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MUDr. Radek Klubal

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What does NCBI do?

Established in 1988 as a national resource for molecular biology information, NCBI creates public databases, conducts research in computational biology, develops software tools for analyzing genome data, and disseminates biomedical information - all for the better understanding of molecular processes affecting human health and disease. More...

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The NCBI Whole Genome Association (WGA) resource provides researchers with access to genotype and associated phenotype information that will help elucidate the link between genes and disease. For more information, click here to see the the WGA resource page and click here to read the press release.

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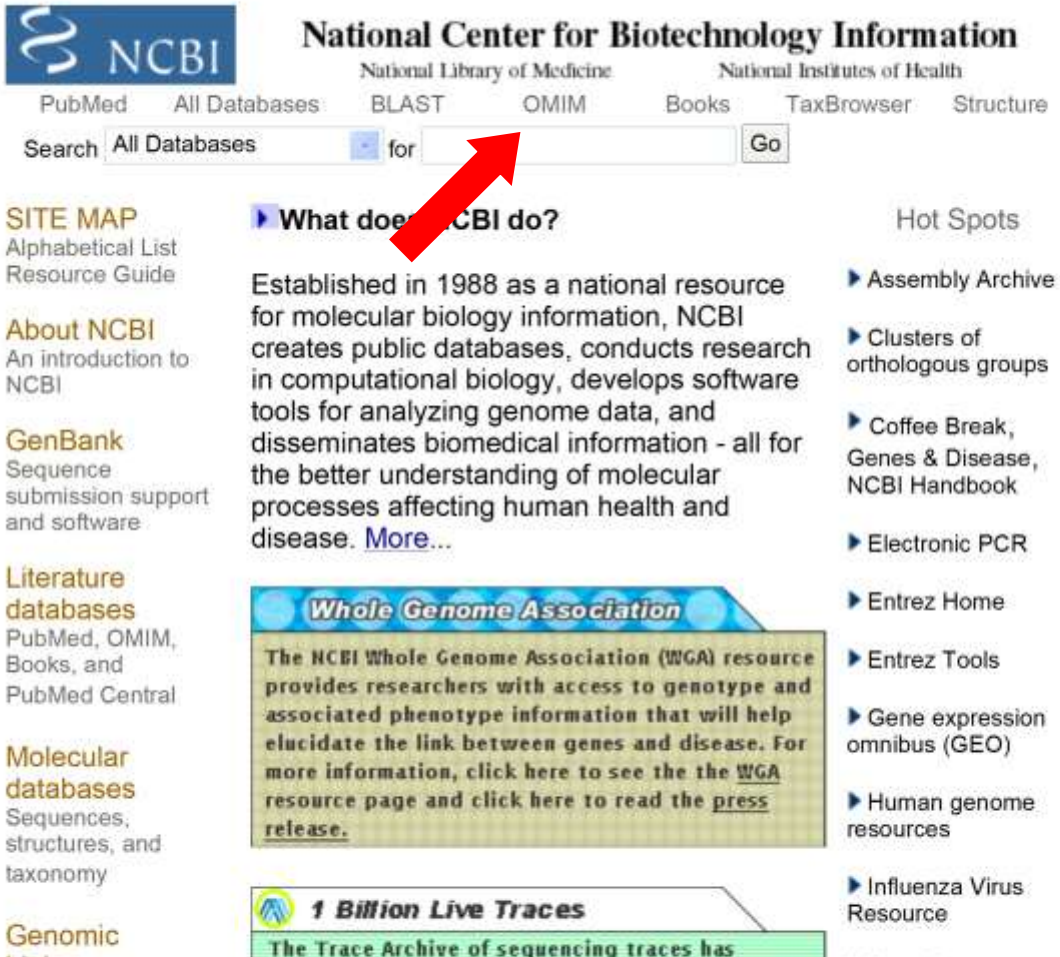

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OMIM™ - Online Mendelian Inheritance in Man™

Welcome to OMIM, Online Mendelian Inheritance in Man. This database is a catalog of human genes and genetic disorders authored and edited by Dr. Victor A. McKusick and his colleagues at Johns Hopkins and elsewhere, and developed for the World Wide Web by NCBI, the National Center for Biotechnology Information. The database contains textual information and references. It also contains copious links to MEDLINE and sequence records in the Entrez system, and links to additional related resources at NCBI and elsewhere.

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The links to the left provide further technical information, searching options, frequently asked questions (FAQ), and information on allied resources. To return to this page, click on the OMIM link in the black header bar or on the graphic at the top of any OMIM page.

NOTE: OMIM is intended for use primarily by physicians and other professionals concerned with genetic disorders, by genetics researchers, and by advanced students in science and medicine. While the OMIM database is open to the public, users seeking information about a personal medical or genetic condition are urged to consult with a qualified physician for diagnosis and for answers to personal questions.

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1: %603165 Links
DERMATITIS, ATOPIC
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Gene map locus 20p, 17q25, 13q12-q14, 5q31-q33, 3q21

2: %605804 Links
DERMATITIS, ATOPIC, 3; ATOD3
Gene map locus 20p

3: %605845 Links
DERMATITIS, ATOPIC, 6; ATOD6
Gene map locus 5q31-q33

4: #605805 Links
DERMATITIS, ATOPIC, 4; ATOD4
Gene map locus 17q25

5: #147050 Links
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ATOPY, SUSCEPTIBILITY TO, INCLUDED
Gene map locus 16p12.1-p11.2, 1q23-q25, 13q14.1, 11q12-q13, 6p21.2-p12, 5q33.2, 5q32

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DERMATITIS, ATOPIC, 5; ATOD5
Gene map locus 13q12-q14

7: 221700 Links
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8: %605803 Links
DERMATITIS, ATOPIC, 2; ATOD2
Gene map locus 1q21

9: *135940 Links
FILAGGRIN; FLG
PROFILAGGRIN, INCLUDED
Gene map locus 1q21

10: #600807 GeneTests, Links
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Gene map locus 13q14.1, 11q12.3-q13.1, 10q11.2, 6p21.2-p12, 6p21.3, 6p21.3, 5q32-q34, 5q31.1-q33.1, 5q31-q34, 5q31-q33, 5q31, 2q22

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INTERLEUKIN 4; IL4
Gene map locus 5q31.1

12: %147060 Links
HYPER-IgE SYNDROME
Gene map locus 4q21

13: *605010 GeneTests, Links
SERINE PROTEASE INHIBITOR, KAZAL-TYPE, 5; SPINK5
Gene map locus 5q32

14: *147781 Links
INTERLEUKIN 4 RECEPTOR; IL4R
Gene map locus 16p12.1-p11.2

15: *147138 Links
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Gene map locus 11q13

16: 606242 Links
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17: 606772 Links
MENTAL RETARDATION, OBESITY, MANDIBULAR PROGNATHISM, AND EYE AND SKIN ANOMALIES

18: *604176 Links
SUPPRESSOR OF CYTOKINE SIGNALING 3; SOCS3
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19: 146840 Links
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20: *147576 Links
INTERFERON REGULATORY FACTOR 2; IRF2
Gene map locus 4q35.1

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21: #115150 GeneTests, Links
CARDIOFACIOCUTANEOUS SYNDROME
Gene map locus 7q34

22: 243700 Links
JOB SYNDROME

23: *607003 Links
THYMIC STROMAL LYMPHOPOIETIN
Gene map locus Chr.5

24: *604833 Links
CHEMOKINE, CC MOTIF, LIGAND 27; CCL27
Gene map locus 9p13

25: *600240 Links
G PROTEIN-COUPLED RECEPTOR 2; GPR2
Gene map locus 17q21.1-q21.3

26: *600953 Links
INTERLEUKIN 18; IL18
Gene map locus 11q22.2-q22.3

27: *173460 Links
PLATELET FACTOR 4; PF4
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28: *609509 Links
INTERLEUKIN 31; IL31

29: #256500 GeneTests, Links
NETHERTON SYNDROME; NETH
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30: #146700 Links
ICHTHYOSIS VULGARIS
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SQUAMOUS CELL CARCINOMA ANTIGEN RECOGNIZED BY T CELLS 1; SART1
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32: #609536 Links

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33: *607796 Links
PHD FINGER PROTEIN 11; PHF11
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34: %257910 Links
OCULOPALATOCEREBRAL SYNDROME
PERSISTENT HYPERPLASTIC PRIMARY VITREOUS, INCLUDED; PHPV, INCLUDED

35: *605084 Links
CALCIUM-BINDING ATOPY-RELATED AUTOANTIGEN 1; CBARA1

36: %609958 Links
ASTHMA-RELATED TRAITS, SUSCEPTIBILITY TO, 3

37: #608584 Links
ASTHMA-RELATED TRAITS, SUSCEPTIBILITY TO, 2
Gene map locus 7p15-p14

38: *608004 Links
INTERLEUKIN 1-INDUCED NUCLEAR ANKYRIN REPEAT PROTEIN
Gene map locus 3q13.11

39: #607277 Links
ASTHMA-RELATED TRAITS, SUSCEPTIBILITY TO, 1
Gene map locus 14q22.1

40: #607154 Links
ALLERGIC RHINITIS
Gene map locus 5q31

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Search for: Find Find Next (from the current location)

- Enter gene symbol, chromosomal location, or disorder keyword to search for, e.g. "CYP1", "5", "1pter", "Xq", or "alzheimer".
- You must capitalize X and Y to search for those chromosomes.

5q31-q33, ATOD6 to 5q31.1, IGES

<<Move Up Move Down>>

Location	Symbol	Title	MIM #	Disorder	Comments	Metl
5q31-q33	ATOD6	Dermatitis, atopic, 6	605845	Dermatitis, atopic, 603165 (2)		Fd
5q31-q33	CDX1	Caudal type homeo box transcription factor 1	600746		100kb distal to CSF1R	REc, REn
5q31-q33	CELIAC2	Celiac disease, susceptibility to, 2	609754	{Celiac disease, susceptibility to, 2} (2)		Fd
5q31-q33	BHR1	Bronchial hyperresponsiveness-1 (bronchial asthma)	600807	Bronchial asthma (2)		Fd
5q31-q33	EOS	Eosinophilia, familial	131400	Eosinophilia, familial (2)		Fd
5q31-q33	HCI, HEMC	Hemangioma, capillary infantile	602089	Hemangioma, capillary infantile (2)	?same as CMAL	Fd
5q31-q33	HTR4	5-hydroxytryptamine (serotonin) receptor-4	602164			A
5q31-q33	PFBI	Plasmodium falciparum blood infection levels	248310	{Malaria, intensity of infection} (2)		Fd
5q31-q33	PPP2R2B	Protein phosphatase 2, regulatory subunit B	604325	Spinocerebellar ataxia		R, Rl

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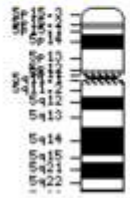

Homo sapiens Build 36.1 (Current)

BLAST The Human Genome

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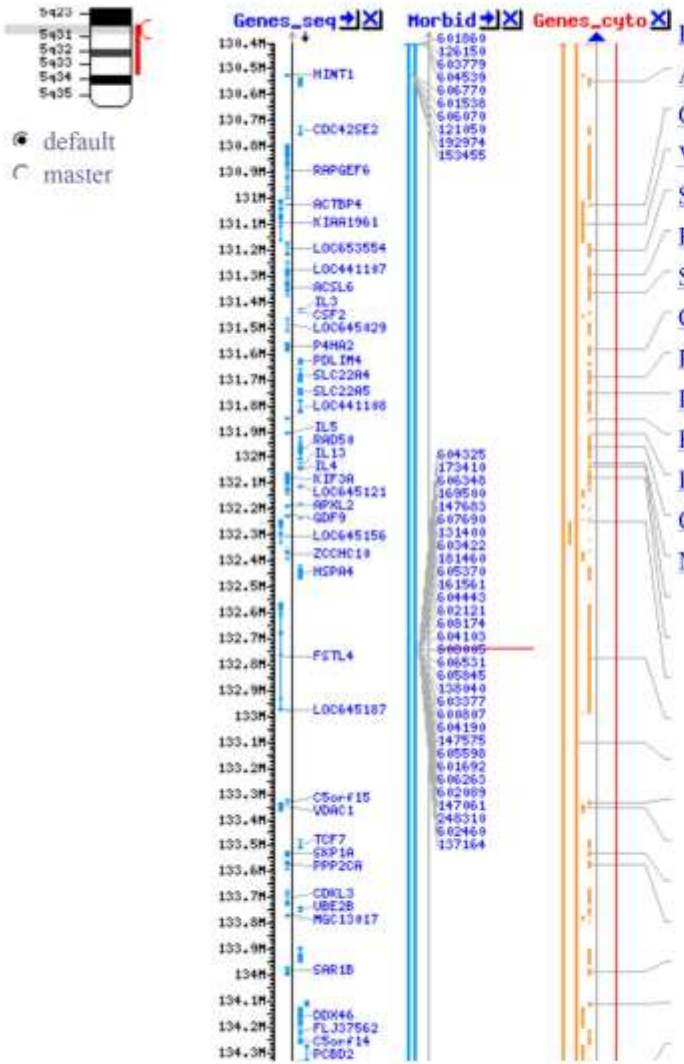
Query: ATOD6 [clear]

Master Map: Genes On Cytogenetic Summary of Maps Maps & Options

Region Displayed: **5q31.1**

Symbol	Links	Cyto	Description
LOC90624	pr hm sts	5q31.1	hypothetical protein LOC90624
ACTBP4		5q31.1	actin, beta pseudogene 4
KIAA1961	pr sts	5q23.3	KIAA1961 gene
LOC653554	pr	5q31.1	similar to acyl-CoA synthetase long-chai
LOC441107	pr hm	5q31.1	LOC441107
ACSL6	OMIM pr hm sts	5q31	acyl-CoA synthetase long-chain family m
P4HA2	OMIM pr hm sts	5q31	procollagen-proline, 2-oxoglutarate 4-dio
SLC22A4	OMIM pr hm sts	5q31.1	solute carrier family 22 (organic cation tr
SLC22A5	OMIM pr hm sts	5q31	solute carrier family 22 (organic cation tr
IRF1	OMIM pr hm sts	5q31.1	interferon regulatory factor 1
IL5	OMIM pr hm sts	5q31.1	interleukin 5 (colony-stimulating factor, ε
RAD50	OMIM pr hm sts	5q31	RAD50 homolog (S. cerevisiae)
IL13	OMIM pr hm sts	5q31	interleukin 13
IL4	OMIM pr hm sts	5q31.1	interleukin 4
KIF3A	OMIM pr hm sts	5q31	kinesin family member 3A
LEAP-2	pr hm sts	5q31.1	liver-expressed antimicrobial peptide 2



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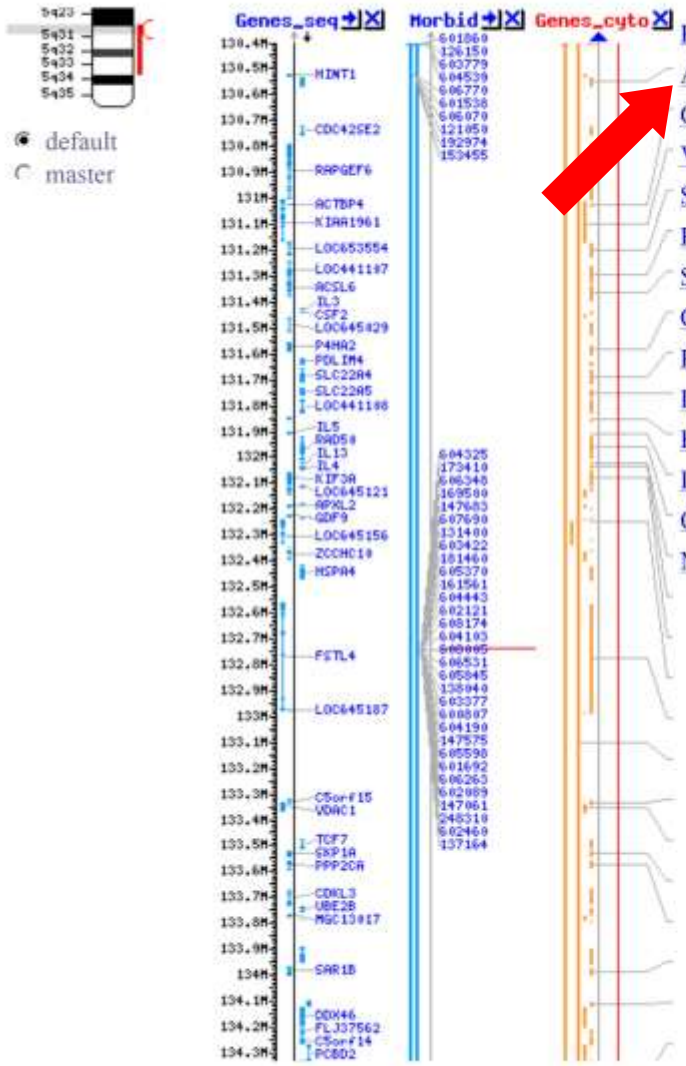
ATOD6	OMIM	sts	5q31-q33	Dermatitis, atopic, 6
C5orf15		pr hm sts	5q31.1	chromosome 5 open reading frame 15
VDAC1	OMIM	pr hm sts	5q31	voltage-dependent anion channel 1
SKP1A	OMIM	pr hm sts	5q31	S-phase kinase-associated protein 1A (p1
PPP2CA	OMIM	pr hm sts	5q31.1	protein phosphatase 2 (formerly 2A), cata
SAR1B	OMIM	pr hm sts	5q31.1	SAR1 gene homolog B (S. cerevisiae)
CAMLG	OMIM	pr hm sts	5q23	calcium modulating ligand
PITX1	OMIM	pr hm	5q31	paired-like homeodomain transcription fa
LOC389328		pr hm	5q31.1	hypothetical LOC389328
H2AFY		pr hm sts	5q31.3-q32	H2A histone family, member Y
LOC645407		pr	5q31.1	hypothetical protein LOC645407
C5orf20	OMIM	pr sts	5q31.1	chromosome 5 open reading frame 20
NEUROG1	OMIM	pr hm sts	5q23-q31	neurogenin 1



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VDAC1	OMIM	pr hm sts	5q31	voltage-dependent anion channel 1
SKP1A	OMIM	pr hm sts	5q31	S-phase kinase-associated protein 1A (p1
PPP2CA	OMIM	pr hm sts	5q31.1	protein phosphatase 2 (formerly 2A), cata
SAR1B	OMIM	pr hm sts	5q31.1	SAR1 gene homolog B (S. cerevisiae)
CAMLG	OMIM	pr hm sts	5q23	calcium modulating ligand
PITX1	OMIM	pr hm	5q31	paired-like homeodomain transcription fa
LOC389328		pr hm	5q31.1	hypothetical LOC389328
H2AFY		pr hm sts	5q31.3-q32	H2A histone family, member Y
LOC645407		pr	5q31.1	hypothetical protein LOC645407
C5orf20	OMIM	pr sts	5q31.1	chromosome 5 open reading frame 20
NEUROG1	OMIM	pr hm sts	5q23-q31	neurogenin 1

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1: %603165 Links

DERMATITIS, ATOPIC
DERMATITIS, ATOPIC, 1, INCLUDED; ATOD1, INCLUDED
Gene map locus 20p, 17q25, 13q12-q14, 5q31-q33, 3q21

2: %605845 Links

DERMATITIS, ATOPIC, 6; ATOD6
Gene map locus 5q31-q33

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%603165 DERMATITIS, ATOPIC

Alternative titles; symbols

ATOD
ECZEMA, ATOPIC
DERMATITIS, ATOPIC, 1, INCLUDED; ATOD1, INCLUDED

Gene map locus 20p, 17q25, 13q12-q14, 5q31-q33, 3q21

TEXT

Many inflammatory diseases, such as atopic eczema, are genetically complex, with multiple alleles at several loci thought to be involved in their pathogenesis. The new occurrence of such diseases after organ transplantation suggests that genetic predisposition may be confined to the particular organs or physiologic systems. A new occurrence of asthma after bone marrow transplantation from a donor who had asthma (Agosti et al., 1998) or new asthma in a recipient who had lungs transplanted from an asthmatic donor (Corris and Dark, 1993) suggests that expression of some inflammatory disorders is a result of both systemic (often immune) influence and end-organ specificity, each under distinct genetic control.

Turner et al. (1998) described an 18-year-old woman who had had an intensely itchy rash on her right leg since childhood. There were no exacerbating factors. Her itch improved with sunlight. Topical corticosteroids gave only a small benefit. The patient also had hayfever, but was otherwise well. Her eczema was in an uninterrupted distribution down the inner aspect of her right leg in a pattern of Blaschko described by Jackson (1976) and thought to be the path of migration of a clone of embryonic keratinocytes. Histology of a biopsy specimen showed features typical of eczema and supported a diagnosis of linear eczema (dermatitic nevus). The rest of her skin was normal. She was found to be atopic with multiple positive prick tests (on normal skin), including house dust mite, feathers and grass, and a raised IgE of 308 IU. In this patient, Turner et al. (1998) suspected that an aberrant clone of cells with either genetic (or epigenetic) change allowed expression of the atopic eczema phenotype in the mutated area only. As conventional treatments were largely unsuccessful and the area of involvement was small, Turner et al. (1998) excised the most itchy area of skin on her thigh as split skin. This produced only transient relief. Full skin thickness was excised from another area, and grafted with split skin from a donor area on her thigh. She had lasting relief for 6 years, although the itch persisted in her untreated skin.

In developed countries, the prevalence of atopic dermatitis is said to be approximately 15%, with a steady increase during the end of the 20th century (Kay et al., 1994; Taylor et al., 1984). To identify susceptibility loci for atopic dermatitis, Lee et al. (2000) ascertained 199 families with at least 2 affected sibs based on established diagnostic criteria. A genomewide linkage study revealed highly significant evidence for linkage on 3q21 at marker D3S3606. Moreover, this locus provides significant evidence for linkage of allergic sensitization under the assumption of paternal imprinting, further supporting the presence of an atopy gene in this region.

Atopic dermatitis (ATOD), also known as eczema, commonly begins in infancy and early childhood, and is typified by itchy inflamed skin. It affects 10 to 20% of children in western societies and shows a strong familial aggregation. Eighty percent of cases of ATOD have elevations of the total serum IgE concentration. Cookson et al. (2001) examined 148 nuclear families recruited through children

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2: %605804 Links
DERMATITIS, ATOPIC, 3; ATOD3
Gene map locus 20p

3: %605845 Links
DERMATITIS, ATOPIC, 6; ATOD6
Gene map locus 5q31-q33

4: #605805 Links
DERMATITIS, ATOPIC, 4; ATOD4
Gene map locus 17q25

5: #147050 Links
IgE RESPONSIVENESS, ATOPIC; IGER
ATOPY, SUSCEPTIBILITY TO, INCLUDED
Gene map locus 16p12.1-p11.2, 1q23-q25, 13q14.1, 11q12-q13, 6p21.2-p12, 5q33.2, 5q32

6: %605844 Links
DERMATITIS, ATOPIC, 5; ATOD5
Gene map locus 13q12-q14

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DEAFNESS, NEURAL, WITH ATYPICAL ATOPIC DERMATITIS

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PROFILAGGRIN, INCLUDED
Gene map locus 1q21

10: #600807 GeneTests, Links
ASTHMA, SUSCEPTIBILITY TO
Gene map locus 13q14.1, 11q12.3-q13.1, 10q11.2, 6p21.2-p12, 6p21.3, 6p21.3, 5q32-q34, 5q31.1-q33.1, 5q31-q34, 5q31-q33, 5q31, 2q22

11: *147780 Links

INTERLEUKIN 4; IL4
Gene map locus 5q31.1

12: %147060 Links
HYPER-IgE SYNDROME
Gene map locus 4q21

13: *605010 GeneTests, Links
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Gene map locus 5q32

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Gene map locus 16p12.1-p11.2

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Gene map locus 11q13

16: 606242 Links
MENTAL RETARDATION, MICROCEPHALY, GROWTH RETARDATION, JOINT CONTRACTURES, AND FACIAL DYSMORPHISM

17: 606772 Links
MENTAL RETARDATION, OBESITY, MANDIBULAR PROGNATHISM, AND EYE AND SKIN ANOMALIES

18: *604176 Links
SUPPRESSOR OF CYTOKINE SIGNALING 3; SOCS3
Gene map locus 17q25.3

19: 146840 Links
IMMUNODEFICIENCY WITH DEFECTIVE LEUKOCYTE AND LYMPHOCYTE FUNCTION AND WITH RESPONSE TO HISTAMINE-1 ANTAGONIST

20: *147576 Links
INTERFERON REGULATORY FACTOR 2; IRF2
Gene map locus 4q35.1

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THYMIC STROMAL LYMPHOPOIETIN
Gene map locus Chr.5

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CHEMOKINE, CC MOTIF, LIGAND 27; CCL27
Gene map locus 9p13

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G PROTEIN-COUPLED RECEPTOR 2; GPR2
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26: *600953 Links
INTERLEUKIN 18; IL18
Gene map locus 11q22.2-q22.3

27: *173460 Links
PLATELET FACTOR 4; PF4
Gene map locus 4q12-q13

28: *609509 Links
INTERLEUKIN 31; IL31

29: #256500 GeneTests, Links
NETHERTON SYNDROME; NETH
Gene map locus 5q32

30: #146700 Links
ICHTHYOSIS VULGARIS
Gene map locus 1q21

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SQUAMOUS CELL CARCINOMA ANTIGEN RECOGNIZED BY T CELLS 1; SART1
Gene map locus Chr.11

32: #609536 Links

COMPLEMENT COMPONENT 5 DEFICIENCY

33: *607796 Links
PHD FINGER PROTEIN 11; PHF11
Gene map locus 13q14.1

34: %257910 Links
OCULOPALATOCEREBRAL SYNDROME
PERSISTENT HYPERPLASTIC PRIMARY VITREOUS, INCLUDED; PHPV, INCLUDED

35: *605084 Links
CALCIUM-BINDING ATOPY-RELATED AUTOANTIGEN 1; CBARA1

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ASTHMA-RELATED TRAITS, SUSCEPTIBILITY TO, 3

37: #608584 Links
ASTHMA-RELATED TRAITS, SUSCEPTIBILITY TO, 2
Gene map locus 7p15-p14

38: *608004 Links
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4th Georg Rajka International Symposium on Atopic Dermatitis Arcachon, France, 15–17 September 2005

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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

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Thursday 15 September 2005

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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

OC12. Intracellular Control Of CTACK/CCL27 (Cutaneous T Cell Attracting Chemokine) in Keratinocytes Through the Nuclear Transcription 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. R. Braathen, 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
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 Dermatitis by Polymerase 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 Comparative Study. 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

Genetics and Epigenetics of Atopic Dermatitis

WOCM Cookson
The Welcome Trust Centre for Human Genetics, Roosevelt Drive, Oxford OX3 7BN, United Kingdom

Atopic Dermatitis is strongly familial and has a genetic as well as an environmental basis. Progress in understanding the genetics of AD is likely to accelerate in the post-genom era. Genetic Mapping studies have identified several chromosomal regions that contain genes predisposing to AD. Four of these regions correspond to known psoriasis loci, and one region in addition schows genetic linkage to psoriasis susceptibility. Detailed mapping of these loci is being carried out in several centres. Our group, in addition, has been carrying out gene expression analyses of keratinocytes during differentiation and the response to inflammatory stimuli. The results from these studies reveal much that is new about keratinocyte biology, as well as indicating key candidate genes for genetic mapping and association.

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The Welcome Trust Centre for Human Genetics, Roosevelt Drive, Oxford OX3 7BN, United Kingdom

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2) J Lokaj keratinocyty
3) P Barták keratinocyty

Keratinocyty

Nethertonův syndrom

→defekt SPINK5 genu
→tento gen kóduje „LEKTI“ *
(inaktivuje/inhibuje aktivitu enzymu, který se podílí na přirozeném „odlupování“ keratinocytů)

LEKTI je defektní → enzym není inhibovaný → kůže se nadměrně olupuje → je porušená bariera → podoba s AD

***lymphoepithelial Kazal-type-related inhibitor**

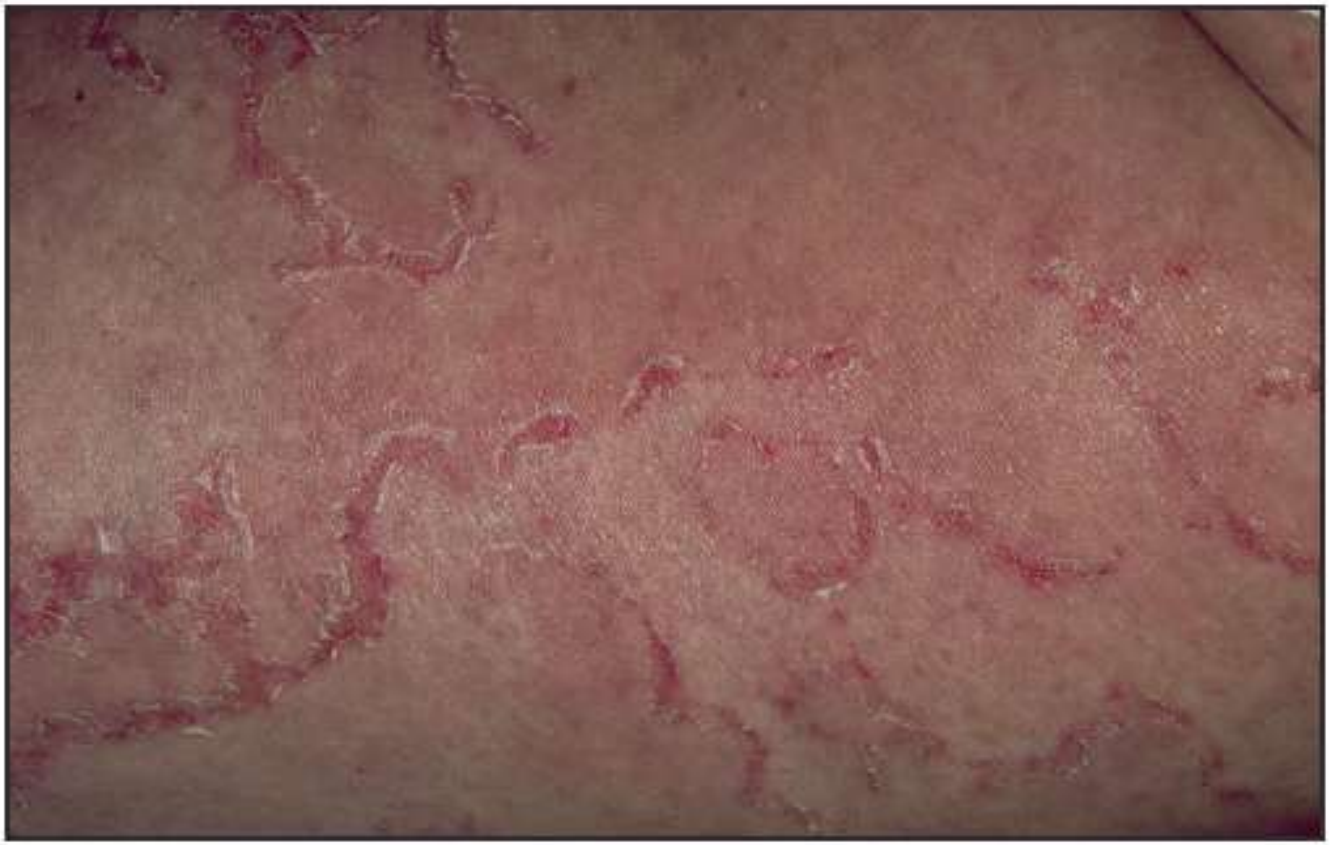

Abb. 51.7. Comèl-Netherton-Syndrom: Ichthyosis linearis circumflexa

Keratinocyty

K dalším genetickým analýzám je v prvé řadě třeba upřesnit diagnostická kritéria pro AD !

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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

OC12. Intracellular Control Of CTACK/CCL27 (Cutaneous T Cell Attracting Chemokine) in Keratinocytes Through the Nuclear Transcription 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. R. Braathen, 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
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 Dermatitis by Polymerase 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 Comparative Study. 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

Elevated Expression and Genetic Association Links the *SOCS3* Gene to Atopic Dermatitis

E. Ekelund,[1] A. Sääf,[9] M. Tengvall-Linder,[2] E. Melen,[4,5] J. Link,[1] J. Barker,[10] N. J. Reynolds,[11] S. J. Meggitt,[12] J. Kere,[6] C.-F. Wahlgren,[3] G. Pershagen,[4,8] M. Wickman,[4,5,8] M. Nordenskjöld,[1] I. Kockum,[1,7] and M. Bradley[1,3]

[1]Department of Molecular Medicine and Surgery, [2]Clinical Allergy Research, and [3]Dermatology Unit, Department of Medicine, [4]Institute of Environmental Medicine, [5]Centre for Allergy Research, and Departments of [6]Bioscience and [7]Clinical Neuroscience, Karolinska Institutet, and [8]Department of Occupational and Environmental Health, Stockholm County Council, Stockholm; [9]Department of Biochemistry, Stanford University School of Medicine, Stanford, CA; [10]St Johns Institute of Dermatology, Kings College, London; and [11]Dermatological Sciences, University of Newcastle, and [12]Department of Dermatology, Royal Victoria Infirmary, Newcastle upon Tyne, United Kingdom

In a systematic analysis of global gene-expression patterns, we found that *SOCS3* messenger RNA was significantly more highly expressed in skin from patients with atopic dermatitis than in skin from healthy controls, and immunohistochemical analysis confirmed a similar elevation of SOCS3 protein. Furthermore, we found a genetic association between atopic dermatitis and a haplotype in the *SOCS3* gene in two independent groups of patients ($P < .02$ and $P < .03$). These results strongly suggest that *SOCS3*, located in a chromosomal region previously linked to the disease (17q25), is a susceptibility gene for atopic dermatitis.

The American Journal of Human Genetics Volume 78 June 2006

The *SOCS3* gene (Locus Link ID 9021; NCBI accession number NM_003955; UniGene cluster ID Hs.527973), located in chromosome region 17q25, was found to be significantly more highly expressed in skin from patients with AD than in skin from healthy individuals ($P < .03$, corrected for multiple testing) (table 1 and fig. 1).

Web Resources

Accession numbers and URLs for data presented herein are as follows:

dbSNP, http://www.ncbi.nlm.nih.gov/projects/SNP/

GenBank, http://www.ncbi.nlm.nih.gov/Genbank/ (for *SOCS3* sequences [accession numbers AA001218, AI922872, and T72915])

NCBI, http://www.ncbi.nlm.nih.gov/ (for *SOCS3* [accession number NM_003955])

Online Mendelian Inheritance in Man (OMIM), http://www.ncbi.nlm.nih.gov/Omim/ (for AD)

RAVEN: Regulatory Analysis of Variations in Enhancers, http://mordor.cgb.ki.se/CONSNP/

Stanford Functional Genomics Facility, http://www.microarray.org/sfgf/

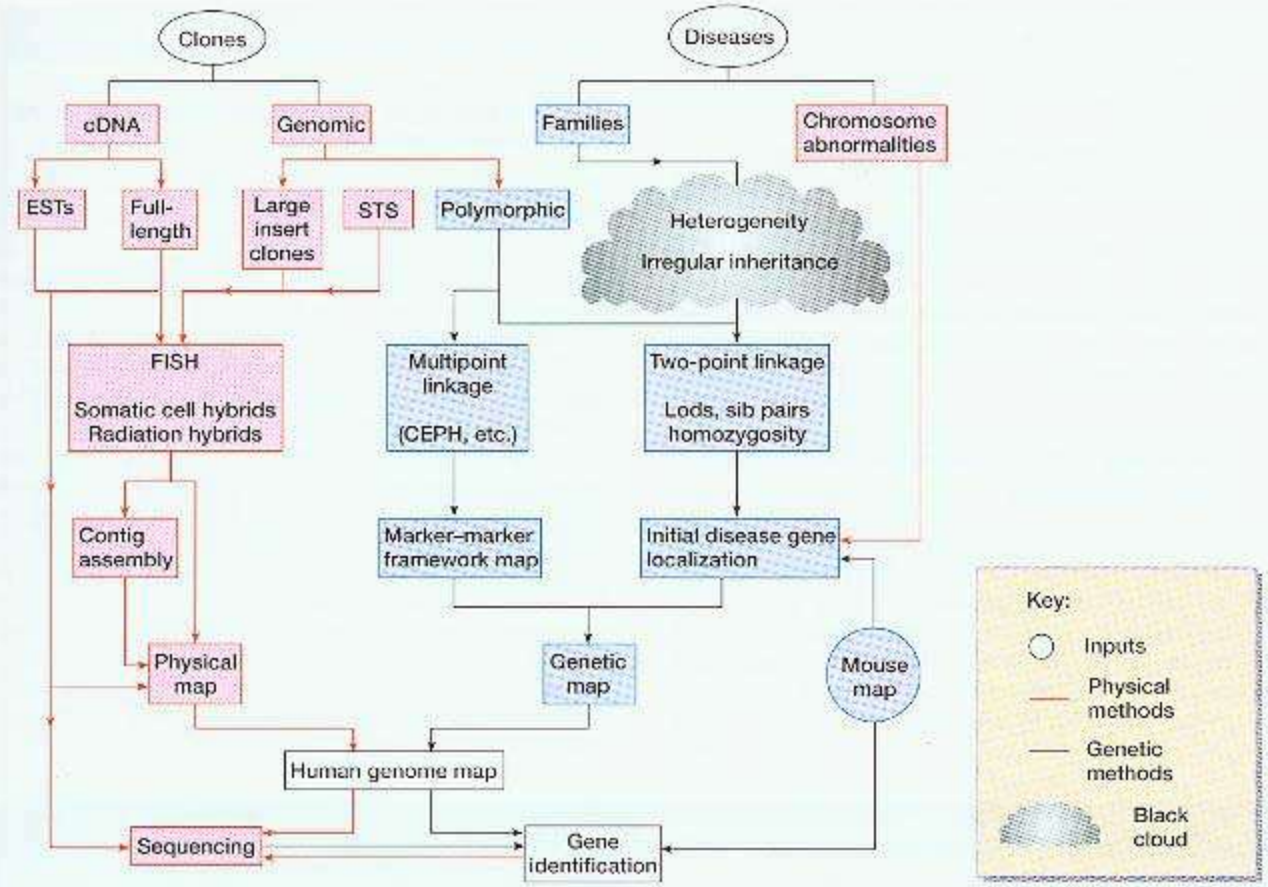


Figure 8.2: Major scientific strategies and approaches used in the Human Genome Project.

The major scientific thrust of the Human Genome Project began with the isolation of human genomic and cDNA clones (by cell-based cloning or PCR-based cloning). These were then used to construct high resolution genetic and physical maps prior to obtaining the ultimate physical map, the complete nucleotide sequence of the 3000-Mb nuclear genome. Inevitably, the project interacted with research on mapping and identifying human disease genes. In addition, ancillary projects included studying genetic variation (the Human Genome Diversity Project – Section 8.3.7), genome projects for model organisms (Section 8.4) and research on ethical, legal and social implications. The data produced were channeled into mapping and sequence databases permitting rapid electronic access and data analysis. EST, expressed sequence tag; STS, sequence tagged site.

Box 8.1: A genomics glossary

centiMorgan (cM). A unit of distance in a ***genetic map*** (see below). In the human genome 1cM corresponds roughly to a physical map distance of 1 Mb.

centiRay (cR). A unit of map distance in a ***radiation hybrid map*** (see below).

Clone. DNA clones are populations of identical DNA molecules which have been purified by cell-based cloning methods (Section 5.3.1) or by PCR (Section 5.2.1).

Contig. A series of DNA clones which have been shown to contain insert DNA molecules that derive from neighboring and *overlapping* regions of a chromosome – see *Box 8.5*.

DNA marker. A general term for a DNA sequence which has been, or can be, placed on a genetic map (in the case of ***polymorphic markers*** – see below) or on a physical map (in the case of all markers).

CpG island. Short stretch of GC-rich DNA, often < 1 kb, containing frequent unmethylated CpG dinucleotides. CpG islands tend to mark the 5′ ends of genes – see *Box 9.3*.

DNA library. A collection of DNA clones which is meant to collectively represent a starting population of DNA. For a ***genomic DNA library***, the starting DNA is the total DNA from a given cell population (which shows little variation between different cell types). In the case of a ***cDNA library***, the starting DNA is ***cDNA*** prepared using reverse transcriptase from single-stranded RNA from a specific tissue (with very considerable variation in the cDNA of different tissues). See Sections 5.3.4 and 5.3.5 for how libraries are made and screened.

EST (expressed sequence tag). An expressed ***STS (sequence tagged site)***; see below) obtained by randomly selecting a cDNA clone for sequencing and designing specific primers for specifically PCR amplifying the corresponding fragment from genomic DNA.

Genetic map. A map which relies on tracing the inheritance of phenotypes and/or polymorphic markers, through generations. Polymorphic loci are positioned relative to one another on the basis of the frequency with which they recombine during meiosis. The unit of distance is ***1 centiMorgan (1 cM)*** which denotes a 1% chance of recombination.

Genome. The collective name for the *different DNA molecules* found in the cells of a particular species. In humans, the genome comprises 25 different DNA molecules: a single type of mitochondrial DNA and 24 different nuclear DNA molecules (see Section 9.1.1). Because the amount of DNA in the nucleus is so large, however, the term genome is often loosely used to mean the set of *nuclear DNA* molecules (more accurately termed the ***nuclear genome***; mitochondrial DNA is often described as the ***mitochondrial genome***).

Hybrid cell mapping. Human DNA markers can be assigned to a specific chromosomal or subchromosomal location by using panels of different hybrid cells containing a full complement of chromosomes from a rodent species (hamster or mouse) and a variable subset of human chromosomes, or of fragments of human chromosomes broken by exposure to X-rays (***radiation hybrids***; see *Box 8.4*).

Microsatellite marker. A type of DNA marker which is commonly used, largely because markers of this type can be very polymorphic. See *Figures 7.7 and 7.8*.

Physical map. A map which provides information on the *linear structure* of DNA molecules. The most detailed physical map is the nucleotide sequence.

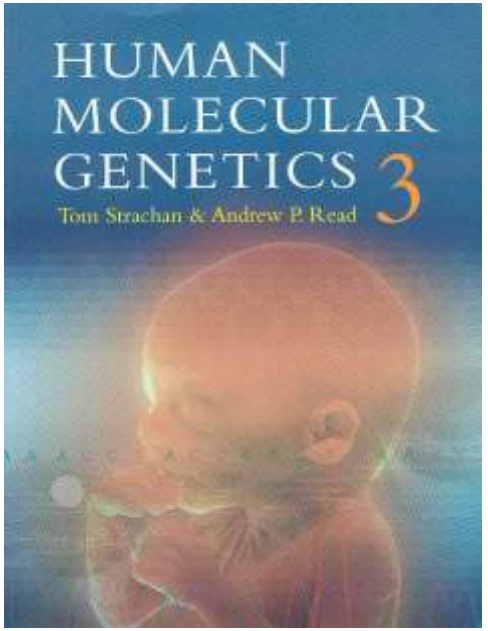
Polymorphic markers. Polymorphic (genetic) markers are DNA sequences which show variation between individuals and which are used in constructing genetic maps by following how alleles segregate in large families. Markers may be located within coding sequences or other gene components but are mostly located in noncoding DNA. Commonly used markers are ***microsatellites*** and ***SNPs***, although in the past ***RFLPs*** were used and even protein polymorphisms.

Radiation hybrid (RH) map. A genome map in which STSs are positioned relative to one another according to the frequency with which they are separated by radiation-induced chromosome breaks. The frequency is assayed by analyzing a panel of **hybrid cell** (human–hamster) lines which contain different patterns of human chromosome fragments initially generated by exposure to X-rays. The unit of map distance is **1 centiRay (1 cR)**, denoting a 1% chance of a break occurring between two loci.

RFLP (restriction fragment length polymorphism). A type of DNA marker used widely in the past but rather infrequently in modern times because they are often not very polymorphic and are not so easy to type. See Section 7.1.3.

SNP (single nucleotide polymorphism). SNPs provide a type of DNA marker which is increasingly being used. They occur very frequently in DNA and can be typed very easily by automated methods, allowing very large numbers of samples to be analyzed at a time. See Section 7.1.3 and *Box 18.2* for how SNPs are typed.

STS (sequence tagged site). Any short (usually < 500 bp) sequence which is uniquely represented in a genome and for which primers have been designed enabling specific PCR amplification of that sequence (see *Box 5.4*). STSs were often designed by randomly sequencing the ends of genomic clones and so were often nonpolymorphic, but a subset of STSs are known to be polymorphic, including ***microsatellite markers*** (see above).



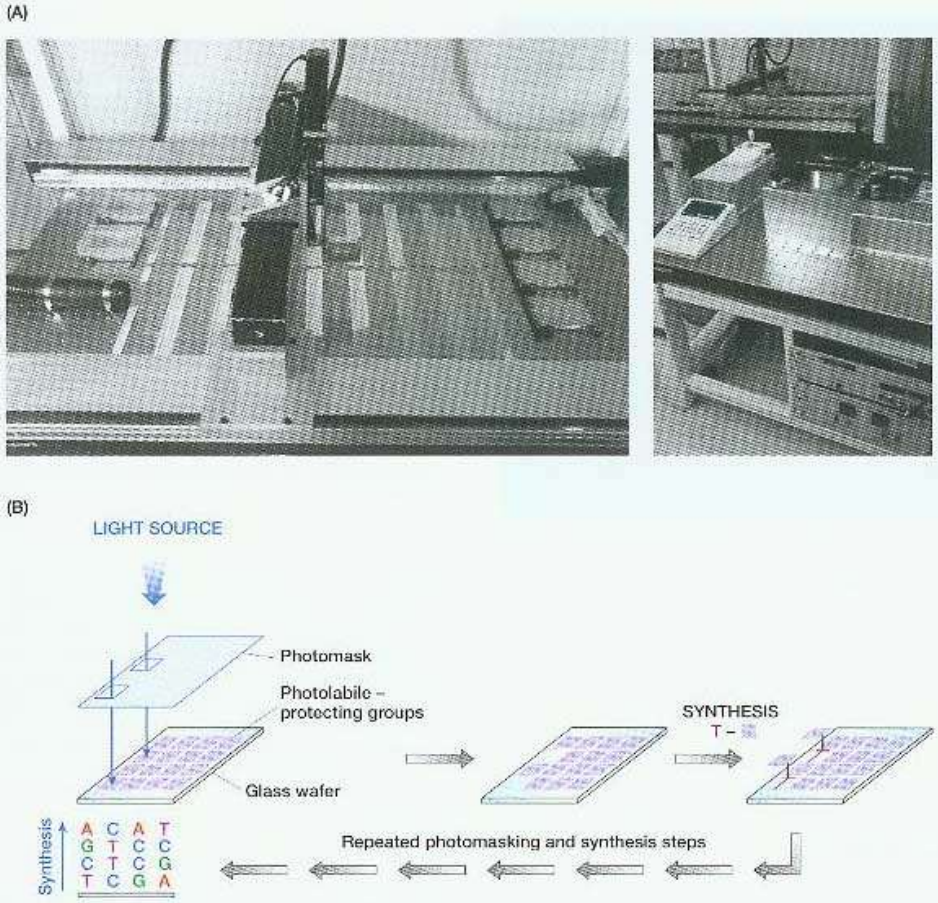


Figure 6.18: Construction of DNA and oligonucleotide microarrays.

(A) ***Robotic spotting for construction of DNA microarrays.*** Left: a microarray robot, with a table configuration which contains 160 slides with four microtitre plates, two wash stations and the dryer. Right: a laser scanner showing the optical table, power supplies for the lasers and photomultiplier tube cooling, the Ludl stage and lenses (see Cheung *et al.*, 1999 for more details). The microspotting of samples by robots can be performed by physical contact between spotting pins and the solid surface (of a microscope slide) or by an *ink-jetting* approach as is used in standard printing (the sample is loaded into a miniature nozzle equipped with a piezoelectric fitting and an electric current is used to expel a precise amount of liquid from the jet onto the substrate). Images kindly supplied by Aldo Massimi, Raju Kucherlapati, and Geoffrey Childs at the Albert Einstein College of Medicine. Reprinted from Cheung *et al.* (1999) *Nature Genet.* **21** (Suppl.), 15–19, with permission from Nature Publishing Group. **(B)** ***Construction of an oligonucleotide microarray*** by combining photolithography and *in situ* synthesis of oligonucleotides. Oligonucleotides are synthesized *in situ* in sequential steps starting from a 3′ mononucleotide which is anchored to the surface of a glass wafer. The photolithography entails modifying the glass wafer with photolabile protecting groups which can be eliminated when exposed to light and the use of carefully constructed *photomasks* which allow light to pass through onto carefully selected spatial co-ordinates. For those areas of the wafer which receive light passing through the photomask, the removal of the photolabile protective groups permits a new synthesis step. In this example thymidine is shown being coupled together with a protective photolabile group.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Genetické aspekty atopické dermatitidy

MUDr. Radek Klubal

Klinické studie rodin a dvojčat jednoznačně potvrzují vrozenou predispozici pro vznik atopické dermatitidy (dále AD) :

1) děti rodičů s AD mají větší sklony opět k AD než k jiné formě atopie
   Arch Dis Child 1992; 67(8): 1018-1022

2) vznik AD u obou jednovaječných dvojčat trojnásobně převyšuje výskyt AD u obou dvojvaječných dvojčat
   Atopic dermatitis : a genetic-epidemiologic study in a population-based twin sample
   J Am Acad Dermatol 1993; 28(5): 719-723

TABLE I. Linkages with asthma and allergy

Chromosome	Candidate genes or products
1p	IL-12 receptor
2q	IL-1, cytotoxic T lymphocyte-associated antigen 4, CD28
3p24	B-cell lymphoma-6 (STAT-6 binding inhibition)
	Chemokine cell receptor 4
5q23-35	IL-3, IL-4, IL-5, IL-9, IL-13, GM-CSF
	LTC4S
	Macrophage colony-stimulating factor receptor
	β_2-Adrenergic receptor
	Glucocorticosteroid receptor
	TIM1, TIM3
6p21-23	MHC
	TNFs
	Transporters involved in antigen processing and presentation (*TAP1* and *TAP2*)
	Large multicatalytic proteolytic particles
7q11-14	T-cell receptor γ chain, IL-6
11q13	High-affinity IgE receptor (FceRI) β chain
	Clara cell protein 16
	Fibroblast growth factor 3
12q14-24	IFN-γ
	Stem cell factor
	Nitric oxide synthetase (constitutive)
	β Subunit of nuclear factor Y (transcription factor for HLA genes)
	Insulinlike growth factor 1
	Leukotriene A_4 hydrolase
	STAT-6 (IL-4 STAT)
13q21-24	Cysteinyl leukotriene 2 receptor
14q11-13	T-cell receptor α and δ chains
	Nuclear factor κB inhibitor
16p11-12	IL-4 receptor
17p12-17	CC chemokine cluster
19q13	CD22, transforming growth factor β_1
20p13	ADAM-33

TABLE II. Candidate genes of atopy and allergy

	Examples
Cytokines influencing allergic phenotype	
Eosinophil growth, activation, and apoptosis-inhibiting factors	IL-5, IL-3, GM-CSF, eotaxin, RANTES
Mast cell growth factors	IL-3, IL-9, IL-10, nerve growth factor, stem cell factor, transforming growth factor β
Histamine-releasing factors	Monocyte chemoattractant protein 1, monocyte chemoattractant protein 3, RANTES
IgE isotype switch factors	IL-4, IL-13
Inhibition of IgE isotype switch	IFN-γ, IL-12, IL-18, IL-23
Lipoxygenase pathway metabolism	*5LO*, 5-lipoxygenase–activating peptide, leukotriene C_4 synthase
Pro-inflammatory cytokines	IL-1α, IL-1β, TNF-α, IL-6
Anti-inflammatory cytokines	Transforming growth factor β, IL-10, interleukin-1 receptor antagonist
Receptors	
Antigen receptors	T-cell receptors (α/β, γ/δ), B-cell receptor (Ig, κ/λ light chain)
IgE	FceRI β chain, FceRII (CD23)
Cytokine gene receptors	IFN-γ receptor β chain, macrophage colony-stimulating factor receptor, IL-1 receptor, IL-4 receptor, TNF receptors
Adhesion molecules	Virus-like agent 4, vascular cellular adhesion molecule 1, intercellular adhesion molecule 1, leukocyte functional activating molecule-1, *TIM1, TIM3*
Corticosteroid receptor	Glucocorticoid receptor–heat shock protein 90
Neurogenic receptors	β_2-Adrenergic, cholinergic receptors
Nuclear transcription factors	Activating protein-1, nuclear factor of interleukin-2, octamer transcription factor-1, STAT-1/2, GATA3, T-box expressed in T cells, nuclear factor κB, inhibitor of nuclear factor kappa B, nuclear factor of activated T cells, STAT-4, STAT-6, BCL-6

Genetics of hypersensitivity
J Allergy Clin Immunol 2003; 111: S495-501

TABLE I. List of SNPs genotyped in the COAST cohort ordered by chromosomal location

Marker	Gene	Chromosome position	Polymorphism	Location	Amino acid exchange	dbSNP rs no.*	Minor allele frequency in sample
1	*VCAM1*	1p32-p31	T→C	Promoter (−1594)		rs1041163	0.167
2	*SELP*	1q21-q24	G→A	Exon 7	S330N	rs6131	0.163
3			G→T	Exon 12	V640L	rs6133	0.092
4	*SELE*	1q22-q25	A→C	Exon 3	S128R	rs5361	0.102
5	*IL10*	1q31-q32	C→A	Promoter (−571)		rs1800872	0.265
6			C→T	Promoter (−854)		rs3021097	0.259
7			G→A	Promoter (−1117)		rs1800896	0.474
8	*IL1A*	2q12-q21	T→C	Promoter (−889)		rs1800587	0.299
9	*IL1B*	2q14	C→T	Promoter (−1418)		rs16944	0.337
10			C→T	Exon 5	F105F	rs1143634	0.216
11	CTLA4	2q33	C→T	Promoter (−318)		rs5742909	0.092
12			A→G	Exon 1	T17A	rs231775	0.374
13	*CCR2*	3p21	G→A	Exon 1	V62I	rs1799864	0.080
14	*CCR3*	3p21	C→T	Exon 1	P39L	rs5742906	0.002
15	*CCR5*	3p21	wt→Δ580-611	Exon 1		rs333	0.107
16			G→A	Promoter (−2454)		rs1799987	0.475
17	*IL5RA*	3p26-p24	G→A	Promoter (−80)		rs2290608	0.261
18	*GC*	4q12-q13	G→T	Exon 3	E416D	rs7041	0.415
19			C→A	Exon 3	T420K	rs4588	0.246
20	*CD14*	5q22-q32	C→T	Promoter (−159)		rs2569190	0.480
21	*IL4*	5q31	C→T	Promoter (−590)		rs2243250	0.138
22	*IL13*	5q31	C→T	Promoter (−1112)		rs1800925	0.186
23			C→T	Intron 3		rs1295686	0.239
24			G→A	Exon 4	R110Q	rs20541	0.220
25	*TCF7*	5q31	C→A	Exon	P19T	rs5742913	0.115
26	*CSF2*	5q31	T→C	Exon 4	I117T	rs25882	0.175
27	*ADRB2*	5q31-q32	A→G	Exon 1	R16G	rs1042713	0.356
28			C→G	Exon 1	Q27E	rs1042714	0.440
29			C→T	Exon 1	T164I	rs1800888	0.007
30	*IL9*	5q31-q35	C→T	Exon 5	T113M	rs2069885	0.132
31	*LTC4S*	5q35	A→C	Promoter (−444)		rs730012	0.257
32	*LTA*	6p21	A→G	Intron A		rs909253	0.303
33	*TNF*	6p21	G→A	Promoter (−308)		rs1800629	0.141
34			G→A	Promoter (−238)		rs361525	0.053
35	*IL6*	7p21-p15	G→C	Promoter (−572)		rs1800796	0.070
36			G→C	Promoter (−174)		rs1800795	0.460
37	*NOS3*	7q35-q36	A→G	Promoter (−922)		rs1800779	0.368
38			G→T	Exon 7	E298D	rs1799983	0.348
39	*C5*	9q32-q34	A→G	Exon 24	I802V	rs17611	0.452
40	*SDF1*	10q11	G→A	3′ UTR (+800)		rs1801157	0.202
41	*CCI6*	11q11-qter	G→A	Exon 1		rs3741240	0.351
42	*FCERB1*	11q13	A→G	Promoter (−109)		rs1441586	0.424
43			A→G	Exon 7	E237G	rs569108	0.040
44	*VDR*	12q13	T→C	Exon 1	M1T	rs2228570	0.399
45			G→A	Intron 8		rs1544410	0.406
46	*IL4RA*	16p12	C→T	Promoter (−3223)		rs2057768	0.300
47			A→G	Exon 5	I50V	rs1805010	0.454
48			C→T	Exon 7	N142N	rs3024571	0.089
49			A→C	Exon 12	E375A	rs1805011	0.114
50			G→T	Exon 12	L389L	rs2234898	0.111
51			T→C	Exon 12	C406R	rs1805012	0.105
52			T→C	Exon 12	S478P	rs1805015	0.164
53			A→G	Exon 12	Q551R	rs1801275	0.196
54			T→C	Exon 12	S761P	rs3024678	0.007
55	*NOS2A*	17q11-q12	C→T	Exon 10 (+231)	D346D	rs1137933	0.217
56	*EOTAXIN*	17q21	G→A	Exon 1	A23T	rs3744508	0.208
57			G→A	Promoter (−1328)		rs4795895	0.175
58	*C3*	19p13	C→G	Exon 3	R102G	rs2230199	0.189
59	*ICAM1*	19p13	A→T	Exon 2	K56M	rs5491	0.000
60			G→A	Exon 4	G214R	rs1799969	0.133
61	*TGFB1*	19q13	C→T	Promoter (−509)		rs1800469	0.284

Genetic variation in immunoregulatory pathways and atopic phenotypes in infancy
J Allergy Clin Immunol 2004; 113: 511-8

TABLE I. Genes with statistically significant, more than two-fold expression in atopic dermatitis as compared with psoriasis

Gene name
Nel-like 2
CCL-18/PARC
CCL-27/CTACK
CCL-13/MCP-4
Osteoblast specific factor 2
Carbonic anhydrase II
PPP1R5
Tenascin precursor
Cysteine-rich protein 1
PPP1R5
Plasminogen activator inhibitor, type I
CD1a
CD26 dipeptidylpeptidase IV
Lymphocyte-specific protein 1
Cysteine-rich protein 2
AQP3 gene for aquaporine 3 (water cha
Collagen VI α-1 C-terminal globular d
N-myc downstream regulated gene 1

TABLE II. Gene with more than two-fold expression and with statistical significant difference in psoriasis as compared with atopic dermatitis

Gene name
A.
Small proline-rich protein 2C, keratin differentiation markers
Transcobalamin I neutrophil granule
CXCL-8/IL-8
Lipocalin 2, α-2-microglobulin-relate of MMP-9
ATPase, H+/K+ transporting, nongast α- polypeptide, found in skin and k
Kallikrein 6
Airway trypsin-like protease
Paraoxonase 3
Kynureninase
iNOS
Kallikrein 13
Serum amyloid A1
CCL-4/MIP-1β
Deoxyribonuclease I-like 2
Aldehyde dehydrogenase 5 family, m
ATPase, Na+/K+ transporting, beta 1
HBD2
Protease inhibitor 3, skin-derived (SK
Arginase
S100 calcium binding protein A12

TABLE II. Continued

Gene name	Probe set	GeneBank accession	Relative intensity (mean ± SEM) AD	Relative intensity (mean ± SEM) Psoriasis	*P* value	Ratio of psoriasis: AD gene expression
B.						
Arylsulphatase	35056_at	X97868	218 ± 60	688 ± 99	.002	3.2
17-β-Hydroxysteroid dehydrogenase	38178_at	L40802	80 ± 27	251 ± 42	.006	3.1
Promyelocytic leukemia zinc finger protein	39681_at	AF060568	53 ± 17	156 ± 35	.028	3.0
CXCL-2/GROβ	37187_at	M36820	50 ± 11	143 ± 38	.050	2.9
L-kynurenine hydrolase	40672_at	U57721	343 ± 49	978 ± 90	.001	2.8
Tissue-type plasminogen activator	33452_at	M15518	231 ± 55	636 ± 129	.020	2.8
G0S2 protein	38326_at	M69199	405 ± 99	1109 ± 281	.049	2.7
Flavin-containing monooxygenase	31806_at	M64082	55 ± 19	149 ± 29	.021	2.7
CCL-20/MIP-3α	40385_at	U64197	54 ± 11	143 ± 31	.030	2.7
GM2 ganglioside activator protein	35820_at	X62078	2484 ± 117	6534 ± 860	.003	2.6
Pre–B-cell colony-enhancing factor	33849_at	U02020	194 ± 19	507 ± 74	.005	2.6
GM2 ganglioside activator protein	33646_g_at	X61094	333 ± 27	860 ± 99	.001	2.6
Arcinoembryonic antigen-related cell adhesion molecule	1582_at	M29540	88 ± 9	226 ± 35	.007	2.6
3-Hydroxy-3-methylglutaryl-coenzyme A synthase 1	34517_at	X66435	85 ± 18	219 ± 41	.018	2.6
Thyroid receptor interactor	269_at	L40387	305 ± 51	742 ± 160	.035	2.4
Aldehyde dehydrogenase 6	36686_at	U07919	165 ± 30	401 ± 91	.043	2.4
Peptide transporter	32906_at	U21936	96 ± 9	226 ± 31	.005	2.4
Paraoxonase 2	40504_at	AF001601	350 ± 15	816 ± 86	.001	2.3
Carcinoembryonic antigen BGPa	988_at	X16354	66 ± 6	152 ± 31	.032	2.3
5-Aminolevulinate synthase	37674_at	Y00451	572 ± 67	1295 ± 208	.012	2.3
C.						
DKFZp586M0723	39616_at	AL050227	370 ± 78	819 ± 43	.001	2.2
Keratinocyte transglutaminase gene	35947_at	M98447	1650 ± 304	3577 ± 470	.006	2.2
Pirin	35724_at	Y07867	118 ± 12	255 ± 19	.001	2.2
LETKI precursor	40315_at	AJ228139	2666 ± 266	5748 ± 593	.001	2.2
Squamous cell carcinoma antigen	1549_s_at	U19557	11,206 ± 2102	24,090 ± 2494	.002	2.1
SWI/SNF complex 60 KDa subunit	32565_at	U66619	229 ± 23	491 ± 85	.021	2.1
G-protein–coupled receptor	649_s_at	L06797	219 ± 19	467 ± 81	.022	2.1
Protein-tyrosine kinase EPHB2v (EPHB2)	41678_at	AF025304	177 ± 21	375 ± 29	.001	2.1
Malic enzyme 1	31824_at	AL049699	199 ± 25	419 ± 73	.023	2.1
Small proline-rich protein	36734_at	M21302	16,149 ± 4040	33,693 ± 3226	.007	2.1
Peroxisome proliferator activated receptor-γ	37104_at	L40904	68 ± 11	142 ± 17	.004	2.1
Metastasis-associated gene	1348_s_at	S79219	152 ± 14	315 ± 23	.001	2.1
Squamous cell carcinoma antigen-serine protease inhibitor	1343_s_at	S66896	3384 ± 293	6972 ± 675	.001	2.1
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	.004	2.1
Small GTP-binding protein	809_at	U57094	169 ± 18	347 ± 40	.004	2.1
Bcell activation gene	36575_at	S59049	75 ± 18	153 ± 23	.023	2.1
CXCR4	649_s_a	L06797	219 ± 19	467 ± 81	.022	2.1
CXCR-2/IL-8R	664_at	L19593	62 ± 9	122 ± 12	.002	2.0
12R-lipoxygenase	33029_at	AF038461	3198 ± 462	6529 ± 841	.007	2.0
Monoamine oxidase A	41772_at	M68840	235 ± 32	477 ± 73	.017	2.0
Normal epithelial cell-specific 1 (NES1)	36838_at	AF055481	1662 ± 224	3363 ± 443	.008	2.0

Distinct patterns of gene expression in the skin lesions of atopic dermatitis and …
J Allergy Clin Immunol 2003; 112: 1195-1202

- uspořádat již získané informace
- začít na novo ?

- zpřesnit diagnozu
- zlepšit kvalitativní hodnocení

  např. SCORAD=SCORing AD

Consensus Conference on Pediatric Atopic Dermatitis

Lawrence F. Eichenfield, MD, Chair
Jon M. Hanifin, MD, Thomas A. Luger, MD
Seth R. Stevens, MD, and Howard B. Pride, MD

J Am Acad Dermatol 2003; 49: 1088-95

A. Essential features (must be present)
    1. Pruritus
    2. Eczema (acute, subacute, chronic)
        a) typical morphology and age-specific patterns
        b) chronic or relapsing history

B Important features (seen in most cases, adding support to the diagnosis)
    1. Early age at onset
    2. Atopy
        a) Personal and/or family history
        b) IgE reactivity
    3. Xerosis

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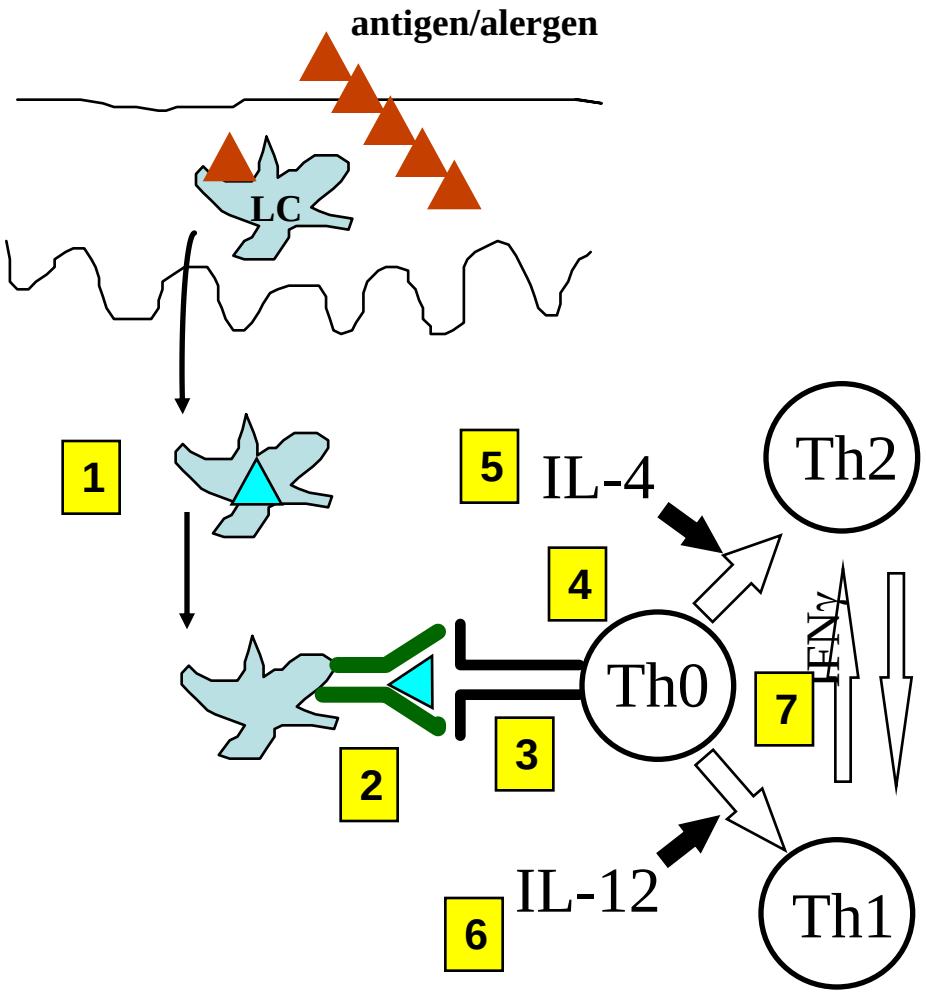
C Associated features (these clinical associations help to suggest the diagnosis of AD but are too nonspecific to be used for defining or detecting AD for research or epidemiologic studies)

1. Atypical vascular responses (eg. facial palor, white dermographism, delayed blanch response)
2. Keratosis pilaris/hyperlinear palms/ichthyosis
3. Ocular/periorbital changes
4. Other regional findings (eg, perioral changes/periauricular lesions)
5. Perifollicular accentuation/lichenification/prurigo lesions
   a) typical morphology and age-specific patterns
   b) chronic or relapsing history

IgE+



IL-4 → B → B +IgE 8

IL-4 → Th0 → Th2

IL-4 → DC → DC +FcεRI 9

IL-4 → MC → MC 10

IL-4,10 → Th1 → Th1

IL-5 → Eo → Eo

GM-CSF → DC → DC 11

IgE$^+$

1. abnormální zpracování antigenu/alergenu (atopický epitop, exprese CD86/B7-2, snížená produkce IL-12)
2. abnormální prezentace antigenu antigen-prezentujícími bb (MHC II a/nebo CD87/B7-2)
3. abnormální rozpoznání antigenu lymfocyty T (TCR)
4. abnormální „pro-Th2“ transkripce DNA u lymfocytů T (STAT 6)
5. abnormální zdroj/množství IL-4 (žírné buňky, bazofily, eosinofily, lymfocyty T, NK bb)
6. snížená produkce IL-12 (makrofágy, dendritické a/nebo Langerhansovy bb)
7. snížená produkce IFNγ (makrofágy, lymfocyty T)
8. zvýšený sklon k produkci IgE (lymfocyty B)
9. abnormální exprese FcεRI (Langerhansovy bb, eosinofily)
10. abnormální složení FcεRI (chybí nebo je málo syntetizovaný β-řetězec)
11. zvýšená produkce dendritických buněk (zvýšená produkce GM-CSF)

TABLE I. Linkages with asthma and allergy

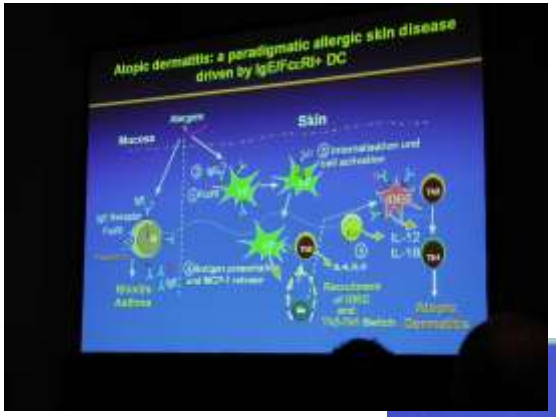
Chromosome	Candidate genes or products
1p	IL-12 receptor
2q	IL-1, cytotoxic T lymphocyte-associated antigen 4, CD28
3p24	B-cell lymphoma-6 (STAT-6 binding inhibition)
	Chemokine cell receptor 4
5q23-35	IL-3, IL-4, IL-5, IL-9, IL-13, GM-CSF
	LTC4S
	Macrophage colony-stimulating factor receptor
	β_2-Adrenergic receptor
	Glucocorticosteroid receptor
	TIM1, TIM3
6p21-23	MHC
	TNFs
	Transporters involved in antigen processing and presentation (*TAP1* and *TAP2*)
	Large multicatalytic proteolytic particles
7q11-14	T-cell receptor γ chain, IL-6
11q13	High-affinity IgE receptor (FcεRI) β chain
	Clara cell protein 16
	Fibroblast growth factor 3
12q14-24	IFN-γ
	Stem cell factor
	Nitric oxide synthetase (constitutive)
	β Subunit of nuclear factor Y (transcription factor for HLA genes)
	Insulinlike growth factor 1
	Leukotriene A_4 hydrolase
	STAT-6 (IL-4 STAT)
13q21-24	Cysteinyl leukotriene 2 receptor
14q11-13	T-cell receptor α and δ chains
	Nuclear factor κB inhibitor
16p11-12	IL-4 receptor
17p12-17	CC chemokine cluster
19q13	CD22, transforming growth factor β_1
20p13	ADAM-33

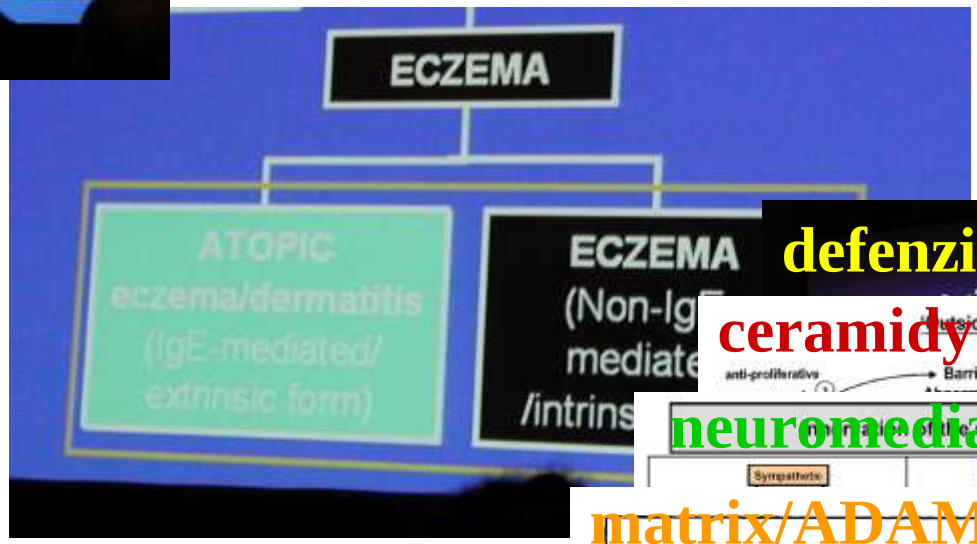
Genetics of hypersensitivity
J Allergy Clin Immunol 2003; 111: S495-501

TABLE II. Candidate genes of atopy and allergy

	Examples
Cytokines influencing allergic phenotype	
Eosinophil growth, activation, and apoptosis-inhibiting factors	IL-5, IL-3, GM-CSF, eotaxin, RANTES
Mast cell growth factors	IL-3, IL-9, IL-10, nerve growth factor, stem cell factor, transforming growth factor β
Histamine-releasing factors	Monocyte chemoattractant protein 1, monocyte chemoattractant protein 3, RANTES
IgE isotype switch factors	IL-4, IL-13
Inhibition of IgE isotype switch	IFN-γ, IL-12, IL-18, IL-23
Lipoxygenase pathway metabolism	*5LO*, 5-lipoxygenase–activating peptide, leukotriene C_4 synthase
Pro-inflammatory cytokines	IL-1α, IL-1β, TNF-α, IL-6
Anti-inflammatory cytokines	Transforming growth factor β, IL-10, interleukin-1 receptor antagonist
Receptors	
Antigen receptors	T-cell receptors (α/β, γ/δ), B-cell receptor (Ig, κ/λ light chain)
IgE	FcεRI β chain, FcεRII (CD23)
Cytokine gene receptors	IFN-γ receptor β chain, macrophage colony-stimulating factor receptor, IL-1 receptor, IL-4 receptor, TNF receptors
Adhesion molecules	Virus-like agent 4, vascular cellular adhesion molecule 1, intercellular adhesion molecule 1, leukocyte functional activating molecule-1, *TIM1, TIM3*
Corticosteroid receptor	Glucocorticoid receptor–heat shock protein 90
Neurogenic receptors	β_2-Adrenergic, cholinergic receptors
Nuclear transcription factors	Activating protein-1, nuclear factor of interleukin-2, octamer transcription factor-1, STAT-1/2, GATA3, T-box expressed in T cells, nuclear factor κB, inhibitor of nuclear factor kappa B, nuclear factor of activated T cells, STAT-4, STAT-6, BCL-6

Genetics of hypersensitivity
J Allergy Clin Immunol 2003; 111: S495-501





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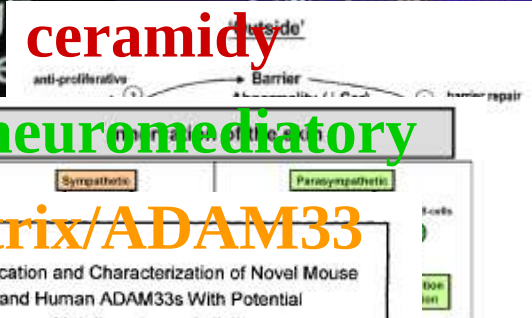

neuromediatory

matrix/ADAM33

Identification and Characterization of Novel Mouse and Human ADAM33s With Potential Metalloprotease Activity

Tsuyoshi Yoshinaka[1], Kazuhiro Nishii[2], Kouji Yamada[2], Hirohide Sawada[2], Eiji Nishiwaki[1], Katherine Smith[3], Kohichiro Yoshino[1], Hiroshi Ishiguro[2], Shigeki Higashiyama[4]

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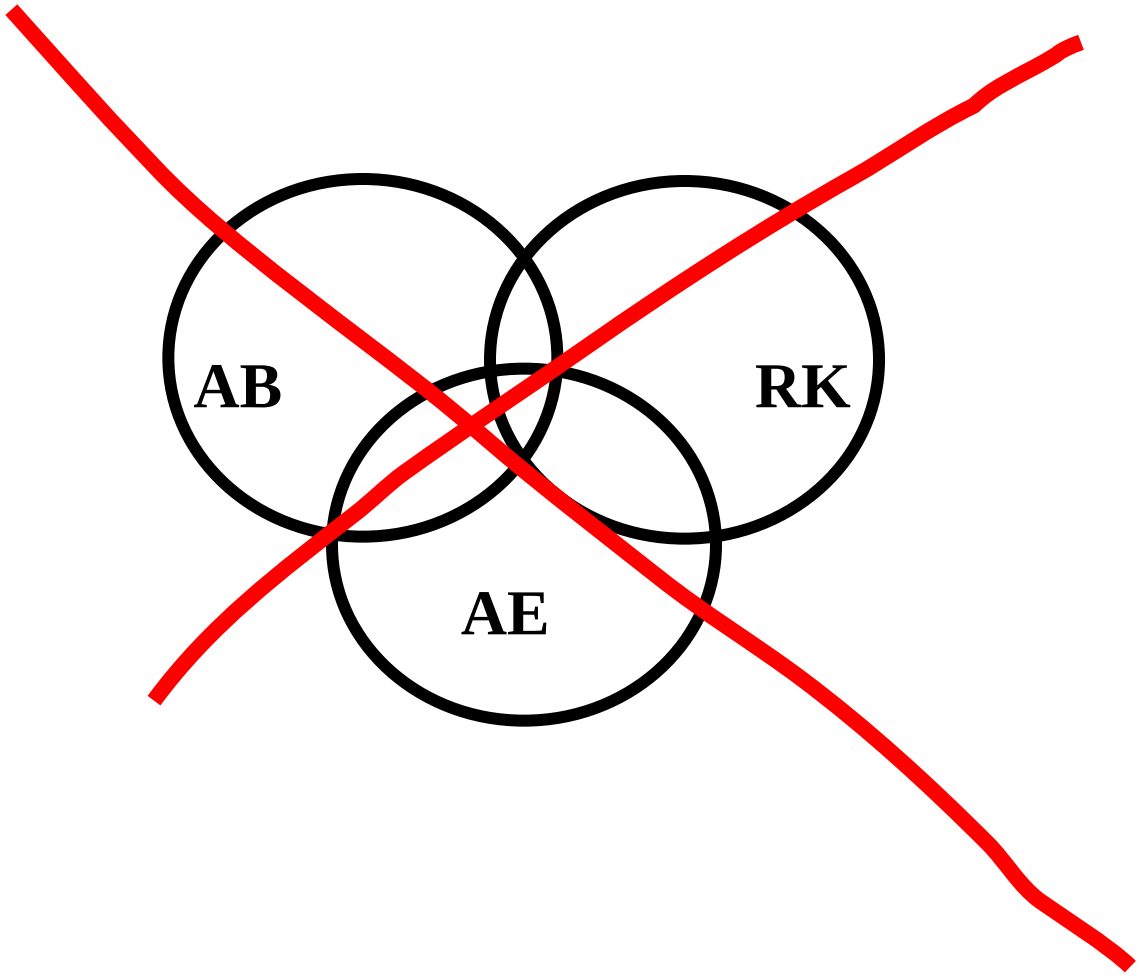
- uspořádat již získané informace
- začít na novo !!!


AB
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AE

TABLE I. List of SNPs genotyped in the COAST cohort ordered by chromosomal location

Marker	Gene	Chromosome position	Polymorphism	Location	Amino acid exchange	dbSNP rs no.*	Minor allele frequency in sample
1	*VCAM1*	1p32-p31	T→C	Promoter (−1594)		rs1041163	0.167
2	*SELP*	1q21-q24	G→A	Exon 7	S330N	rs6131	0.163
3			G→T	Exon 12	V640L	rs6133	0.092
4	*SELE*	1q22-q25	A→C	Exon 3	S128R	rs5361	0.102
5	*IL10*	1q31-q32	C→A	Promoter (−571)		rs1800872	0.265
6			C→T	Promoter (−854)		rs3021097	0.259
7			G→A	Promoter (−1117)		rs1800896	0.474
8	*IL1A*	2q12-q21	T→C	Promoter (−889)		rs1800587	0.299
9	*IL1B*	2q14	C→T	Promoter (−1418)		rs16944	0.337
10			C→T	Exon 5	F105F	rs1143634	0.216
11	CTLA4	2q33	C→T	Promoter (−318)		rs5742909	0.092
12			A→G	Exon 1	T17A	rs231775	0.374
13	*CCR2*	3p21	G→A	Exon 1	V62I	rs1799864	0.080
14	*CCR3*	3p21	C→T	Exon 1	P39L	rs5742906	0.002
15	*CCR5*	3p21	wt→Δ580-611	Exon 1		rs333	0.107
16			G→A	Promoter (−2454)		rs1799987	0.475
17	*IL5RA*	3p26-p24	G→A	Promoter (−80)		rs2290608	0.261
18	*GC*	4q12-q13	G→T	Exon 3	E416D	rs7041	0.415
19			C→A	Exon 3	T420K	rs4588	0.246
20	*CD14*	5q22-q32	C→T	Promoter (−159)		rs2569190	0.480
21	*IL4*	5q31	C→T	Promoter (−590)		rs2243250	0.138
22	*IL13*	5q31	C→T	Promoter (−1112)		rs1800925	0.186
23			C→T	Intron 3		rs1295686	0.239
24			G→A	Exon 4	R110Q	rs20541	0.220
25	*TCF7*	5q31	C→A	Exon	P19T	rs5742913	0.115
26	*CSF2*	5q31	T→C	Exon 4	I117T	rs25882	0.175
27	*ADRB2*	5q31-q32	A→G	Exon 1	R16G	rs1042713	0.356
28			C→G	Exon 1	Q27E	rs1042714	0.440
29			C→T	Exon 1	T164I	rs1800888	0.007
30	*IL9*	5q31-q35	C→T	Exon 5	T113M	rs2069885	0.132
31	*LTC4S*	5q35	A→C	Promoter (−444)		rs730012	0.257
32	*LTA*	6p21	A→G	Intron A		rs909253	0.303
33	*TNF*	6p21	G→A	Promoter (−308)		rs1800629	0.141
34			G→A	Promoter (−238)		rs361525	0.053
35	*IL6*	7p21-p15	G→C	Promoter (−572)		rs1800796	0.070
36			G→C	Promoter (−174)		rs1800795	0.460
37	*NOS3*	7q35-q36	A→G	Promoter (−922)		rs1800779	0.368
38			G→T	Exon 7	E298D	rs1799983	0.348
39	*C5*	9q32-q34	A→G	Exon 24	I802V	rs17611	0.452
40	*SDF1*	10q11	G→A	3′ UTR (+800)		rs1801157	0.202
41	*CCI6*	11q11-qter	G→A	Exon 1		rs3741240	0.351
42	*FCERB1*	11q13	A→G	Promoter (−109)		rs1441586	0.424
43			A→G	Exon 7	E237G	rs569108	0.040
44	*VDR*	12q13	T→C	Exon 1	M1T	rs2228570	0.399
45			G→A	Intron 8		rs1544410	0.406
46	*IL4RA*	16p12	C→T	Promoter (−3223)		rs2057768	0.300
47			A→G	Exon 5	I50V	rs1805010	0.454
48			C→T	Exon 7	N142N	rs3024571	0.089
49			A→C	Exon 12	E375A	rs1805011	0.114
50			G→T	Exon 12	L389L	rs2234898	0.111
51			T→C	Exon 12	C406R	rs1805012	0.105
52			T→C	Exon 12	S478P	rs1805015	0.164
53			A→G	Exon 12	Q551R	rs1801275	0.196
54			T→C	Exon 12	S761P	rs3024678	0.007
55	*NOS2A*	17q11-q12	C→T	Exon 10 (+231)	D346D	rs1137933	0.217
56	*EOTAXIN*	17q21	G→A	Exon 1	A23T	rs3744508	0.208
57			G→A	Promoter (−1328)		rs4795895	0.175
58	*C3*	19p13	C→G	Exon 3	R102G	rs2230199	0.189
59	*ICAM1*	19p13	A→T	Exon 2	K56M	rs5491	0.000
60			G→A	Exon 4	G214R	rs1799969	0.133
61	*TGFB1*	19q13	C→T	Promoter (−509)		rs1800469	0.284

Genetic variation in immunoregulatory pathways and atopic phenotypes in infancy
J Allergy Clin Immunol 2004; 113: 511-8

TABLE I. Genes with statistically significant, more than two-fold expression in atopic dermatitis as compared with psoriasis

Gene name	Probe set	GeneBank accession	Relative intensity (mean ± SEM)		*P* value	Ratio of AD: psoriasis gene expression
			AD	Psoriasis		
Nel-like 2	32598_at	D83018	2825 ± 491	374 ± 81	.004	7.56
CCL-18/PARC	32128_at	Y13710	3078 ± 874	422 ± 108	.028	7.30
CCL-27/CTACK	31644_at	AJ243542	5972 ± 1283	972 ± 326	.010	6.14
CCL-13/MCP-4	37454_at	AJ001634	1100 ± 327	203 ± 36	.041	5.41
Osteoblast specific factor 2	1451_s_at	D13666	2278 ± 436	463 ± 53	.009	4.92
Carbonic anhydrase II	40095_at	J03037	1074 ± 171	223 ± 31	.004	4.82
PPP1R5	39366_at	N36638	1095 ± 205	243 ± 30	.008	4.50
Tenascin precursor	32818_at	X78565	1951 ± 381	572 ± 95	.014	3.41
Cysteine-rich protein 1	33232_at	AI017574	9515 ± 1545	2939 ± 389	.007	3.24
PPP1R5	39364_s_at	Y18207	480 ± 87	167 ± 23	.014	2.88
Plasminogen activator inhibitor, type I	38125_at	M14083	305 ± 52	111 ± 18	.012	2.76
CD1a	34926_at	M28825	1246 ± 264	475 ± 58	.032	2.62
CD26 dipeptidylpeptidase IV	34823_at	X60708	207 ± 19	86 ± 19	.001	2.41
Lymphocyte-specific protein 1	36493_at	M33552	1210 ± 174	549 ± 68	.011	2.20
Cysteine-rich protein 2	35828_at	D42123	5314 ± 923	2450 ± 249	.026	2.17
AQP3 gene for aquaporine 3 (water channel)	39249_at	AB001325	16,912 ± 1418	8053 ± 1008	.001	2.10
Collagen VI α-1 C-terminal globular domain	38722_at	X15880	4872 ± 905	2369 ± 200	.039	2.06
N-myc downstream regulated gene 1	36933_at	D87953	5605 ± 770	2749 ± 213	.013	2.04

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