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The American Society for Histocompatibility and Immunogenetics

30th Annual Meeting Abstracts

October 2–6, 2004

San Antonio Convention Center
San Antonio, Texas

Meeting Attendees: This is the Only Copy of the Abstracts You Will Receive

The Official Journal of ASHI
American Society for Histocompatibility & Immunogenetics

Human Immunology

Aims and Scope

Human Immunology will publish research in Immunogenetics, Cellular, Molecular and Clinical Immunology. Section editors for each of these fields have been selected to expedite the review process. The immunogenetics section will present findings on structural polymorphism of HLA genes in healthy and diseased populations, their function, regulation and expression in normal and malignant cells. Our NEW VISION is to improve the quality of human health through the understanding and application of immunogenetics. The molecular immunology section will present research on the structure and function of molecules that play a role in the activation and regulation of the immune system. Cellular immunology will encompass the broad areas of *in vitro* and *in vivo* studies of cellular immune responses in transplantation, autoimmunity and infectious diseases. The clinical immunology section will present studies on the immunology of cell, tissue and organ transplantation, autoimmune, allergic and infectious diseases, and anti-tumor responses. Manuscripts will be reviewed and published rapidly. The chief criteria for publication are originality and quality. Pertinent information about immunologic studies in animal systems may also be considered. Topics of general interest form the basis for editorials and special issues.

ABSTRACTS

The American Society for Histocompatibility and Immunogenetics

30th Annual Meeting
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The American Society for Histocompatibility and Immunogenetics (ASHI) is pleased to provide you with the 2004 Abstract Edition of *Human Immunology*. This special edition contains abstracts selected for oral and poster presentations at ASHI's 30th Annual Meeting to be held October 2-6, 2004, at the San Antonio Convention Center in San Antonio, Texas.

ASHI will distribute this issue at the 30th Annual Meeting. Additional copies will be available at the meeting site for a $30 charge. Plan to refer to the enclosed abstracts during the educational and poster sessions.

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FAX: (510) 749-5200
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Leiden, 2300RC
Netherlands
PHONE: 011-31-71-526-3800
FAX: 011-31-71-521-6751
E-MAIL: F.H.J.Claas@lumc.nl

John M. Hart, MBA, CHS (2005)
Johns Hopkins University
Immunogenetics Lab
2041 East Monument Street
Baltimore, MD 21205
PHONE: (410) 955-3600
FAX: (410) 955-0431
E-MAIL: hartjm@jhmi.edu

Doreen Jezek, MS, CHS, CLSP(MB)
1403 Crystal Lake Circle West
Pearland, Texas 77584
PHONE: (281) 997-7854
E-MAIL: Doreen.jezek@sbcglobal.net

Robert Lewis, PhD
University of Mississippi Medical Center
Department of Pathology
2500 North State Street
Jackson, MS 39216-4505
PHONE: (610) 984-1562
FAX: (610) 984-1835
E-MAIL: rlewis@pathology.umsmed.edu

Peter Nickerson, MD, FRCPC
Canadian Blood Services
Winnipeg Centre
777 William Avenue
Winnipeg, MB R3E 3R4
Canada
PHONE: (204) 789-1130
FAX: (204) 783-6780
E-MAIL: peter.nickerson@bloodservices.ca

Lori D. Osowski, MS, CHS (2005)
American Red Cross
National Histocompatibility Lab
22 South Greene Street, Box 173
Baltimore, MD 21201-1595
PHONE: (410) 328-2973
FAX: (410) 328-2967
E-MAIL: osowskil@usa.redcross.org

Anajane G. Smith, MA (2005)
Seattle Cancer Care Alliance
Clinical Immunogenetics Lab
825 Eastlake Avenue East G7-200
Seattle, WA 98109
PHONE: (206) 288-1147
FAX: (206) 288-1169
E-MAIL: asmith@seattlecca.org

Victoria Turner, PhD, Diplomate, ABHI (2004)
St. Jude Hospital
HLA Lab C4035
322 North Lauderale
Memphis, TN 38105
PHONE: (901) 495-3468
FAX: (901) 495-3100
E-MAIL: vicky.turner@stjude.org

4
130

IDENTIFICATION OF THE NEW VARIANT HLA-A2911 IN TWO UNRELATED ZULUS Khaled R. Alkharsah,[1] Holger-Andreas Elsner,[2] Martin Dedicot,[1] Thomas F. Schulz,[1] Rainer Blasczyk[3], [1]Virology, Hannover Medical School, Hannover, Germany; [2]MMD, Medical Molecular Diagnostics GmbH, Dresden, Germany; [3]Transfusion Medicine, Hannover Medical School, Hannover, Germany

We here report the identification of the new allele HLA-A2911 in two healthy, unrelated Zulus (Hlabisa, South Africa). The allele was detected by sequencing-based typing (PCR-SBT) in the course of a disease association study. Its presence in two unrelated individuals suggests that it may be not uncommon in Zulu population. Compared to A290201, to which it is closest, the new variant is characterized by a nonsynonymous nucleotide exchange (A C) at nucleotide position 151 of exon 3. It results in the amino acid exchange Gln Pro at position 141 of the mature polypeptide. This position, located in the helix H1 of the $\alpha 2$ domain, is probably not directly involved in peptide binding. Yet, since proline is a helix breaker, the structure of the neighbouring pocket F may be altered, which might result in a change of the peptide binding characteristics compared to the A2902 molecule.

4
131

MICA EXON 5 MICROSATELLITE ALLELES IN TURKISH PATIENTS WITH FMF E. Yigitbas,[1,2] A. Uner,[2] M. Tevfik Dorak,[3] W.Z. Ding,[1] G. Tatayoglu,[2] B. Balci,[4] A. Duzova,[5] F. Ozaltin,[5] S. Ozen,[5] N. Besbas,[5] R. Topaloglu,[5] E. Yilmaz,[4] A. Bakkaloglu,[5] M. Ozguc,[4] E. Kansu,[2] P.A. Fraser[1], [1]CBR Institute for Biomedical Research, Harvard Medical School, Boston, MA, USA; [2]Oncology Institute, Hacettepe University, Hacettepe, Turkey; [3]Clinical Medical Sciences, Hacettepe, Turkey; University of Newcastle-upon-Tyne, Newcastle-upon-Tyne, United Kingdom; [4]Medical Biology, Hacettepe University, Hacettepe, Turkey; [5]Pediatric Nephrology-Rheumatology, Hacettepe University, Hacettepe, Turkey

MICA encodes a stress inducible ligand for the NKG2D, located 46 kb centromeric to HLA-B. Familial Mediterranean Fever (FMF) is an autoinflammatory disease, with a carrier rate of 20% in Turks. It was suggested that MICA exon 5 alleles may modulate the course of FMF. Therefore we investigated MICA exon 5 microsatellite alleles and linkage disequilibrium (LD) with HLA-B alleles in 101 unrelated donors and 114 FMF patients in Turkey.

Microsatellite alleles were defined by size sequencing (ABI Prism). HLA-B alleles were determined by Labtype SSO assay (One Lambda Inc.). MICA exon 5 microsatellite frequencies were: A4 (15.84 %), A5 (15.34 %), A5.1 (21.78 %), A6 (31.68 %), A9 (15.34 %) in control subjects, and A4 (9.64 %), A5 (17.10 %), A5.1 (23.24 %), A6 (35.96 %), A9 (14.03 %), in FMF patients. Both loci were in Hardy-Weinberg equilibrium. HLA-B*52 was in weak LD with MICA*A6 (D=0.0153, P=0.02). HLA-B*52 was significantly increased in cases, (10.5% vs 2.0%; P=0.01, OR=5.82, 95% CI=1.24 to 54.5), while the slight increase in MICA*A6 s (OR=1.44, 95% CI=0.80 to 2.57) was not statistically significant.

Our data are more likely due to population stratification, rather than associations between MHC loci and risk of FMF. Formal analyses to detect and quantitate population stratification will be needed to test this hypothesis. These data will provide the basis for our future studies of MICA as a modifier in FMF. Research was partially supported by The Scientific and Technical Research Council of Turkey and Research Fund of Hacettepe University. Gratitude is expressed to the staff at the Immunogenetics Laboratory, Hacettepe Oncology Institute, MEGA Ltd., ODITAS AS., and One Lambda Inc.