WORKSHOP ON ATOPIC DERMATITIS
PRAGUE 2006

26. – 28.5.2006

Odborný program konference:

pátek 26. května

sobota 27. května

neděle 28. května

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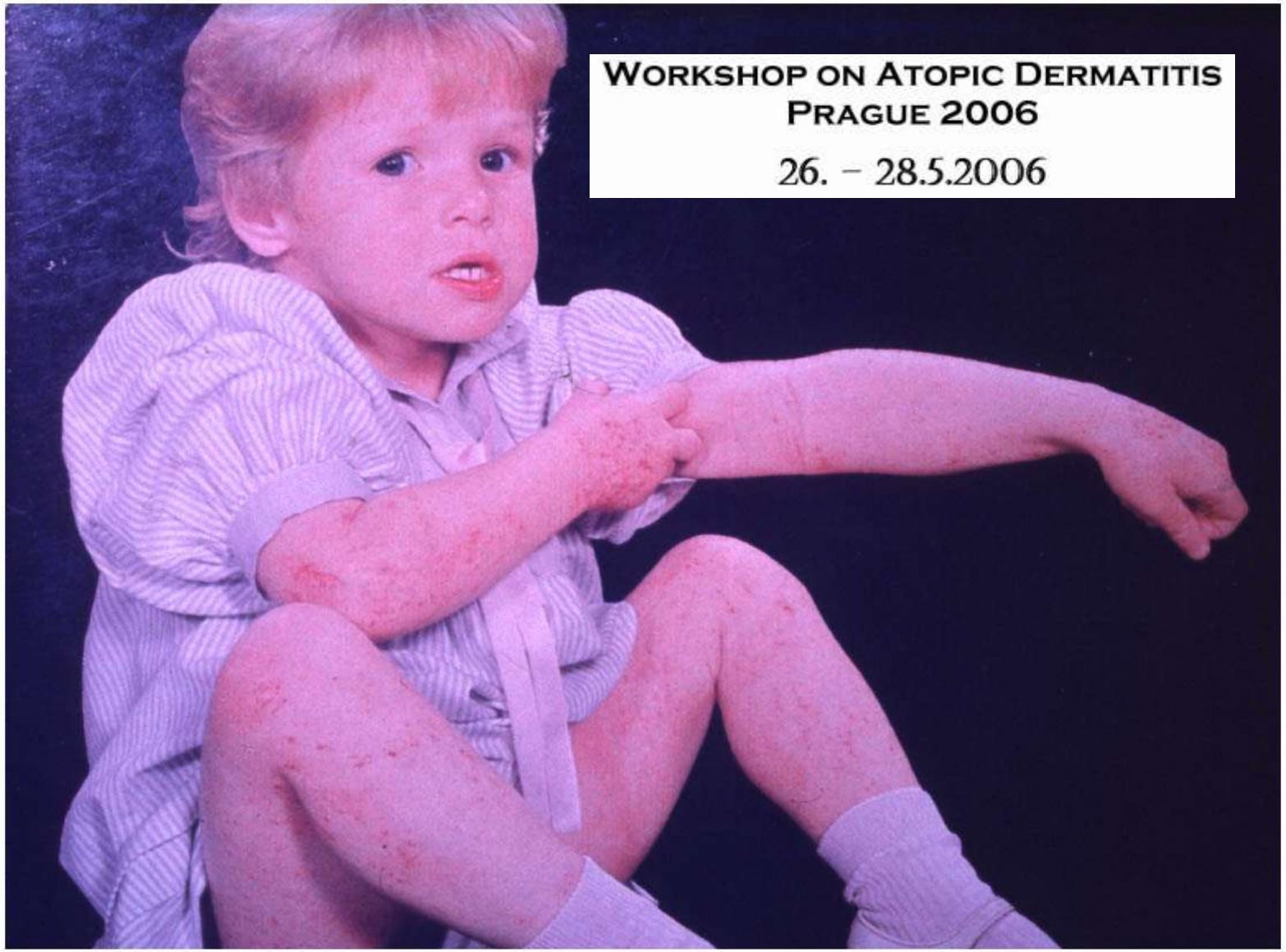
WORKSHOP ON ATOPIC DERMATITIS
PRAGUE 2006
26. – 28.5.2006

Časový program přednášek

Pátek 26. května • Friday May 26th

Čas • Time

15:00-15:15 OFICIÁLNÍ ZAHÁJENÍ KONFERENCE

15:15-15:30 Úvodní slovo k atopické dermatitidě (MUDr. R. Klubal)

15:30-16:15 Prof.MUDr.J.Lokaj, CSc
„Kůže – kompartment imunitního systému"

16:15-17:00 Prof.MUDr.P.Barták, DrSc
„Langerhansovy buňky a atopická dermatitida"

17:00-17:45 Prof.MUDr.H.Tlaskalová-Hogenová, DrSc
„Normální bakteriální flora, alergie a účinky probiotik"

17:45-18:30 As.MUDr.Š.Čapková
„Léčba atopického ekzému z pohledu dermatologa"

18:30-19:15 Prof. RNDr. V. Hořejší, DrSc.
„Regulační lymfocyty T"

19:15 Ukončení přednáškové části 1. dne konference

19:30 Odjezd přistaveným autobusem na společnou večeři v plzeňské restauraci Olympia v Praze 1.

Občanské Sdružení pro alergiky, astmatiky a ekzematiky Máša (MášAlergii?)
Národní 9, 110 00 Praha 1
Tel.: 222 075 133, Fax: 222 075 132
www.masa.cz - info@masa.cz

Sobota 27. května • Saturday, May 27th

Čas • Time

8:45 Zahájení 2. dne konference

9:00-9:30 MUDr. R. Klubal
„Genetické aspekty atopické dermatitidy"

9:30-10:00 Prim.MUDr.N.Benáková
„Diagnostická kriteria pro atopickou dermatitidu".

10:00-10:30 Prim.MUDr.V.Gutová
„SCORAD – klinické hodnocení tíže atopické dermatitidy".

10:30-11:00 **Coffeebreak** /káva, čaj, minerálka, chlebíčky/

11:00-11:30 MUDr.R.Klubal
„Laboratorní vyšetření u atopické dermatitidy".

11:30-12:00 MUDr.I.Nentwich, PhD
„Imunologie mateřského mléka"

12:00-12:30 Prim.MUDr.M.Fuchs
„Alergie na kravské mléko"

12:30-13:30 **Oběd** /formou bufetového menu vč. nápojů/

13:30-14:00 Prof.MUDr.F.Novotný, DrSc
„Balneoterapie - imunomodulační nástroj".

14:00-14:30 Prim.MUDr.J.Nebesař
„Význam komplex. Lázeň. léčby dětí – alergiků, astmatiků a ekzematiků"

14:30-15:00 D.J.Atherton MA MB BChir FRCP
„Systemic treatment for severe atopic eczema in children".

15:00-15:30 Mgr.I.Kudlíková/J.Hubert, PhD
„Přehled o roztočích".

15:30-16:00 Prof.RNDr.J.Krejsek, DrSc
„Staphylococcus aureus a atopický ekzém".

16:00-16:30 MUDr.R.Klubal
„Lymfocyty B, žírné buňky, eosinofily"

16:30-17:00 **Coffeebreak** /káva, čaj, minerálka, koláčky/croisant/

17:00-17:30 Prim.MUDr.M.Selerová
„Psychosomatické aspekty atopické dermatitidy"

17:30-18:00 Uwe Gieler
"Atopic Dermatitis - a neurogenic disease?"
MUDr.R.Klubal/MUDr.A.Vocilková
„Kosmetické aspekty atopické dermatitidy"

18:00-18:30 OFICIÁLNÍ UKONČENÍ KONFERENCE

18:45 Odjezd přistaveným autobusem na koncerty Pražského jara.

Máš alergii?

Máš alergii?
květen 2001

Máš alergii?
květen 2001

Máš alergii?
květen 2001

- ✓ **Máš a**lergii?
- ✓ květen 2001
- ✓ dobrovolníci

M Á Š A

- ✓ **Máš a**lergii?
- ✓ květen 2001
- ✓ dobrovolníci

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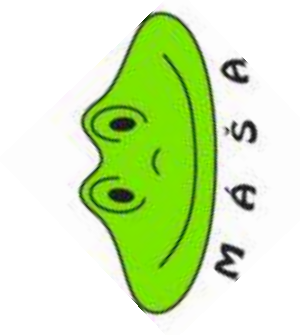
- ✓ květen 2001
- ✓ dobrovolníci

- ✓ **Máš a**lergii?

✓ **Máš a**lergii?

- ✓ **Máš a**lergii?
- ✓ **duben 2005**


MÁŠA

- ✓ Máš alergii?
- ✓ duben 2005
- ✓ Eva Görtlerová

MÁŠA

MÁŠA

Občanské sdruženi pro ekzematiky,
astmatiky a alergiky

Sídlo:
Národní 9
110 00 Praha 1

Spojení:
Tel.: 222 075 133
Fax: 222 075 132

č.ú. u ČSOB:
175640595 / 0300
E-mail: masa@masa.cz
Internet: www.masa.cz

Občanské sdružení „Máša" (Máš alergii) je určeno všem,
kdo mají něco společného s alergiemi, ekzémy nebo astmatem.

Cílem občanského sdružení „Máša" je:

1) usnadnit alergikům jejich každodenní život
   - ➔ poskytování informací (publikace, telefonicky, e-mailem, osobní konzultací, odkazy na odborníky)
   - ➔ pořádání pravidelných seminářů a přednášek pro laickou veřejnost
   - ➔ pořádání pravidelných seminářů a přednášek pro odbornou veřejnost
   - ➔ organizování ozdravných pobytů v ČR i v zahraničí
   - ➔ prosazování a hájení zájmů pacientů ve společnosti
   - ➔ garance kvality výrobků pro alergiky
2) vytvořit skupinu lidí ochotných pomáhat nemocným (lékaři, pacienti, dobrovolníci)
3) podpora výzkumu alergických nemocí

Občanské sdružení pro ekzematiky, astmatiky a alergiky „Máša"
registrované u MV ČR dne 4.5.2001, IČ: 26531640
předseda sdružení a garant projektu: MUDr. Radek Klubal


sevapharma


NUTRICIA


COMMERZBANK


TOMEX


MediPool®


SCHERING
making medicine work


Biopath

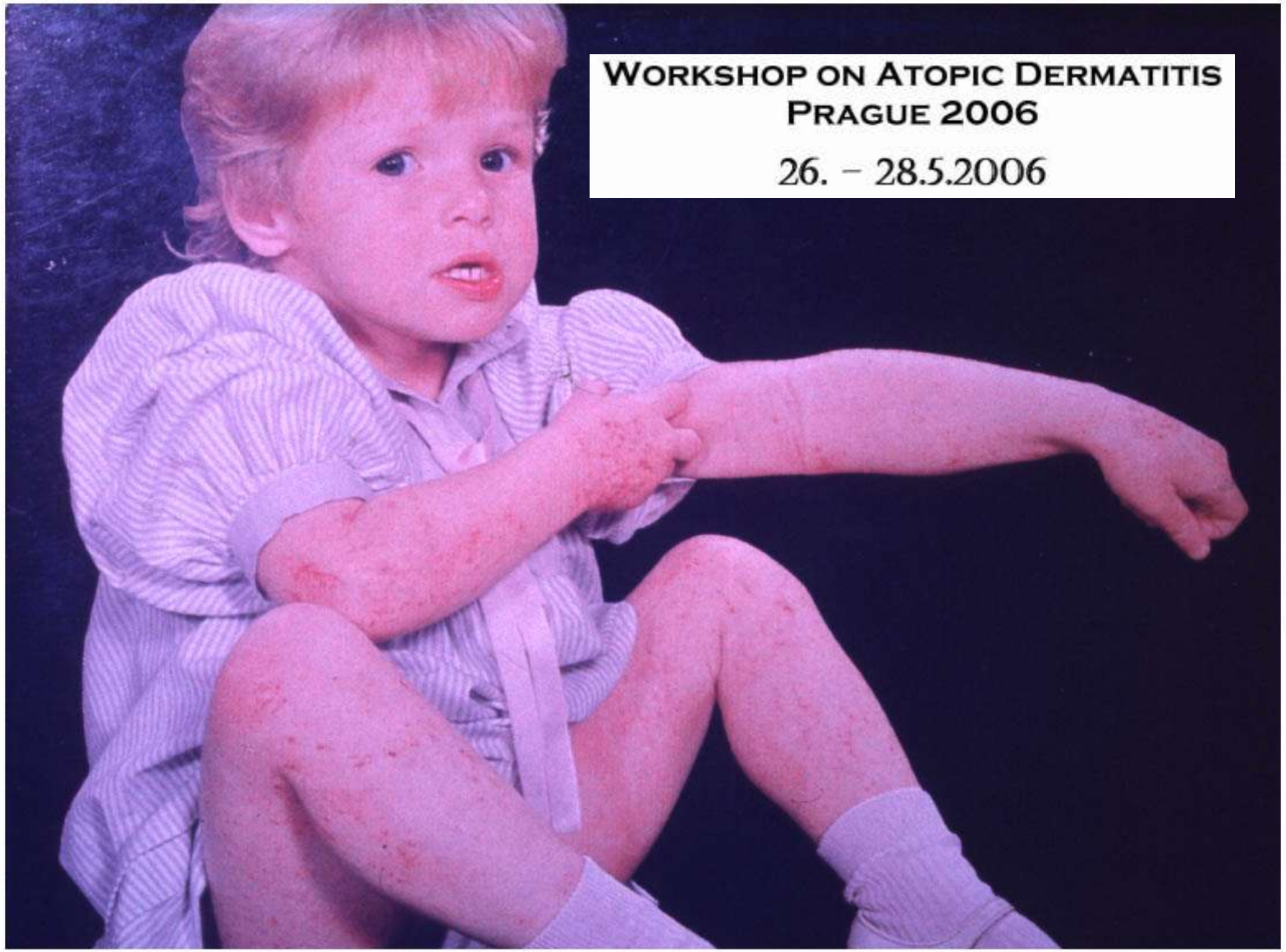

HERMAL


GEUM


nadace duhová energie


MINISTERSTVO
ZDRAVOTNICTVÍ
České republiky


ČESKÁ
SPOŘITELNA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Synonyma

- ✓ **Neurodermitis** (1891)
- ✓ Besnier´s **prurigo** (1903)
- ✓ **Neurodermitis** diffusa (1928)
- ✓ Atopic dermatitis (1933)
- ✓ Endogenous eczema (1954)
- ✓ **Neurodermitis** constitutionalis sive atopica (1967)

- ✓ Fitzpatrick – Atopic Dermatitis (Atopic Eczema) (2003)
- ✓ Braun-Falco – Atopie und atopisches Ekzem (2005)
- ✓ Rajka – 4th Georg Rajka Symposium on Atopic Dermatitis (září 2005)

- ✓ **atopie**
- ✓ **dermatitida/ekzém**
- ✓ **nervový systém**

- ✓ atopie
- ✓ dermatitida/ekzém
- ✓ nervový systém

1990 ----------

- ✓ **atopie**
- ✓ **dermatitida/ekzém**
- ✓ **nervový systém**

1990 ----------

atopie
- astma, rinokonjunktivitida
- imunita
  - IgE
  - antigen-prezentující buňky (LCs)
  - lymfocyty T (Th2 vs. Th1)
  - žírné buňky, eosinofily, basofily
  - Staphylococcus aureus

- ✓ **atopie**
- ✓ **dermatitida/ekzém**
- ✓ **nervový systém**

1990 ----------

atopie
- astma, rinokonjunktivitida
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  - IgE
  - antigen-prezentující buňky (LCs)
  - lymfocyty T (Th2 vs. Th1)
  - žírné buňky, eosinofily, basofily
  - Staphylococcus aureus

dermatitida
- kožní bariera (xeroza)
- nenasycené mastné kyseliny (GLA)

- ✓ **atopie**
- ✓ **dermatitida/ekzém**
- ✓ **nervový systém**

1990 ----------

atopie	- astma, rinokonjunktivitida	
	- imunita	- IgE
		- antigen-prezentující buňky (LCs)
		- lymfocyty T (Th2 vs. Th1)
		- žírné buňky, eosinofily, basofily
		- Staphylococcus aureus
dermatitida	- kožní bariera (xeroza)	
	- nenasycené mastné kyseliny (GLA)	
nervový systém	- pruritus	
	- psychická labilita (neurodermitis)	
	- autonomní nervová dysbalance (bílý dermografismus)	

1990 ----------

2006 ----------

atopie	- astma, rinokonjunktivitida	
	- imunita	- IgE
		- antigen-prezentující buňky (LCs)
		- lymfocyty T (Th2 vs. Th1)
		- žírné buňky, eosinofily, basofily
		- Staphylococcus aureus
dermatitida	- kožní bariera (xeroza)	
	- nenasycené mastné kyseliny (GLA)	
nervový systém	- pruritus (neuropetidy)	
	- psychická labilita (neurodermitis)	
	- autonomní nervová dysbalance (bílý dermografismus)	

1990 ----------

2006 ----------

atopie
- astma, rinokonjunktivitida
- imunita
  - IgE **(pouze 66% AD pacientů)**
  - antigen-prezentující buňky (LCs) **(IDEC, pDCs)**
  - lymfocyty T (Th2 vs. Th1) **(Treg)**
  - **tolernace (probiotika)**
  - žírné buňky, eosinofily, basofily
  - **nespecifická imunita (defensiny, cathelicidiny, TLRs, chemokiny)**
  - Staphylococcus aureus **(Treg)**

dermatitida
- kožní bariera (xeroza) **(ceramidy, fosfolipidy, mastné kyseliny, cholesterol, pH, humidita, profilaggrin, keratohyalin, corneodesmosomy, nespec. imunita)**
- **enzymy (Netherton syndrom LEKTI 1 – anti-proteaza)**
- nenasycené mastné kyseliny (GLA)

nervový systém
- pruritus (neuropeptidy,**neurotrophins NGF,BDNF,NT3,4/5,6,7**)
- psychická labilita (neurodermitis)
- autonomní nervová dysbalance (bílý dermografismus)

4th Georg Rajka International Symposium on Atopic Dermatitis Arcachon, France, 15–17 September 2005

Honorary Presidents: G Rajka (Oslo), K Yamamoto (Tokyo)

Chair: A. Taleb (Bordeaux)

Co-chairs: A. Giannetti (Modena), J. Ring (Munich), K. Thestrup Pedersen (Aarhus)

Local Organizing Committee: A. Taleb, F. Boralevi, C Labrèze (Bordeaux)

Scientific Advisory Board: T Bieber (Bonn), JD Bos (Amsterdam), KD Cooper (Cleveland), M Furue (Fukuoka), G Girolomoni (Verona), J Hanifin (Portland), A Kapp (Hannover), TL Diepgen (Heidelberg), DN Leung (Denver), T Luger (Münster), JF Nicolas (Lyon), AP Oranje (Rotterdam), Y de Prost (Paris), T Reunala (Tampere), JF Stalder (Nantes), M Takigawa (Hamamatsu), K Tamaki (Tokyo), U Wahn (Berlin), HC Williams (Nottingham), B Wüthrich (Zürich)

Scientific Secretariat: F Boralevi, C Labrèze, Pediatric Dermatology Unit, Bordeaux Children's Hospital, France

Website: www.isad2005.org

Wednesday, 14 September 2005: 19.00–21.00: Welcome cocktail

Thursday 15 September 2005

Session 1: Opening/From History To Genetics 8.00–10.30

Opening Session: Chairs: G Rajka & K Yamamoto

Welcome: A Taleb

Session 1: Chairs: J Ring, K Cooper, J Harper

KL1. 8h15–8.45: D Wallach (France): Atopic Dermatitis And Dermatological Doctrines: An Historical Approach

KL2. 8.45–9.15 WOCM Cookson (UK): Genetics And Epigenetics of Atopic Dermatitis

IC1. 9.15–9.35 TL Diepgen (Germany): Worldwide Variation Of Risk Factors In Infants With AD

Oral Communications: Genomics-Epidemiology 9.35–10.30

OC1. Microarray Analysis Of Atopic Skin Lesions. H Sugiura, T Ebise, T Tazawa, K Tanaka, Y Sugiura, M. Uehara, K. Kikuchi, T. Kimura

OC2. Gene-expression Profiling of Lesional and Atopy Patched Tested Skin in Patients with Atopic Dermatitis Using cDNA Microarrays. A Sääf, M Bradley, M Tengvall-Linder, HY Chang, C-F Wahlgren, A Scheynius, M Nordenskjöld, PO Brown

OC3. Positional Cloning of a Susceptibility Gene for Childhood Eczema from Chromosome 1q21. P Edser, T Street, M Taylor, J Broxholme, JI Harper, WOC Cookson, MF Moffatt.

OC4. The Links Between Gut Worms, Malaria, and Atopic Dermatitis: A Study in Rural Vietnam. C Flohr

Related Posters:

P1. Epidemiological Characteristics of Atopic Dermatitis in I[redacted] Kanwar

P2. Worldwide Changes in the Prevalence of Eczema Symptoms. H Williams, A Stewart, E von Mutius, W Cookson, R Anderson and the ISAAC Study Team.

Session 2: Maturation of the Immune System 11h–13h
Chairs: K Thes[redacted]rsen, T Bieber

KL3. 11.00–11.30 PJ Holt (Australia): **Perinatal Maturation of the Immune System: Implications for the Aetiology and Pathogenesis of Atopic Dermatitis and Related Diseases**

KL4. 11.30–12.00 E Isolauri (Finland): **Gut Microbiota and Atopic Dermatitis** [redacted]

Oral Communications: Immunology I (12.00–13.00)

OC5. Numerical and Functional Abnormality of Circulating Plasmacytoid DC in Atopic Dermatitis. H Hashizume, T Horibe, N Ito, T Ito, M Takigawa.

OC6. The Severity of Atopic Dermatitis Correlates to Thymic Function. H Just, M Deleuran, C Vestergaard, B Deleuran, K Thestrup-Pedersen

OC7. Probiotics Provide Clinical Benefit in Moderate and Severe Atopic Dermatitis: A Randomised Controlled Trial. S Weston, JA Dunstan, J Roper, L Breckler, A Halbert, P Richmond, SL Prescott

OC8. Involvement of Innate Conditioning of Antigen Presenting Cells by *Staphylococcus Aureus* Toxin B in Immune Response of Atopi[redacted]s Patients. M Mandron, M-F Aries, F Boralevi F, M Charveron, A Taleb, C Davinche

Related Posters:

P3. Probiotics in the Management of Atopic Dermatitis. JH Y[redacted]n, M-Y Kim, HO Kim, YM Park

P4. The IgE-Bearing B-Cell Receptor Repertoire of Atopic Dermatitis Patients Shows Unbiased VH-Usage but Patient-Specific Clonal Expansions Regardless of Serum Ige Levels. M Mempel, A Gauger, C Schnopp, J Ring, M Ollert, P Kourilsky, A Lim

Session 3: Infection and Immunity 14.30–16.00
Chairs: JD[redacted]icolas, G Imokawa

KL5. 14.30–15.00 D Leung (USA): The Role of Infection in Atopic Dermatitis

IC2. 15.00–15.20 T Werfel (Germany): Inflammatory Reactions To *S Aureus* in AD

Oral Communications: Immunology II 15.20–16.00

OC9. Th2 Cytokines Down-Regulate Cathelicidin Expression and Increase Skin Susceptibility to Viral Infection in Atopic Dermatitis (AD) Patients. MD Howell, M Boguniewicz, JE Streib, C Wong, RL Gallo, DYM Leung

OC10. Microanalysis of Anti-Microbial Peptide, βDefensin-2, in the Stratum Corneum from Atopic Dermatitis (AD) Patients. S Asano, M Kawashima, Y Ichikawa, G Imokawa

OC11. The Balance Between Langerhans Cells and Inflammatory Dendritic Epidermal Cells as a Regulator of Immunogenic and Tolerogenic Immune Responses in Atopic Eczema. N Novak, J-PAllam, B Schlütter-Böhmer, T Bieber, B Kwiek

Related Poster:

P5. Aberrant Blood Dendritic Cells in Atopic Dermatitis. C Lebre, T van Capel, M Kapsenberg, J Bos, E de Jong

Session 4: Epidermal Inflammation Including Neurogenic Inflammation and Pruritus 16.30–18.30
Chairs: A Giannetti, T Luger, U Gieler

KL6. 16.30–17.00: B Homey (Germany): Chemokines in AD

IC3. 17.00–17.20 : U Gieler (Germany) Is Atopic Dermatitis a Neurogenic Inflammatory Disease?

IC4. 17.20–17.40: M Takigawa, S Shirahama, T Sakamoto, H Hashizume (Japan): Anxiety and Atopic Dermatitis

Oral Communications: Epidermal/Neurogenic Inflammation 17.40–18.30 [redacted]

OC12. Intracellular Control Of CTACK/CCL27 (Cutaneous T Cell Attracting Chemokine) in Keratinocytes Through the Nuclear Tran[redacted]Factor Kappa B (NF-kB). C Vestergaard, C Johansen, K Otkjaer, L Iversen, M Deleuran.

OC13. Brain-Derived Neurotrophic Factor Exerts Immunomodulatory Functions in Atopic Dermatitis. U Raap, A Kapp, B Wedi

OC14. Increased Expression and a Potential Anti-Inflammatory Role of TRAIL in Atopic Dermatitis. E. Vassina, M. Leverkus, L. H. [redacted], H-U. Simon and D. Simon

OC15. Graphology and Atopic Dermatitis. C. Gelmetti, G Fabrizi, C Colonna, C Guerriero, P Vizziello, V Tarantino, C. Centofanti and G. Galdo.

Friday 16 September 2005

Session 5: Clinical Research, Prognostic and Severity Markers 8.00–10.30 [redacted]
Chairs: J Hanifin, T David, C Gelmetti

KL7. 8.00–8.30: T Bieber (Germany): A Novel View on the Natural History of Atopic Dermatitis

IC5. 8.30–8.50: P Schmid-Grendelmeier et al (Switzerland): Autoreactivity in Atopic Dermatitis—Induced by Skin Fungi?

Oral Communications: Clinical Research 8.50–10.30

OC16. Expression of Thymic Stromal Lymphopoietin (TSLP) in Keratinocytes of Atopic Dermatitis Patients and Normal Controls. CO Park, WW Hao, J H Lee, KH Lee

OC17. Identification of *Malassezia Sympodialis* in Patients with Atopic Dermati[redacted]ymerase Chain Reaction and its Impact on Disease Activity. A Roll, N Juricevic, P Schmid-Grendelmeier

OC18. High Concentrations of Circulating Macrophage Migration Inhibitory Factor in Patients with 'Extrinsic' Atopic Dermatitis. J-S Kim, D-S Yu, J-W Kim

OC19. Elevated Serum Levels of I-309/CCL1 in Patients with Severe Atopic Dermatitis. N Higashi, Y Niimi, Y Kato, S Kawana

OC20. Serum Levels of IL-16 and Disease Activity in Children with Atopic Dermatitis. B Pigozzi, E Tonin, A Belloni Fortina

OC21. Effect of Caring for a Child with Atopic Dermatitis and Asthma on Parental Sleep, Depression and Anxiety Scores: A Prospective Comparati[redacted]y. K Moore, TJ David, CS Murray, HF Child, PD Arkwright

OC22. Flare Cycles, Itch-Scratch Loops and Associated Downturns in QoL: The Human and Economic Burden of Atopic Dermatitis on Patients and Caregivers F Turk

Related Posters:

P6. Comparative Efficacy of Hanifin and Rajka's Criteria and U.K. Working Party's Diagnostic Criteria in Diagnosis of Atopic Dermatitis in a Hospital Setting. A.J. Kanwar

P7. Atopic Dermatitis & The Adolescent Patient. A Taïeb on behalf of the ISOLATE study group

Session 6: Animal Models 11.00–12.30
Chairs: A K[redacted]rue, H de Verneuil

KL8. 11.00–11.30: T Olivry (USA): Canine AD

IC6. 11.30–11.50: P Verzaal, AP Oranje, L van der Fits, P Jäger, P Rensen, L Havekes, E Prens, L Nagelkerken (The Netherlands): Spontaneous Dermatitis in Mice Transgenic for Human Apolipoprotein C1

Oral Communications: Mouse Models 11.50–12.30

OC23. Establishment of a Mouse Model for Atopic Dermatitis : Getting New Insights into the Role of T Cells. A Hennino, J Benetière, K Rodet, F Berard, M. Vocanson, A-M Schmitt, M-F Aries, JF Nicolas

OC24. Collared Mice: A Model to Assess the Effects of Scratching. S Takeuchi, F Takeuchi, M Furue, SI Katz

OC25. Rapid and Specific Acoustic Analysis of Itch in AD Model Mouse. H Mizutani, K Umeda, K Tokime, Y Omoto

Session 7: Skin Barrier 14.00–16.00
Chairs: JF S[redacted]irolomoni

KL9. 14.00–14.30 A. Hovnanian (France): Netherton Syndrome as a Model for Skin Barrier Dysfunction

IC7. 14.30–14.50 JP Hachem (Belgium): Stratum Corneum pH Regulates Permeability Barrier Homeostasis

IC8. 14.50–15.10 M Brattsand, K Stefansson, T Egelrud. (Sweden): Kallikreins in the Stratum Corneum

Oral Communications: Skin Barrier 15.10–16.00

OC26. Epicutaneous Sensitization to Aeroallergens in Infantile Atopic Dermatitis: Determining the Role of Epidermal Barrier Impairment. F Boralevi, T Hubiche, C Léauté-Labrèze, E Saubusse, S Maurice-Tison, A Taïeb.

OC27. Re-Characterization of the Non-Lesional Skin in Association with Barrier Function and the Severity of Atopic Dermatitis. H Matsuki, K Kiyokane, T Matsuki, S Sato, G Imokawa

OC28. Re-Evaluation of the Importance of Barrier Dysfunction in the Non-Lesional Dry Skin of Atopic Dermatitis: Analysis by Topical Application of a Barrier Cream. T Matsuki, S Sato, H Matsuki, K Kiyokane, G Imokawa,

Related Posters:

P8. Skin Barrier Damage: Cause or Conseq Atopic Dermatitis ? MJ Cork, D Robinson, Y Vas A Ferguson, M Moustafa, R Tazi-Ahnini, SJ Ward

P9. Epidermal Abnormalities Underlying Defec rier in Netherton Syndrome. P Descargues, S Fraitag, J Mazereeuw, G Zambruno, C Bo Hovnanian

Session 8: Evidence-Based Therapy, Educa Quality of Life 16.30–18.30
Chairs: Y de Prost, P Schmid-Grendelmeier, T D

KL10. 16.30–17.00 H Williams (UK): Updating the Systematic Review on Evidence-Based Treatment ic Dermatitis

IC9. 17.00–17.20 T Werfel, U Gieler (Germany) an man Atopic Dermatitis Intervention Study (GADI The German Multicenter Trial of Education in At matitis.

Oral Communications: Education/Eviden Treatments 17.20–18.00

OC29. Parental Education in the Long-Term ment in Childhood Atopic Dermatitis. K-B Suhr J-S Yoon, Y-K Kim, M-S Jang, J-H Lee, J-K Park

OC30. An Audit of the Impact of a Consultati Paediatric Dermatology Team on Quality of L fants with Atopic Eczema and Their Families Validation of the Infants' Dermatitis Quality of l and Dermatitis Family Impact Score. PE Be Lewis-Jones

OC31. Comparative Trial of Topical Corticost Atopic Dermatitis. T Uenishi, H Sugiura, T Tanaka,

Related Posters:

P10. Atopic Dermatitis and Cancer Risk. H Diepgen

P11. Methotrexate Treatment of Atopic Derr Goujon, N Saad, I Guillot, A Hennino, F Bérard, J

Gala Evening at the "Tir au Vol", Arcacho Pereire

Saturday 17 September

Session 9: European Task Force on Atopic D (ETFAD) Workshop on Allergy Testing in Atopi titis D 8.00–10.00
Chairs: U Darsow, F Rancé, T Werfel

OC32. Atopic Eczema And Malassezia. A (Sweden)

OC33. Studies with Aeroallergen Atopy Patch Tests. U Darsow, J Ring (Germany) [redacted]

OC34. Food Atopy Patch Test and Repeated Food Challenge. S Seidenari, F Giusti (Italy)

OC35. SAFT and APT Using Fresh Foods in Children with Atopic Dermatitis and Food Allergy. AP Oranje, ACA Devillers, PG Mulder, FB de Waard-van der Spek. (The Netherlands)

OC36. Atopy Patch Test with Foods. K Turjanmaa (Finland)

OC37. The Geneva experience with Epicutaneous Tests 2000–2005 in Atopic Children. J. Lübbe, A. Gkomouzas, A-M. Calza (Switzerland)

OC38. The Labial Food Challenge in Children with Atopic Eczema. F Rancé[redacted]

Discussion of Related Posters (10.00–10.30)

P12. Is the Labial Food Challenge a Useful Tool in the Management of Food Allergy in Children with Atopic Dermatitis? F Boralevi, T Hubiche, S Roul, C Léauté Labrèze, A Taïeb.

P13. The Comparison of Atopy Patch Test with Skin Prick Test in Korean Patients with Atopic Dermatitis. K-B Suhr, Y-S Kim, J-S Yoon, E-J Oh, E-H Lee, J-H Lee, J-K Park

P14. Gene Expression Change of PBMC using IGEC (Immune [redacted]ression Chip) by Milk Stimulation Test in Milk Allergy of Atopic Dermatitis. GW Noh, S Choi

P15. Follow-Up Study of 92 Infants with Multiple Food Allergies. K Turjanmaa, A-R Ketvell

P16. Alternaria Alternata Patch Tests on a Study Population of 500 Atopic Dermatitis Patients. F Giusti, S Seidenari

P17. Allergic Sensitization to Common Inhalant Allergens and the Association with Atopic Diseases: Results of a Population Based in Elderly. M Wolkewitz, D Rothenbacher, M Löw, C Stegmaier, H Ziegler, H Wang, H Brenner, TL Diepgen

Session 10: New Frontiers in Therapy 11.00–12.30
Chairs: D Atherton, A Oranje, M Takigawa

KL 11. 11.00–11.30 M Cork (UK): Treatment of Atopic Dermatitis from a Skin Barrier Perspective

Oral Communications: Therapy 11.30–12.30 [redacted]

OC39. What Causes Flares of Atopic Eczema? SM Langan, HC Williams

OC40. Differences in Percutaneous Absorption in Normal and Atopic Dermatitis Skin in Relation to the Molecular Weight. I Jakasa, M Verberk, M Esposito, JD Bos, S Kezic

OC41. N-3/N-6 Polyunsaturated Fatty Acids in a Group of Patients with Recalcitrant Atopic Dermatitis and the Influence of Balanced Japanese Traditional Diet. H Kobayashi, D Tsuruta, M Nanatsue, K Hirai, M Ishii

Related Posters:

P18. Evaluation of the Corticosteroid-Sparing Effect of an Emollient Milk in a Large Population of Infants Affected by Atopic Dermatitis. M Josse, V Mengeaud, V Durosier, V Sibaud, R Grimalt, F Cambazard

P19. Refractory Atopic Dermatitis Associated with Cobalamine Deficiency Treated with a Single B12 Injection. M Nabavi

P20. Does Prolonged Topical Application of Tacrolimus in Children Result in Systemic Accumulation? E Tonin, B Pigozzi and A Belloni Fortina

Close of Meeting

The page costs of the printing of the program and abstracts have been sponsored by an educational grant of Novartis Pharma.

Časový program přednášek

Pátek 26. května • Friday May 26th

Čas • Time

15:00-15:15 OFICIÁLNÍ ZAHÁJENÍ KONFERENCE

15:15-15:30 Úvodní slovo k atopické dermatitidě (MUDr. R. Klubal)

15:30-16:15 Prof.MUDr.J.Lokaj, CSc
„Kůže – kompartment imunitního systému"

16:15-17:00 Prof.MUDr.P.Barták, DrSc
„Langerhansovy buňky a atopická dermatitida"

17:00-17:45 Prof.MUDr.H.Tlaskalová-Hogenová, DrSc
„Normální bakteriální flora, alergie a účinky probiotik"

17:45-18:30 As.MUDr.Š.Čapková
„Léčba atopického ekzému z pohledu dermatologa"

18:30-19:15 Prof. RNDr. V. Hořejší, DrSc.
„Regulační lymfocyty T"

19:15 Ukončení přednáškové části 1. dne konference

19:30 Odjezd přistaveným autobusem na společnou večeři v plzeňské restauraci Olympia v Praze 1.

Občanské Sdružení pro alergiky, astmatiky a ekzematiky Máša (MášAlergii?)
Národní 9, 110 00 Praha 1
Tel.: 222 075 133, Fax: 222 075 132
www.masa.cz - info@masa.cz

Sobota 27. května • Saturday, May 27th

Čas • Time

8:45 Zahájení 2. dne konference

9:00-9:30 MUDr. R. Klubal
„Genetické aspekty atopické dermatitidy"

9:30-10:00 Prim.MUDr.N.Benáková
„Diagnostická kriteria pro atopickou dermatitidu".

10:00-10:30 Prim.MUDr.V.Gutová
„SCORAD – klinické hodnocení tíže atopické dermatitidy".

10:30-11:00 **Coffeebreak** /káva, čaj, minerálka, chlebíčky/

11:00-11:30 MUDr.R.Klubal
„Laboratorní vyšetření u atopické dermatitidy".

11:30-12:00 MUDr.I.Nentwich, PhD
„Imunologie mateřského mléka"

12:00-12:30 Prim.MUDr.M.Fuchs
„Alergie na kravské mléko"

12:30-13:30 **Oběd** /formou bufetového menu vč. nápojů/

13:30-14:00 Prof.MUDr.F.Novotný, DrSc
„Balneoterapie - imunomodulační nástroj".

14:00-14:30 Prim.MUDr.J.Nebesař
„Význam komplex. Lázeň. léčby dětí – alergiků, astmatiků a ekzematiků"

14:30-15:00 D.J.Atherton MA MB BChir FRCP
„Systemic treatment for severe atopic eczema in children".

15:00-15:30 Mgr.I.Kudlíková/J.Hubert, PhD
„Přehled o roztočích".

15:30-16:00 Prof.RNDr.J.Krejsek, DrSc
„Staphylococcus aureus a atopický ekzém".

16:00-16:30 MUDr.R.Klubal
„Lymfocyty B, žírné buňky, eosinofily"

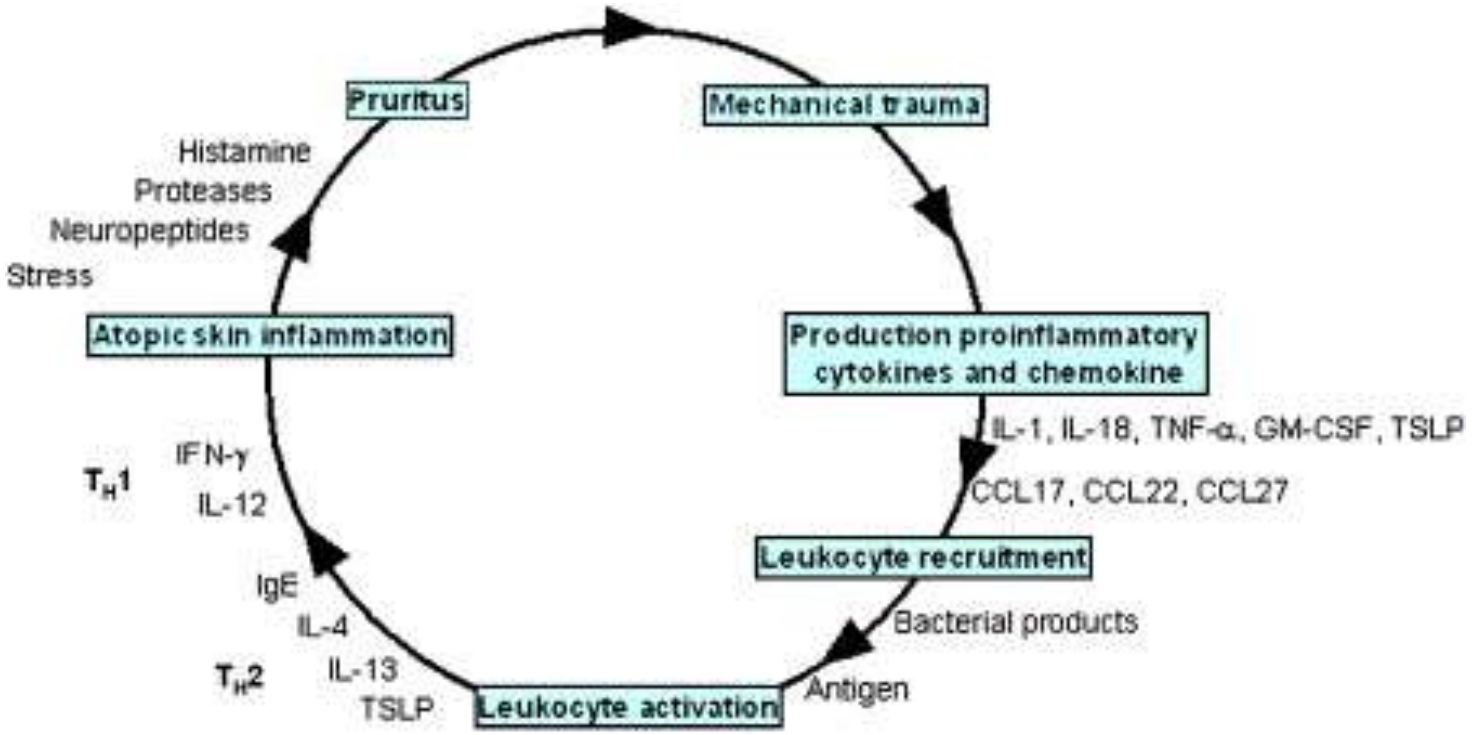
16:30-17:00 **Coffeebreak** /káva, čaj, minerálka, koláčky/croisant/

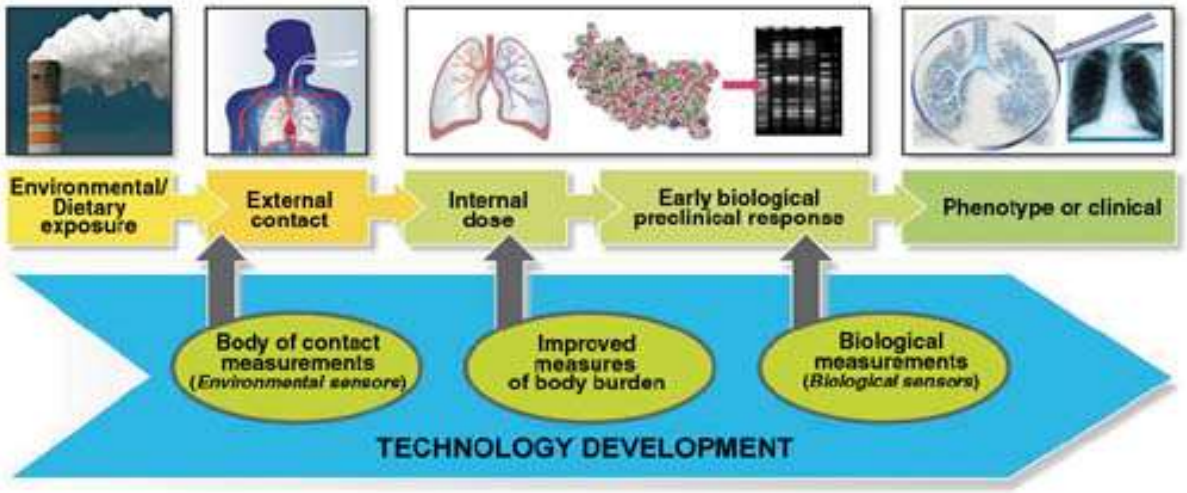
17:00-17:30 Prim.MUDr.M.Selerová
„Psychosomatické aspekty atopické dermatitidy"

17:30-18:00 Uwe Gieler
"Atopic Dermatitis - a neurogenic disease?"
MUDr.R.Klubal/MUDr.A.Vocilková
„Kosmetické aspekty atopické dermatitidy"

18:00-18:30 OFICIÁLNÍ UKONČENÍ KONFERENCE

18:45 Odjezd přistaveným autobusem na koncerty Pražského jara.


Pruritus
Mechanical trauma
Histamine
Proteases
Neuropeptides
Stress
Atopic skin inflammation
Production proinflammatory cytokines and chemokine
IL-1, IL-18, TNF-α, GM-CSF, TSLP
CCL17, CCL22, CCL27
Leukocyte recruitment
Bacterial products
Antigen
Leukocyte activation
IgE
IL-4
IL-13
TSLP
T_H2
IFN-γ
IL-12
T_H1


Environmental/ Dietary exposure
External contact
Internal dose
Early biological preclinical response
Phenotype or clinical
Body of contact measurements (Environmental sensors)
Improved measures of body burden
Biological measurements (Biological sensors)
TECHNOLOGY DEVELOPMENT

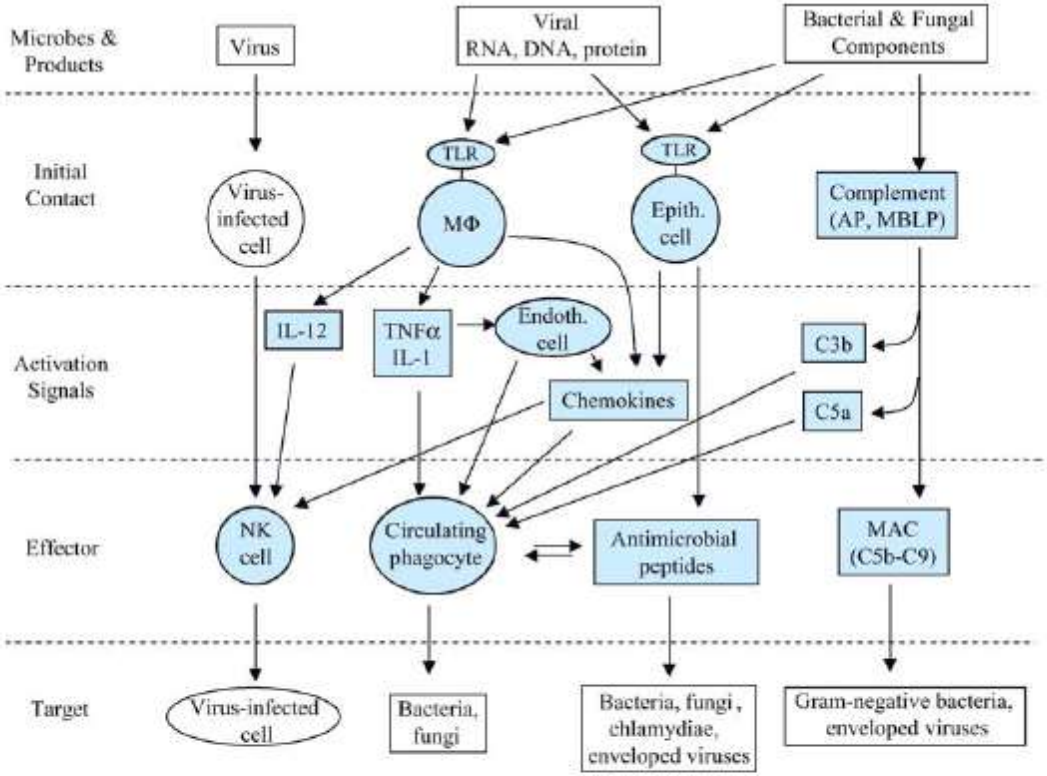


FIG 1. Innate immunity: responses to first contact. Diagrammed are important host responses to infection that are independent of specific cell-mediated immunity or antibodies. Initial contact between the host and microbes or their products results in a range of activating signals that mobilize both cellular and humoral effectors for attack on their respective microbial targets. Components of the host response are highlighted in blue. *MΦ*, Macrophages; *AP*, alternative pathway; *MBLP*, mannose-binding lectin pathway.

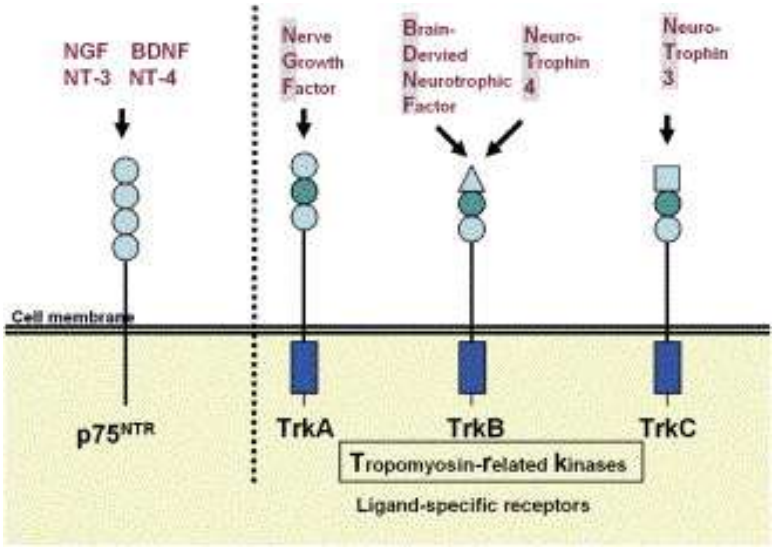
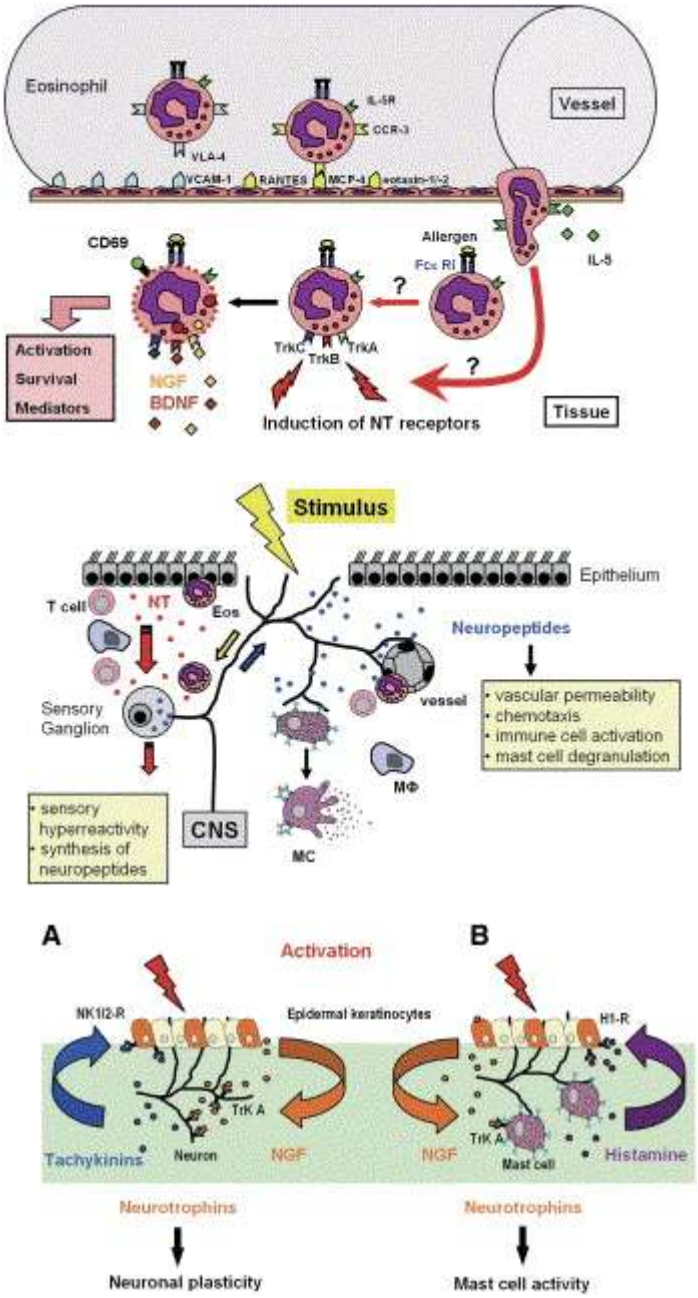
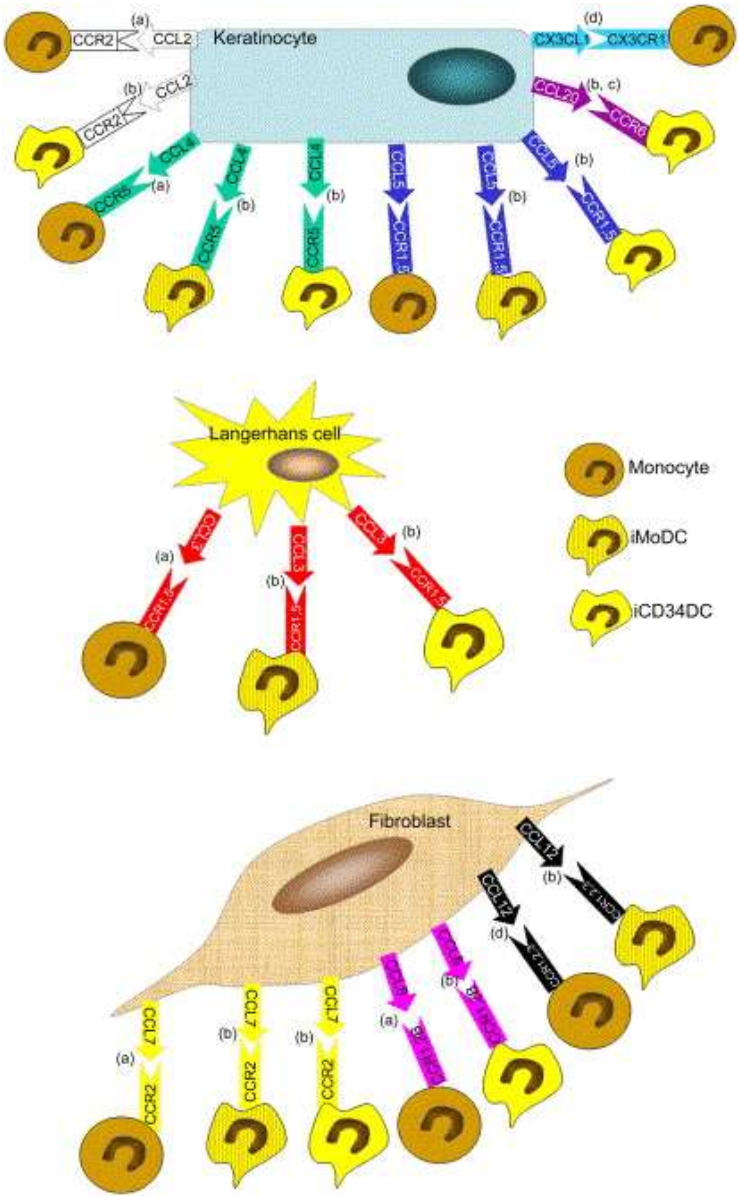
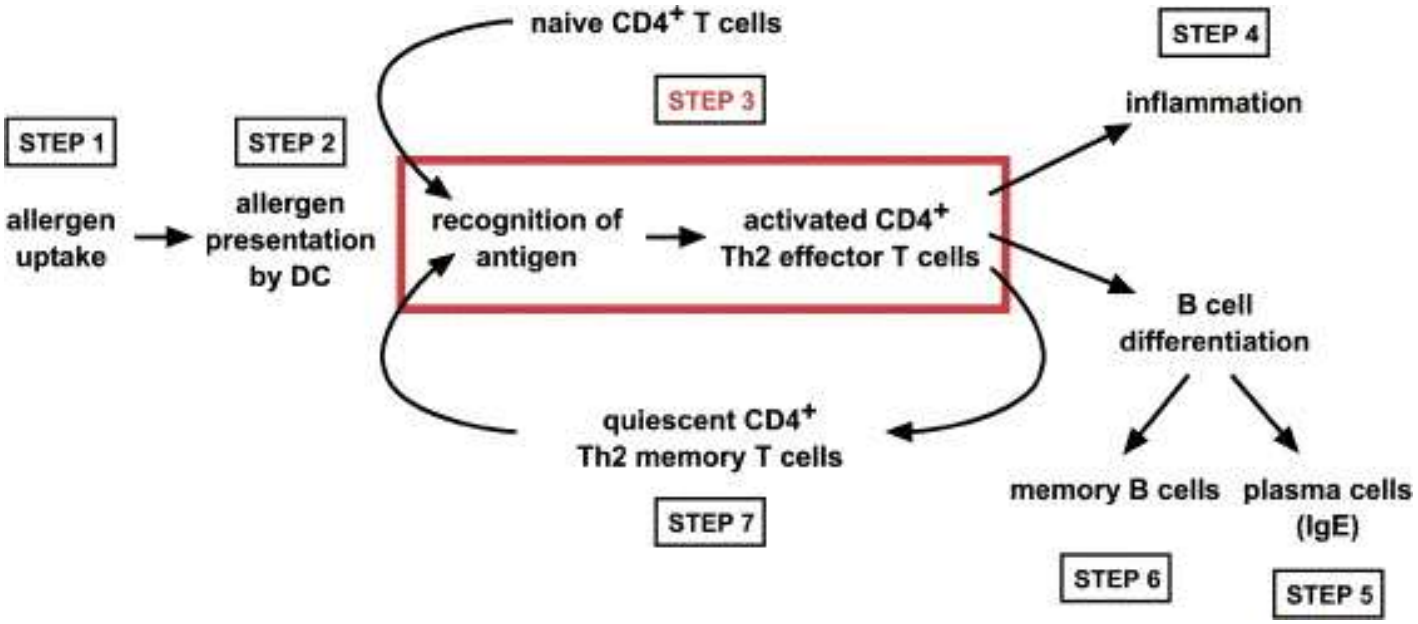


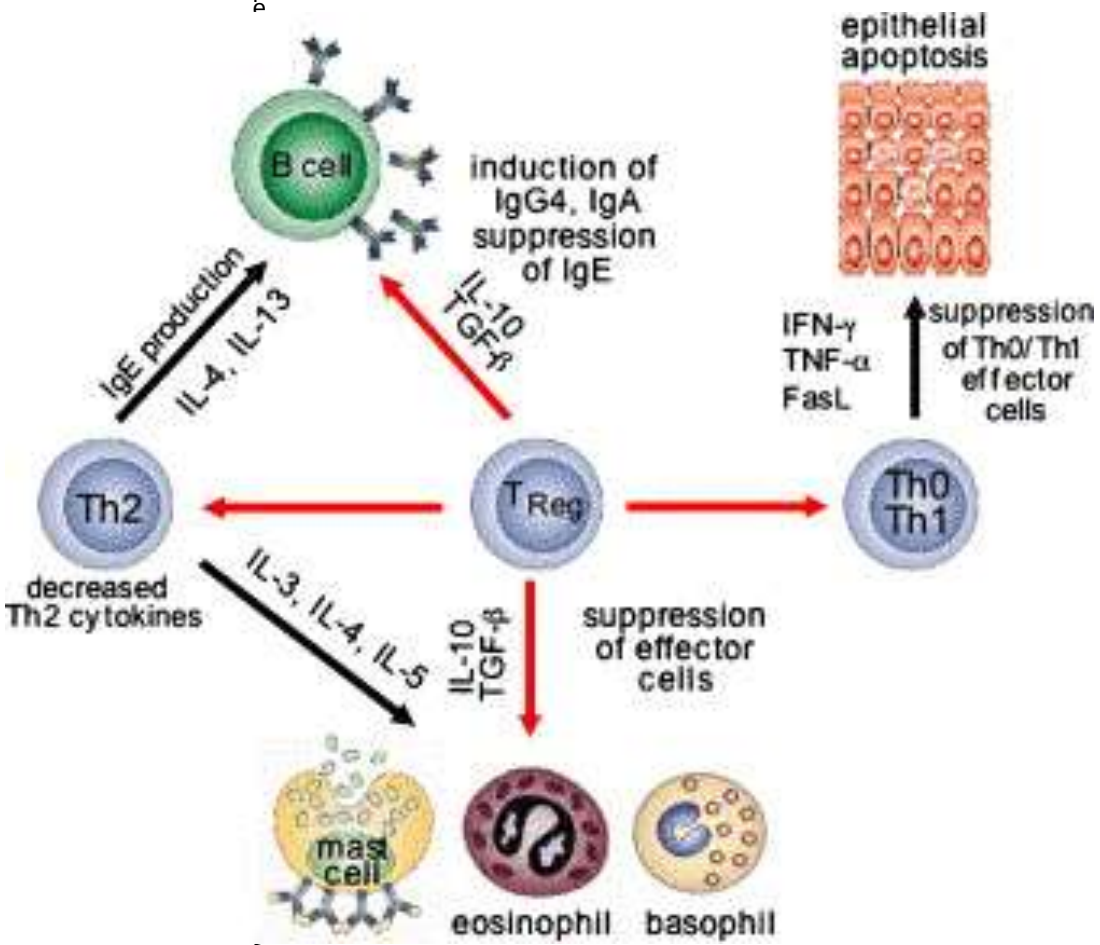
TABLE I. Neurotrophins in allergic diseases

Neurotrophin	Allergic disease	Effects and observations
NGF	Asthma	Increased expression in the lung tissue Elevated levels in • blood • bronchoalveolar fluid Enhancement of • airway inflammation • neural hyperresponsiveness • early-phase reaction
	Rhinitis	Elevated levels in nasal fluid Enhancement of neural hyperresponsiveness
	Contact eczema	Nerve fiber sprouting
	Atopic dermatitis	Elevated blood levels Increased expression in skin lesions
BDNF	Asthma	Elevated levels in • blood • bronchoalveolar fluid Correlation of blood levels with airflow limitation Increased expression in airway epithelia Enhancement of neural hyperresponsiveness
	Atopic dermatitis	Elevated blood levels
NT-3	Asthma	Elevated levels in bronchoalveolar fluid
NT-4	Atopic dermatitis	Increased expression in keratinocytes


Eosinophil
Vessel
VLA-4
IL-5R
CCR-3
VCAM-1
RANTES
MCP-4
eotaxin-1/-2
IL-5
Allergen
FcεRI
?
CD69
TrkC
TrkB
TrkA
NGF
BDNF
Activation
Survival
Mediators
Induction of NT receptors
Tissue
Stimulus
Epithelium
T cell
NT
Eos
Neuropeptides
vascular permeability
chemotaxis
immune cell activation
mast cell degranulation
Sensory Ganglion
vessel
MΦ
sensory hyperreactivity
synthesis of neuropeptides
CNS
MC
A
Activation
B
NK1/2-R
Epidermal keratinocytes
H1-R
TrK A
Tachykinins
Neuron
NGF
NGF
TrK A
Mast cell
Histamine
Neurotrophins
Neurotrophins
Neuronal plasticity
Mast cell activity


Keratinocyte
CCR2
CCL2
(a)
(b)
(d)
CX3CL1
CX3CR1
CCL20
(b, c)
CCR6
CCL4
CCR5
CCL5
CCR1,5
Langerhans cell
Monocyte
iMoDC
iCD34DC
Fibroblast
CCL7
CCR2


naive CD4+ T cells
STEP 4
STEP 3
inflammation
STEP 1
STEP 2
allergen uptake
allergen presentation by DC
recognition of antigen
activated CD4+ Th2 effector T cells
B cell differentiation
quiescent CD4+ Th2 memory T cells
memory B cells
plasma cells (IgE)
STEP 7
STEP 6
STEP 5


epithelial apoptosis
B cell
induction of IgG4, IgA suppression of IgE
IL-10 TGF-β
IgE production
IL-4, IL-13
IFN-γ
TNF-α
FasL
suppression of Th0/Th1 effector cells
Th2
T Reg
Th0 Th1
decreased Th2 cytokines
IL-3, IL-4, IL-5
IL-10 TGF-β
suppression of effector cells
mast cell
eosinophil
basophil

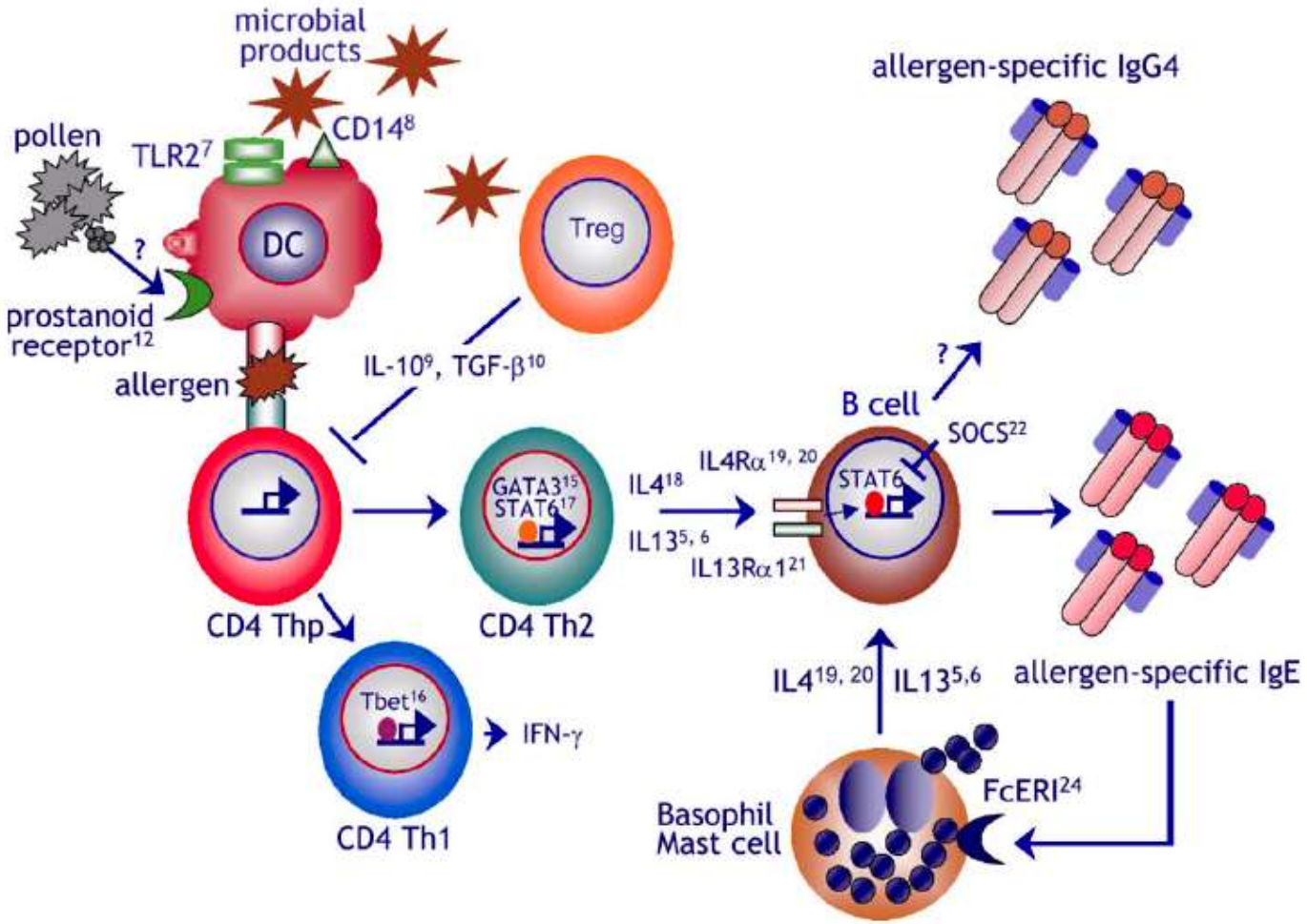


FIG 1. Gene-gene interactions and the pathogenesis of allergic inflammation: a working roadmap. Groups of interactions are color coded: the regulatory and sensing interfaces are in *red/orange*, T_H differentiation is in *blue/green*, and the effector phase is in shades of *brown*. See text for an in-depth discussion of individual pathways and genes. References are numbered as in the text.

Absence of T-regulatory cell expression and function in atopic dermatitis skin

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Background: The role of regulatory T cells has been widely reported in the suppression of T-cell activation. A dysfunction in $CD4^+CD25^+$ T-regulatory cell–specific transcription factor FoxP3 leads to immune dysregulation, polyendocrinopathy, enteropathy X-linked syndrome, often associated with atopic dermatitis. Increasing the number and activity of regulatory T cells in affected organs has been suggested as a remedy in various inflammatory diseases, including allergy.
Objective: To determine the presence and function of regulatory T cells in atopic dermatitis.
Methods: Immunohistochemistry of lesional atopic dermatitis skin and control skin conditions was used to demonstrate regulatory cells and cytokines in situ. The role of effector and regulatory T cells as well as their specific cytokines in apoptosis in human keratinocyte cultures and artificial skin equivalents was investigated.
Results: Human T-regulatory type 1 cells, their suppressive cytokines, IL-10 and TGF-β, as well as receptors for these cytokines were significantly expressed, whereas $CD4^+CD25^+FoxP3^+$ T-regulatory cells were not found in lesional and atopy patch test atopic dermatitis or psoriasis skin. Both subsets of regulatory T cells suppress the allergen-specific activation of T_H1 and T_H2 cells. In coculture and artificial skin equivalent experiments, subsets of T-regulatory cells neither induced keratinocyte death nor suppressed apoptosis induced by skin T cells, T_H1 cells, IFN-γ, or TNF-α.
Conclusion: A dysregulation of disease-causing effector T cells is observed in atopic dermatitis lesions, in association with an impaired $CD4^+CD25^+FoxP3^+$ T-cell infiltration, despite the expression of type 1 regulatory cells in the dermis. (J Allergy Clin Immunol 2006;117:176-83.)

Key words: Regulatory T cell, atopic dermatitis, apoptosis, suppression, regulation, skin, human, inflammation

Abbreviations used
AD: Atopic dermatitis
APT: Atopy patch test
FasL: Fas ligand
HDM: House dust mite
NAD: Nonallergic type of dermatitis
Tr1: T-regulatory type 1
Treg: T-regulatory

Atopic dermatitis (AD) is a chronic relapsing skin disorder with an interplay of migrating lymphocytes and epidermal keratinocytes (KC).[1,2] Lesional AD skin is histologically characterized by dermal mononuclear infiltration and spongiosis in the epidermis. At the initial stages of inflammation, T_H2 cells migrate to the dermis, where they acquire a T_H0/T_H1 phenotype under the influence of IL-12, produced by antigen-presenting cells or activated keratinocytes.[3-5] These T_H0/T_H1 cells are characterized by the expression of Fas ligand (FasL) and secretion of significant amounts of the effector cytokines TNF-α and IFN-γ.[2,4,5] The secreted IFN-γ induces apoptosis of keratinocytes, leading eventually to the eczematous lesions characteristic of AD.[6,7] In response, keratinocytes upregulate the production of IFN-γ–inducible chemokines,[8] which in turn promotes the further infiltration of T cells into the epidermis, thereby augmenting the severity of inflammation and keratinocyte apoptosis.

After their initial discovery in the early 1970s, the concept of T-regulatory (Treg) cell–mediated immune suppression has been extensively explored. Two main groups of Treg cells have been defined. One comprises the natural Treg cells, which are characterized by their $CD4^+CD25^+$ phenotype. These cells have been suggested to develop under the control of the transcription factor FoxP3.[9] The other group of Treg cells, the adaptive Treg or T-regulatory type 1 (Tr1), are characterized by the secretion of high levels of IL-10 with or without TGF-β.[10-12] They develop in the periphery under the influence of presumably immature dendritic cells[13] and/or the presence of IL-10 and TGF-β, but also immunosuppressive drugs like glucocorticoids and vitamin D3,[14] and operate in a cytokine-mediated manner.

Most research on the inhibitory capacities of Treg cells has focused on their ability to suppress proliferation of effector T cells. It has been tempting to speculate that migration of increased numbers of Treg cells to the inflammation area, or the induction of their local proliferation, might be beneficial in the treatment of several

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Authors' laboratories supported by Swiss National Science Foundation grants No. 31-105865 and 32-100266 and the Global Allergy and Asthma European Network (GA2LEN).
Received for publication July 11, 2005; revised October 20, 2005; accepted for publication October 28, 2005.

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0091-6749/$32.00

doi:10.1016/j.jaci.2005.10.040

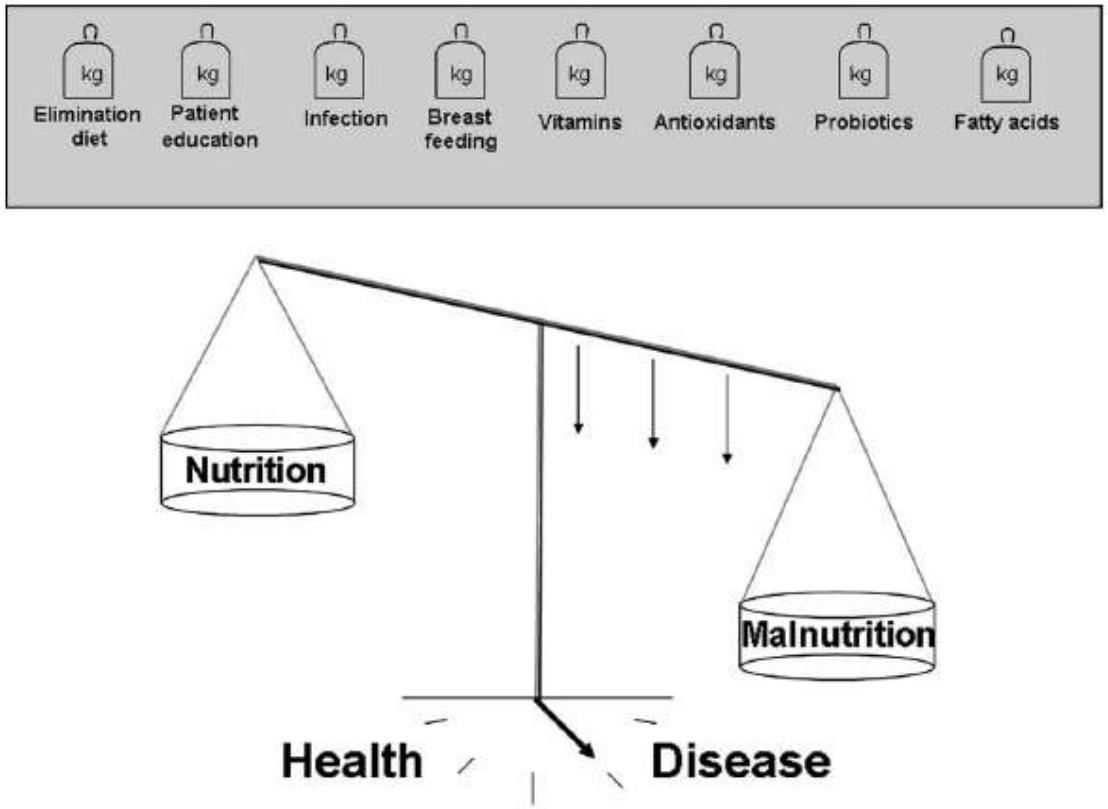

FIG 1. The balance between nutrition and malnutrition and the factors that preponderate on the outcome of health and disease in this interplay are shown.