



11th International
Congress on

Autoimmunity

Lisbon, Portugal, 16-20 May 2018

Program Book



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Table of Contents

Welcome Message	3
Committee	4
Timetable	5
General Information	12
Information for Presenters - Oral and Poster Presenters	14
Discover Lisbon	16
Networking Events	17
CME/CPD Accreditation	18
Venue Maps	20
Awards	22
5th International Symposium on Vaccines	25
Academy of Autoimmunity	26
Zandman/Colton Patient Forum for Autoimmunity	28
Editorial Meetings	29

Scientific Program

Monday, 14 May	31
Tuesday, 15 May	35
Wednesday, 16 May	39
Thursday, 17 May	45
Friday, 18 May	75
Saturday, 19 May	111
Sunday, 20 May	143
Index of Authors	155

Acknowledgements & Industry Support

Acknowledgements	181
Industry Supported Sessions	184
Exhibition Map	188
List of Exhibitors	188
Supporter & Exhibitor Profiles *	189

Friday, 18 May 2018

10:30 - 12:30

Pavilion 3C

PSORIASIS AND PSORIATIC ARTHRITIS

Chairs: J. Scher (USA)

398

R. Perricone (Italy)

399

10:30 PATHOGENESIS AND TARGETS IN PSORIATIC ARTHRITIS

400

J. Scher (USA)

SMALL MOLECULES BEYOND PSORIASIS AND PSORIATIC ARTHRITIS

401

A. Selmi (Italy)

MEASUREMENT OF FRACTION OF EXHALED NITRIC OXIDE (FeNO)
MEASUREMENT IN PSORIATIC PATIENTS

402

A. Damiani, M. Rizzi, R. Dejan, A. Airolidi, A. Cristiano, S. Petrou, P. Santus (Italy)

11:20 CORRELATIONS BETWEEN HLA-Cw6 AND IL-23 IN PSORIASIS VULGARIS

403

E. Darwin, S.L. Kuncorowati, D. Elvira (Indonesia)

11:30 ALEXITHYMIA IN INFLAMMATORY ARTHRITIS: PRELIMINARY INVESTIGATION
IN A CLINICAL SAMPLE OF PATIENTS AFFECTED BY RHEUMATOID AND
PSORIATIC ARTHRITIS

404

G.L. Fonti, M.S. Chimenti, P. Conigliaro, J. Hitaj, M. Galluzzo, M. Talamonti, B. Kroegler,
E. Greco, R. Perricone (Italy)

11:40 ASSOCIATIONS OF ELEMENTS OF ANTIGEN-PRESENTING MACHINERY WITH
PSORIASIS VULGARIS IN POLISH POPULATION

405

P. Kusnierczyk, A. Wiśniewski, Ł. Matusiak, A. Szczerkowska-Dobosz, I. Nowak,
W. Łuszczek, M. Jasek, M. Wagner, W. Niepiekło-Miniewska, K. Wilczyńska (Poland)

11:50 PIGMENTARY CHANGES IN SYSTEMIC SCLEROSIS: A WINDOW TO SEVERITY

406

B. Khaitan, S. Mittal (India)

12:00 SERUM MATRIX METALLOPROTEINASE-3 NORMALIZATION IS A POTENTIAL
PREDICTOR FOR LESS ONE-YEAR RADIOGRAPHIC PROGRESSION IN
RHEUMATOID ARTHRITIS

407

L.F. Chen, J.D. Ma, Y.Q. Mo, X.Y. Du, D.H. Zheng, L. Dai (China)

12:30 - 14:00

Auditorium II

INDUSTRY SUPPORTED SESSION 2

Not included in main CME/CPD credit

Please refer to page 187 of the book for full information

12:30 - 14:00

Exhibition Hall

LUNCH BREAK, EXHIBITION, POSTERS AND SHORT E-POSTERS DISCUSSIONS

Correlations between HLA-Cw6 and IL-23 with psoriasis vulgaris



Eryati Darwin, Sri Lestari Kuncorowati, and Dwitya Elvira
Faculty of Medicine, Andalas University, Padang-Indonesia
May, 2018



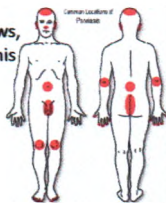
Faculty Disclosure

☒ No, nothing to disclose
☐ Yes, please specify:

Company Name	Honorary/ Expenses	Consulting/ Advisory Board	Funded Research	Royalties/ Patent	Stock Options	Ownership/ Equity Position	Employee	Other (please specify)
Example: company XYZ	X		X		X			

Introduction

- Psoriasis is a complex, chronic, multifactorial, inflammatory disease of unknown etiology
- bilaterally symmetric, non-pruritic lesion of elbows, knees, umbilicus, lower back, scalp and glans penis
- affects 1-3% of population, all ages, equal sex
- Subtype: plaque, guttate, pustular, and erythrodermic
- Common type: plaque psoriasis (90%)→ psoriasis vulgaris



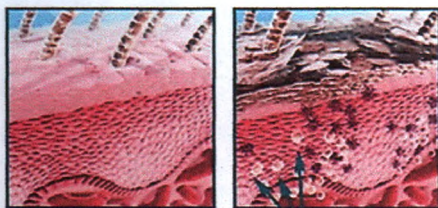
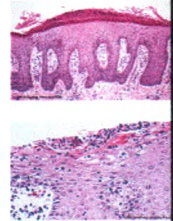
Clinical features

- Well demarcated erythematous plaques covered by fine, loosely adherent, silvery-white scales
- Auspitz sign: bleeding when scale is lifted from plaque
- Koebner phenomenon: new lesions at site of trauma



Histologic Appearance

- Hyperproliferation of keratinocytes with parakeratosis
- elongation of rete ridges
- thin/no granular cell layer
- microabscesses
- Increased mitotic figures above basal layer
- increased angiogenesis
- infiltrate of T lymphocytes, macrophages, neutrophils and dendritic cells

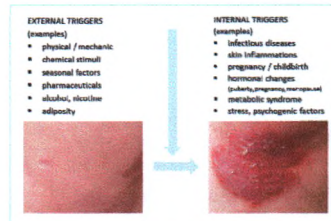


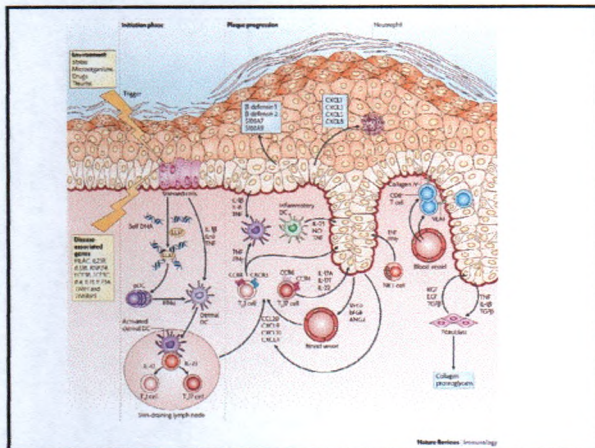
T-cells



Pathogenesis

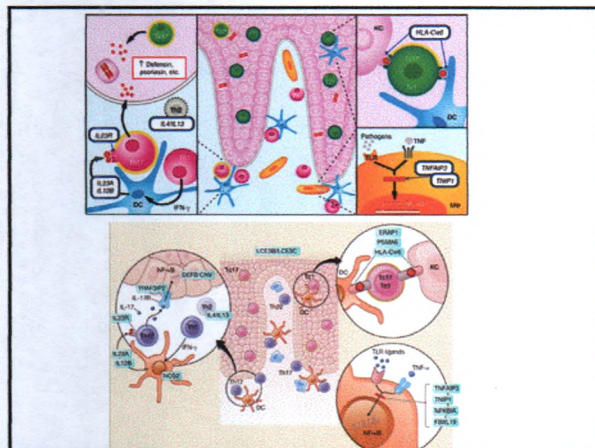
- not yet fully understood
- autoimmune basis, mediated by the Th1 lymphocytes
- chronic systemic inflammatory disorder resulting from the combination of predisposing genetic factors and environmental triggers





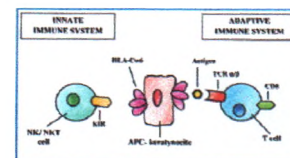
Model integrating the genetics and immunology of psoriasis

- Genetic -Immune response→ Psoriasis
- Genetic-Infection-immune response→ Psoriasis
- Stress-Immune response→ Psoriasis



HLA-Cw6

- The MHC region on chromosome 6 contain a risk allele for psoriasis most notably HLA Cw6
- HLA-Cw6 expressed on APCs
 - trigger specific immune responses by presentation of processed antigen to the TCR of CD8+ T cells.
 - trigger innate immune response by interaction of HLA-Cw6 NK and NKT cells.



IL-23

- IL-23 was discovered in 2000 by Oppmann and co-workers
- Since its, IL-23 has been linked to the pathogenesis of autoimmune inflammation.
- IL-23 expressed by activated monocytes, macrophages, dendritic cells, T cells, B cells, and endothelial cells
- mediate the epidermal hyperplasia, keratinocyte and inflammation



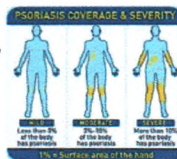
Aim of Study

- To determine the role of IL-23 in psoriasis vulgaris we studied its correlation with HLA-Cw6 in psoriasis vulgaris



Methods

- Cross-sectional study
- 30 unrelated patients with psoriasis vulgaris and 30 non-psoriatic from Dermatology and Venereology Department of M.Djamil Hospital outpatients
- Dermatological examination with psoriasis severity assessment using PASI score
- IL-23 by using ELISA methods
- HLA-Cw6 allele by PCR-SSP method.
- Data were analyzed statistically
- Approved by Ethics Committee on Health Research at Medical Faculty, Andalas University



Results

Table 1.1: Characteristic of Psoriasis patients and control group

Characteristics	Psoriasis (n=30)	Kontrol (n=30)	%	p
Age				
< 40	14	15	48,3	1,000
≥ 40	16	15	51,7	
Gender				
Male	16	16	53,3	1,000
Female	14	14	46,7	

Kolmogorov-Smirnov: Normal distribution

Psoriasis may begin at any age, it is most likely to appear between the ages of 15 and 30 years.



Table 1: Severity, Onset and Duration of Disease of Psoriasis Vulgaris Patients

Characteristic	Psoriasis Vulgaris (n=30)	
	N	%
Severity (PASI)		
Mild (PASI <8)	23	76,7
Moderate (PASI >8 to < 12)	5	16,7
Severe (PASI >12)	2	6,6
Onset of Disease		
< 40 years	23	76,7
> 40 years	7	23,3
Duration of disease		
< 5 years	14	46,7
> 5 years to < 15 years	14	50
>15 years	1	3,3

Possession of certain HLA Class I antigens, particularly HLA-Cw6, is associated with an earlier age of onset and with a positive family history



Table 3: Expression of HLA-Cw6v allele in psoriasis and control group

Groups	HLA Cw-6				p
	Positive		Negative		
	N	%	N	%	
Psoriasis (n=30)	6	20	24	80	0,024
Control (n=30)	0	0	30	100	

- HLA Cw-6 found in 20% of psoriatic patients
- No correlation with duration and severity of disease
- Ikaheimo et al, 1996: Cw6-positive patients have an earlier disease onset
- Guðjónsson, et al., 2002: Patients who are Cw6 positive had a lower age at onset
- Chen and Tsai, 2017: The worldwide frequency of the HLA-Cw6 allele varies greatly, with it being generally higher in white people than in Asians.
- Chen and Tsai, 2017: HLA-Cw6 has been found to be associated with guttate psoriasis



Table 4: The level of IL-23 in psoriasis and control group

Groups	IL-23 (pg/ml) (Mean±SD)	p
Psoriasis	31,2088±10,5809	0,013
Control	25,5839±5,3964	

- Fitch et al., 2007: Several recent studies suggest that psoriasis is a Th17 cell-mediated disease driven by IL-23



Table 5: Correlation between HLA Cw-6 with IL-23 in Psoriasis Vulgaris

HLA-Cw-6 in Psoriasis	IL-23 (pg/ml) (Mean±SD)	p
Positive (n=6)	32,2126±11,2846	0,791
Negative (n=24)	30,9033±10,6036	

IL-23 in Severe Psoriatic patients (PASI >12): - 50,43
- 51,46

HLA-Cw6, is associated with an earlier age of onset and with a positive family history
Burlando et al, 2016: HLA-Cw6 allele is associated with a faster and better clinical response rate to human monoclonal antibody directed against interleukin 12 and interleukin 23

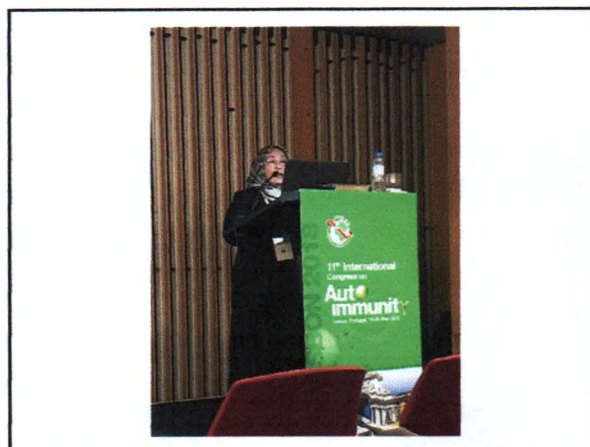


- Psoriasis as a consequence of dysregulated immunity that lead inflammation
- HLA Cw-6 is a diseases-associated gene, which together with other factors stimulates inflammation
- IL-23 as pro-inflammatory cytokine, play a crucial role in the pathogenesis of psoriasis
- IL-23 activation stimulates production of IL-17 and IL-22, which directly affect skin inflammation, may promote keratinocyte proliferation and differentiation



Conclusion

IL-23 is associated with psoriasis vulgaris, and HLA-Cw6 may play a role in the occurrence of psoriasis inflammation through IL-23.



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CERTIFICATE OF ORAL PRESENTATION

This is to certify that

Prof. Dr. Eryati Darwin

presented the abstract entitled

CORRELATIONS BETWEEN HLA-Cw6 AND IL-23 IN PSORIASIS VULGARIS

as an oral presentation at the

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Yehuda Shoenfeld, MD, FRCP, MaACR
Congress President

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