 4

Reunanen, Niina Kotimäenkatu 29b A 4 FIN-20540 TURKU Sandell-Laaksonen, Vappu Palotie 27 FIN-02760 ESPOO

Savolainen, Leena Kirvesmiehentie 16 FIN-81 100 KONTIOLAHTI

Schreck-Purola, Ilona Hongisto FIN-08500 LOHJA as

Siberg, Rauha-Lea Kristianinkatu 5 A 3 FIN-00170 Helsinki

Sihvonen, Tuula Kalliokuja 6 FIN-40900 SÄYNÄTSALO

Sillantaka, Irmeli Lomatie 20 FIN-30600 PÄLKÄNE

Snellman, Erna Rautatienk. 6 A 21 FIN-15100 LAHTI

Soini, Marja Tiilimäki 5 C FIN-00330 HELSINKI

Somerma, Simo Kirkkokatu 1 A 8 FIN-18100 HEINOLA

Somerola-Kunnari, Kirsti Vastarannankatu 35 FIN-33610 TAMPERE

Sonck, Carl Eric Hämeentie 2 A 20 FIN-00530 HELSINKI

Sten, Marja Malikkakuja 14 FIN-78870 VARKAUS

Stenberg, Anneli Relanderinaukio 2 F 44 FIN-00570 HELSINKI

Strand, Ritva Värtöntie 2 A 1 FIN-90230 OULU

Stubb, Sakari Poijutie 22 A 1 FIN-00980 HELSINKI

Suhonen, Raimo Kalevankatu 22 FIN-50100 MIKKELI

Suomela, Sari* Paneliantie 29 B FIN-00940 Helsinki

Suramo, Marja-Liisa Ylisrinne 1 as 29 FIN-02210 ESPOO Riekki, Riitta* Orsitie 1 A 1 FIN-90240 OULU

Riepponen, Senja Lana 6 FIN-40520 JYVÄSKYLÄ

Rinne, Eliisa Veräjänkorva 3 FIN-00650 HELSINKI

Rosenblad, Eija Dalagatan 78A S-113 24 Stockholm

Rostila, Timo Vahverotie 10 B FIN-02730 ESPOO

Rouhunkoski, Sirkka Hietalahdenranta 15 A FIN-00180 HELSINKI

Räsänen, Liisa Maljalahdenkatu 14 C 29 FIN-70100 KUOPIO

Saarelainen, Ilkka O. Pohjoisranta 6 A 1 FIN-00170 HELSINKI

Saari, Salli Letonlaidantie 7 FIN-90440 KEMPELE

Saari, Seppo Viitakantie 10 FIN-20320 TURKU

Saarialho-Kere, Ulpu Lahnatie 1 A FIN-02170 ESPOO

Saarinen, Jari Kalliokatu 31 FIN-70600 KUOPIO

Saarinen, Kari Consultant Dermatologist Tawan Hospital P.O.BOX:15258 Al Ain, Abu Dhabi

Saarni, Heikki Kotimäenkatu 16 FIN-20540 TURKU

Saksela, Olli Morbacka FIN-02430 MASALA

Salminen, Mirja-Liisa Auvaisberg FIN-20760 PIISPANRISTI

Salo, Heikki Ihotautipoliklinikka Lapin KS FIN-96101 ROVANIEMI Susitaival, Päivikki Kuopion aluetyöterveyslaitos PL 93 FIN-70701 KUOPIO

Sysilampi, Marja-Liisa Ribäcksvägen 1039 FIN-66140 ÖVERMALAX

Särkkä, Sylvi Kasabergsvägen 12 D 14 FIN-02700 GRANKULLA

Söderqvist, Sirkku Koskelantie 25 B 18 FIN-00610 HELSINKI

Taipale, Anja Ulvilantie 21 C 28 FIN-00350 HELSINKI

Talve, Lauri Kontionk. 3-5 A FIN-20760 PIISPANRISTI

Tammi, Raija Karhonsalmi FIN-71130 KORTEJOKI

Tarvainen, Kyllikki Kovelipolku 12B FIN-00430 HELSINKI

Tasanen, Kaisa Oulun yliopisto Ihotautien osasto FI-90220 Oulu

Taskila, Kati* Rovastinkatu 7 C 26 FIN-70600 KUOPIO

Timonen, Kaisa Hämeentie 30 E 56 FIN-00530 HELSINKI

Tuohimäki, Jorma Cygnaeuksenkatu 10 B 16 FIN-00100 HELSINKI

Tuomi, Marja-Leena Laalahdenkatu 6 K FIN-33560 TAMPERE

Turjanmaa, Kristiina Palomäentie 7B

FIN-33230 TAMPERE Tuukkanen, Inga

Kristianinkatu 5 A 7 FIN-00170 HELSINKI Työlathi, Harri*

Lyhdekuja 3 E FIN-02200 ESPOO Uggeldahl, Paul-Erik

69

Suvikatu 8 FIN-80200 JOENSUU Unnérus, Viveca Marieberg gård FIN-10210 INGÅ

Uurasmaa, Tutta* Soinimäentie 65 FIN-21110 NAANTALI

Vaalamo, Maarit Urkupillintie 7 I 86 FIN-00420 Helsinki

Vaalasti, Annikki Huhmarenkatu 13 FIN-33560 TAMPERE

Vainio, Eeva Jalustinkatu 7 E 42 FIN-20880 TURKU

Vaismaa, Ulla-Kaija Palomäentie 32 A 3 FIN-33230 TAMPERE

Valkiala, Seija Kylynkatu 14 FIN-33730 Tampere

Valle, Sirkka-Liisa Kammantekijäntie 2 C FIN-00640 HELSINKI

Valmari-Kankkunen, Saara Museokatu 17 A 4 FIN-00100 HELSINKI

Varjonen, Elina Munkinpolku 18 as 2 FIN-00330 HELSINKI Viikari, Marjukka FIN-20540 TURKU

Viljanen, Pertti* Miniäntie 11 FIN-28330 PORI

Vimpari-Kauppinen, Leena Kirkkokatu 8 A 7 FIN-87100 KAJAANI

Virolainen, Susanna Jollaksentie 44 A FIN-00850 HELSINKI

Virrankoski, Terttu Bredantie 10 A 2 FIN-02700 KAUNIAINEN

Virtanen, Erkki Raatihuoneenkatu 20A 30 FIN-68620 PIETARSAARI

Visa, Kirsti Pollentie 14 FIN-13500 HÄMEENLINNA

Wuokko, Pentti Lönnrotinkatu 7 B 14 FIN-00120 HELSINKI

Vuorela, Anna-Maija Kulosaaren puistotie 38 as 3 FIN-00570 HELSINKI

Vuorio, Tuula Seikonkatu 1 as. 22 FIN-20610 TURKU

Väkevä, Liisa Rakovalkeantie 15 B 5 FIN-00670 Helsinki

Väänänen, Antti Kauppaseurantie FIN-90520 OULU

Väätäinen, Niilo Ilvolankatu 45 FIN-74120 IISALMI

Ylitalo, Leena Houvarinkatu 6 B 5 FIN-33270 TAMPERE

ICELAND

Baldursson, Baldur T Durovägen 102 S-806 28 GÄVLE

Davidsson, Steingrimur Hateigsvegur 1 IS-105 Reykjavik

Gudgeirsson, Jon Hrisholt 2 IS-210 Gardaber

Johannesdottir, Hanna Safamyri 75 IS-108 Reykjavik

Mooney, Ellen Nordurvangur 30 IS-220 Hafnafjördur

Olafsdottir, Elin* Department of Dermatology University Hospital IS-105 Reykjavik

Olafsson, Jón Hjaltalin Hrauntun v. Alftanesveg IS-210 Gardabaer

Pálsdóttir, Rannveig Noatun 31 IS-105 Reykjavik

Sigurgeirsson, Bárdur Háaberg 39 IS-220 Hafnafjördur

Steinsson, Jon Thrándur Blesugróf 16

Sveinsson, Birkir Mithskógar 1 IS-225 Bessastatharhrepp

Thorhallsdottir, Helga Hrönn Selbraut 34 IS-170 Seltjarnarnes

Torisdottir, Kristin Hólahjalli 12 IS-200 Kopavogur

Valdimarsson, Reynir Höfdalid 15 IS-603 Akureyri

NORWAY

Aandahl, Dag* Otto Ruges vei 80 N- Østerås

Aarebrot, Steinulf Hudavd. Haukeland sykehus N-5021 Bergen

Aarset, Harald Avd. for patologi Regionsykehuset N-7006 Trondheim

Aarstein, Kjetil Hudavdelingen Ullevål sygehus N-0407 Oslo

Aase, Karl Blekholmsterassen 11 S-111 64 STOCKHOLM

Aulie, Line A. Kirkevej 64B N-0364 Oslo

Austad, Joar Hudavdelingen Rikshospitalet N-0027 Oslo

Bachmann, Ingeborg* Hudavdelningen Haukelands sykehus N-5021 Bergen

Barlinn, Christa Olafiaklinikken Olafigangen 7 N-0188 Oslo

Benestad, Bjørg Prof. Kohtsv. 84 N-1320 Stabekk

Bengtsson, Helge Moss hudlegekontor Jeløygt. 8 N-1500 Moss

Bjerke, Jens Roar Hudavdelingen Ullevål sykehus N-0407 Oslo

Bjørnstad, Roar Hjørnungvn. 6 N-0375 Oslo

Bohmann, Peter 8000 Bodø, Linderud C. Erich Mogenssonsv. 38 N-0598 Oslo

Bonesrønning, Jon Helge Hudavdelingen Regionsykehuset N-7006 Trondheim

70

Borup, Mårten Hudpolikl., Revmatismesykehuset Karmsundsgt. 134 N-5500 Haugesund

Braathen, Lasse R. Dermatologische Klinik Inselspital CH-3010 Bern

Braun, Rosemarie Hudavd., Regionsykehuset, Postboks 1 N-9038 Tromsø

Bull Berg, Jacob Landøystranda 6 N-1360 Nesbru

Bø, Kristine Hudavdelingen Haukelands sykehus N-5021 Bergen

Christensen, Eidi

Trondheim

Christensen, Ole B Villavägen 21 S-216 11 MALMÖ

Dahle, John S. Sandesundsvn. 23 Dermatologisk poliklinikk N-1704 Sarpsborg

Dalaker, Morten Avd. for patologi Regionsykehuset N-7006 Trondheim

Danielsen, Helge Utfrågt. 19 N-5700 Voss

Dotterud, Lars Kåre Nymosvingen 2 N-2600 Lillehammer

Ek, Lorens* EK-Stjärnborg Hagalundsvägen 30D S-302 74 HALMSTAD

Elset, Kjersti Hudlegekontoret Bullsgt. 2 A N-3100 Tønsberg

Engelsen, Arne Slettebakkveien 36 N-5030 Landås

Eriksen, Bjørn Hudavdelingen Regionsykehuset N-9038 Tromsø

Falk, Edvard S. Hudavdeln., Regionsykehuset Postboks 1 N-9038 Tromsø

Faye, Ragnar

N- Oslo

Fiskestrand, Eli Janne Hudlegekontoret Kjøpmansg. 28 N-7500 Stjørdal

Flint-Hansen, Henrik Bogstadsveien 51 N- Oslo

Formoe, Torill Hudavdelningen Ullevål sykehus N-0407 Oslo

Foss, Madela Hartitzalleen 18 A N-0275 Oslo

Frølich, Karin W. Hudavdelningen Haukeland sykehus N-5021 Bergen

Funk, Jürgen Dr Funk's Hudklinikk as Prinsensgate 9 N-1530 Moss

Fyrand, Ole Hudavdelingen Rikshospitalet N-0027 Oslo

Gasior-Chrzan, Barbara Hudavdelingen Regionsykehuset Postboks 4 N-9038 Tromsø

Gedde-Dahl, Tobias Rettsmed. inst.. Rikshospitalet N-0027 Oslo

Gjersvik, Petter Jensen Hudavdelningen Rikshospitalet N-0027 Oslo

Gjertsen, Bjørn Tore Hudavdelingen, Sentralsjukehuset i Sogn og Fjordane N-6900 Florø

Gjørud, Magnar Hegdehaugsvn. 36 A N-0352 Oslo Granholt, Astri Kongsveien 94 N-1177 Oslo

Gundersen, Thor Haakon den Godesvei 7 N-0373 Oslo

Haaland, Björn Bondevägen 5A S-227 64 LUND

Haavelsrud, Odd Oslo Behandlingsinst. Grensevn. 36 B N-0663 Oslo

Hafsahl, Martin Gamle Asheimsvei 37 N-4300 Sandnes

Halsos, Arne M. Olafiaklinikken, Klin for foreb.med, Postutak Grønland P.k. N-0133 Oslo

Hannuksela-Svahn, Anna* Westye Egebergs gate 1C N-0177 OSLO

Hansen, Even Th. Angellsgt. 22 N-7011 Trondheim

Hanssen, Leif Ivar Hudseksjonen, Sentralsykehuset i Møre og Romsdalen N-6026 Ålesund

Hanstad, Inger Amtmann Blomsgt. 1 N-3000 Drammen

Haug, Sidsel*

N- Oslo

Haugstvedt, Åse Hudavdelingen Ullevål sykehus N-0407 Oslo

Heir, Martin Asalvn. 1 N-0870 Oslo

Helgesen, Anne Lise O. Hudavdelingen Rikshospitalet N-0027 Oslo

Helland, Svein Hudavdelingen Haukeland sykehus N-5021 Bergen Hellgren, Lars Bronsgjutaveg 13 S-421 63 V. Frølunda

Helme, Per Boks 743 N-1755 Halden

Helsing, Per Hudavdelingen Rikshospitalet N-0027 Oslo

Hestholm, Freddy* Hudavdelningen Haukelands sykehus N-5021 Bergen

Hilleren, Per K. Hudavdelingen Fylkessjukehuset i Lærdal N-5890 Lærdal

Hohlbrugger, Herbert Langggt. 16 N-4013 Stavanger

Holm, Jan Ø. Trosterudvn. 14 N-0778 Oslo

Holsen, Dag Sollesnes Hudavdelningen Haukeland sykehus N-5021 Bergen

Holst, Tormod Hudlegekontoret Bullsgt. 2 A N-3100 Tønsberg

Holtestaul, Aslak Dragvn. 28 N- Høvik

Huldt-Nystrøm, Theis Hud-poliklinikken Innherrad sykehus N-7600 Levanger

Husebø, Åsa L. Markevn. 2 A N-5012 Bergen Norway Høviskeland, Aase N- Høvik

Ingvarsson, Gisli Hudavdelingen Regionsykehuset N-9038 Tromsø

Jensen, Ada Hjaltlandsgt. 19 N-4009 Stavanger

Jensen, Tor S. Fantoftveien 34 Blokk D N-5072 Fantoft

71

Johansen, Arne G. Bjørnefaret 24 N-1270 Oslo

Johnsen, James Køpmannsgt. 34 N-7011 Trondheim

Johnsen, Paul Otto Skippergt. 21 N-N-4611 Kristiansand

Johnsson, Margareta K. Hudavdelingen Regionsykehuset N-7006 Trondheim

Jørgensen, Hans P Valnes Storgt. 15 N-1607 Fredrikstad

Kalgaard, Ole Magne Røde Kors klinikk Frederik Stangsgt. 11-13 N-0264 Oslo

Kampman, Petra T

N- Oslo

Kavli, Gunnar Ilderveien 18 N-2400 Elverum

Kjus, Trine

N- Oslo

Koss-Harness, Dörte S.H. Lundhs Vei 7 N-0287 Oslo

Kramer, Mette Asker hudlegekontor Gamle Drammensv. 227 N-1370 Asker

Kramer, Patrick Bekkestua

Kristensen, Terje Legesenteret Lundegt. 11 N-3724 Skien

Kristiansen, Frode

N- Oslo

Krogh, Hans-Kristian Ths. Heftyesgt. 54 B N-0267 Oslo

Krohn, Susanne* PB 8100 N- Stavanger

Kråkenes, Anine G. Bergvegen 54 N-5152 Bønes

Kummels, Marianne* Hudavdelingen Fylkessjukehuset i Kristiansund N- Kristiansund

Laastad, Olav Porsgrunn Hudlegekontor Hovengasentret, Hovengagt. 35 N-3915 Porsgrunn

Langeland, Berit Thomas Heftyes Gate 37 N-0264 Oslo

Langeland, Jon Hudklinikken Postboks 8764, Youngstorget N-0028 Oslo

Langeland, Tor Fritjof Nansens Plats 6 N-0160 Oslo

Larsen, Maria*

N- Tromsø

Larsen, Tove Eeg Pat.anat. Lab. Ullevål sykehus N-0407 Oslo

Leite, Kari Stortingsgt. 28 N-0160 Oslo

Lier, Anders Storgaten 107 N-2390 Moelv

Lium, Marit S. Solhaug Einarsvei 14 B N-575 Oslo

Livden, John Karsten Teatergaten 37 N-5010 Bergen

Loven, Anne Marie Hudavdelningen Ullevål sykehus N-0407 Oslo

Lund-Hanssen, Kristin* Hudavdelingen Regionsykehuset N-7006 Trondheim

Lützow-Holm, Claus Hovsetervn. 98 N-0768 Oslo Løken, Per A. Pettersandåsen 20 N-1614 Fredrikstad

Marcusson, Jan Danav. 7A 181 31 Lidingö

Martin-Odegard, Brit Hudavdelingen Ullevål sykehus N-0407 Oslo

McFadden, Noel Volvat med. senter Postboks 5280 Majorstua N-0303 Oslo

Midelfart, Kjell Køpmannsgatan 31 N-7011 Trondheim

Moi, Harald Håkonsvej 63 N-1470 Lörenskog

Morken, Tore Hudavdelingen Haukeland sykehus N-5016 Bergen

Moseng, Dagfinn Hudavdelingen Regionsykehuset N-9038 Tromsø

Myhre, Knut Adm. Børresensv. 25 N-0281 Oslo

Mørk, Cato Hudavdelingen Rikshospitalet N-0027 Oslo

Mørk, Gro Igland Hudavdelingen Rikshospitalet N-0027 Oslo

Mørk, Nils-Jørgen Hudavdelingen Rikshospitalet N-0027 Oslo

Nielsen, Morten Brekne Hudavdelingen Regionsykehuset N-9038 Tromsø

Nilsen, Arvid Hudavdelingen Haukeland sykehus N-5021 Bergen

Nilsen, Tore Specialistläkargruppen Trädgårdsgatan 10 S-352 34 VÄXJÖ

72

Nordahl, John S Storgaten 60 N-8000 Bodø

Nordal, Eli J. Hudavdelingen Ullevål sykehus N-0450 Oslo

Nyfors, Allan Yrkesmed. avd. Haukeland sykehus N-5021 Bergen

Nyfors, Birgit Forskjønnelsen 3 N-5018 Bergen

Nyrud, Morten Trollåsveien 25 N-1414 TROLLÅSEN

Ochremenko, Pavel Henrik Wergelandsvei 19 N-4612 Kristiansand

Odu, Solveig Oskarsgatan 24 S-331 41 VÄRNAMO

Olsen, Anne Olaug Hudavdelingen Ullevål sykehus N-0407 Oslo

Olsen, Pål Derm. avd. Nordland sentralsykehus N-8000 Bodø

Rajka, Georg Frederik Stangs G. 44 N-0264 OSLO

Ree, Arve Ragnhildsvei 9 N-2800 Gjøvik

Ree, Kristian Kallerudvn. 7 N-2800 Gjøvik

Ree, Sidsel Kallerudvn. 7 N-2800 Gjøvik

Refsum, Hedevig Midtsugrenda 15 N-0787 Oslo

Remaut, Kinia Hudavdelingen Ullevål sykehus N-0407 Oslo

Roscher, Ingrid Stiernvejen 12A N-0779 Oslo Rustad, Lisbeth Hudavd. Haukeland sykehus N-5021 Bergen

Rustenberg, Berit Sigurdsgt. 4 N-3256 Larvik

Rutle, Oddvar Grønnegt.33 N-2300 Hamar

Ryggen, Kristin Hudavd. Regionsykehuset N-7006 Trondheim

Rødland, Ole Valkendorfsg. 9 N-5012 Bergen

Røen, Svein Aksel Hamrevn. 18 N-5210 Kalandseidet

Rønnevig, Jørgen Lersolveien 22 N-0876 Oslo

Sandberg, Morten Oppsal hudlegekontor Oppsalstubben 3 N-0685 Oslo

Saunes, Marit*

N-7006 Trondheim

Schmittenolf, Janine A* Hudavdelingen Regionsykehuset N-7006 Trondheim

Schmittgeolf, Markus O.* Hudavdelingen Regionsykehuset N-7006 Trondheim

Selvaag, Edgar Department of Dermatology Bispebjerg hospital DK-2400 Köpenhamn

Serup, Jørgen Ingeborgvej 42 DK-2900 Hellerup

Sitek, Jan Cezary*

N- Oslo

Sjøborg, Steinar Trädgårdsgatan 10 S-352 34 Växjö

Smith, Anne Klemeyer Ruglandsveien 14 A N-1342 Jar

Stang, Henning J*

N- Oslo

Steinkjer, Bjarte Hudpol Sentralsykehuset N-4011 Stavanger

Stenersen, Merete Hudavdelingen Regionsykehuset N-7006 Trondheim

Stenvold, Svein E. Hudavdelingen Regionsykehuset N-9038 Tromsø

Strand, Arnfinn Kirkevn. 98 A N-0361 Oslo

Stray, Per A. Kongensgt. 28 N-4610 Kristiansand

Strøm, Sonja Rute 1039 N-2480 Koppang

Stærfelt, Frode Havblikk helsesenter Kystvn. 154 N-4800 Arendal Norway

Stærnes Tambs, Kari Elisas Smiths vei 10, PB 63 N- Sandivika

Sviland, Lisbet* Hudavdelingen Haukelands sykehus N-5021 Bergen

Søyland, Elisabeth Hudavdelingen Rikshospitalet N-0027 Oslo

Teigen, Nina* Hudavdelingen Regionsykehuset i Tromsø N-9038 Tromsø

Thorvaldsen, Johannes Olafiaklinikken Postutak Grønland pk N-0133 Oslo

Thune, Per O. Storgata 4 N-0184 Oslo

Thune, Turid J*

N- Oslo

Tigalonova, Maya Oslo Hudklinikk Hegdehaugsv. 36 B N-0352 Oslo

Todal, Anders Hudavd. Regionsykehuset N-7006 Trondheim

Tveit, Kåre S. Hudavd. Haukeland sykehus N-5021 Bergen

Tørud, Erik Islandsgata 7 N-0658 OSLO

Utne, Leif Fjellvn. 64 N-5019 Bergen

Vadla, Jan Harald Hudavdelningen Innhered sykehus Levanger

Vatne, Øystein Hud. polikl., Sentralsyke huset i Sogn og Fjordane N-6800 Førde

Wereide, Knut Elisenbergvein 35 B N-0265 Oslo

Vik, Ingeborg Lyngstad Olafiaklinikken Postutak Grønland pk N-0133 Oslo

Volden, Gunnar Parkveien 19 B N-1405 Langhus

Wollan, Terje Jernbanealleen 30 N-Sandefjord

Ødegaard, Kristian Svenstuvn. 4 B N-0781 Oslo

Østensjø, Brynjulf Karmsundsgt. 272 N-5500 Haugesund

SWEDEN

Aase, Karl Blekholmsterassen 11 S-111 64 STOCKHOLM

Abrahamsson, Gudrun Överbyn 2110 S-834 00 BRUNFLO

Agdell, Jan Regimentsgatan 50 S-217 48 MALMÖ

Agrup, Gun Klöverv. 13 S-227 38 LUND

Ahnlide, Ingela Neptunivägen 32 S-237 35 BJÄRRED

Al-Sabori, Sam* Storskogsvägen 79, 1tr S-144 32 Rönninge

Anagrius, Carin Hans Järtas väg 12 S-791 32 FALUN

Anderson, Christopher c/o Jessica Andersson Stureg. 12, 4tr SE-582 21 LINKÖPING

Andersson, Anna-Carin Stenbergsvägen 12 S-752 41 Uppsala

Andersson, Bertil Börjesons väg 56 S-161 55 BROMMA

Andersson, Karin Lekattvägen 7 S-783 31 SÄTER

Andersson, Karin Hantverkareg. 12D SE-302 42 Halmstad

Andersson, Thomas Gustaf Smiths Plats S-583 47 LINKÖPING

Arenlind, Lars Salängsg 14 S-502 43 BORÅS

Arnamo, Anna-Maria Pilfinksvägen 18 S-183 51 TÄBY Aronsson, Annika

Hasselgången 3 S-241 00 ESLÖV Aspegren, Nils Tyska Skolgränd 4-6

S-111 31 STOCKHOLM

Augustsson, Agneta Torggatans Hudläkarmottagning Torggatan 8 S-411 05 Göteborg

Bartosik, Jacek* Skallgången 11 S-226 52 LUND

Beckman-Nahmias, Susa Houston Mill Road Atlanta GA 30329

Beebe, Bruce Västra Ekbacken 632 22 Eskilstuna

Beitner, Harry Bisittargatan 46 S-129 44 HÄGERSTEN

Bendsöe, Niels Vardavägen 249 F S-224 71 LUND

Bengtsson, Helge Hudlegekontoret Jelogatan 8 N-1500 MOSS

Berg, Jan Löjvägen 4B S-281 35 HÄSSLEHOLM

Berg, Mats Musseronvägen 1 S-633 58 Eskilstuna

Berg, Peter Norra vägen 31 S-163 41 SPÅNGA

Bergbrant, Ing-Marie Melongatan 69 S-426 56 VÄSTRA FRÖLUNDA

Bergdahl, Kjell Fagerövägen 23 B S-791 53 FALUN

Bergfelt, Louise Norra Vaktmansgatan 37 S-426 68 VÄSTRA FRÖLUNDA

Berggren, Gudrun Romansv.6 PL 13 S-131 40 NACKA

Berglund, Lena Eriksbergsgatan 4, 2tr S-114 30 STOCKHOLM

Bergman, Anita Strömgatan 27 S-856 43 SUNDSVALL

73

Bergqvist-Karlsson, Annika Furudalsvägen 20 B S-752 60 UPPSALA

Bergstedt, Kristina* Vadarvägen 7 731 42 Köping

Bergström, Marianne Arthur Engbergsv. 8 S-852 40 SUNDSVALL

Berne, Berit Döbelnsgatan 28 G S-752 37 UPPSALA

Berntsson, Matilda* Citrusgatan 22 426 54 V'ästra Frölunda

Bihi, Mohamed Söndrumsv.51 S-302 39 HALMSTAD

Bjarke, Torsten Hallbäcksgatan 3 S-252 34 HELSINGBORG

Bjarnason, Bolli Hudkliniken Karolinska sjukhuset S-171 76 STOCKHOLM

Bjellerup, Mats Pinnhättevägen 15 S-246 57 BARSEBÄCK

Björkner, Bert Regementsgatan 52 C S-217 48 MALMÖ

Björnberg, Alf Apgränd 10A S-271 42 YSTAD

Björnelius, Eva Ivar Hallströms väg 30 S-129 38 HÄGERSTEN

Björngren, Heléne Box 4259 S-203 14 MALMÖ

Björntorp, Elisabeth Södra Rosenbergsgatan 13 S-426 76 VÄSTRA FRÖLUNDA

Bleeker, Johan Linjevägen 11 S-531 50 LIDKÖPING

Bleeker, Thor Kållandsgatan 38 S-531 50 LIDKÖPING

Blom, Inga Väståstrand 1 S-702 32 ÖREBRO

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

Bohn, Jörg Verkstadsgatan 1A SE-211 42 Malmö

Bojs, Gunnel Näsbychaussen 14 S-291 35 KRISTIANSTAD

Boman, Anders Yrkes- och miljöderm enh. Karolinska sjukhuset S-171 76 STOCKHOLM

Borglund, Erik Ulvögatan 15 S-162 23 VÄLLINGBY

Boström, Christina Hudmottagningen Kullberska sjukhuset S-641 22 KATRINEHOLM

Boström, Åsa Statarvägen 16 S-752 45 UPPSALA

Bremer-Andersson, Eva Värtav.17, 2tr S-115 53 STOCKHOLM

Broberg, Ann Fjällbackag. 27 S-416 74 GÖTEBORG

Brodd, Astrid Bigatan 10 S-431 39 MÖLNDAL

Brundin, Göran Slåttervägen 25 S-614 00 SÖDERKÖPING

Bruze, Magnus Lotsgatan 8 S-216 42 Limhamn

Burian, Elzabieta Postlåda 8224 S-541 91 SKÖVDE

Burström, Eva Opalvägen 11 S-961 46 BODEN

Byrenius-Mellström, Birgitta Björnögatan 5 S-761 40 NORRTÄLJE

Båmstedt, Halina Odeng. 69, 6tr S-113 22 STOCKHOLM

Bäck, Ove Otto Lindblads väg 4 S-224 65 LUND

Cadova, Vera Näsby Allé 3, 1 tr S-183 55 TÄBY Carlberg, Hans Hudkliniken Södersjukhuset

S-118 83 STOCKHOLM

Carstam, Ragnar Adelgatan 15 S-223 50 LUND

Cederroth, Susanne Karlaplan 10, 7tr S-115 20 STOCKHOLM

Christensen, Ole B Villavägen 21 S-216 11 MALMÖ

Christiansen, Julie Hudkliniken Lasarettet S-221 85 LUND

Coble, Britt-Inger Kopparstigen 3 S-582 58 LINKÖPING

Dahl, Jens Christian Saltværksvej 168 DK-2770 Kastrup

Dahlberg, Erik Snorres väg 34 S-832 55 FRÖSÖN

Dahlbäck, Karin Regementsgatan 8 S-211 42 Malmö

Dahlman Ghozlan, Kristina Tamanaco 3 17 Ch. de Lérmitage F-06160 Juan Les Pins

Davidsson, Steingrimur Hateigsvegur 1 IS-105 REYKJAVIK

Dobrescu, Justin Tulipanvägen 23 S-227 38 Lund*

Domar, Margareta Slagrutevägen 17 S-756 47 UPPSALA

Dunér, Kari Roparebergsvägen 1A S-371 42 KARLSKRONA

Edeland-Odd, Brita Qvarntorps herrgård S-667 00 FORSHAGA

Edgardh, Karin* Thulstrupsg. 5A N-0450 Olso Edmar, Birgitta Strandplatsgt 8 S-426 76 VÄSTRA FRÖLUNDA

Egelrud, Torbjörn Generalsg. 11 S-903 36 UMEÅ

Ek, Lorens EK-Stjärnborg Hagalundsvägen 30D S-302 74 HALMSTAD

Ekbäck, Maria Fältspatsv 28 S-703 74 ÖREBRO

Ekelund, Anna-Greta Snapperupsgatan 12 S-211 35 MALMÖ

Ekholm, Elisabeth Hudkliniken Regionsjukhuset i Umeå S-901 85 UMEÅ

Eklind, Jan* Kalmgatan 29, 4tr S-121 45 Johanneshov

Ekmehag, Björn* Mesangatan 6 S-234 42 Lomma

Elmros, Theodor Silvervägen 62 S-907 50 UMEÅ

Emtestam, Lennart Ingemansvägen 4A S-141 41 HUDDINGE

Enerbäck, Charlotta* Sundhagsgatan 8 S-414 76 Göteborg

Enfors, Wera Sandelsgatan 23 S-115 34 STOCKHOLM

Engman, Christina Nicoloviusgatan 10B S-217 57 Malmö

Enhamre, Anders Läkargruppen Mörby Box 89 S-182 11 DANDERYD

Enström, Ylva Arkivgatan 5, 2tr S-411 34 GÖTEBORG

Ericsson, Gunnel Kungsholms kyrkopl 1 S-112 24 STOCKHOLM Eriksson, Tomas Räfsarstigen 38 S-954 34 GAMMELSTAD

Faergemann, Jan Apotekarg. 7 Sahlgrenska sjukhuset S-413 19 GÖTEBORG

Fagerblom, Lena* Falkenbergsvägen 34 S-857 32 Sundsvall

Falk, Anna-Maria Bärsta S-715 92 Stora Mellösa

Falk, Lars Oskarsplatsen 9 S-702 13 ÖREBRO

Fast, Kristian Furuv. 3 S-440 90 HENÅN

Fernström, Åke Fredhällsg 12 S-112 54 STOCKHOLM

Fischer, Torkel Hensvik 253 S760 49 Herräng

Fjellner, Bo Box 5701 S-114 87 STOCKHOLM

Flur, Barbara Genvägen 3B S-141 37 Huddinge

Forneus, Anders Wiks gård S-755 91 UPPSALA

Forsberg, Annika* Norrbackagatan 44, 1tr S-113 41 Stockholm

Forslind, Bo Kungsholmsgatan 22 S-112 27 STOCKHOLM

Forsman, Sten Dermatolog Lilla Risåsgatan 20 S-413 04 GÖTEBORG

Fransson, Jessica Öregrundsgatan 10 6tr S-115 59 STOCKHOLM

Fregert, Sigfrid Mellanvångsvägen 5 S-223 55 LUND

Friedman, Marie Hantverkargatan 34, 2tr S-112 21 Stockholm Frithz, Anders Börjesons väg 49 S-161 55 BROMMA

Frohm-Nilsson, Margareta Grönviksvägen 191 S-167 76 BROMMA

Frödin, Thomas Lästgatan 4 S-582 66 LINKÖPING

Fåhraeus-Morin, Lena Åsvägen 13 S-818 33 VALBO

Färm, Gunilla Olof Skötkonungsv 34 S-126 50 HÄGERSTEN

Gajecki, Maria Norrskensbacken 9, 2tr S-146 00 TULLINGE

Gamborg-Nielsen, Poul Hudkliniken Lasarettet S-301 85 HALMSTAD

Gerdén, Barbro Torgnygatan 9 S-752 31 UPPSALA

Gezelius-Strausser, Birgitta c/o Gezelius Klövervägen 29 S-161 36 BROMMA

Gilboa, Ruth 617 Ridgeline Place Solana Beach CA 92075 CALIFORNIA

Gisslén, Peter Brotorpsvägen 36 S-163 59 SPÅNGA

Gjede, Uffe Simons Bakke 66 DK-7700 THISTED

Granger, Katri Runstens Prästgård 4294 S-386 94 FÄRJESTADEN

Groth, Ove Ådalagt. 8 S-582 26 LINKÖPING

Grängsjö, Anders Celsiusgatan 9 S-752 31 UPPSALA

Gånemo, Agneta Gånarp 2517 S-266 92 Munkaljungby Gäfvert, Elisabeth Pharmacia & Upjohn SG30-7 S-112 87 Stockholm

Gärtner, Lena Fack 2101 S-331 02 VÄRNAMO

Haaland, Björn Bondevägen 5A S-227 64 LUND

Hackzell-Bradley, Maria Igelkottsvägen 12 S-161 37 BROMMA

Hagerman, Gösta Limhamnsvägen 22 A S-217 59 MALMÖ

Hallén, Anders Fyrisgatan 14 S-753 15 UPPSALA

Hamnerius-Olofsson, Nils Östra Strandgatan 1A S-371 36 KARLSKRONA

Hansen, Bodil Stenvang Langdyssevej 15 DK-7700 Thisted

Hansson, Carita Borgåsvägen 20 S-438 32 LANDVETTER

Hansson, Christer Småviltsg. 13 S-226 52 LUND

Hashim, Firouz Kungsgatan 3B S-553 31 JÖNKÖPING

Hedblad, Mari-Anne Ulvögatan 15 S-162 23 VÄLLINGBY

Hedenborg, Lennart Östgötagatan 22 S-116 25 STOCKHOLM

Hedström, Sven-Åke Arvid Karlssons väg S-311 92 Falkenberg

Heijer, Arne Safirvägen 13 S-451 62 UDDEVALLA

Hellgren, Lars Bronsgjutaregatan 13 S-421 63 VÄSTRA FRÖLUNDA

Hellström, Clas Drottningsgatan 56 S-111 21 STOCKHOLM

75

Herczka, Olga Götgatan 21 S-116 46 STOCKHOLM

Herdenstam, CG Pontus Erikslundsv. 5, 6tr S-611 61 NYKÖPING

Hersle, Kjell Kedjestigen 3 S-436 50 HOVÅS

Hesser, Göran Skandiavägen 26 S-474 31 ELLÖS

Hestner, Anna* Regementsg. 3A S-541 46 Skövde

Hillström, Lars Lövens Tä 29 S-802 57 GÄVLE

Hindsén, Monika Östervångsvägen 9 S-224 60 LUND

Hoasseing, Sirn* Hudkliniken Gävle lasarett S-802 85 Gävle

Hofer, Per-Åke Hudkliniken Umeå lasarett S-901 85 UMEÅ

Holm, Joanna Hudkliniken Uddevalla sjukhus S-451 80 UDDEVALLA

Holm, Lena Hornsgatan 57 3tr/16 S-118 49 STOCKHOLM

Holm, Pelle Rörviksv. 16 S-451 77 UDDEVALLA

Holmberg, Jadwiga Brushanevägen 35 S-556 25 JÖNKÖPING

Holmdahl, Mejrav Hudkliniken Universitetsjukhuset S-221 85 Lund

Holmdahl-Källén, Katarina Falkenbergsv. 10 S-392 44 Kalmar

Holst, Rolf Roskildevägen 17C S-217 46 Malmö

Holt, Ingebjörg Hudkliniken Lasarettet S-262 81 ÄNGELHOLM

Hornmark, Anne-Marie Korsgatan 2A S-702 27 ÖREBRO

Horova, Vera Västra Boängsv 50 S-691 44 KARLSKOGA

Hovmark, Anders Ruriks väg 13 S-186 50 VALLENTUNA

Hradil, Eva Rektorsv. 20 S-224 67 LUND

Hård af Segerstad, Stig Svartkärrsvägen 10 S-133 32 SALTSJÖBADEN

Hägermark, Östen Kungsvägen 5 S-182 75 STOCKSUND

Häggarth, Ingrid Lövgatan 26 S-169 32 SOLNA

Hägglund, Gun Kolonigatan 11 B S-852 39 SUNDSVALL

Hällgren, Jenny* Hudkliniken Karolinska Sjukhuset S-171 76 Stockholm

Hök, Monika Älvgatan 17, 1tr S-652 26 KARLSTAD

Hörnqvist, Rune Södra Gimonäsvägen 72 A S-907 42 UMEÅ

Inerot, Annica Rådastocksvägen 6 S-435 44 MÖLNLYCKE

Irestedt, Magnus Villav. 32 C Centralsjukhuset S-296 38 ÅHUS

Isaksson, Marlene Rösträttsgatan 3 S-227 60 LUND

Itman, Sofia Odengatan 24 S-113 51 STOCKHOLM Iversen, Normann HudDoktorn i Örebro Slottsg 8 S-703 61 ÖREBRO

Jansen, Elin Roskildegatan 4 3tr S-252 21 Helsingborg

Jansson, Kerstin Sunnanvindsvägen 6 S-582 72 LINKÖPING

Jeansson, Irene Box 114 S-386 00 FÄRJESTADEN

Jekler, Jan Lill-Jans Plan 3 S-114 25 STOCKHOLM

Jeppsson, Eva Ringvägen 75 S-235 93 Vellinge

Jerner, Björn Eddagatan 6 S-802 54 GÄVLE

Johannesson, Anders Loviselundsvägen 112 S-165 70 HÄSSELBY

Johannisson, Gunnar Berzeliigatan 5 S-412 53 GÖTEBORG

Johansson-Lange, Margaretha Ripvägen 13 S-351 42 VÄXJÖ

Johnsen, Paul Otto Skippergatan 21 N-4611 Kristiansand S

Johnsson, Margareta Hudavdelningen Regionsykehuset N-7000 TRONDHEIM

Jonell, Ragnar Hästviksgången 16 S-426 71 VÄSTRA FRÖLUNDA

Jonsson, Lennart Däldernav. 8 S-541 47 SKÖVDE

Josefson, Anna Hudkliniken Regionssjukhuset S-701 85 ÖREBRO

Josefsson, Kaj* Stenhuggarevägen 1 S-30240 Halmstad Juhlin, Lennart Döbelnsgatan 30 H S-752 37 UPPSALA

Jurkas, Beatrice Ölandsvägen 5 S-826 39 SÖDERHAMN

Jörgensen, Esben Tegelviksvägen 9B S-392 43 KALMAR

Jörgensen, Hans-Petter V Storgatan 15, 4.etg. N-1614 FREDRIKSTAD

Kaaman, Ann-Catrin Herr Stens väg 35 S-125 30 ÄLVSJÖ

Kaaman, Taavi Hudkliniken Södersjukhuset S-118 83 STOCKHOLM

Karlberg, Ann-Therése Bågspännarvägen 10 S-125 30 ÄLVSJÖ

Karlsson, Pia Ekängsvägen 17 S-582 75 LINKÖPING

Karltorp, Nils Långhemsgatan 13 S-502 50 BORÅS

Karnå, Eva Karin Helgonabacken 7 S-451 32 UDDEVALLA

Karpe, Barbro Fördelningsgatan 18 S-633 41 ESKILSTUNA

Kehler Rosenlind, Simone Juvelvägen 11 S-541 42 Skövde

Kelfve, Birgitta Didriksgatan 3 S-722 18 VÄSTERÅS

Kinnman, Kristel Östbovägen 6A S-182 56 Danderyd

Klintberg, Per Östermalmsgatan 50 S-903 32 Umeå

Kogan, Michael Drottninggatan 33 2tr S-252 21 Helsingborg

Kristensen, Berit Hudklinikken Bredgade 50 DK-4400 Kalundborg

76

Kristensen, Ove Hudklinikken Bredgade 50 DK-4400 Kalundborg

Kristoffersson, Per Olof Fåborgvägen 19 S-311 45 FALKENBERG

Krogh, Geo von Bondegt 1 C S-116 23 STOCKHOLM

Kronberg, Anna* Hudkliniken Centrallasarettet S-721 89 Västerås

Krook, Klas Sibyllegatan 81 S-114 43 STOCKHOLM

Krupicka, Pavel Stenbov. 13 S-735 35 SURAHAMMAR

Kuylenstierna, Maria-Pia Lindallévägen 19 S-554 54 JÖNKÖPING

Kuylenstierna, Nils* Gahns väg 2A S-791 32 Falun

Laestadius, Anette Hudkliniken Norrlands Universitetssjukhus S-901 85 UMEÅ

Lagerholm, Björn Tors väg 11 S-182 35 DANDERYD

Lagmo, Kenneth Persiljevägen 4 S-585 91 LINKÖPING

Landegren, Johan Värtavägen 16, 5tr S-115 24 Stockholm

Lapins, Jan Yttersta Tvärgränd 10D S-118 23 STOCKHOLM

Larkö, Olle Arkivgatan 5, 2tr S-411 34 GÖTEBORG

Larsen, Allan Örnäsv 87 S-302 40 Halmstad

Larsson, Per-Åke Lorichvägen 4 S-791 37 FALUN

Larsson, Åke Tandläk. högksolan S-214 21 MALMÖ

Larsson-Stymne, Birgitta Nygatan 73B S-702 13 ÖREBRO

Laszlo, Csilla Essingeringen 82/853 S-112 64 STOCKHOLM

Laurell, Hans Kattsfotsvägen 9 S-459 32 LJUNGSKILE

Lengstam, Ingvar Roslagsgatan 7 S-113 55 STOCKHOLM

Leppert, Anna Schenströmsgatan 5A S-724 62 VÄSTERÅS

Liander, Wendela Hudmottagningen Borgmästargatan 5 S-434 32 KUNGSBACKA

Lidbrink, Peter Plantskolevägen 39 S-122 38 ENSKEDE

Lidén, Carola Upplandsgatan 58 S-113 28 Stockholm

Lidén, Sven Margretelundsgt 12 S-412 67 GÖTEBORG

Lidman, Hjördis Karlavägen 5 S-114 24 STOCKHOLM

Liljestrand Husebö, Åsa Dermatolog Markevei 2a N-5012 BERGEN

Lindberg, Lena Lundåsvägen 12 B S-502 60 BORÅS

Lindberg, Magnus Idrottsv 4 B S-192 66 SOLLENTUNA

Lindberg-Wollmar, Bo Strandg. 5 S-621 57 Visby

Lindborg, Lena* Bygatan 29 S-171 49 Solna

Linde, Ylva Igelkottsv 4 S-161 37 BROMMA

Lindelöf, Bernt Fornuddsvägen109 S-135 52 TYRESÖ Lindemalm-Lundstam, B Askims Hovslagarväg 10 S-436 00 ASKIM

Linder-Carlsson, Gerd Nydalavägen 18 A S-903 39 UMEÅ

Lindeskog, Gerhard Ekåsvägen 18 B S-433 62 PARTILLE

Lindewall, Gertrud Banergt 10 3 tr S-115 23 STOCKHOLM

Lindgren, Sören Platensgatan 10B S-582 20 LINKÖPING

Lindholm, Ann-Charlotti* Trädgårdsgatan 10 S-633 55 Eskilstuna

Lindström, Börje Blockhusvägen 24 S-791 45 FALUN

Linse, Ulla-Britta Kometvägen 21, 6tr S-183 48 TÄBY

Lirwall, Margareta Uvebergsvägen 26 S-582 60 LINKÖPING

Ljunggren, Bo Marieholmsvägen 8 S-217 63 MALMÖ

Ljunghall, Kerstin Tallbacksv 49 S-756 45 UPPSALA

Lodén, Marie Illerstigen 32, 2tr S-170 71 SOLNA

Lundeberg, Lena Höjdstigen 7 S-181 31 LIDINGÖ

Lundqvist, Katarina PL 2613 Lackagården S-244 60 FURULUND

Lundström, Anita Magnusdalsv. 1 S-903 39 UMEÅ

Löfberg, Helge Stora Tomegatan 29 S-223 51 LUND

Löwhagen, Gun-Britt Ingegärdsvägen 2 B S-421 68 VÄSTRA FRÖLUNDA Mabergs, Nils Ringvägen 131,2 tr S-116 61 Stockholm

Magnusson, Britt-Louise Doktor Forseliusgata 12 S-413 26 GÖTEBORG

Malmkvist Padoan, Sigrid Kristianstadkliniken Ö Boulevarden 56 S-291 31 KRISTIANSTAD

Malmros-Enander, Ingergärd Lunds Östra 7 S-621 48 VISBY

Marcusson, Jan Danav. 7A S-181 31 LIDINGÖ

Maroti, Marianne Högabergsgatan 37 S-554 46 JÖNKÖPING

Martin, Peter Värtavägen 1 S-183 63 TÄBY

Meding, Birgitta Ankdammsgatan 40, 1tr S-171 43 SOLNA

Michaelsson, Eva* Strandvägen 223 S-234 32 Lomma

Michaëlsson, Gerd Skogsmyrsv 9

S-756 45 UPPSALA Mikkelsen, Henrik

Box 17 Bergdalen S-310 70 TORUP

Mjörnberg, Per Anders Vårvägen 31 S-541 33 SKÖVDE

Mobacken, Håkan Lars Hägers väg 20 S-443 32 LERUM

Moberg, Sven Stensvägen

Box 2057

S-433 02 PARTILLE Modée, Jan Ellen Keys g. 37 S-129 52 HÄGERSTEN

Modén, Margareta Snöflingegatan 5 S-723 50 VÄSTERÅS

Mohammadi, Noushin* St Eriksgatan 7 S-112 39 Stockholm Moi, Harald Håkonsvej 63 N-1470 Lörenskog

Molin, Lars Hudkliniken Regionsjukhuset S-701 85 ÖREBRO

Molnar, Christina Hudkliniken Centrallasarettet S-721 89 VÄSTERÅS

Montelius, Henning* Pehr Thomassons v 27 S-371 63 Lyckeby

Montelius, Johan Arbetslivsinstitutet S-171 84 SOLNA

Munksgaard, Ingrid Coldinuvägen 6 S-371 42 Karlskrona

Månesköld, Anna* Bögatan 37A S-412 72 Göteborg

Månsson, Tore Krusbärsvägen 40 S-262 92 Båstad

Mårtensson, Kerstin* Hudmottagningen Länssjukhuset S-301 85 Halmstad

Möller, Halvor Ledungsgt 21 S-217 74 MALMÖ

Mölne, Lena Hudkliniken Sahlgrenska sjukhuset S-413 45 GÖTEBORG

Nagy, Veronika Källsprångsgatan 2 S-413 20 GÖTEBORG

Neumann, Emil Odenvägen 4 B S-133 38 SALTSJÖBADEN

Niklasson, Eva Skördegatan 7 S-602 12 NORRKÖPING

Nilsen, Tore Specialistläkargruppen Trädgårdsgatan 10 S-352 34 VÄXJÖ

Nilsson, Eskil Jakobsensväg 5 S-856 34 SUNDSVALL



Niordsson, Ann-Mari Gammelhovedgaden 14 DK-2970 Hörsholm

Nohlgård, Christina Nacka hudmottagning Box 4133 S-131 04 NACKA

Norborg-Iversen, Lise Lindö S-611 93 NYKÖPING

Nordin, Håkan Rundgatan 6 S-243 30 HÖÖR

Nordin, Leif Tråkärrslättsvägen 57 S-427 50 BILLDAL

Nordin, Peter P L 714 Box 138 S-436 00 HOVÅS

Nordin-Björklund, Karin Björnövägen 36 A S-723 48 VÄSTERÅS

Nordlind, Klas Riddargt 72 S-114 57 STOCKHOLM

Norén, Peter Edov. 13 S-741 93 KNIVSTA

Norman, Anders Gårdsvägen 10 S-182 75 STOCKSUND

Nourbakht, Ali Luftvärnsg. 32 S-587 34 Linköping

Nyberg, Filippa Skeppsvägen 2 S-182 76 Stocksund

Nygren, AnnCharlotte Slottsv 2 S-191 51 SOLLENTUNA

Nylander-Lundqvist, Elisabeth Pilgatan 8D S-903 31 UMEÅ

Nyman, Gunnar Sälggatan 2 S-510 54 BRÄMHULT

Nyrén, Miruna Banérg. 25 S-115 22 Stockholm

Nyrud, Morten Trollåsveien 25 N-1414 TROLLÅSEN Ochremenko, Pavel Henrik Wergelandsgt 19 N-4600 KRISTIANSAND

Odd Löland, Einar Branders lovvej 28 DK-9900 FREDRIKSHAVN

Odu, Solveig Oskarsgatan 24 S-331 41 VÄRNAMO

Ohlsson, Erik Visby lasarett S-621 84 VISBY

Ohlsson, Ylva Hud/STD kliniken Borås lasarett S-501 82 BORÅS

Okrasinksi, Henryk Drottninggatan 16 S-411 14 Göteborg

Olivecrona, Eva Tegnergatan 7 S-111 40 STOCKHOLM

Olson, Kerstin Kasten Rönnowsgatan 3G Länssjukhuset S-302 36 HALMSTAD

Olsson, Elisabeth Grytholmsg. 4 S-572 40 OSKARSHAMN

Olsson, Ingegerd Vanadisvägen 31A S-113 23 STOCKHOLM

Overgaard Petersen, Hans Sjögrensv. 11 S-593 34 VÄSTERVIK

Pawlik, Ewa Hudkliniken Regionsjukhuset i Umeå S-901 85 UMEÅ

Pedersen, Niels B Carl Krooks gata 1A S-252 25 HELSINGBORG

Persson, Bertil Kastanjegatan 21:29 S-224 56 LUND

Persson, Lill-Marie Hamngatan 6 S-542 30 MARIESTAD

Persson-Lindgren, Gun Bjurnäs 6659 S-388 96 LJUNGBYHOLM

Pettersson, Arne Överbyn 2110 S-834 00 BRUNFLO Pettersson, Karl-Göran Hudkliniken Lasarettet S-851 86 SUNDSVALL

Pettersson, Lars Liegatan 7 S-802 70 GÄVLE

Piechowicz, Malgorzata Storkstigen 5 S-393 59 KALMAR

Plá Arlés, Ulla-Britt Urbanización el Mirador Pasaje San José nr 9 ES-12600 Vall de Uxó

Pompe, Jarmila Mikrog 62 S-502 47 BORÅS

Pontén, Fredrik Patologi C Lab Akademiska sjukhuset 751 85 Uppsala

Popova, Irina Tolvmannagatan 3A S-392 35 KALMAR

Poulsen, Jens Søgade 16 DK-4100 RINGSTED

Preisler-Häggqvist, Anna Älvans väg 186 S-907 50 Umeå

Probierz-Zak, Hanna Ragnar Jändelsvägen 7 S-371 63 LYCKEBY

Pålsdottir, Rannveig Noatun 31 IS-IS-105 REYKJAVIK

Qvarner, Hans Grev Turegatan 75 S-114 38 STOCKHOLM

Radecka, Maria Galleasvägen 3 S-352 55 VÄXJÖ

Ramstedt, Gunnar Siglajvs Rute

Reidhav, Inga Alnarpsv. 34 S-232 53 Åkarp

S-620 34 LÄRBRO

Richtnér, Tomas Döbelnsgatan 83

S-113 52 STOCKHOLM Ridderström, Eva Tegnergatan 17A S-752 26 Uppsala

78

Rietz, Hélène* BML, Liljeholmstorget S-117 94 Stockholm

Rollman, Ola Billvägen 12 S-756 48 UPPSALA

Rorsman, Hans Pålsjövägen 26 S-223 63 LUND

Ros, Anne-Marie Mälartorget 13 S-111 27 STOCKHOLM

Roscher, Ingrid Stiernvejen 12A N-0779 Oslo

Rosdahl, Inger Djurgårdsgatan 58 S-582 29 LINKÖPING

Rosén, Karin Norra Grindekärrsvägen 24

S-436 56 HOVÅS

Rosenberg, Inger Turevägen 14 S-191 47 SOLLENTUNA

Rosenblad, Eija Dalagatan 78A S-113 24 Stockholm

Rossman-Ringdahl, Ingrid Tallboängen 66 S-436 00 ASKIM

Roupe, Gösta Apotekarg 6 S-413 19 GÖTEBORG

Rudén, Ann-Kerstin Ringvägen 124, 3tr S-116 64 STOCKHOLM

Ruhnek-Forsbeck, Margit Nybergsgt 4, 2tr S-114 45 STOCKHOLM

Ryberg, Kristina Björkvägen 4B S-459 31 LJUNGKILE

Rystedt, Ingela Majorsg 4 S-114 47 STOCKHOLM

Rönnerfält, Lena Sturegatan 18, II S-114 36 STOCKHOLM

Saighani, Simor* Hackspettsvägen 7 S-746 35 Bålsta

Sandström, Erik Bataljvägen 27 S-133 33 SALTSJÖBADEN

Sandström, Mari Helen Gibraltargatan 42 S-412 58 GÖTEBORG

Schmidtchen, Artur Göingegatan 4 222 41 Lund

Schou, Marie Notvägen 9 S-711 35 LINDESBERG

Schön, Jenny* Hud - och STD-kliniken Universitetsjukhuset S-901 85 Umeå

Seideman, Peter Kungsholmstorg 3B 7tr S-112 21 Stockholm

Seller, Heinz Strandgatan 5 S-432 44 Varberg

Sigurgeirsson, Bárdur Haaberg 39 IS-220 HAFNARFJÖRBUR

Sjöborg, Steinar Kalendervägen 7 S-352 60 VÄXJÖ

Sjölin-Forsberg, Gunilla Tors väg 20 A S-754 40 UPPSALA

Sjöström, Karin Ekorrstigen 3 S-703 75 ÖREBRO

Sjövall, Peter Nordmannagatan 11 S-217 74 Malmö

Skawski, Annette Hudkliniken Centrallasarettet S-721 89 VÄSTERÅS

Skog, Erik Banergatan 49 S-115 22 STOCKHOLM

Skogh, Marcus Järdalav. 52D SE-589 21 LINKÖPING

Skoglund, Curt Grev Turegatan 75 S-114 38 STOCKHOLM

Skoog, Marja-Leena Domherrevägen 11 S-585 95 LINKÖPING Sköld, Gunilla Kaprifolv. 20 S-603 66 NORRKÖPING

Sokolski, Jan G:a Övägen 1D S-603 53 NORRKÖPING

Sommerfeld, Beatrice Danavägen 7 B S-181 31 LIDINGÖ

Sonesson, Björn Hudkliniken Lasarettet S-221 85 LUND

Spångberg, Sune Odengatan 16 B S-753 13 UPPSALA

Starck-Romanus, Vera Vedhuggaregatan 16 S-412 61 GÖTEBORG

Stark, Hans Ulrik Grindstugev 4 S-663 41 HAMMARÖ

Stempa, Marian Högbyv 146 S-175 46 JÄRFÄLLA

Stenberg, Berndt Rönnbärsstigen 25 D S-903 46 UMEÅ

Stenberg, Åsa Falsterbovägen 35 S-857 30 SUNDSVALL

Stenlund, Kajsa Orrspelsvägen 42 S-167 66 BROMMA

Stenmark-Särhammar, Gunnel Videgatan 1 S-652 30 KARLSTAD

Stenström, Monika Brändövägen 62 S-165 72 Hässelby

Strand, Anders Geijersgatan 17C S-752 26 UPPSALA

Ståhle-Bäckdahl, Mona Bastug 33 S-118 25 STOCKHOLM

Sund Böhme, Maria Västmannagatan 27 S-113 25 STOCKHOLM

Sundberg, Karin Hudmottagningen Länssjukhuset Ryhov S-551 85 JÖNKÖPING Surakka, Jouni* Lavettvägen 31 S-174 59 Sundbyberg

Swanbeck, Gunnar Trollåsvägen 29 436 42 Askim

Svedman, Cecilia Västra Rönneholmsvägen 36A S-217 41 Malmö

Sveinsson, Birkir

Mithskógar 1 IS-225 Bessastatharhrepp

Svensson, Louise Bagaregatan 13, 2 tr S-611 31 Nyköping

Svensson, Margareta Leksandsv 4 S-192 67 SOLLENTUNA

Svensson, Åke Norregatan 17 S-289 00 KNISLINGE

Synnerstad, Ingrid* Hudliniken Universitetssjukhuset S-581 85 Linköping

Szpak, Ewa Björkängsvägen 15 S-141 38 HUDDINGE

Söderberg, Björn Höstvägen 106 S-976 33 LULEÅ

Sörensen, Dennis Kongens gade 30 A st DK-4800 NYKÖPING

Talme, Toomas Porsvägen 19 S-146 37 TULLINGE

Tammela, Monica Flogstavägen 18 S-752 73 Uppsala Tarras-Wahlberg, Casper

Skånegatan 15 S-571 40 Nässjö

Tarstedt, Mikael Gjutaregatan 9 S-703 57 Örebro

Tegner, Eva Markaskälsvägen 8 S-226 47 LUND

Thelin, Ingrid Astrakansvägen 21 S-224 56 LUND

79

Thórarinsson, Hannes Sóleyjargötu 27 IS-101 REYKJAVIK

Thorgeirsson, Arnar Bellmansgatan 3 S-254 40 HELSINGBORG

Thornéus, Inga-Britt* Hud- och STD-kliniken Norrlands Universitetssjukhus S-901 85 Umeå

Thurfjell, Christina Trekantsgatan 1 S-652 20 Karlstad

Thyresson, Nils Norbyvägen 41 S-752 39 UPPSALA

Thörneby-Andersson, Kirsten Svenshögsvägen 3 S-222 41 LUND

Tjernlund, Ulla Torphagsvägen 2 2tr S-104 05 Stockholm

Tollesson, Anders Saltsjövägen 15 A S-181 62 LIDINGÖ

Torssander, Jan Thun Ollevägen 7 S-134 34 Gustavsberg

Troilius, Agneta Råbelövsgt 56 S-216 19 MALMÖ

Trokenheim, Alina Furuängsgången 2 S-129 52 HÄGERSTEN

Trolin, Ingrid Albertsväg 14 A S-752 60 UPPSALA

Trovallius, Cecilia Hallonbergsvägen 21 S-172 43 SUNDBYBERG

Tunbäck, Petra Mossgatan 16 S-413 21 Göteborg

Törmä, Hans Hudkliniken Akademiska sjukh S-751 85 UPPSALA

Törngren, Mats Pliggvägen 7 S-126 39 HÄGERSTEN

Ustupski, Janina Rödklövervägen 80 S-165 73 HÄSSELBY

Wahbi, Abdelhabi Grödingev 3 S-147 30 Tumba

Wahlberg, Jan Norrbacka Yrkesderm klin S-171 76 STOCKHOLM

Wahlgren, Carl-Fredrik Oviksgatan 83 S-162 21 VÄLLINGBY

Vahlquist, Anders St Olofsgatan 1A S-753 10 UPPSALA

Vahlquist, Carin St Olofsgatan 1A S-753 10 UPPSALA

Wallberg, Peter Spångavägen 13 3tr/1089 S-168 75 Bromma

Wallengren, Joanna Beleshögsvägen 42 S-217 74 MALMÖ

Wallenhammar, L-M Nackagatan 20 S-116 47 Stockholm

Wanger, Lena Granestigen 8 S-182 65 DJURSHOLM

Vatne, Øystein Sentralsjukehuset Sogn og Fjordane N-6800 FØRDE

Wedén, Ulla Cedervägen 5 S-141 45 HUDDINGE

Wenger, Helena Björnebergsvägen 8B S-553 12 JÖNKÖPING

Wengström, Claes S.Hamngatan 37-41 S-411 06 GÖTEBORG

Wennberg, Ann-Marie Skjutbanegatan 14 S-413 21 GÖTEBORG

Wennberg, Kerstin S:t Eriksplan 4, v S-113 20 STOCKHOLM

Wennersten, Göran Grev Tureg. 59-61 S-114 38 STOCKHOLM

Went, Maria Starrängsringen 26 3tr S-115 50 STOCKHOLM Westerberg, Per Skårsgatan 45 S-412 69 GÖTEBORG

Westerhoff, Karin Aspö 8003A S-541 91 Skövde

Wictorin, Åsa* Iliongränden 245 S-224 72 Lund

Widén-Karlsson, Veronica* Larsbergsgatan 28 S-181 39 Lidingö

Wiegleb-Edström, Desiree Älghagsstigen 30 S-165 76 HÄSSELBY

Wikström, Arne Kungstensg. 28 uppg. 2, 4tr S-113 57 STOCKHOLM

Wikström, Kjell Stenåsen, Skärv S-532 92 AXVALL

Wilson-Clareus, Birgitta Louiselundsvägen 137 S-165 70 HÄSSELBY

Vinnerberg, Åsa Sandbergsgt 4 S-603 55 NORRKÖPING

Wirestrand, Lars-Erik Torsekevägen 113 S-291 94 KRISTIANSTAD

Virtanen, Marie* Hudkliniken Akademiska sjukhuset S-751 85 Uppsala

Wolff, Hélène Fyradalersgatan 32 S-413 19 Göteborg

Voog, Eva Sofiagatan 85 S-416 72 GÖTEBORG

Wrangsjö, Karin Garvargatan 19 2tr S-112 21 STOCKHOLM

Wærsted, Atle Höjbjerggårdsvej 22 DK-2840 Holte

Zak, Richard Ragnar jändels v 7 S-371 63 LYCKEBY

Ågren-Jonsson, Siv PL 1490 Ålabodarna S-261 63 GLUMSLÖV

80

Forum for Nord Derm Ven Vol. 6, 2001 - Suppl. 2

Åsbrink-Hovmark, Eva Ruriks väg 13 S-186 50 VALLENTUNA

Öhman, Hans Hjalmar Svenfelts v 15 S-590 60 LJUNGSBRO

Öhman, Sven Banergt 12 B S-752 37 UPPSALA

Öjner, Barbara Plaza Alcazar No 3, Local 3 Centro Magna Plaza E-07015 Portals Nous CALVIA, Mallorca

Örsmark, Kerstin Kullagatan 1 Baskemölla S-272 94 SIMRISHAMN

Postbesørget blad (8245 ARC) Blad nr. 50629 Produced in Denmark by Knud Gr@phic Consult + 45 6618 0711

Eftersendes ikke ved vedvarende adresseforandring. men tilbagesendes med oplysning om den nye adresse

Golden Main Sponsors





Main Sponsors



