New Discoveries

Tri-Society Annual Conference

LISBON | 2009

Cellular and Cytokine Interactions in Health and Disease

• Society for Leukocyte Biology • International Cytokine Society
• International Society for Interferon and Cytokine Research

October 18-21, 2009

Monument to the Age of Discovery, Lisbon, Portugal

Vancouver

The Three R's of Immunity

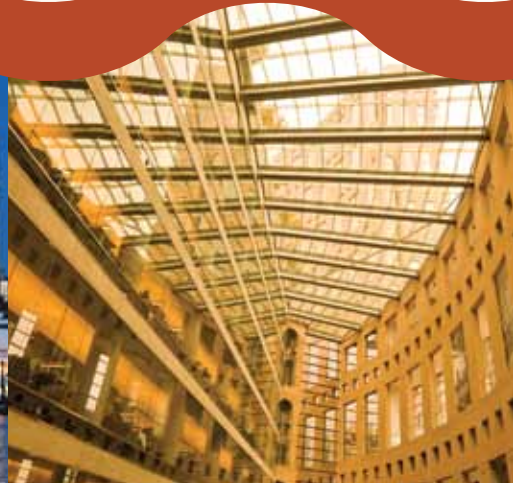
Recognition, Response & Resolution

Annual Meeting

Society for Leukocyte Biology

International Endotoxin & Innate Immunity Society

October 6-9, 2010



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Explore with us

Dear 2009 Meeting Attendees,

It is with great pleasure that we welcome you to the 2009 Tri-Society Meeting of SLB, ICS, and ISICR: Cellular and Cytokine Interactions in Health and Disease. The gathering of our three societies at one meeting is a very special event, providing rare opportunities for linkages and networking.

The three of us, Luis Montaner (SLB), Scott Durum (ICS), and Michael Tovey (ISICR) have been working together on the program for the better part of two years and are extremely gratified to see the level of participation from all three societies.

We have more than 700 registrants, 63 invited speakers, more than 50 selected talks from abstracts and over 500 poster presentations. Given the world economy and all of the other challenges facing us in gathering together in Lisbon, we couldn't be more pleased with the turnout and we expect that each of you will be thrilled by the scientific program that we have put together.

We think you will also find that Lisbon is a city truly worth exploring and we are excited to be able to hold our meeting in this beautiful and historical city. Please take advantage of the opportunities to explore and enjoy Lisbon, a UNESCO World Heritage Site, while you are here.

We would also like to sincerely thank all of our sponsors without whom this program would not have been possible.

This is an excellent opportunity to reconnect with old colleagues and forge new relationships. Thank you again for your participation and enjoy your stay in beautiful Lisbon.

Welcome to Lisbon! We wish you a wonderful and educational experience.

Best regards,

Luis Montaner (Chair), Scott Durum, Michael Tovey

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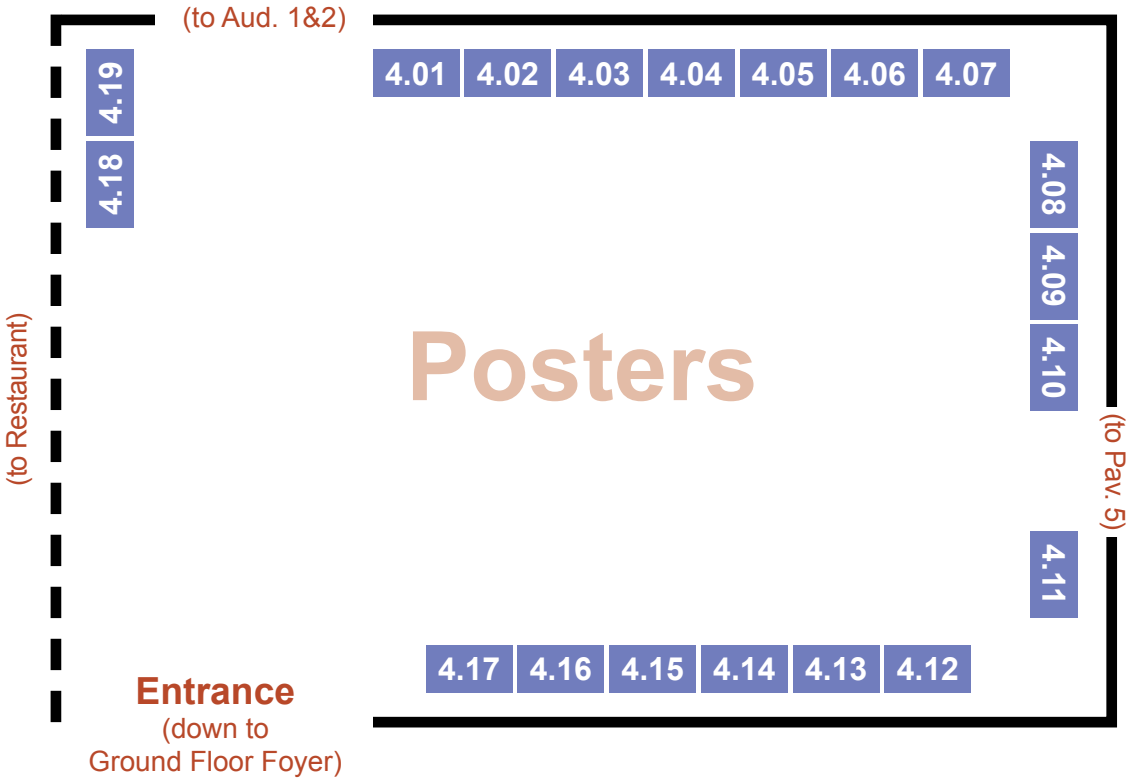
Exhibitor Listings

Pavilion 4



Booth 4.01
eBioscience, Inc.

10255 Science Center Drive
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Email: contact@eBioscience.com
Web: www.eBioscience.com
Representatives: Chris Oakley & Alasdair Stewart

eBioscience provides innovative, high quality reagents to researchers worldwide that empower the process of scientific discovery in the areas of cellular immunity and oncology. Our extensive portfolio of leading edge cell analysis products and technologies, focused on flow cytometry and immunodetection, position our customers to be at the forefront of science.

Booth 4.02
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Email: diaclone@gen-probe.com
Web: www.gen-probe.com
Representatives: Eric Cairns, Dr. Warren Higgs, and Laurence Ringenbach

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San Diego, CA 92121
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Tel: US Toll-Free: 1-877-Bio-Legend (1-877-246-5343)
Fax: 858.455.9587
Email: info@biolegend.com
Web: biolegend.com
Representatives: Brad Kraft and Dzung Nguyen

BioLegend offer optimal immunological reagents at an outstanding Value. More Sizes, More Colors (PerCP/Cy5.5, Pacific Blue™, Alexa Fluor®,…). New markers for Th17 (IL-17A, RORγt), Treg (FOXP3, FR4), and Stem Cell (SSEA-1, SSEA-4). Bulk Discounts on Low Endotoxin, Azide-Free (LEAF™) Antibodies for Functional Analyses. Sample testing services. Custom assay development for ELISA, Luminex, and more.

Booth 4.04
Bender MedSystems GmbH

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Web: www.bendermedsystems.com
Representatives: Dr. Claudia Jursik, Estela Rodriguez

Bender MedSystems´ product portfolio includes ELISA kits, FlowCytomix bead based multiplexing systems, antibodies and proteins targeted for research in various fields of immunology and cellular biology: Adhesion, Apoptosis, Cytokine Research and Tumor Biology. Close collaboration with research institutes all over the world ensure Bender MedSystems is always up to date with the latest trends and discoveries. Our goal is to provide innovative tools to the scientific community. **Bender MedSystems GmbH** holds certificates for ISO9001:2000 and ISO13485:2003 Quality Standards. Our products are regularly tested by internal and external experts and we provide the highest standards of technical and scientific service.

Booth 4.05
Biomonitor A/S

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Web: www.biomonitor.dk
Representatives: Arsalan Kharazmi, CEO, Klaus Bendtzen, CSO, Winie Nielsen, VP Sales & Marketing

Biomonitor A/S is a Danish state-of-the-art, GLP compliant clinical reference laboratory. Biomonitor has extensive experience in analyzing protein drugs and in developing cell-based assays, culture of different cell lines and measurement of cell products in particular cytokines by different assays such as MxA, ELISA, RIA, CPE, iLite™ and EIA. Our focus areas are interferons, anti-TNFalpha drugs and antibody drugs including monoclonals. Biomonitor performs PK analysis for drug concentration and/or biological activity and antibodies to these drugs.

Booth 4.06
PeproTech, Inc.

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Email: info@peprotech.co.uk
Web: www.peprotech.com
Representatives: Elaine Prpa, Dagmar Prien, Brigitte Ricard

PeproTech manufactures an extensive line of Recombinant Human, Murine and Rat Cytokines as well as a complementary line of Monoclonal Antibodies, Affinity Purified Polyclonal Antibodies, Affinity Purified Biotinylated Polyclonal Antibodies and Elisa Development Kits.

Booth 4.07
Cell Signaling Technology®

3 Trask Lane
Danvers, MA, 01923
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Phone: 978.867.2300
Fax: 978.867.2400
Email: info@cellsignal.com
Web: www.cellsignal.com
Representatives: Jessica Switzer and Susan Rogers

Cell Signaling Technology, the leader in the production of activation-state antibodies, now offers a growing selection of cytokines and growth factors. These products are produced in-house with the highest possible purity and bioactivity. Stringent product specifications and quality control ensure maximum lot-to-lot consistency. Product validation includes the use of our antibodies to demonstrate biological effectiveness. As with all of our products, technical support is provided by the scientists who produce the products and know them best.

Booth 4.08
R&D Systems Europe Ltd

19, Barton Lane
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Abingdon
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Fax: +44(0)1235.551115
Email: info@rndsystems.co.uk
Web: www.RnDSystems.com
Representative: Bradley Mabbutt

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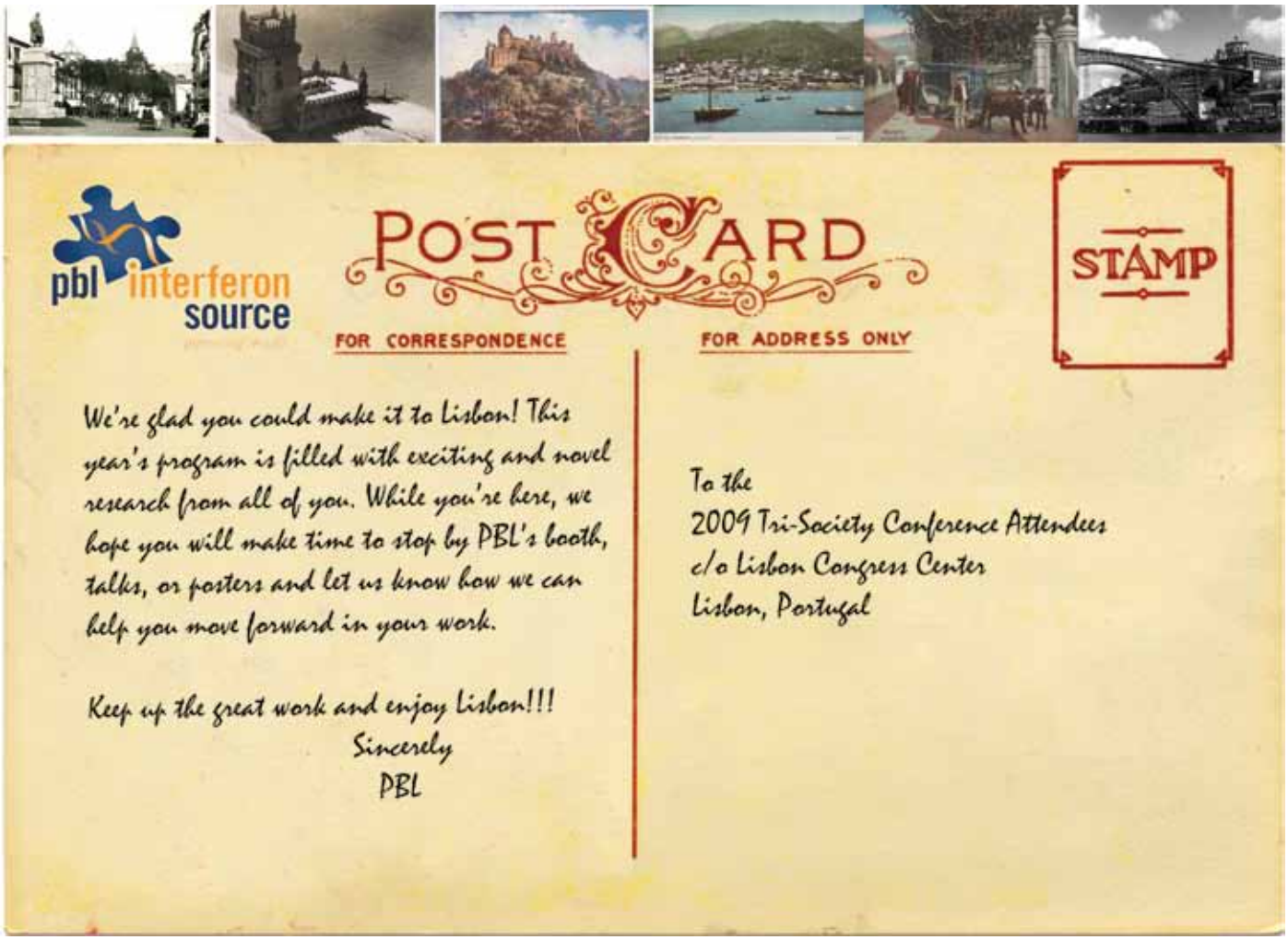
Booth 4.09
Mabtech AB

Box 1233
131 28 Nacka Strand
Sweden
Tel: +46.8.716.27.00
Fax: +46.8.716.27.01
Email: mabtech@mabtech.com
Web: www.mabtech.com
Representatives: Alexandre Antoni (sales), Sten Braesch-Andersen (scientific)

Mabtech AB is a research focused biotech company that emerged from the immunology department at Stockholm University in 1986. Mabtech develops, manufactures and markets high quality ELISpot and ELISA kits for analysis of cytokines and other immunological effector molecules. We also provide our antibodies as separate reagents for other applications including flow cytometry and immunocytochemistry. We continuously strive to develop the ELISpot method and can now offer designated B-cell ELISpot kits for enumeration of B cells secreting immunoglobulin and/or antigen-specific antibodies. Mabtech has also recently launched kits for Fluorospot; a new sensitive assay, which enables detection of cells secreting multiple cytokines.

Booth 4.10
PBL InterferonSource

131 Ethel Road West
Suite 6
Piscataway, NJ 08854-5900
USA
Tel: 732.777.9123
Fax: 732.777.9141
Email: info@interferonsource.com
Web: www.interferonsource.com
Representatives: Robert Pestka, Timothy Doris, Jaleel Shujath, Ronald Jubin, Thomas Lavoie, Mike Skawinski, Doranelly Koltchev



PBL InterferonSource is the leading supplier of interferon research tools to the life science researcher. Founded in 1990 by Dr. Sidney Pestka, PBL is the company scientists turn to for their interferon-related needs: products, services, information and know-how. With over 100 combined years of interferon experience, PBL strives to aid researchers around the world in our common quest to help humanity.

Booth 4.11
Invitrogen

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Email: techsupport@invitrogen.com
Web: www.invitrogen.com
Representatives: Kyle Miller, Thao Sebata, and Chris Brotski

Invitrogen Corporation provides products and services that support academic and government research institutions and pharmaceutical and biotech companies worldwide in their efforts to improve the human condition. Invitrogen is your source for cellular pathway exploration tools, including ELISAs, Luminex® assays, kinase activity assays, protein arrays, antibodies and recombinant proteins. Our products help researchers improve their understanding of the role of both extracellular proteins and intracellular proteins and their function in the disease process. **Invitrogen** is committed to providing the most innovative pathway solutions along with personalized customer support.

Booth 4.12
BD Biosciences

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Fax: 0032.2.401.70.94
Email: help.biosciences@europe.bd.com
Web: www.bdbiosciences.com
Representatives: Jean-Francois Mathieu

BD Biosciences, a segment of Becton, Dickinson and Company, is one of the world's leading businesses focused on bringing innovative tools to life science researchers and clinicians. Its product lines include: flow cytometers, cell imaging systems, monoclonal antibodies, research reagents, diagnostic assays, and tools to help grow tissue and cells.

Booth 4.13
Meso Scale Discovery

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USA
Tel: 240.631.2522
Fax: 240.632.2219
Email: events@mesoscale.com
Web: www.mesoscale.com
Representatives: Richard Dennis and Michel Popielarz (Account Managers)

Meso Scale Discovery® (MSD®) develops and markets solutions for singleplex and multiplex biological assays, including assays for toxicology biomarkers, metabolic biomarkers, cytokines, and phosphoproteins. MSD's platform is based on MULTI-ARRAY® technology, a proprietary combination of patterned arrays and electrochemiluminescence detection, enabling large numbers of measurements with exceptional sensitivity, wide dynamic range, and convenience.

Booth 4.14
Shenandoah Biotechnology, Inc.

101 Camars Drive
Warwick, PA 18974
USA
Tel: 215.672.7550
Fax: 215.672.7552
Email: ricr@shenandoah-bt.com
Web: www.shenadoah-bt.com
Representative: Michael Jones, President & CSO and
David Sehy, Director, Business Development

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Booth 4.15
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Email: info.eu@stemcell.com
Web: www.stemcell.com
Representatives: Lydie Guyon and Karin Gaisbauer

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Booth 4.16
Quansys Biosciences

365 N 600 W
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Fax: 435.750.6869
Email: info@quansysbio.com
Web: www.quansysbio.com
Representatives: Matthew Groll (General Manager) and Chris Lyman (R&D Product Manager)

Quansys Biosciences is the leader in the development and manufacture of planar based protein arrays. With over 10 years of experience in multiplex technologies, Quansys Biosciences has the expertise and ability to produce high quality arrays with high sensitivity and low variability. Our products and services include custom array development and manufacture, Q-Plex retail kits, sample testing, custom printing, and Q-View Imager and Software.

Booth 4.17
Symansis NZ Ltd

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Email: leanne.daly@symansis.com
Web: www.symansis.com
Representative: Leanne Daly

Symansis specializes in the development, manufacture and marketing of human cell expressed (hcx) human cytokines, chemokines, adhesion molecules and growth factors, polyclonal antibodies for cell signalling and inhibitors in the life science and drug discovery areas. Over 120 recombinant human proteins (hcx), including both ligands and receptors are available. ELISA kits with human recombinant protein standards expressed from human cells (hcx) are also available. Symansis production methods using human cell lines ensure natural human post-translational modifications. Post-translational modifications such as glycosylation assist with protein folding, stability and antigenicity, and vary from species to species.

Booth 4.18
Journal of Leukocyte Biology

FASEB AdNet
9650 Rockville Pike
Bethesda, MD 20814-3998
USA
Tel: 800.433.2732 ext. 7117
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Email: adnet@faseb.org
Web: www.jleukbio.org
Representative: Amy Huter-Imming

The Journal of Leukocyte Biology, established in 1981, is published by the Society for Leukocyte Biology. JLB publishes peer-reviewed manuscripts on original investigations focusing on the cellular and molecular biology of leukocytes and on the origins, the developmental biology, biochemistry and functions of granulocytes, lymphocytes, mononuclear phagocytes and other cells involved in host defense and inflammation.

Booth 4.19
Chimera Biotec GmbH

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Fax: +49 (0)231.9742.844
Email: info@chimera-biotec.com
Web: www.imperacer.com
Representative: Jan Detmers

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General Information

Registration

The Registration Counters are located on the Ground Floor Foyer of the Congress Centre. Staff will be available to provide conference materials and process check-ins during the following hours:

Sunday, October 18th: 11:00 AM – 5:30 PM
Monday, October 19th: 7:30 AM – 5:00 PM
Tuesday, October 20th: 8:00 AM – 5:00 PM
Wednesday, October 21st: 8:00 AM – 2:30 PM

The fees for onsite for registration are as follows and payment can be made via check or credit card (VISA, MC, AmEx)

Registration Fees (USD $)
Academic/Government (Member): $650
Academic/Government (Non-Member): $750
Industry (Member): $850
Industry (Non-Member): $900
Students*: $400
One Day Registration**: $200
Extra Opening Reception Ticket: $50
Extra Banquet Ticket: $100
Accompanying Guests***: $375

* Must provide advisor's name and email address
** Includes entrance to scientific sessions only
*** Includes banquet and reception tickets and entrance to scientific sessions for guest residing in the same household

The registration fee for participants includes:
- Admission to all scientific sessions
- Admission to the commercial exhibition
- Conference Program & Abstract Books, and tote
- Opening Reception at the Lisbon Congress Centre, Pavilion 4, on Sunday, October 18th from 7:30 – 8:30 PM
- Conference Banquet at the National Agronomy Pavilion on Tuesday, October 20th from 7:30 – 10:30 PM

Exhibition

The exhibition will be open in Pavilion 4 at the following times:
Sunday, October 18th: 4:30 PM – 8:30 PM
Monday, October 19th: 8:30 AM – 6:30 PM
Tuesday, October 20th: 8:30 AM – 6:30 PM
Wednesday, October 21st: 8:30 AM – 2:30 PM

Please note the Exhibitor Information located on page 6 for further information about the companies. Use coffee breaks, lunch hours and reception times to visit the exhibition booths.

Badges
Participants are asked to wear their name badges at all times during the conference.

Coffee Breaks
Refreshments will be available in Pavilion 4 at the following times:
Monday, October 19th: 9:30 – 10:00 AM & 3:00 – 3:30 PM
Tuesday, October 20th: 9:30 – 10:00 AM
Wednesday, October 21st: 9:00 – 9:30 AM

Receptions
Receptions will be hosted with light fare and cash bars during the following times in Pavilion 4:
Opening Reception, Sunday, October 18th, 7:30 – 8:30 PM
Poster Session I, Tuesday, October 20th, 3:00 – 5:00 PM

Banquet
A Conference Banquet will be hosted on Tuesday, October 20th, at the National Agronomy Pavilion from 7:30 – 10:30 PM with dinner and a cash bar. Bus service will be provided from the conference hotels. See the Conference Bus Schedule on page 67 for details.

Student Mixer
A Student Mixer Social Sponsored by SLB will be held on Monday, October 19th from 7:30 – 10:00 PM at the Trindade. Light fare and refreshments will be provided. While the event is free, you must visit the JLB Booth (4.18) to pick up a complimentary ticket for attendance. Please feel free to come and join your fellow junior scientists for this fun networking event.

Lunch
Lunch break is "On Your Own" at the following times:
Monday, October 19th: 12:00 PM – 1:30 PM
Tuesday, October 20th: 12:15 PM – 1:30 PM
Wednesday, October 21st: 11:30 AM – 12:30 PM

A restaurant and coffee/sandwich bar is available on site, plus many more options in the surrounding area. Boxed lunches will be available for purchase on Wednesday via ticket purchase at the bar located near Pavilion 4 to provide a convenient alternative so attendees can participate in their society business meetings.

Speaker Ready Room
The Speaker Ready Room is located near the entrance to Pavilion 4 in room 1.13 and will be open during the listed registration hours.

Speakers should visit this office as soon as possible to upload presentations to ensure they are properly loaded well in advance of their lecture times. Please allow a minimum of two hours prior to presentation time for loading all presentation files. Files in MAC and PC will be accepted and AV technicians will be available to assist with loading presentations as well as any other AV needs. It is recommended that you bring your presentation on a USB flash drive. Speakers should arrive in their session rooms at least 30 minutes prior to the session to ensure all materials are properly loaded prior to the start of the session.

Parking
The Lisbon Congress Centre has two parking lots with 1,100 spaces available. The cost of parking per day is 12,40€ (VAT included). When arriving to the center, park inside the garage and pick up a ticket. When paying, ask for a parking day ticket instead of per hour rate.

Handicap Accessibility
We are pleased to provide any assistance you may require. Please visit the registration desk to let us know of any accommodations you may require.

Medical Care
Clinics and hospitals provide 24 hour emergency services. The national emergency phone number is 112. Nursing staff is provided on site at the conference during main conference hours. Hotels have a doctor on call.

Membership
Members of all three societies are invited and encouraged to attend the individual business meetings for each society held on Wednesday, October 21st from 11:30 – 12:30 PM. Boxed lunches will be available (via tickets) for purchase at the bar located near Pavilion 4. Please attend the meetings to learn more about the happenings, structure and future directions of your society. If you are not currently a member of any society, please feel free to visit the society information table on the Ground Floor Foyer, near the Registration Counters, for membership applications.

Poster Set-Up
All Poster Presentations will be posted in Pavilion 4 and 5 for the entire conference and available for "browsing" during main conference hours. Highlighted Poster Sessions are on Tuesday, October 20th from 3:00 – 5:00 PM (during which light refreshments will be provided) and on Wednesday, October 21st from 12:30 – 2:30 PM (during which boxed lunches will be available for purchase). Poster Presenters are asked to be available at their poster display to discuss their work with visitors during designated Poster Session times as follows:

Poster Session A: Tuesday, October 20th from 3:00 PM – 5:00 PM
Poster Session B: Wednesday, October 21st from 12:30 PM – 2:30 PM
(See Poster section starting on page 27 for A/B designation next to topic title.)

For display, please look for the proper section based on category as well as poster board number as provided in your acceptance letter (or refer to the Poster section of this program book starting on page 27) in order to display your poster in the proper location. Posters are to be affixed on the boards using double sided tape (provided). If you require assistance, please contact one of the staff located in the Pavilion 4 and Pavilion 5 poster board areas or visit the registration counters. **Please post your materials no later**

than Monday, October 19th 8:00 AM and remove materials by Wednesday, October 21st 5:00 PM. The Conference organizers are not responsible for loss or damage to any materials posted.

Bank
Lisboa Congress Centre has 2 ATM machines available.

Multibanco ATMs are widespread throughout the city.
Barclays Bank (217 911 100; Avenida da República 50)
Cota Câmbios (213 220 480; Rossio 41; 8.30am-10pm)
Grupo Deutsche Bank (210 001 200; Rua Castilho 20)
Top Atlântica (213 108 800; Avenida Duque de Loulé 108) Lisbon's American Express representative.

Currency and Credit Cards
The Euro (€) is the official currency of Portugal. Foreign currency can be exchanged at the airports, hotels, banks or exchange offices. Most hotels, restaurants and shops accept internationally valid credit cards.

Electricity
The electricity in Portugal runs on 220 volts. The frequency is 50Hz and the plugs have two male contact pins.

Society Information Table
Please visit the Society Information Table near the Registration Counters on the Ground Floor Foyer to view the three participating society's membership information, newsletters and other materials.

Internet
There are several different ways for delegates to connect to the internet. Wireless is available in the Lisbon Congress Centre at the conference "Hot Spot" in Pavilion 5 (bring your own laptop). All Conference hotels have business centers with internet access and in private accommodations for an additional fee. Check your specific hotel for rates.

Language
English is the official language of the conference. No simultaneous translation will be provided.

Insurance, Liability
Neither the organizers nor the Lisbon Congress Centre can be held responsible for any personal injury, loss, damage, accident to private property or additional expenses incurred as a result of delays or changes in air, rail, sea, road or other services, strikes, sickness, weather, acts of terrorism and any other cause. All participants are encouraged to make their own arrangements for health and travel insurance.

CONFERENCE SECRETARIAT
John Lord, Executive Director
Society for Leukocyte Biology
9650 Rockville Pike
Bethesda, MD USA
Tel: 301-634-7453
Fax: 301-634-7455
Email: slb@faseb.org

CONGRESS HOME PAGE
www.trisociety2009.org

Invited Speakers

The Tri-Society Conference Organizers wish to thank all of our invited speakers who have enriched our extensive program with their valuable participation and contributions.

Shizuo Akira, Osaka University, Japan
Antonio Alcami, Centro de Biologia Mol. Severo Ochoa CSIC-UAM, Spain
David Artis, University of Pennsylvania, PA, USA
Darren Baker, Biogen Idec, MA, USA
Franck Barrat, Dynavax Technology Corporation, CA, USA
Klaus Bendtzen, Rigshospitalet University Hospital, Denmark
Rod Bremner, University of Toronto, Canada
Vincenzo Bronte, Universita Degli Studi Di Padova, Italy
Gordon D. Brown, University of Aberdeen, UK
Silvia Bulfone-Paus, Research Center Borstel, Germany
Marco A. Cassatella, University of Verona, Italy
Andrew Caton, The Wistar Institute, PA, USA
Ajay Chawla, Stanford University, CA, USA
Fabio Cominelli, University of Virginia, VA, USA
Lisa Coussens, UCSF, CA, USA
Anthony Coyle, MedImmune, MD, USA
Ed Croze, Bayer HealthCare Pharmaceuticals, Inc., USA
Michael David, UCSD, CA, USA
Mariapia Degli-Esposti, Centre for Exp. Immunology, Lions Institute, AU
Vishva Dixit, Genentech, CA, USA
Charles (Chuck) Drake, John Hopkins University, MD, USA
Sir Gordon Duff, University of Sheffield Molecular Medicine, UK
Matthias Ernst, Ludwig Institute for Cancer Research, AU
Takashi Fujita, Kyoto University, Japan
Laurie Glimcher, Harvard, MA, USA
Steve Goodbourn, Division of Medial Sciences, St.Georges's University, UK
Maureen M. Goodenow, University of Florida, College of Medicine, FL, USA
Siamon Gordon, NCI, SAIC-Frederick, MD, USA
Chris Hunter, University of Pennsylvania, Medicine, PA, USA
Michael Hutchinson, St. Vincent's University Hospital Dublin, IE
Lionel Ivashkiv, Cornell University, NY, USA
Michael Karin, UCSD, CA, USA
Sergei V. Kotenko, New Jersey Medical School, NJ, USA
Steven L. Kunkel, University of Michigan Medical School, MI, USA
Dan Littman, NYU School of Medicine, NY, USA
Richard Locksley, UCSF, CA, USA
Domenico Mavilio, Istituto Clinico Humanitas, Italy
Judy Mikovits, University of Nevada, Reno, NV, USA
Cesar Munoz-Fontela, Mount Sinai, NY, USA
Phil Murphy, NIH-LMI/NIAID, MD, USA
Anne O'Garra, The National Institute for Medical Research, UK
John O'Shea, NIAMS/NIH, MD, USA
Suzanne Ostrand-Rosenberg, University of Maryland, BC, MD, USA
Manolis Pasparakis, University of Cologne, Germany
Leon C. Platanias, Northwestern University Medical School, IL, USA
Amanda Proudfoot, Merck Serono Geneva Research Centre, Switzerland
Bali Pulendran, Emory Vaccine Center, GA, USA
Boris Reizis, Columbia University Medical Center, NY, USA
Luigina Romani, University of Perugia, Italy
Sasha Rudensky, Univ. of Wash. School of Medicine Imm., WA, USA
Federica Sallusto, Institute for Research in Biomedicine, Switzerland
Robert W. Sauerwein, NCLMS-Medial Microbiology, Netherlands
Ke Shuai, Biological Chemistry-UCLA, CA, USA
Charalampos G. Spilianakism, Inst. of Mol. Biology and Biotech., Greece
Ira Tabas, Columbia University, NY, USA
Jenny Ting, UNC CCBC, NC, USA
Jurg Tschopp, Universite de Lausanne, Switzerland
Dario Vignali, St. Judes Children's Research Hospital, Immunology, TN, USA
Peter Weller, Harvard University, MA, USA
John Wherry, The Wistar Institute, PA, USA
Hao Wu, Cornell University, NY, USA
Tom Wynn, NIAID-NIH, MD, USA
Dong Zhang, Scripps Research Institute, CA, USA

Awards

2009 ICS AWARDS

Outstanding Scholar Award
First Place: Frank van de Veeerdonk, Radboud University, Netherlands
Second Place: Swaidani Shadi, Cleveland Clinic, USA
Third Place: Jamie Flammer, Cornell, USA
Fourth Place: Christoph Menzel, University of Pittsburgh, USA

Post-Doctoral Investigator Award
First Place: Julie Ribot, Instituto de Medicina Molecular, Portugal
Second Place: Tracy Putoczki, Ludwig Institute, Australia
Third Place: Annalisa Camporeale, University of Turin, Italy
Fourth Place: Tilmann Buerckstuemmer, Research Center for Molecular Medicine, Austria

Young Investigator Award
First Place: Tao Lu, Cleveland Clinic, USA
Second Place: Bruno Silva-Santos, Instituto de Medicina Molecular, Portugal
Third Place: Yasuo Yoshioka, Osaka University, Japan

2009 ISICR AWARDS

Honorary Membership
Sidney Grossberg, Medical College of Wisconsin, USA
Charles Weissmann, Scripps, USA

2009 Seymour & Vivian Milstein Award
Glen Barber, University of Miami, USA
Peter Staeheli, University of Freiburg, Germany

2009 Milstein Young Investigator Award
Hiroki Ishikawa, University of Miami, USA
Xiao-Ling Li, University of Maryland, USA
Niamh Mangan, Monash University, Australia
Ramtin Rahbar, University of Toronto, Canada
Benjamin Tenoever, Mount Sinai School of Medicine, USA

2009 Christina Fleischmann Award
Caini Liu, Cleveland Clinic, USA

2009 Outgoing President
Eleanor Fish, University Health Network, Canada

2009 ISICR Milstein Travel Award Winners
Manel Amri, University USTHB
Joseph Ashour, Mount Sinai School of Medicine
Betsy Barnes, UMDNJ
Brigitte Blanchard Sury, CNRS
Viviana Blank, University of Buenos Aires
Daniel Burke, University of Toronto
Lally Chan, The University of Hong Kong
Olivia Chan, University of Toronto
Mounira Chelbi-Alix, CNRS
Jieliang Chen, Shanghai Med. Col. Fudan Univ.
Wanjun Chen, NIDCR/NIH
Ahmet Civas, Paris Descartes University
Ann Cornish, Walter and Eliza Hall Institute
Alexandre Corthay, University of Oslo
Marco De Andrea, Medical School of Turin
Heather Ezelle, University of Maryland, Baltimore
Brenda Fredericksen, University of Maryland
Nir Friedman, Weizmann Institute of Science
Ka Yee Fung, Monash Institute of Medical Research
Carole Galligan, Toronto General Research Institute
Yiwei Gao, Stony Brook University
Sanjukta Ghosh, Harvard
Alan Goodman, University of Washington
Nathalie Grandvaux, Université de Montréal
Simon-Pierre Gravel, Université de Montréal
Claire Greenhill, Monash Institute of Medical Research
Francesca Gugliesi, University of Turin
Bret Hassel, University of Maryland School of Medicine
Deborah Hodge, NDI-Frederick
Markus Hofer, University Hospital Marburg
Teresa His, University of Maryland Sch. of Medicine
Nadia Kavrochorianou, Hellenic Pasteur Institute
Hiu (Jessie) Kiu, The Walter and Eliza Hall Institute
Christophe Kraus, RWJMS - UMDNJ
Thomas Kuri, Inst. for Med. Microbio. & Hygiene
Christophe Lallemand, CNRS
Andrew Larner, Virginia Commonwealth University
Chien-Kuo Lee, National Taiwan University
Jana Liskova, Charles University
Barbora Lubyova, Institute of Immunology and Microbiology
Katherine Martin, Monash University
Jenny Miu, McGill University
Reem Mohamed, Institue of Endemic Diseases
Markus Mordstein, University of Freiburg
Bei Morrison, Taussig Cancer Institute
Kazuhide Onoguchi, Kyoto University
Anna Overby, University of Freiburg
Leesa Pennell, University of Toronto
Hongwei Qin, University of Alabama at Birmingham
Nupur Raychaudhuri, Kellogg Eye Center at University of Michigan
Shlomit Reich-Zeliger, Weizmann
Erin Rogers, Department of Immunology
Giovanna Romeo, Sapienza University of Rome
Saleela Ruwanpura, Monash University
Martina Schroeder, National University of Ireland Maynooth
Marc Servant, University of Montreal
Martina Severa, Istituto Superiore di Sanità
Ha Youn Shin, Stony Brook University
Håkan Steen, Temple University
Shadi Swaidani, Cleveland Clinic-Lerner Reseach
Emmanuel Thomas, NIDDK-NIH
Chafia Touil-Boukoffa, USTHB
Shawna Wall, CTRC, UTHSCSA
Marta Wlodarska, University of British Columbia
Jae-Kwang Yoo, University of Toronto
Raza Zaidi, National Cancer Institute, NIH

2009 SLB AWARDS

Marie T. Bonazinga Award
Peter Ward, University of Michigan Medical School, USA

Jean Thorbecke Award
Julie Margarian Blander, Mount Sinai School of Medicine, USA

Outgoing Council Awards
Luis Montaner, The Wistar Institute, USA
Michelle Swanson, University of Michigan Medical School, USA

2009 SLB Travel Award Winners
Shaheed Abdulhaqq, The Wistar Institute
Jessica Allen, Cincinnati Children's Hospital
Seyeon Bae, Seoul National University College of Medicine
Shashi Bala, U Mass. Medical School
Andre Boonstra, Erasmus Medical Center
Natalija Budimir, UMC Groningen
Lynn Butler, University of Birmingham
Ilaria Cervellini, Brighton and Sussex Medical School
Nor Fazila Che Mat, Queen's University
Okki Cho, Ajou University
Jessica Cohen, Cleveland Clinic
Irazu Contreras, McGill University
Chrysoula Deligianni, IMBB-FORTH
Senad Divanovic, Cincinnati Children's Hospital Medical Center
Daniel Eklund, Linköping University
Julia Foldi, Weill Graduate School of Cornell University
Ka Yee Fung, Monash Institute of Medical Research
Bethsebah Gekonge, The Wistar Institute
Mallary Greenlee, University of Notre Dame
Christina Guzzo, Queen's University
Marc Hanschen, Brigham and Women's Hospital
Marieke Hoeve, University of Edinburgh
Evan Jacobs, UMDNJ
Joanna Jaworska, Universite Laval
Vladimir Jurisic, University of Kragujevac
Kanstantsin Katlinksi, The Res. Cntr. for Hem. & Transfusiology
Yuliya Katlinskaya, The Res. Cntr. for Hem. & Transfusiology
Hyemin Kim, Seoul National University College of Medicine
Hiu Kiu, The Walter and Eliza Hall Institute of Medical Science
Hsin-Ni Li, Centre for Inflammation Research/QMRI
Mohlopheni Marakalala, University of Cape Town
Helen McGettrick, University of Birmingham
Peyman Nakhaei, McGill University/ Lady Davis Institute
Rossella Parrotta, Universita' Degli Studi di Torino
Oscar Pello, Istituto Clinico Humanitas
Maya Poffenberger, University of British Columbia
Michele Pritchard, Cleveland Clinic
Suhkneung Pyo, Sungkyunkwan
Christiane Quiniou, University of Montreal
Megha Rajasekhar, The Centenary Institute
Michael Schmohl, University of Tübingen
Marina Tiemi Shio, McGill University
Alex Shnyra, Kansas City University of Medicine and Biosciences
Shadi Swaidani, Cleveland Clinic
Goro Tajima, Brigham and Women's Hospital
Costin Tomescu, The Wistar Institute
Silvia Uriarte, University of Louisville
Timothy Welliver, University of Michigan
Bin Wen, John Curtin School of Medical Research, ANU
Todd Wuest, University of Oklahoma HSC
Shiyan Yu, Fudan University Shanghai Medical College

Scientific Program Schedule

(Note: The code preceding the title indicates its location in the Abstract Book. If no code appears, there is no abstract provided.)

Sunday, October 18th

11:00 AM – 5:30 PM

Registration – Ground Floor Foyer

12:30 PM – 2:15 PM

SLB Presidential Awards Presentations - Auditorium 1
Chairs: Matthew Fenton and William Nauseef

12:30 – 12:45 PM
Introduction: Matthew Fenton and William Nauseef

Post – Doc and Junior Faculty Finalists

12:45 – 1:00 PM
(SLBAW1-A) c-Myc Triggers Macrophage Alternative Activation and Controls Macrophage Activity and Survival in Tumour
Oscar Pello, Istituto Clinico Humanitas, Milano, IT

1:00 – 1:15 PM
(SLBAW1-B) Granule Exocytosis Contributes to TNF-Alpha and PAF-Induced Priming in Human Neutrophils
Silvia M. Uriarte, University of Louisville, Louisville, KY, USA

1:15 – 1:30 PM
(SLBAW1-C) Heightened Activation of Plasmacytoid Dendritic Cells and Increased NK Activity in HIV-1 Exposed, Uninfected Intra-venous Drug Users
Costin Tomescu, The Wistar Institute, Philadelphia, PA, USA

Student Finalists

1:30 – 1:45 PM
(SLBAW2-A) A Diffusion Barrier in the Plasma Membrane During the Closure Stage of Macropinocytosi
Timothy P. Welliver, University of Michigan, Ann Arbor, MI, USA

1:45 – 2:00 PM
(SLBAW2-B) Long Range Genomic Cytokine-Receptor Interaction Regulates Gene Expression
Chrysoula Deligianni, IMBB-FORTH, Heraklion, GR

2:00 – 2:15 PM
(SLBAW2-C) Soluble Human CXCR2: Structure, Properties, Bioactivity
Kanstantsin Katlinski, The Research Center for Hematology & Transfusiology, Minsk, BY

2:20 PM – 5:30 PM

Plenary Award Session - Auditorium 1
Chairs: Eleanor Fish, Scott Durum, William M. Nauseef, and John Sims

2:20 – 2:25 PM
Awards and Recognitions Introduction: Luis Montaner

2:25 – 2:35 PM
Tri-Society Recognition of Joe Oppenheim: Scott Durum

2:35 – 2:50 PM
G. Jeanette Thorbecke Award, SLB: William Nauseef
Julie Margarian Blander, Mount Sinai School of Medicine, New York, NY, USA

2:50 – 3:10 PM
ISICR Awards Presentations: Robert Silverman
Honorary Membership – Sidney Grossberg and Charles Weissmann
2009 Seymour & Vivian Milstein Award – Glen Barber and Peter Staeheli
2009 Milstein Young Investigator Award – Hiroki Ishikawa, Xiao-Ling Li, Niamh Mangan, Ramtin Rahbar, Benjamin Tenover
2009 Christina Fleischmann Award - Caini Liu
2009 Outgoing President – Eleanor Fish

3:10 – 3:30 PM
ICS Awards Presentations: John Sims
Outstanding Scholar Awards:
First Place – Frank van de Veerdonk
Second Place – Swaidani Shadi
Third Place – Jamie Flammer
Fourth Place – Christoph Menzel

Post-Doctoral Investigator Award:
First Place – Julie Ribot
Second Place – Tracy Putoczki
Third Place – Annalisa Camporeale
Fourth Place – Tilmann Buerckstuemmer

Young Investigator Award:
First Place – Tao Lu
Second Place – Bruno Silva-Santos
Third Place – Yasuo Yoshioka

3:30 – 4:00 PM
Introduction of ICS Lifetime Achievement Award: John Sims
ICS – Lifetime Award Lecture: Nancy Ruddle, Yale University School of Medicine, New Haven, CT, USA
(PP2-099) Lymphotoxin: From Inflammation to Lymphoid Organs and Back

4:00 – 4:30 PM
Introduction of ISICR – Milstein Award Lecture I: Eleanor Fish
ISICR Milstein Award Lecture: Glen N. Barber University of Miami, FL, USA
Innate Immune Signaling Pathways that Regulate Type I Interferon Production

4:30 – 5:00 PM
Introduction of ISICR – Milstein Award Lecture II: Michael Tovey
ISICR Milstein Award Lecture: Peter Staeheli, University of Freiburg, Freiburg, DE
Role of IFN and Mx genes in Influenza Virus Defense

5:00 – 5:30 PM
Introduction of SLB Bonazinga Award: William Nauseef
SLB – Bonazinga Award Lecture: Peter Ward, University of Michigan Medical School, Ann Arbor, MI, USA
Molecular Determinants of Sepsis

4:30 PM – 8:30 PM

Exhibits – Pavilion 4

5:30 PM – 7:30 PM

Joint Plenary Session 1:
Opening Keynote Lectures - Auditorium 1
Chairs: Scott Durum and Luis Montaner

5:30 – 6:10 PM
(PL1-1) Requirement For Mature T Cells, Type I Interferon and STAT1 in Negative T Cell Selection
Michael David, UCSD, CA, USA

6:10 – 6:50 PM
(PL1-2) Tracking Cytokine Expression in vivo: Getting to the Root of the Matter
Richard Locksley, UCSF, CA, USA

6:50 – 7:30 PM
(PL1-3) Interchromosomal Cytokine Gene Expression
Charalampos G. Spilianakism, Institute of Molecular Biology and Biotechnology, GR

7:30 PM – 8:30 PM

Opening Reception – Pavilion 4

Monday, October 19th

7:30 AM – 5:00 PM

Registration – Ground Floor Foyer

8:30 AM – 6:30 PM

Exhibits – Pavilion 4

8:00 AM – 9:30 AM

Joint Plenary Session 2:
Pattern Recognition Receptors & Inflammation - Auditorium 1
Chairs: Luke O'Neill and Leon Platanias
Sponsored by PBL Interferon Source

8:00 – 8:30 AM
(PL2-1) Zc3h12a, a Negative Regulator in the TLR Response
Shizuo Akira, Osaka University, Osaka, Japan

8:30 – 9:00 AM
(PL2-2) Activation of an Antiviral Program through the Cytoplasmic Recognition of Non-Self RNA Patterns by RLR
Takashi Fujita, Kyoto University, Kyoto, JP

9:00 – 9:30 AM
(PL2-3) The Inflammasome
Jurg Tschopp, Universite de Lausanne, Epalinges, CH

9:30 AM – 10:00 AM

Coffee Break - Pavilion 4
Sponsored by Invitrogen

10:00 AM – 12:00 PM

Concurrent Basic Science Symposia 1:
Immunoregulation I – Auditorium 1
Chairs: Richard Locksley and Rachel Caspi

10:00 – 10:30 AM
(CBSS1-1) IL-35 and Regulatory T Cell Function
Dario Vignali, St. Judes Children's Research Hospital, Memphis, TN, USA

10:30 – 11:00 AM
Modulating Vaccine Responses with Innate Immunity
Bali Pulendran, Emory Vaccine Center, Atlanta, GA, USA

11:00 – 11:30 AM
(CBSS1-3) The Transcription Factor XBP1 Regulates Hepatic Lipogenesis, Immunity and Inflammation
Laurie Glimcher, Harvard School of Public Health, Boston, MA, USA

Selected Talks

11:30 – 11:45 AM
(CBSS1-4) A Critical Function of TGF-beta in the Generation of Adaptive and Natural CD4+Foxp3+ Regulatory T Cells
Wanjun Chen (Award Recipient), NIDCR/NIH, Bethesda, MD, USA

11:45 – 12:00 PM
(CBSS1-5) Dual Function for a Vision-Related Molecule: Retinoic Acid in the Eye May Contribute to Ocular Immune Privilege by Inducing T Regulatory Cells
Rachel R. Caspi, NIH, Bethesda, MD, USA

10:00 AM – 12:00 PM

Concurrent Basic Science Symposia 2:
Inflammation and Cancer – Auditorium 2
Chairs: Thomas Decker and Giorgio Trinchieri
Sponsored by Meso Scale Discovery

10:00 – 10:30 AM
Inflammation as an Inducer of Tumor-Induced Immune Suppression
Suzanne Ostrand-Rosenberg, University of Maryland, Baltimore, MD, USA

10:30 – 11:00 AM
(CBSS2-2) Protumor Immunity and Breast Cancer Development
Lisa Coussens, UCSF, CA, USA

Selected Talks

11:00 – 11:15 AM
(CBSS2-3) GRIM-19: A Novel Growth Regulator that Inhibits STAT3 and Beyond
Dhan V. Kalvakolanu, University of Maryland School of Medicine, Baltimore, MD, USA

11:15 – 11:30 AM
(CBSS2-4) IL-11 Mediated STAT3 Activation in Inflammation and Cancer
Tracy Putoczki (Award Recipient), Ludwig Institute for Cancer Research, Melbourne, AU

11:30 – 11:45 AM
(CBSS2-5) Origin, Phenotype and Function of Monocyte/Macrophage Subsets in Distinct Mammary Tumor Microenvironments
Jo A. Van Ginderachter, VIB-Vrije Universiteit Brussel, Brussels , BE

11:45 – 12:00 PM
(CBSS2-6) Interferon-Alpha Boosts Anti-Tumor Immunity Through Effects on T Cells and Dendritic Cells and Augments the Clinical Efficacy of Regulatory T Cell Depletion
Tyler J. Curiel, CTRC, San Antonio, TX, USA

10:00 AM – 12:00 PM

Concurrent Immunopathogenesis Symposia 1:
Immunopathogenesis I - Pavilion 5 A&B
Chairs: Nancy Reich and Michael Parkhouse

10:00 – 10:40 AM
(CIS1-1) Viral Pathogenesis and Interactions with Toll-Like Receptors: HIV
Maureen M. Goodenow, University of Florida College of Medicine, Gainesville, FL, USA

10:40 – 11:20 AM
Viral pathogenesis and interactions with Toll-like Receptors: CMV
Mariapia Degli-Esposti, Lions Institute Centre for Experimental Immunology, CITY, AU

11:20 – 12:00 PM
(CIS1-3) TLR Recognition of Self Nucleic Acids Hampers Glucocorticoids Anti-Inflammatory Activity in Lupus
Franck Barrat, Dynavax Technology Corporation, Berkeley, CA, USA

12:00 PM – 1:30 PM

Lunch on your own

1:30 PM – 3:00 PM

Joint Plenary Session 3:
Anti-Tumor Immunity – Auditorium 1
Chairs: Eleanor Fish and Ana Costa-Pereira
1:30 – 2:00 PM
(PL3-1) Role of Myeloid Cells and Inflammation in Cancer Progression and Metastasis
Michael Karin, UCSD, La Jolla, CA, USA

2:00 – 2:30 PM
Innate and Adaptive Inflammation in Prostate Cancer
Charles (Chuck) Drake, John Hopkins University, MD, USA

2:30 – 3:00 PM
Late Breaking Abstract: Detection and Immune Correlates of an Infectious Retrovirus, XMRV, in Blood Cells of Patients with Chronic Fatigue Syndrome and Cancer
Judy Mikovits, University of Nevada, Reno, NV, USA

3:00 PM – 3:30 PM

Coffee Break - Pavilion 4
Sponsored by Amgen

3:30 PM – 6:00 PM

Concurrent Basic Science Symposia 3:
Gene Activation – Auditorium 1
Chairs: Tom Hamilton and Ana Gamero

3:30 – 4:00 PM
(CBSS3-1) IKK/NK-kappaB Signaling in Chronic Inflammation
Manolis Pasparakis, University of Cologne, Cologne, DE

4:00 – 4:30 PM
Transcriptional and Epigenetic Regulation of Helper T Cell Differentiation
John O'Shea, NIAMS/NIH, MD, USA

4:30 – 5:00 PM
New Mechanisms in the Interferon-Gamma Signaling Network
Rod Bremner, University of Toronto, Toronto, CA

Selected Talks

5:00 – 5:15 PM
(CBSS3-4) Inhibition of Dynamin-Dependent Endocytosis Interferes with Type III IFN Expression in Bacteria-Infected Human Dendritic Cells
Taija E. Pietilä, National Institute for Health and Welfare, Helsinki, FI

5:15 – 5:30 PM
(CBSS3-5) Regulation of c-maf by IL-2
Susan John, Kings College London, London, UK

5:30 – 5:45 PM
(CBSS3-6) Single-Stranded RNA Viruses Inhibit P53 Transcriptional Activity by Post Translational Activation of ΔNP63
Christophe Lallemand (Award Recipient), CNRS, Paris, FR

5:45 – 6:00 PM
(CBSS3-7) Interferon Signaling is Activated in Response to DNA Damage
Nancy Reich, Stony Brook University, Stony Brook, NY, USA

3:30 PM – 6:00 PM

Concurrent Immunopathogenesis Symposia 2: Immunopathogensis II – Auditorium 2
Chairs: Michele Somes Swanson and Lee-Ann H. Allen
Sponsored by Celgene

3:30 – 4:00 PM
Immune Responses in Experimental Human Malaria Infections
Robert W. Sauerwein, NCLMS-Medial Microbiology, Nijmegen, NL

4:00 – 4:30 PM
(CIS2-2) Dendritic Cell-Derived Notch Ligand, Delta-Like 4, Regulates the Immune Environment via TLR9 during Mycobacterial Challenge
Steven L. Kunkel, University of Michigan Medical School, Ann Arbor, MI, USA

Selected Talks

4:30 – 4:45 PM
(CIS2-3) p53 Regulates TLR3 Expression and Function in Human Epithelial Cells
Manabu Taura, Graduate School of Pharmaceutical Sciences, Kumamoto University, Kumamoto, JP

4:45 – 5:00 PM
(CIS2-4) The Anti-Inflammatory Cytokine IL-10 Inhibits miR-155 in Response to Toll-like Receptor Signaling
Claire E. McCoy, Trinity College Dublin, Dublin, IE

5:00 – 5:15 PM
(CIS2-5) Activation of NK Cells in vivo Following Leishmania Infection Requires Myeloid Dendritic Cells, TLR9 and a Unique Set of Cytokines
Ulrike Schleicher, University Hospital Erlangen, Erlangen, DE

5:15 – 5:30 PM
(CIS2-6) A Medium-Throughout, Microplate-Based ex vivo Model for Measuring Intramacrophage Growth of Mycobacterium Tuberculosis
Daniel Eklund (Award Recipient), Linköping University, Linköping, SE

5:30 – 5:45 PM
(CIS2-7) NLRP3 Inflammasome in Malaria: Role of Hemozoin-Induced Signaling on Inflammasome Activation
Marina Tiemi Shio, McGill University, Montreal, CA

5:45 – 6:00 PM
(CIS2-8) Subversion of Human CD4+CD25+ Regulatory T Cells to IL-17-Producing T Cells by an Inflammatory Milieu
Behdad Afzali, Kings College London, London, UK

3:30PM – 6:00 PM

Concurrent Immunopathogenesis Symposia 3: Pathogen Manipulation of Cytokine Responses - Pavilion 5 A&B
Chairs: Eleanor Fish and Christopher Karp

3:30 – 4:00 PM
(CIS3-1) The IL-6/12 Family in Resistance to Infection
Chris Hunter, University of Pennsylvania, Philadelphia, PA, USA

4:00 – 4:30 PM
(CIS3-2) Virus Manipulation of the Interferon Response
Steve Goodbourn, St. Georges's University, London, UK

Selected Talks

4:30 – 4:45 PM
(CIS3-3) Inhibition of Type I Interferon Transcription by IRF3/7 Sumoylation
Keiko Ozato, NICHD, NIH, Bethesda, MD, USA

4:45 – 5:00 PM
(CIS3-4) Interferon and Influenza Viruses: The Yin and Yang of Survival
Eleanor N. Fish, Toronto General Research Institute, Toronto, CA

5:00 – 5:15 PM
(CIS3-5) Microbial Immune Evasion Through Exploitation of Macrophage Pattern-Recognition Receptors
George Hajishengallis, University of Louisville , Louisville, KY, USA

5:15 – 5:30 PM
(CIS3-6) A Novel Function of the Crohn's Disease–Associated NOD2Mutant 1007fs in the Regulation of Human IL10 Gene Transcription
Xiaojing Ma, Weill Medical College of Cornell University, New York, NY, USA

5:30 – 5:45 PM
(CIS3-7) V Protein-Mediated Block of MX Transcription is Essential for Morbillivirus Virulence
Nicholas Svitek, INRS-Institut Armand-Frappier, Quebec, CA

5:45 – 6:00 PM
(CIS3-8) C1q Enhances Phagocytosis of Mycobacterium Avium through a Pertussis Toxin Sensitive Pathway
Suzanne S. Bohlson, Indiana University School of Medicine, South Bend, IN, USA

6:00 PM – 8:30 PM

Focus Workshop:
A Spotlight on: Interferon-lambda (IL-29) – Pavilion 5 A&B
Chairs: Raymond Donnelly and Eleanor Ramos
Sponsored by Bristol-Myers Squibb

6:00 – 6:25 PM
Introduction and Brief Overview of IFN-lambda/IL-29
Raymond Donnelly, Center for Drug Evaluation & Research, FDA, Bethesda, MD, USA

6:25 – 6:50 PM
IFN-lambda is Functionally an Interferon but Structurally More Related to the IL-10 Family
Rune Hartmann, University of Aarhus, Aarhus, Denmark

6:50 – 7:15 PM
What Have We Learned From the IL-28R Knock-Out Mouse?
Markus Mordstein and Peter Staeheli (Award Recipient), University of Freiburg, Freiburg, Germany

7:15 – 7:40 PM
Expression and Function of Type III IFNs During Viral Infection in vitro and in vivo
Søren Paludan, University of Aarhus, Aarhus, DK

7:40 – 8:05 PM
Mechanisms of IFN-lambda Antiviral Activity Against HBV and HCV
Michael Robek, Yale University School of Medicine, New Haven, CT, USA

8:05 – 8:30 PM
Preclinical and Clinical Development of Pegylated-IFN-lambda
Eleanor Ramos, Zymogenetics, Inc. Seattle, WA, USA

7:30 PM – 10:00 PM

Student/Post-Doc Mixer – Trindade
Sponsored by The Society for Leukocyte Biology (SLB).
Pick up complimenatry tickets for this event at the JLB Booth (4.18).

Tuesday, October 20th

8:00 AM – 5:00 PM

Registration – Ground Floor Foyer

8:30 AM – 6:30 PM

Exhibits – Pavilion 4

8:00 AM – 9:30 AM

Joint Plenary Session 4:
New T-helper Subsets – Auditorium 1
Chairs: Warren Leonard and Elizabeth J. Kovacs
Sponsored by Biogen Idec, Inc.

8:00 – 8:30 AM
(PL4-1) Role of Microbiota and Transcription Factors in Control of Th17 Cell Differentiation
Dan Littman, NYU School of Medicine, New York, NY, USA

8:30 – 9:00 AM
Control of Immune-Mediated Inflammation by Regulatory T Cells
Sasha Rudensky, University of Washington School of Medicine Immunology, WA, USA

9:00 – 9:30 AM
IL-10 Producing Th I Cells
Anne O'Garra, The National Institute for Medical Research, London, UK

9:30 AM – 10:00 AM

Coffee Break - Pavilion 4
Sponsored by Biomonitor

10:00 AM – 12:00 PM

Concurrent Basic Science Symposia 4:
Signaling Session I - Auditorium 1
Chairs: Martha Cathcart and John Schrader

10:00 – 10:30 AM
(CBSS4-1) Structural and Functional Analyses of Protein Complexes in Immune and Inflammatory Pathways
Hao Wu, Weill Cornell Medical College, New York, NY, USA

10:30 – 11:00 AM
(CBSS4-2) Transcriptional Control of Dendritic Cell Development and Homeostasis
Boris Reizis, Columbia University Medical Center, New York, NY, USA

Selected Talks

11:00 – 11:15 AM
(CBSS4-3) Essential Regulatory Role of NOX2 in RIG-I-Mediated Innate Immune Responses
Nathalie Grandvaux (Award Recipient), University of Montreal, Montreal, CA

11:15 – 11:30 AM
(CBSS4-4) Regulation of NFκB by NSD1/FBXL11-Dependent Reversible Lysine Methylation of p65
Tao Lu (Award Recipient), Cleveland Clinic Foundation, Cleveland, OH, USA

11:30 – 11:45 AM
(CBSS4-5) TRAF3 Recruitment to Sec16A and p115 Reveals a New Role for the ER-TO-GOLGI Transport Compartments in Innate Immunity
Marc Servant, Université de Montréal, CA

11:45 – 12:00 PM
(CBSS4-6) Tank is a Negative Regulator of TLR Signaling and Critical for Preventing Autoimmune Nephritis
Osamu Takeuchi, IFReC, Osaka University, Suita, JP

10:00 AM – 12:05 PM

Concurrent Special Symposia 1:
IFN in the Clinic: Immunotherapy of Multiple Sclerosis - Auditorium 2
Chairs: Michael Tovey and John Hiscott
This special session is made possible through the generous support of Bayer Schering Pharma AG, Biogen Idec, Biomonitor, and Celgene

10:00 – 10:25 AM
Role of Interferons in the Pathogenesis of Autoimmune Disease
Anthony Coyle, MedImmune, Gaithersburg, MD, USA

10:25 – 10:50 AM
(CSS1-2) Pharmacokinetic, Pharmacodynamic, and Safety Profiles of Pegylated Interferon Beta-1A in Healthy Volunteers: Results from Two Phase 1 Clinical Studies
Darren Baker, Biogen Idec, Cambridge, MA, USA

10:50 – 11:15 AM
(CSS1-3) Clinical Significance of Antibodies to Interferon beta Therapy in Patients with Relapsing-Remitting Multiple Sclerosis
Klaus Bendtzen, Rigshospitalet University Hospital, Copenhagen, DK

11:15 – 11:40 AM
(CSS1-4) Translational Approaches and Patient Profiling to Identify Relevant Biomarkers of Interferon beta Activity in MS
Ed Croze, Bayer HealthCare Pharmaceuticals, Inc., Richmond, CA, USA

11:40 – 12:05 PM
(CSS1-5) Natalizumab Therapy of MS
Michael Hutchinson, St Vincent's University Hospital, Dublin, IE

10:00 AM – 12:00 PM

Concurrent Special Symposia 2:
Recent Advances - Pavilion 5 A&B
Chair: Howard Young

Selected Talks

10:00 – 10:15 AM
(CSS2-1) Thymic CD70-CD27 Signals Promote the Differentiation of ab and gd T Cell Subsets
Julie C. Ribot (Award Recipient), Instituto de Medicina Molecular, Lisbon, PT

10:15 – 10:30 AM
(CSS2-2) Characterization of a New Population of CD4+ Innate Spleen Cells that Produce IL-22 During Inflammatory Processes
Laure Dumoutier, Universite Catholique de Louvain , Brussels, BE

10:30 – 10:45 AM
(CSS2-3) Caspase-8 Regulates Cellular Response to Pattern Recognition Receptors and Prevents Spontaneous Triggering of Chronic Inflammation by their Endogenous Activators
David Wallach, Weizmann Institute of Science, Rehovot , IL

10:45 – 11:00 AM
(CSS2-4) TGF-b1 Signals TIAF1 Self-Association, Amyloid Superinduction and Apoptosis
Nan-Shan Chang, National Cheng Kung University Medical College, Tainan, TW

11:00 – 11:15 AM
(CSS2-5) Genetic Variants and Disease-Associated Factors Contribute to IRF-5 Expression in Primary Blood Cells of SLE Patients
Betsy J. Barnes, University of Medicine and Dentistry of New Jersey, Newark, NJ, USA

11:15 – 11:30 AM
(CSS2-6) Acute T Cell Leukemia: An in vivo Struggle Between HTLV-1-Production and Type 1 Interferon Production
Francis Ruscetti, NCI Laboratory of Experimental Immunology, Frederick, MD, USA

11:30 – 11:45 AM
(CSS2-7) T Cell Receptor Agonist and Tumor Biomarkers For Gamma-Delta T-Cell-Based Immunotherapy of Lymphomas and Leukemias
Bruno Silva-Santos (Award Recipient), Instituto de Medicina Molecular, Lisbon, PT

11:45 – 12:00 PM
(CSS2-8) Intracellular Inhibitors of Cysteine Cathepsins in Activated Macrophages
Natasa Kopitar-Jerala, Institute Jozef Stefan, Ljubljana, SI

10:00 AM – 12:15 PM

Concurrent Basic Science Symposia 5:
Immunoregulation II – Pavilion 5 C
Chairs: David Artis and Anne O'Garra
Sponsored by PBL Interferon Source

Selected Talks

10:00 – 10:15 AM
(CBSS5-1) Immediate Mediators of the Inflammatory Response are Poised for Rapid Gene Activation Through RNA Polymerase Stalling
Inez Rogatsky, HSS and Weill Cornell, New York, NY, USA

10:15 – 10:30 AM
(CBSS5-2) LOX-1 as Natural IFN-ƒÑƒn Mediated Signal for Apoptotic Cell Uptake and Antigen Presentation in Dendritic Cells
Filippo Belardelli, Istituto Superiore di Sanita , Rome, IT

10:30 – 10:45 AM
(CBSS5-3) Novel Gene Expression Patterns in IFN-GAMMA 3'Untranslated Region Au-Rich Element-Deleted Mice
Deborah L. Hodge (Award Recipient), NCI/CCR, Frederick, MD, USA

10:45 – 11:00 AM
(CBSS5-4) The IL-27 P28 Subunit Binds CLF to form a Cytokine Regulating NK and T Cell Activities Requiring IL-6R for Signaling
Sandrine Crabé, Université de Montréal, Montreal, CA

11:00 – 11:15 AM
(CBSS5-5) The Type I Interferon (IFN) a Mediates a More Severe Neurological Disease in the Absence of the Canonical Signaling Molecule Interferon Regulatory Factor (IRF) 9
Markus J. Hofer, (Award Recipient) University of Marburg, Marburg, DE

11:15 – 11:30 AM
(CBSS5-6) Unc93 Homolog B1 Regulates the Balance of Toll-Like Receptor 7 and Toll-Like Receptor 9 Responses Reciprocally in Dendritic Cells
Ryutaro Fukui, The Institute of Medical Science, The University of Tokyo, Tokyo, JP

11:30 – 11:45 AM
(CBSS5-7) Identification of a Novel Antigen Presenting Cell Population Modulating Anti-Influenza Type-2 Immunity
Jae-Kwang Yoo, University of Toronto, Toronto, CA

11:45 – 12:00 PM
(CBSS5-8) Antiviral Effects of Cytokines
Thomas Lavoie, PBL Interferonsource, Piscataway, NJ, USA

12:00 – 12:15 PM
(PP2-173) Act1: A Novel U-box E3 Ubiquitin Ligase for IL-17R-Mediated Signaling
Caini Liu (Award Recipient), Cleveland Clinic, Cleveland, OH, USA

12:15 PM – 1:30 PM

Lunch On Your Own

1:30 PM – 3:00 PM

Concurrent Basic Science Symposia 6:
Neutrophil Biology – Auditorium 1
Chairs: Marco Cassatella and William Nauseef

1:30 – 2:00 PM
(CBSS6-1) Neutrophils as Active Participants in Cross-Talks with Other Cells of the Immune System
Marco A. Cassatella, University of Verona, Verona, IT

Selected Talks

2:00 – 2:15 PM
(CBSS6-2) Inter-Kingdom Signalling: A Quorum-Sensing Molecule of Pseudomonas Aeruginosa Activates Human Polymorphonuclear Neutrophils (PMN)
Gertrud Maria Hänsch, University of Heidelberg, Heidelberg, DE

2:15 – 2:30 PM
(CBSS6-3) Functional Cooperation between Fc Gamma RIIa AND Fc Gamma RIIIb on Human Neutrophils
Louis Marois, Laval University, Québec, CA

2:30 – 2:45 PM
(CBSS6-4) A Critical Role of Nitric Oxide in the Resolution of Inflammation
Yoshiro Kobayashi, Toho University, Funabashi, JP

2:45 – 3:00 PM
(CBSS6-5) Leishmania Promastigotes Induce the Formation of Neutrophil Extracellular Traps
Albert Descoteaux, Institut Armand-Frappier, Laval, CA

1:30 PM – 3:00 PM

Concurrent Basic Science Symposia 7:
IFN-Stimulated Genes – Auditorium 2
Chairs: Moira K. B. Whyte and Ganes Sen

1:30 – 2:00 PM
(CBSS7-1) Signaling Pathways Controlling mRNA Translation of Interferon Regulated Genes
Leon C. Platanias, Northwestern University Medical School, Chicago, IL, USA

2:00 – 2:30 PM
UBP43 and ISG15 in the Innate Immune Response
Dong Zhang, The Scripps Research Institute, La Jolla, CA, USA

Selected Talks

2:30 – 2:45 PM
(CBSS7-3) A Novel Small RNA Regulatory Mechanism Employed by Interferons for Regulating Growth
Dhan V. Kalvakolanu, University of Maryland School of Medicine, Baltimore, MD, USA

2:45 – 3:00 PM
(CBSS7-4) Cooperation of Stat and NFkB in the Assembly of a Transcription Competent Initiation Complex
Thomas Decker, University of Vienna, Vienna, AT

1:30 PM – 3:00 PM

Concurrent Clinical Symposia:
Biological Therapeutics – Pavilion 5 A&B
Chairs: Kathy Zoon and John Sims

1:30 – 2:00 PM
(CCS1-1) Interferons and Interferon Antagonists in Disease and Clinical Applications
Sergei V. Kotenko, New Jersey Medical School, Newark, NJ, USA

2:00 – 2:30 PM
(CCS1-2) Where are we with Anti-Chemokine Therapies?
Amanda Proudfoot, Merck Serono, Geneva, CH

2:30 – 3:00 PM
Developing Safe and Effective Immunotherapy: Lessons from Anti-CD28 Trials (TGN1412)
Sir Gordon Duff, University of Sheffield Molecular Medicine, London, UK

1:30 PM – 3:00 PM

Concurrent Immunopathogenesis Symposia 4:
Inflammation & Pathogenesis - Pavilion 5 C
Chairs: Sanna M. Goyert and Amanda Proudfoot

1:30 – 2:00 PM
(CIS4-1) Immune Modulation by Virus-Encoded Cytokine Binding Proteins Antonio Alcami, Centro de Biologia Molecular
Severo Ochoa, CSIC-UAM, Madrid, Spain

2:00 – 2:30 PM
(CIS4-2) Molecular Pathogenesis of West Nile Virus Encephalitis
Phil Murphy, NIH-LMI/NIAID, Bethesda, MD, USA

2:30 – 3:00 PM
(CIS4-3) Dissecting "Alternative Macrophage Activation": The Role of L-Arginine Metabolism in Chronic Inflammation and Fibrosis
Tom Wynn, NIAID-NIH, Bethesda, MD, USA

3:00 PM – 5:00 PM

Poster Session A – Pavilion 4&5

7:30 PM – 10:30 PM

Conference Banquet
National Agronomy Pavilion – Bus service provided from hotels

Wednesday, October 21st

8:00 AM – 2:30 PM

Registration – Ground Floor Foyer

8:30 AM – 2:30 PM

Exhibits – Pavilion 4

8:00 AM – 9:00 AM

Joint Plenary Session 5:
The Macrophages in Health and Disease – Auditorium 1
Chair: Alberto Matovani
Sponsored by BD Biosciences

8:00 – 8:30 AM
(PL5-1) The Macrophage's Dual Role in Health and Disease
Siamon Gordon, NCI, SAIC-Frederick, MD, USA

8:30 – 9:00 AM
(PL5-2) Learning Tolerance from Cancer: Lessons from Myeloid-Derived Suppressor Cells
Vincenzo Bronte, Universita Degli Studi Di Padova, Padova, IT

9:00 AM – 9:30 AM

Coffee Break – Pavilion 4

9:30 AM – 11:30 AM

Concurrent Immunopathogenesis Symposia 5: The Role of Tissue-Specific Macrophages in Chronic Disease Processes – Auditorium 1
Chairs: Joe Oppenheim and Jill Suttles

9:30 – 10:00 AM
(CIS5-1) Mechanisms and Consequences of Macrophage Apoptosis and Efferocytosis in Atherosclerosis
Ira Tabas, Columbia University, New York, NY, USA

10:00 – 10:30 AM
(CIS5-2) Regulation of Macrophage Activation and Function by PPARs
Ajay Chawla, Stanford University, Stanford, CA, USA

Selected Talks

10:30 – 10:45 AM
(CIS5-3) Macrophage Effector Function in Anti-Filarial Nematode Immunity is Independent of Arginase 1, Relma and YM-1
Stephen Jenkins, University of Edinburgh, Edinburgh, UK

10:45 – 11:00 AM
(CIS5-4) Stabilin-1 –Multifunctional Receptor Linking Endocytosis and Secretion in Macrophages
Alexei Gratchev, University of Heidelberg, Mannheim, DE

11:00 – 11:15 AM
(CIS5-5) Expression of the Inhibitory CD200 Receptor is Associated with Alternative Macrophage Activation
Jörg Hamann, Academic Medical Center, University of Amsterdam, Amsterdam, NL

11:15 – 11:30 AM
(CIS5-6) Control of RSV-Induced Lung Injury by Alternatively Activated macrophages is IL-4Ralpha-, TLR4-, and IFN-beta-dependent
Jorge C. Blanco, Virion Systems Inc., Rockville, MD, USA

9:30 AM –11:30 AM

Concurrent Basic Science Symposia 8: Signaling Session II – Auditorium 2
Chairs: Sara Gaffen and Rui Victorino

9:30 – 10:00 AM
(CBSS8-1) NLR Genes and Adaptive and Innate Immunity
Jenny Ting, UNC CCBC, Chapel Hill, NC, USA

10:00 – 10:30 AM
(CBSS8-2) Signaling Through SUMO Ligases to Regulate Immune Responses
Ke Shuai, Biological Chemistry-UCLA, Los Angeles, CA, USA

Selected Talks

10:30 – 10:45 AM
(CBSS8-3) Deregulated Activation of Cytokine Signaling by Interleukin-6 (IL-6) in the Pathogenesis of Emphysema
Saleela M Ruwanpura (Award Recipient), Monash Univ., Clayton, AU

10:45 – 11:00 AM
(CBSS8-4) IL-22, a TH17 Cytokine, Induces a Systemic Acute Phase Response
Lynette A. Fouser, Wyeth Research, Cambridge, MA, USA

11:00 – 11:15 AM
(CBSS8-5) Interleukin-6 Induces Translocation of the Adapter Protein GAB1 by MAPK-Dependent Phosphorylation of GAB1 on SERINE 552
Fred Schaper, RWTH Aachen University, Aachen, DE

11:15 – 11:30 AM
(CBSS8-6) Role of Small RNAS Generated by RNASE L in Signaling Innate Immunity against Hepatitis C Virus
Robert H. Silverman, Cleveland Clinic, Cleveland, OH, USA

9:30 AM –11:30 AM

Concurrent Immunopathogenesis Symposia 6: Immunopathogensis III – Pavilion 5 A&B
Chair: Patricia Fitzgerald-Bocarsly

9:30 – 10:00 AM
(CIS6-1) HIV-1 Escape from Innate Immune Response
Domenico Mavilio, Istituto Clinico Humanitas, Milano, IT

10:00 – 10:30 AM
(CIS6-2) Tumor Suppressors in Antiviral Immunity
Cesar Munoz-Fontela, Mount Sinai, New York, NY, USA

10:30 – 11:00 AM
Transcriptional Regulation of CD8 T Cell Differentiation During Chronic Viral Infection
John Wherry, The Wistar Institute, Philadelphia, PA, USA

Selected Talks

11:00 – 11:15 AM
(CIS6-4) Alpha-1-Antitrypsin Inhibits Influenza in vitro, Reduces Influenza Disease in vivo, and Genetic Deficiency is a Risk Factor for Human Influenza Infection
K. Scott Beard, University of Colorado Denver, Denver, CO, USA

11:15 – 11:30 AM
(CIS6-5) Lethal Viral Infection Results from STAT1 but not STAT2 or IRF9 Deficiency in Mice and is Mediated by CD4+ T-Cells
Markus J. Hofer, University of Marburg, Marburg, DE

11:30 AM – 12:30 PM

Lunch On Your Own - Boxed Lunches Available for Purchase at the Bar located near Pavilion 4

ISICR General Society Meeting – Auditorium 1
ICS General Society Meeting – Auditorium 2
SLB General Society Meeting – Pavilion 5 A&B

12:30 PM – 2:30 PM

Poster Session B – Pavilion 4&5

2:30 PM – 4:30 PM

Concurrent Basic Science Symposia 9:
Allergy and Mast Cells – Auditorium 1
Chairs: Matthew Fenton and Keiko Ozato

2:30 – 3:00 PM
(CBSS9-1) Mechanisms of Human Eosinophil Cytokine Secretion
Peter Weller, Harvard University, Boston, MA, USA

3:00 – 3:30 PM
(CBSS9-2) New Facets in Mast Cell Activation
Silvia Bulfone-Paus, Research Center Borstel, Borstel, DE

Selected Talks

3:30 – 3:45 PM
(CBSS9-3) S100A8 – An Oxidant Scavenger and Immune Modulator in Allergic Inflammation
Carolyn L. Geczy, University NSW, Sydney, AU

3:45 – 4:00 PM
(CBSS9-4) T Cell-Specific Act1 Deficiency Leads to Attenuated Cellular and Humoral Allergic Responses
Swaidani Shadi (Award Recipient), Cleveland Clinic, Cleveland, OH, USA

4:00 – 4:15 PM
(CBSS9-5) Mast Cell Degranulation Requires Activation of PI3Kg by PKCb
Romy Walser, University of Basel, Basel, CH

4:15 – 4:30 PM
(CBSS9-6) The Antimicrobial Peptides Human beta-Defensins Mediate Secretion of Pruritogenic Factors in Human Mast Cells
Francois Niyonsaba, Juntendo University School of Medicine, Tokyo, JP

2:30 PM – 4:30 PM

Concurrent Immunopathogenesis Symposia 7:
Sensing of Fungal & Parasitic Infection and Host Response - Auditorium 2
Chairs: Michael Tovey and Christian Bogdan

2:30 – 3:00 PM
(CIS7-1) Sensing Danger Signals and Pathogen-Associated Molecular Patterns Defines Binary Signaling Pathways in Mammalian Response to Fungi
Luigina Romani, University of Perugia, Perugia, IT

3:00 – 3:30 PM
Role of Beta-Glucan in Anti-Fungal Immunity
Gordon D. Brown, University of Aberdeen, Aberdeen, UK

Selected Talks
3:30 – 3:45 PM
(CIS7-3) Vitamin A Derived Retinoic Acid Signaling Mediates Intestinal Immune Homeostasis and Immunity
Jason A. Hall, National Institutes of Health/ U Penn Partnership, Bethesda, MD, USA

3:45 – 4:00 PM
(CIS7-4) The Induction of IL-10 by Fungi in Dendritic Cells Depends on CREB Activation by the Coactivators CBP and TORC2 and Autocrine PGE2
Mariano Sánchez Crespo, CSIC, Valladolid, ES

4:00 – 4:15 PM
(CIS7-5) Th17/IL-17 Receptor Signaling and not Th1 Cells are Essential for Mucosal Host Defense Against Oral Candidiasis
Sarah L. Gaffen, University of Pittsburgh, Pittsburgh, PA, USA

4:15 – 4:30 PM
(CIS7-6) Origin, Phenotype and Function of Monocyte/Macrophage Subsets in Distinct Mammary Tumor Microenvironments
Shinobu Saijo, The University of Tokyo, The Institute of Medical Science, Tokyo, JP

2:30 PM – 4:30 PM

Concurrent Immunopathogenesis Symposia 8:
Chronic Inflammatory Disease – Pavilion 5 A&B
Chairs: Sir Gordon Duff and Otto Haller

2:30 – 3:00 PM
Cytokines and Innate Immunity in Intestinal Inflammation
Fabio Cominelli, University of Virginia, Richmond, VA, USA

3:00 – 3:30 PM
(CIS8-2) Linking Inflammation to Cancer – A Novel Role for Stat3
Matthias Ernst, Ludwig Institute for Cancer Research, Melbourne, AU
3:30 – 4:00 PM
Regulation of Immunity and Inflammation in the Gut
David Artis, University of Pennsylvania, Philadelphia, PA, USA

4:00 – 4:30 PM
Regulation of Macrophage Signaling and Function During Chronic Inflammation
Lionel Ivashkiv, Cornell University, New York, NY, USA

4:45 PM – 6:15 PM

Joint Plenary Session 6:
Closing Keynote Lectures – Auditorium 1
Chairs: Luis Montaner and David Wallach

4:45 – 5:15 PM
Violation of the Sanctity of the Cytosolic Compartment Provokes the Wrath of the Inflammasome
Vishva Dixit, Genentech, South San Francisco, CA, USA

5:15 – 5:45 PM
(PL6-2) How Specificity for Self-Peptides Shapes the Development and Function of Regulatory T Cells
Andrew Caton, The Wistar Institute, Philadelphia, PA, USA

5:45 – 6:15 PM
T Lymphocyte Trafficking in Immunity and Autoimmunity
Federica Sallusto, Institute for Research in Biomedicine, Switzerland

Posters

Poster Presenters are asked to be available at their poster display to discuss their work with visitors during designated Poster Session times as follows (See A/B designation next to topic titles):
Poster Session A: Tuesday, October 20th from 3:00 PM – 5:00 PM
Poster Session B: Wednesday, October 21st from 12:30 PM – 2:30 PM

A Allergy and Mast Cells

CBSS9-3 S100A8 – AN OXIDANT SCAVENGER AND IMMUNE MODULATOR IN ALLERGIC INFLAMMATION. Carolyn L. Geczy, Lincoln Gomes, *Mark Raftery, Jing Zhao, Ikuko Endoh, Yasumi Endoh and Paul Thomas.

CBSS9-4* T CELL-SPECIFIC ACT1 DEFICIENCY LEADS TO ATTENUATED CELLULAR AND HUMORAL ALLERGIC RESPONSES. Shadi Swaidani, Katarzyna Bulek, Zizhen Kang, Caini Liu, Mark Aronica, Xiaoxia Li.

CBSS9-5 MAST CELL DEGRANULATION REQUIRES ACTIVATION OF PI3KG BY PKCB. Romy Walser, Peter Küenzi, Daniel Hess, Michael Leitges, Emilio Hirsch, Muriel Laffargue, Matthias P. Wymann.

CBSS9-6 THE ANTIMICROBIAL PEPTIDES HUMAN BETA-DEFENSINS MEDIATE SECRETION OF PRURITOGENIC FACTORS IN HUMAN MAST CELLS. Francois Niyonsaba, Hiroko Ushio, Isao Nagaoka, Hideoki Ogawa and Ko Okumura.

PP2-001 SECRETION OF IFN GAMMA AND INFLAMMATORY CHEMOKINES BY HUMAN MACROPHAGES STIMULATED WITH IL-12 AND IL-18. Margarita Bofill, Laila Darwich , Gema Comma, Esther Jimenez, Robert ME Parkhouse.

PP2-003 THE EFECTS OF CHCL3 SOLVENT SUB-FRACTIONS FROM CARPINUS TSCHONOSKII ON THE INFLAMMATORY CHEMOKINES, MDC AND TARC, IN THE HACAT KERATINOCYTES. Gyeoung-jin Kang.

LB-01 ROLE OF AIRWAY EPITHELIUM IN ENGULFING APOPTOTIC EOSINOPHILS. Faris Q. Alenzi, Ph.D.

B Anti-Tumor Immunity

PP1-001 THE EXPRESSION OF TOLL-LIKE RECEPTOR PATHWAY MOLECULES IN PERIPHERAL BLOOD MONONUCLEAR CELLS AND ITS USE AS A POTENTIAL BIOMARKER FOR TUMOR BEHAVIOR IN LARYNGEAL CARCINOMA. Katarzyna Starska, Ewa Forma, Magdalena Brys, Ewa Glowacka, Olga Stasikowska, Iwona Lewy-Trenda, Wanda M. Krajewska, Marek Lukomski.

PP1-002 INTERFERON-ALPHA EXHIBITS DUAL EFFECTS IN A RAT LIVER TUMOR MODEL WITH LIVER CIRRHOSIS. Hao-Tien Wang, Yung-Chi Huang, Hsin-I Lee, and Lih-Hwa Hwang.

PP1-003 THE BLOCKADE OF TIM-3 PATHWAY ENHANCES IMMUNE RESPONSE AGAINST TUMOR IN MOUSE. Mi-Jin Lee, Min Y Woo, Sun Park.

PP1-004 THE NKG2D LIGAND ULBP1 DETERMINES LYMPHOMA AND LEUKEMIA CELL SUSCEPTIBILITY TO HUMAN GD T CELL CYTOTOXICITY. T. Lança, A. Q. Gomes, D. V. Correia, L. F. Moita and B. Silva-Santos.

PP1-005* PROSTAGLANDIN E2-DEPENDENT MODULATION OF MACROPHAGES' RESPONSES BY COLON CANCER CELLS. Brandy M. Conner, Julia Ahn, Edith Chang, and Alex Shnyra.

PP1-006 EXHIBITION OF ENHANCED IMMUNO-REGULATORY POTENTIAL IN HUMAN DENDRITIC CELLS TREATED WITH GAMMA-IRRADIATED COLON CANCER CELLS. Sun Kyung Kim, Cheol-Heui Yun, Seung Hyun Han.

PP1-007 DEVELOPMENT OF RECOMBINANT VESICULAR STOMATITIS VIRUS FOR USE AS AN ONCOLYTIC VECTOR IN CANCER THERAPY. Joshua Heiber, Jinhee Hyun, Masatsugu Obuchi, Glen N. Barber.

LB-02 IL-12 INHIBITS DIRECTLY THE GROWTH OF HUMAN PRIMARY ACUTE MYELOID LEUKEMIA CELLS. Elisa Ferretti, Claudia Cocco, Emma Di Carlo, Daniela Montagna, Emanuela Ognio, Franco Locatelli, Irma Airoldi and Vito Pistoia.

A Biological Therapeutics

PP2-005 GREEN TEA REGULATES CELL SIGNALLING BY INDUCING SOCS1 GENE EXPRESSION VIA ITS CONSTITUENT POLYPHENOL EGCG. Barry Ripley, Minoru Fujimoto, Satoshi Serada, Tomoharu Ohkawara, Teppei Nishikawa, Fumitaka Terabe, Yuko Matsukawa, Anastasis Stephanou, Richard Knight, David Isenberg, David Latchman, Tadamitsu Kishimoto and Tetsuji Naka.

PP2-006 DETECTION AND BIOLOGICAL ACTIVITY OF THERAPEUTICALLY INDUCED ANTIBODIES TO PEG-IFN-a-2a IN HEPATITIS C VIRUS INFECTED PATIENTS. Zahra Alvandi, Lisette Provacia, Byron E.E. Martina, Albert D.M.E Osterhaus and Bart L. Haagmans.

PP2-007 ANTI-CANCER DRUGS POTENTIATE TLR3-DEPENDENT APOPTOSIS BY REGULATING TLR3 EXPRESSION VIA DUAL P53-DEPENDENT AND -INDEPENDENT PATHWAYS. Fukuda R., Taura M., Eguma A., Suico M.A., Koga T., Shuto T., Kai H.

PP2-008 ANTI-INTERLEUKIN-21 MONOCLONAL ANTIBODY REDUCES DISEASE SEVERITY AND INFLAMMATORY CYTOKINES IN A MURINE MODEL OF COLITIS AND PSORIASIS-LIKE SKIN INFLAMMATION. Katherine E. Lewis, Kristen Bontadelli, Mark Maurer, Felecia Wagener, Kimberly Waggie, Cecile M. Krejsa, and Stacey R. Dillon.

PP2-009 THE ORAL HISTONE DEACETYLASE INHIBITOR ITF2357 REDUCES CYTOKINE PRODUCTION, CYTOKINE ACTIVITIES AND PROTECTS ISLET β-CELLS IN VIVO AND IN VITRO. Eli C. Lewis, Lykke Blaabjer, Joachim Størling, Sif G. Ronn, Paolo Mascagni, Charles A. Dinarello, Thomas Mandrup-Poulsen.

PP2-010* CHARACTERIZATION OF NEUTRALIZING ANTIBODIES TO INTERFERON α USING A NOVEL REPORTER-GENE ASSAY: Neutralization Titer is Dependent upon the Specific Activity of the Interferon a Subtype. Brigitte Blanchard, Christophe Lallemand, Jean-François Meritet, Pierre Lebon, and Michael G. Tovey.

PP2-011 HUMAN RECOMBINANT INTERLEUKIN-1 RECEPTOR ANTAGONIST LOCAL APPLICATION FOR THE THERAPY OF INFLAMMATORY AND ALLERGIC AIRWAY PATHOLOGY. Andrey Simbirtsev, Alexander Petrov, Natalia Pigareva, Ludmila Solovieva, Alexander Ischenko, Sergey Ketlinsky.

*Travel Award Recipient

PP2-012 ACTIVATED COLLAGEN ACCELERATES WOUND REPAIR AND MODULATES CYTOKINE PRODUCTION IN WHOLE BLOOD AND PBMC CULTURES. Gregory B. Pott, K. Scott Beard, Matthew Regulski, and Leland Shapiro.

PP2-013 SAFETY AND EFFICACY OF ORAL ITF2357 IN PATIENTS WITH ACTIVE SYSTEMIC ONSET JUVENILE IDIOPATHIC ARTHRITIS. Jelena Vojinovic, Charles A. Dinarello, Antonio Furlan, Nemanja Damjanov, Carmine D'Urzo, Tiziano Oldoni.

PP2-014* VISUALIZING ENGINEERED INTERFERON-PRODUCING CELLS WITH MULTICISTRONIC VECTORS AND FLUORESCENT PROTEINS. Lara S. Izotova, Christopher D. Krause, Gwangwen Ren, Zeng-Rong Yuan, Yufang Shi, and Sidney Pestka.

PP2-015 ANTI-CYTOKINE VACCINES USING LIVING CELLS PRESENTING ENDOGENOUS CYTOKINES IN FUSION WITH A HUMAN TRANSMEMBRANE PROTEIN. Muriel M. Lemaire, Aurélie Vanhaudenarde, Yannick Nizet, Laure Dumoutier, and Jean-Christophe Renauld.

LB-03 ADALIMUMAB IN SEVERE ACUTE SCIATICA. A RANDOMIZED,DOUBLE BLIND, PLACEBO CONTROLLED CLINICAL TRIAL. Genevay S, Viatte S, Finckh A, Zufferey P, Balagué F, Gabay C.

LB-04* APG2305 - A NEW ORALLY-ACTIVE SELECTIVE PEPTIDIC ANTAGONIST OF IL-23R. Christiane Quiniou, Isabelle Lahaie, Jean-Claude Honoré, Mark Kaufmann, Sylvain Chemtob

LB-05* TNR1-14 AND TNR1-23 - NEW POTENT PEPTIDIC ORALLY ACTIVE ANTAGONISTS OF TNFR1. Christiane Quiniou, Isabelle Lahaie, Jean-Claude Honoré, Mark Kaufmann, Shawn Barney, Sylvain Chemtob.

LB-42 TLR3 SIGNALING IN SYNOVIOCYTES IS NEGATIVELY REGULATED BY EXTRACELLULAR ATP THROUGH INHIBITION OF NF-κB Thusitha Gajanayake, Jakub Siednienko & Sinead Miggin

B Chronic Inflammatory Disease

PP2-016* QUANTITATIVE ANALYSES OF THE DYNAMIC SIGNALLING PROCESSES BETWEEN INFLAMED GUT EPITHELIUM AND THE IMMUNE SYSTEM. Michael Schmohl, Nicole Schneiderhan-Marra, Michael Blum, Gerburg Stein, Manfred Schmolz, Thomas O. Joos.

PP2-017 CIGARETTE SMOKE CONDENSATE EXTRACTS INDUCE PROINFLAMMATORY CYTOKINES FROM SYNOVIAL CELLS AND EXACERBATE COLLAGEN-INDUCED ARTHRITIS IN MICE. Kikuo Onozaki, Satomi Chujo, Shosuke Okamoto, Ryohei Sunahara, Yuka Itoh, Hidetoshi Hayashi, Takemasa Takii, Kazuichi Hayakawa.

PP2-018* NITRIC OXIDE IMMUNOMODULATION BY IL-17 AND IL-10 IN ALGERIAN PATIENTS WITH INFLAMMATORY BOWEL DISEASE. Hayet Rafa, Mourad Belkhelfa, Oussama Medjeber, Samia Bouaziz, Zineb Djerraba, Amina Lammali, Katia Abdelouaheb, Houria Saoula, Amira F Boutaleb, Aftiss, M'hamed Nakmouche, Chafia Touil-Boukoffa.

PP2-019 A COMPARATIVE INVESTIGATION OF CELLULAR RESPONSES INDUCED BY CYTOKINES IL-17 AND IL-32 IN HUMAN MONOCYTIC CELLS AND FIBROBLASTS-LIKE SYNOVIAL CELLS. Emily Turner-Brannen, Ka-Yee (Grace) Choi and Neeloffer Mookherjee.

PP2-020* PRO-ATHEROGENIC FACTORS MODULATE THE FATE OF MOUSE VASCULAR SMOOTH MUSCLE CELLS. Hye-Jin Park, Hye-Eun Byun, Suhkneung Pyo.

PP2-021 INTERFERON-A CORRELATES POSITIVELY WITH DISEASE SEVERITY IN DANISH PATIENTS WITH SLE. Dorthe Lundsgaard , Søren Jacobsen, Inger L. Pedersen, Lone Hummelshøj, Lars K. Poulsen, Pernille Keller, Jan Fleckner and Christian Ross.

PP2-023 IDENTIFICATION OF LEUCINE RICH ALPHA 2 GLYCOPROTEIN AS A NOVEL BIOMARKER ASSOCIATED WITH DISEASE ACTIVITY OF INFLAMMATORY AUTOIMMUNE DISORDERS. Satoshi Serada, Fumitaka Terabe, Minoru Fujimoto, Teppei Nishikawa, Tadamitsu Kishimoto, Tetsuji Naka.

PP2-025 GLUCOCORTICOIDS PARTICIPATE IN THE DEVELOPMENT OF THYMUS ATROPHY FOUND DURING INFECTION WITH A VIRULENT STRAIN OF MYCOBACTERIUM AVIUM. Margarida Borges, Manuela Flórido, Margarida Correia-Neves and Rui Appelberg

PP2-026 IFNg PROMOTES FIBROBLAST-LIKE SYNOVIOCYTES MOTILITY. T Karonitsch, K Dalwigk, R Byrne, B Niedereiter, E Cetin, A Wanivenhaus, C Scheinecker, JS Smolen, HP Kiener.

PP2-027* INFLAMMATORY VERSUS ANTI-INFLAMMATORY IL-6 DURING BEHCET DISEASE: DUAL EFFECT ON NITRIC OXIDE AND TGF-BETA. Houda Belguendouz, Djamel Messsaoudene , Mohammed L. Ahmedi , Karima Lahmar, Fifi Otmani, Djennat Hakem and Chafia Touil-Boukoffa.

LB-06 A NOVEL IMMUNE ORALLY ACTIVE MODULATOR ISOXAZOLINE INHIBITS INFLAMMATORY CYTOKINE PRODUCTION FROM TH-17 CELLS. Muthumani, K, Choo, AY, Fagone P, Chung CW, Kawalekar, OU, Sardesai NY, White J, Kim JJ, Weiner DB.

LB-07 CROSS-TALK BETWEEN IFNγ AND HEDGEHOG SIGNALING RESTORES ADIPOGENESIS IN 3T3-L1 CELLS. Jelena Todoric, Oswald Wagner, Harald Esterbauer.

LB-08 CYTOKINES MODULATION OF INTERFERON GAMMA-1B THERAPY IN IDIOPATHIC PULMONARY FIBROSIS (USUAL INTERSTITIAL PNEUMONIA). S. Marinari1, V. Dadorante, A.L. Di Mele, M.R. Flacco, R. Faricelli, S. Martinotti, M.Amitrano, A. Sanduzzi, F. De Benedetto and E. Toniato.

A Gene Activation

SLBAW2-B LONG RANGE GENOMIC CYTOKINE-RECEPTOR INTERACTION REGULATES GENE EXPRESSION. Chrysoula Deligianni and Charalampos G. Spilianakis.

CBSS3-4 INHIBITION OF DYNAMIN-DEPENDENT ENDOCYTOSIS INTERFERES WITH TYPE III IFN EXPRESSION IN BACTERIA-INFECTED HUMAN DENDRITIC CELLS. Taija E. Pietilä, Sinikka Latvala, Pamela Österlund, and Ilkka Julkunen.

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CBSS3-5 REGULATION OF C-MAF BY IL-2. Aradhana Rani, Audrey Kelly, Lemlem Tewolde Berhan, Stipo Jurevic, Jack Ragheb, Paul Lavender and Susan John.

CBSS3-6* SINGLE-STRANDED RNA VIRUSES INHIBIT P53 TRANSCRIPTIONAL ACTIVITY BY POST-TRANSLATIONAL ACTIVATION OF ΔNP63. Lallemand C., Blanchard B., May E. and Tovey M.G.

CBSS3-7 INTERFERON SIGNALING IS ACTIVATED IN RESPONSE TO DNA DAMAGE. Sabrina Brzostek-Racine, Chris Gordon, Sarah VanScoy, and Nancy C. Reich.

PP2-029 CYTOKINE GENE POLYMORPHISMS AS RISK FACTORS IN ACUTE REJECTION IN RENAL TRANSPLANTATION. Pourfathollah A.A.

B IFN in the Clinic: Immunotherapy of Multiple Sclerosis

PP1-106 DETECTION OF INTERFERON BETA IN MULTIPLE SCLEROSIS PATIENT SERA REVEALS DISPARITY BETWEEN ELISA AND FUNCTIONAL ASSAY QUANTIFICATION. Michael A. Skawinski, Sara Crisafulli, Yognandan Pandya, Steven Carbone, Matt Carroll, Sidney Pestka, William A. Clark, Thomas B. Lavoie, and Ronald G. Jubin.

PP1-107* IFN-β LIMITS TH17 CELL LINEAGE DEVELOPMENT: IMPLICATIONS IN MULTIPLE SCLEROSIS. Leesa Pennell, Carole Galligan, Ramtin Rahbar, Beata Majchrzak, Thomas Murooka, Ehtesham Baig, and Eleanor N. Fish.

PP1-114 INTERFERON (IFN)-STIMULATED GENES (ISGs) AS A RESISTANCE MECHANISM IN CANCER CELL DEATH. Venugopalan Cheriyath, Wioletta Luszczek, Barabara S. Jacobs, Ernest C. Borden.

PP1-117 SERUM LEVELS OF INTERLEUKIN-4 AND INTERFERON-GAMMA IN RELATION TO SEVERE LEFT VENTRICULAR DYSFUNCTION IN PATIENTS WITH ACUTE MYOCARDIAL INFACRTION UNDERGOING PERCUTANEOUS CORONARY INTERVENTION. Janusz Szkodziński, Bartosz Hudzik, Marcin Osuch, Wojciech Romanowski, Bozena Szygula-Jurkiewicz, Lech Polonski, Barbara Zubelewicz-Szkodzinska.

PP1-118* THE APOPTOTIC EFFECT INDUCED BY A CHIMERIC CYCLIC INTERFERON-ALPHA2b PEPTIDE IS MEDIATED BY STAT1, STAT3 AND p38MAPK SIGNALING. Viviana C. Blank, Clara Peña and Leonor P. Roguin.

PP1-123* BOTH IFN-γ ENDOCYTOSIS AND THE IFN-γ RESPONSIVE PROMOTER ACTIVATION ARE DEPENDENT ON CHOLESTEROL. Okki Cho, Seung-Ho Hong, Jungsik Kim, Sun Park.

PP1-124* ANTIPROLIFERATIVE ACTIVITY OF INTERFERON-β IN MUCOSAL AND CUTANEOUS HUMAN PAPILLOMA VIRUS – TRANSFORMED KERATINOCYTES. M.V. Chiantore, S. Vannucchi, R. Accardi, M. Tommasino, E. Affabris, G. Fiorucci and G. Romeo.

A IFN-stimulated genes

CBSS7-3 A NOVEL SMALL RNA REGULATORY MECHANISM EMPLOYED BY INTERFERONS FOR REGULATING GROWTH. Shreram C. Nallar, Limei Lin, Padmaja Gade, Edward R. Hofmann, Dhan V. Kalvakolanu.

CBSS7-4 COOPERATION OF STAT AND NFkB IN THE ASSEMBLY OF A TRANSCRIPTION COMPETENT INITIATION COMPLEX. Matthias Farlik, Thomas Decker.

PP1-171* THE TYPE I INTERFERON RECEPTOR PROTECTS AGAINST INFLUENZA VIRUS REPLICATION WHILE THE TYPE II RECEPTOR IS DISPENSIBLE. Alan G. Goodman, Hui Zeng, Cristian Cilloniz, Victoria S. Carter, Xinxia Peng, Sean C. Proll, Terrence M. Tumpey, Michael G. Katze.

PP1-172 PROMYLEOCYTIC ZINC FINGER PROTEIN REGULATES INTERFERON MEDIATED INNATE IMMUNITY. Bryan RG Williams, Dakang Xu, and Anthony J Sadler.

PP1-173* INTERFERON-LAMBDA MEDIATES RESISTANCE AGAINST VARIOUS RESPIRATORY VIRUSES. Mordstein Markus, Neugebauer Eva, Ditt Vanessa, Jessen Birthe, Rieger Toni, Günther Stephan, Wolff Thorsten, Klucher Kevin, Kochs Georg, Ehl Stephan, Michiels Thomas, Drosten Christian, Staeheli Peter.

PP1-174 IFNBETA INDUCES SECRETED IL-1 RECEPTOR ANTAGONIST PRODUCTION THROUGH A MEK2/PI3KDELTA-DEPENDENT, ERK1/2-INDEPENDENT PATHWAY IN HUMAN MONOCYTES. Karim J. Brandt, Rakel Carpintero, Lyssia Gruaz, Nicolas Molnarfi, and Danielle Burger.

PP1-175* AN ESSENTIAL ROLE FOR THE ANTIVIRAL ENDORIBONUCLEASE, RNASE-L, IN ANTIBACTERIAL IMMUNITY. Bret A. Hassel, Xiao-Ling Li, Heather J. Ezelle, Tae-Jin Kang, Lei Zhang, Kari Ann Shirey, Janette Harro, Jeffrey D. Hasday, Saroj K. Mohapatra, Oswald R. Crasta, Stefanie N. Vogel, and Alan S. Cross.

PP1-176* TICK-BORNE ENCEPHALITIS VIRUS DELAYS INTERFERON INDUCTION AND IS VERY SENSITIVE TO THE INTERFERON-STIMULATED GENE VIPERIN. Anna K Överby, Ju-Tao Guo and Friedemann Weber.

PP1-178 CONNECTING THE DOTS ON INTERFERON RESPONSIVENESS IN HCV/HIV CO-INFECTION: THE VIRUS, THE CYTOKINE, THE RECEPTOR AND THE GENE. Kottilil S, Kim C, Schmeisser H, Lempicki RA, Yang J, Zoon K, Young H., Polis MA, Fauci AS.

PP1-179 STRUCTURAL INSIGHTS IN THE ANTIVIRAL MxA PROTEIN: IMPORTANCE OF MxA OLIGOMERIZATION FOR ITS FUNCTION. Alexander von der Malsburg, Song Gao, Susann Paeschke, Joachim Behlke, Oliver Daumke, Georg Kochs, Otto Haller

PP1-180 IMAGING RESOLVES THE TEMPORAL AND SPATIAL PROPAGATION OF IFN ACTION IN VIVO. Mario Köster, Julia Pulverer, Ulfert Rand, Stefan Linienklaus, Daniela Kugel, Natalia Zietara, Siegfried Weiss, Peter Staeheli and Hansjörg Hauser.

PP1-181* THE INTERFERON-INDUCIBLE GENE IFI16, A MEMBER OF THE HIN200 FAMILY, TRIGGERS PRIMARY ENDOTHELIAL CELL APOPTOSIS THROUGH CASPASE 2 AND CASPASE 3 PATHWAY. Francesca Gugliesi, Marco De Andrea, Michele Mondini, Paola Cappello, Mirella Giovarelli, Marisa Gariglio, and Santo Landolfo.

PP1-182 CHARACTERIZATION OF GENE INDUCTION AND ANTIVIRAL EFFECTS ON HCVCC FOLLOWING RIBAVIRIN, INTERFERON AND POLYIC STIMULATION. Emmanuel Thomas, Qisheng Li, Shauna A. Clark, Jordan J. Feld, T. Jake Liang.

*Travel Award Recipient

PP1-184 POLYMERIZATION OF STAT1 DIMERS IS REQUIRED FOR STAT1 NUCLEAR RETENTION AND IFNGAMMA TARGET GENE INDUCTION. Filipa Antunes and Uwe Vinkemeier.

PP1-185 INTERFERON-INDUCED 2',5'-OLIGOADENYLATE SYNTHETASES VERSUS THOSE FROM SPONGES, EVOLUTIONARILY LOWEST MULTICELLULAR ANIMALS. Anne Kuusksalu, Annika Lopp, Mailis Päri, Tõnu Reintamm, Merike Kelve.

PP1-186 TYPE I INTERFERON-DEPENDENT GENE EXPRESSION IS A TARGET FOR GLUCOCORTICOID INHIBITION. Jamie R. Flammer, Megan A. Kennedy, Yurii Chinenov, Lionel B. Ivashkiv, and Inez Rogatsky.

PP1-187* DIVERGENT SUSCEPTIBILITIES OF HUMAN HERPESVIRUS 6 VARIANTS TO TYPE I INTERFERON. Joanna Jaworska, Louis Flamand.

PP1-188 DIFFERENTIAL DESENSITIZATION OF CELLS TO IFNα AND IFNβ. Véronique Francois, Gabriel Magno de Freitas Almeida, Ignacio Moraga, Danièle Monneron, Gilles Uze, Sandra Pellegrini.

PP1-189* IKAROS, TRANSCRIPTION FACTOR, REGULATES CIITA GENE EXPRESSION IN VASCULAR SMOOTH MUSCLE CELLS AND MACROPHAGES. Hye-Sook Choi, Kyung-Ho Kim, Eunhwa Sohn, Suhkneung Pyo.

PP1-190* LNX IS A NOVEL PROTEIN INTERACTOR FOR THE ANTIVIRAL ENDORIBONUCLEASE, RNASE-L. Heather J. Ezelle, Jesper B. Andersen, Irene Hao, and Bret A. Hassel.

PP1-191 IDENTIFICATION OF IFN-ALPHA-INDUCED GENES AND PROTEINS ASSOCIATED WITH ANTIVIRAL ACTIVITY IN DAUDI CELLS. Hana Schmeisser, Josef A. Mejido, Corey Balinsky, Kathryn C. Zoon.

PP1-192 PHYLOGENETIC ANALYSIS OF INTERFERON INDUCIBLE TRANSMEMBRANE GENE FAMILY AND FUNCTIONAL ASPECTS OF IFITM3. Fredy Siegrist, Martin Ebeling and Ulrich Certa.

PP1-193 RECIPROCAL REGULATION OF TRISTETRAPOLINE AND RNASE-L MODULATES THE INDUCTION OF PROINFLAMMATORY CYTOKINES. Xiao-Ling Li, Sarah E. Brennan, Gerald M. Wilson, Bret A. Hassel.

PP1-194* REGULATION OF THE ENDORIBONULEASE RNASE-L BY MICRORNAS. Teresa Y. Hsi, Xiao-Ling Li, and Bret A. Hassel.

PP1-195 NOVEL BIOASSAYS FOR MOUSE TYPE I AND TYPE III INTERFERONS. Daniela Kugel, Julia Elisabeth Pulverer, Mario Köster, Hansjörg Hauser and Peter Staeheli.

PP1-196 OCT-6 IS AN INTERFERON INDUCIBLE PROTEIN AND CONTRIBUTES TO THE TRANSCRIPTIONAL RESPONSES TO POLY(I:C). Elisabeth Hofmann, Ursula Reichart, Christian Gausterer, Christian Gülly, Dies Meijer, Mathias Müller and Birgit Strobl.

PP1-197 INFLAMMATION IN THE LIVER IS ASSOCIATED WITH DECREASED EXPRESSION OF IMMUNOHISTOCHEMICALLY DETECTED MYXOVIRUS RESITANCE PROTEIN B. Milen Vassilev, Diana Kyoseva, Jechka Vassileva, Georgi Mutafov, Vili Pashev, Lubomir Spassov, Ivan Mihailov.

PP1-198 EXPRESSION OF INTERFERON-INDUCED microRNAs IN PATIENTS WITH CHRONIC HEPATITIS C VIRUS INFECTION TREATED WITH PEGYLATED INTERFERON ALPHA. Carolina Scagnolari, Simona Cicetti, Carla Selvaggi, Pompea Zingariello, Jacopo Vecchiet, Delia Racciatti, Gloria Taliani, Ombretta Turriziani, Eligio Pizzigallo, Guido Antonelli.

PP1-199 ANTIVIRAL PROTECTION OF CELLS TREATED WITH EXOGENOUS OLIGOADENYLATE SYNTHETASE 1 (OAS1) IS MEDIATED THROUGH A NOVEL RNASE L-INDEPENDENT PATHWAY. Helle Kristiansen, Susanne Vends, Karthiga Thavachelvam, Søren Paludan, & Rune Hartmann.

PP1-200 SEQUESTRATION OF RB/E2F COMPLEX INTO PML-NBS PROVIDES A DISTINCT MECHANISM TO CONTROL THE EXPRESSION OF E2F TARGET GENES. Mathieu Vernier, Véronique Bourdeau, Marie-France Gaumont-Leclerc, David Beaudry, Olga Moiseeva, Valérie Forest, Fred Saad, Anne-Marie Mes-Masson, and Gerardo Ferbeyre.

PP1-201* CHARACTERISATION OF A NOVEL, CONSTITUTIVE CYTOKINE THAT REGULATES MUCOSAL IMMUNITY IN THE REPRODUCTIVE TRACT. Ka Yee Fung.

PP1-202 NFAR-1 AND NFAR-2 MODULATE TRANSLATION AND ARE REQUIRED FOR EFFICIENT HOST DEFENSE. Ai Harashima, Ingrid Pfeifer, Rachel Elsby, Hiroyasu Konno and Glen N. Barber.

LB-09 VIRUS INDUCED IRF-1 MEDIATES INTERFERON-INDEPENDENT ANTIVIRAL EFFECTS THROUGH THE INDUCTION OF VIPERIN. Anja Stirnweiss, Antje Ksienzyk, Hansjörg Hauser and Andrea Kröger.

B Immunopathogenesis

PP1-060 SUPER INTERFERONS FOR THE TREATMENT OF BIRD FLU. BJ Zheng, Johnny Sze, William KC Cheung, Marie C. Lin and Hsiang-fu Kung.

PP1-061 ROLE OF ISG15 IN VACCINIA VIRUS INFECTION. Susana Guerra, Ana Cáceres Núñez and Mariano Esteban.

PP1-062 THE PRODUCTION, PROCESSING AND SECRETION OF IL-1β IN AFRICAN SWINE FEVER VIRUS INFECTED PORCINE ALVEOLAR MACROPHAGES. Fuquan Zhang and Linda K. Dixon.

PP1-063 DIETHYLDITHIOCARBAMATE INDUCES APOPTOSIS VIA SUPPRESSION OF NF-KB PATHWAY IN HHV-8 INFECTED CELLS. Takashi Matsuno, Saori Morino, Shuichiro Yano, Mary Ann Suico, Tsuyoshi Shuto, Hirofumi Kai, Seiji Okada.

PP1-064* EMPLOYING THE METHOD OF RNA INTERFERENCE TO STUDY THE FUNCTION OF KAPOSI'S SARCOMA-ASSOCIATED HERPESVIRUS-ENCODED VIRF-3. Barbora Lubyova, Jose A. Frisancho, Paula M. Pitha.

PP1-065 ANALYSIS OF CHIKUNGUNYA VIRAL PROTEIN INTERACTIONS WITH THE INTERFERON RESPONSE PATHWAY. Matthew Tangeman, Thomas Briese, W. Ian Lipkin, David E. Levy.

PP1-066 GAMMA-INTERFERON (INGARON®) FOR TREATMENT OF HERPES SIMPLEX PATIENTS. Dmitriy S.Orekhov, Tamara B.Getiya, Irina M.Shapoval, Marina V.Mezentseva, Marina I.Tregubova, Alexey A.Khaldin, Alexander N.Narovlyansky.

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PP1-067 RIBOSOMAL PROTEIN L22 INHIBITS INDUCTION OF TYPE I IFN AND FACILITATES THE REPLICATION OF VSV. Eun-Jeong Yang.

PP1-068 THE HISTONE DEACETYLASE INHIBITOR ITF2357 DECREASES SURFACE CXCR4 AND CCR5 EXPRESSION IN CD4+ T-CELLS AND MONOCYTES AND INDUCES LATENT HIV-1 EXPRESSION IN VITRO. Shay Matalon, Brent E. Palmer, Marcel F. Nold, Antonio Furlan, Gianluca Fossati, Paolo Mascagni and Charles A. Dinarello.

PP1-070* ACTIVATION OF CASPASES IN CELLS LYTICALLY INFECTED WITH VACCINIA VIRUS. Jana Liskova, Jarmila Zajicova, and Zora Melkova.

PP1-071 POSITIVE ROLE OF MITOGEN-ACTIVATED PROTEIN KINASE PHOSPHATASE-1 IN REGULATING THE ANTI-MYCOBACTERIAL IMMUNE RESPONSES. Benny K.W. Cheung, Howard C.H. Yim, and Allan S.Y. Lau.

PP2-196 NONSTRUCTURAL PROTEIN 4B OF HEPATITIS C VIRUS INHIBITS INTERFERON RESPONSES. Hui-Ju Wu, Pong-Yu Huang, and Lih-Hwa Hwang.

PP2-197* ROLE OF FUSION ACTIVITY IN CROSS-PRESENTATION OF INFLUENZA NUCLEOPROTEIN-DERIVED ANTIGENS. Natalija Budimir, Tjarko Meijerhof, Jan WIlschut, Anke Huckriede, Aalzen de Haan.

PP2-198 HUMAN METAPNEUMOVIRUS BLOCKS THE INDUCTION OF TYPE I INTERFERON. B.van den Hoogen, M. Zahira, F. van Hagen, A. Andeweg, A. Osterhaus and R. Fouchier.

PP2-199 TYPE III INTERFERON ACTIVITY IN THE BRAIN. Prasanthi Bandi, Nyree Maes, Minjung Han, Anthony van den Pol, Michael D. Robek.

PP2-200 INTERFERON RESPONSE IN MURINE PLASMACYTOID DENDRITIC CELLS AFTER SARS CORONAVIRUS INFECTION. Anna de Lang, Corine H. Geurts van Kessel, Albert D.M.E Osterhaus and Bart L. Haagmans.

PP2-201 IDENTIFICATION OF NEW REGULATORS OF THE INNATE ANTIVIRAL RESPONSE USING A GENOME-SCALE LENTIVIRAL–BASED SHRNA SCREEN. Martin Baril and Daniel Lamarre.

PP2-202 THE RELATIVE ANTIVIRAL ACTIVITY OF HUMAN ALPHA INTERFERONS ON PRIMATE AND MOUSE ALPHA INTERFERONS ON HAMSTER AND RAT CELL LINES. Thomas B. Lavoie, Sara Crisafulli, Jessica Esposito, Karlene Moolchan, Lara Isotova, Sidney Pestka.

LB-10 INNATE RECOGNITION OF HERPES SIMPLEX VIRUS BY HUMAN PRIMARY MACROPHAGES IS MEDIATED BY INTRACELLULAR PATTERN RECOGNITION RECEPTORS. Jesper Melchjorsen, Johanna Rintakaha, Lars Østergaard, Ilkka Julkunen, Søren R. Paludan, Sampsa Matikainen.

LB-11 TOPICAL DELIVERY OF NOVEL DRUG FORMULATION "VIFERON®, GEL FOR LOCAL TREATMENT" IN GENITAL HERPES GUINEA PIG MODEL. Vyzhlova E.N. , Malinovskaya V.V. , Polesco I.V. , Parfenov V.V.

LB-12 TRANSCRIPTIONAL REGULATION OF T CELL DIFFERENTIATION DURING CHRONIC VIRAL INFECTION. E. John Wherry.

A Immunopathogenesis II

CIS2-3 P53 REGULATES TLR3 EXPRESSION AND FUNCTION IN HUMAN EPITHELIAL CELLS. Taura M., Fukuda R., Eguma A., Suico M.A., Koga T., Shuto T., Kai H.

CIS2-4 THE ANTI-INFLAMMATORY CYTOKINE IL-10 INHIBITS MIR-155 IN RESPONSE TO TOLL-LIKE RECEPTOR SIGNALLING. Claire E. McCoy and Luke A. J. O'Neill.

CIS2-5 ACTIVATION OF NK CELLS IN VIVO FOLLOWING LEISHMANIA INFECTION REQUIRES MYELOID DENDRITIC CELLS, TLR9 AND A UNIQUE SET OF CYTOKINES. Ulrike Schleicher, Simone Haeberlein, Heidi Sebald, and Christian Bogdan.

CIS2-6* A MEDIUM-THROUGHPUT, MICROPLATE-BASED EX VIVO MODEL FOR MEASURING INTRAMACROPHAGE GROWTH OF MYCOBACTERIUM TUBERCULOSIS. Daniel Eklund, Amanda Welin, Maria Lerm.

CIS2-7 NLRP3 INFLAMMASOME IN MALARIA: ROLE OF HEMOZOIN-INDUCED SIGNALING ON INFLAMMASOME ACTIVATION. Marina Tiemi Shio, Stephanie C. Eisenbarth, Myriam Savaria, Adrien F. Vinet, Marie-Josée Bellemare, Kenneth W. Harder, Fayyaz S. Sutterwala, D. Scott Bohle, Albert Descoteaux, Richard A. Flavell and Martin Olivier.

CIS2-8 SUBVERSION OF HUMAN CD4+CD25+ REGULATORY T CELLS TO IL-17-PRODUCING T CELLS BY AN INFLAMMATORY MILIEU. B Afzali 1, PJ Mitchell, A Rani, W Khamri, SY Kordasti, KB Bamford, B Grimbacher, Susan John, RI Lechler, G Lombardi.

B Immunopathogenesis III

CIS6-4 ALPHA-1-ANTITRYPSIN INHIBITS INFLUENZA IN VITRO, REDUCES INFLUENZA DISEASE IN VIVO, AND GENETIC DEFICIENCY IS A RISK FACTOR FOR HUMAN INFLUENZA INFECTION. K. Scott Beard, Sam MaWhinney, Martin Zamora, Rebecca E. Oberley-Deegan, James D. Crapo, Gregory B. Pott, Claudia Nold-Petry, Andrew Churg, Eli C. Lewis, Charles L. Edelstein, Charles A. Dinarello, and Leland Shapiro.

CIS6-5 LETHAL VIRAL INFECTION RESULTS FROM STAT1 BUT NOT STAT2 OR IRF9 DEFICIENCY IN MICE AND IS MEDIATED BY CD4+ T-CELLS. Markus J. Hofer, Peter Manders, Sue Ling Lim, Rachael L. Terry, Meghann T. Getts, Daniel R. Getts, Nicholas J.C. King and Iain L. Campbell.

A Immunoregulation

SLBAW2-C SOLUBLE HUMAN CXCR2: STRUCTURE, PROPERTIES, BIOACTIVITY. Kanstantsin Katlinski, Sviatlana Akalovich, Yuliya Katlinskaya, Anton Sholukh, Tatyana Doroshenko, Yury Chaly, Nikolai Voitenok.

CBSS5-8 ANTIVIRAL EFFECTS OF CYTOKINES. Thomas B. Lavoie, Sara Crisafulli, Herwig Moll, Christine Brostjan, Sidney Pestka.

PP2-030 IL-27 ABROGATES RANKL-MEDIATED OSTEOCLASTOGENESIS THROUGH STAT1- DEPENDENT INHIBITION OF C-FOS. Hiroki Yoshida, Mitsuru Furukawa, and Hironari Takaishi.

PP2-031 IL-17/TH-17 PROMOTES TYPE-I T CELL IMMUNITY AGAINST PULMONARY INTRACELLULAR BACTERIAL INFECTION THROUGH MODULATING DC FUNCTION. Hong Bai, Jianjun Cheng, Xiaoling Gao, Antony George Joyee, Yijun Fan, Shuhe Wang, Lei Jiao, Zhi Yao and Xi Yang.

PP2-032* ROLE OF MULTIPLE REGULATORY T CELL POPULATIONS IN CONTROLLING PERIPHERAL BLOOD AND LIVER IMMUNITY TO HUMAN HEPATITIS C VIRUS INFECTIONS. Mark Claassen, Rob de Knegt, Duygu Turgut, Anthonie Groothuismink, Harry Janssen, André Boonstra.

PP2-033 MONOCYTES FROM CHRONIC HCV PATIENTS ARE FUNCTIONALLY ALTERED WITH DISTINCT REGULATION OF THE TLR4 AND TLR7/8 PATHWAYS. Bisheng Liu, Harry L.A. Janssen, Andre Boonstra.

PP2-034 THE ANTI-CD20 ANTIBODY RITUXIMAB REDUCES THE TH17 RESPONSE. Frank L. van de Veerdonk, Bernard Lauwerys, Franco DiPadova, Renoud J. Marijnissen, Marije I. Koenders, Ilse Gutierrez-Roelens, Patrick Durez, Mihai G. Netea, Jos W.M. van der Meer, Wim B. van den Berg, Leo A.B. Joosten.

PP2-036* EPSTEIN-BARR VIRUS AND PLASMACYTOID DENDRITIC CELLS: A POSSIBLE DUET IN AUTOIMMUNITY. M Severa, B Serafini,V Gafa, E Anastasiadou, E Giacomini, P Trivedi, F Aloisi, EM Coccia.

PP2-037 IMMUNOMODULATION VIA TLR3/IRF3 BY MISPLACED U1-SNRNA AS DETECTED IN HUMAN A549 LUNG EPITHELIAL CELLS. Christan D. Sadik, Malte Bachmann, Josef Pfeilschifter, Heiko Mühl.

PP2-038 A REWIEW OF THE CYTOKINE NETWORK IN MULTIPLE MYELOMA: DIAGNOSTIC, PROGNOSTIC AND THERAPEUTIC IMPLICATIONS. Vito Michele Lauta.

PP2-039 METABOLIC PARAMETERS AND CELLULAR ACTIVATION ARE DETERMINANT OF IMMUNE RECONSTITUTION IN ARV-TREATED HIV-1-INFECTED SOUTH AFRICANS. Livio Azzoni, Cynthia Firnhaber, Xiangfan Yin, Andrea Foulkes, Deborah Glencross, Wendy Stevens, Nigel Crowther, Emmanouil Papasavvas, Ian Sanne and Luis Montaner.

PP2-040 IFN-g INDUCES IL-23 EXPRESSION IN PRIMARY HUMAN MONOCYTES VIA THE P38 MAPKS INDEPENDENTLY OF THE JAK/STAT SIGNALLING. Maria A Blahoianu, Ali A Rahimi, Jonathan G Boucher, Niranjala Gajanayaka, Jonathan B Angel and Ashok Kumar.

PP2-041 DISSECTING IN VIVO INNATE IMMUNE RESPONSES TO TLR7/9 AGONISTS IN PRIMATES. Montserrat Puig, Lucja T. Grajkowska, Joseph J. Mattapallil, and Daniela Verthelyi.

PP2-042* CD93 REGULATES INFLAMMATION IN VIVO. Mallary C. Greenlee and Suzanne S. Bohlson.

PP2-044 UPREGULATION OF AUTOPHAGY BY INHIBITORS OF mTOR OR CASPASES DECREASES SPLENIC T CELL APOPTOSIS IN SEPSIS. Ya-Ching Hsieh, Hsiang-Wei Hsueh, Chi-Hsun Hsieh, Shyng-Shiou Yuan.

PP2-045 POTENTIALLY PROBIOTIC BACTERIA INDUCE CYTOKINE PRODUCTION AND SUPPRESSOR OF CYTOKINE SIGNALING 3 GENE EXPRESSION IN HUMAN MONOCYTE-DERIVED MACROPHAGES. Sinikka Latvala, Minja Miettinen, Riina Kekkonen, Riitta Korpela, Ilkka Julkunen.

PP2-046 BIOACTIVITY-GUIDED IDENTIFICATION AND CELL SIGNALING ANALYSIS TO INVESTIGATE THE IMMUNOMODULATORY EFFECTS OF GINSENG ON U937 CELLS. Davy Lee, Cindy Yang, Stanley Chik, James Li, Allan Lau.

PP2-047 EXPRESSION OF IL-22R1 ON LYMPHOCYTES INDUCES LETHAL INFLAMMATION. Ram Savan, Della Reynolds, Adelle McFarland, Lionel Feigenbaum, Raymond P. Donnelly, Howard A. Young.

PP2-048* REGULATORY FUNCTION OF SOCS-3 IN ASTROCYTES. Hongwei Qin, Stephanie L. Reynolds and Etty N. Benveniste.

PP2-049* REGULATORY T CELLS DEMONSTRATE AN INJURY-SPECIFIC RECALL RESPONSE. Goro Tajima, Fionnuala O'Leary, Marc Hanschen, Kimiko Ikeda, Adam Delisle, Mohamed Oukka, Vijay Kuchroo, James Lederer.

PP2-050* ADMINISTERED G CSF BOOSTS LOW AFFINITY ANTIBODY PRODUCTION DURING T CELL DEPENDENT RESPONSES. Ann L. Cornish, Jo L. Eyles, Jane Murphy, Sarah F. Drake, Donald Metcalf, Andrew W. Roberts, David M. Tarlinton, Ian P. Wicks.

PP2-051 ROLE OF C/EBPB AND CREB IN IL-10 PRODUCTION BY TOLEROGENIC DENDRITIC CELLS. Chantal Guindi, Alexandre Cloutier, Michaël Ménard, Patrick P. Mcdonald, Abdelaziz Amrani.

PP2-052* MULTIPARAMETER PHOSPHO-FLOW ANALYSIS OF PERIPHERAL BLOOD IN EARLY RHEUMATOID ARTHRITIS. Carole L. Galligan, Janet Siebert, Katherine Siminovitch, Edward Keystone, Vivian Bykerk, Omar Perez and Eleanor N. Fish.

PP2-054* MODELING AUTOREGULATION BY IL-2 IN T-CELL ACTIVATION: A TIME TO COOPERATE, A TIME TO COMPETE. Nir Waysbort, Yonatan Savir, Yaron Antebi, Tsvi Tlusty, and Nir Friedman.

PP2-055 GALECTIN-3, A β-GALACTOSIDE BINDING LECTIN AFFECTS CYTOKINE SECRETION BY HUMAN MACROPHAGES. Rudjer Novak, Sanja Dabelic, Adriana Lepur and Jerka Dumic

PP2-056* REGULATION OF ENERGY METABOLISM BY THE RP105/TLR AXIS. Senad Divanovic, Aurelien Trompette, Paul T. Pfluger, Stuart P. Weisberg, Isaac T.W. Harley, Leah M. Flick, Jessica L. Allen, Deborah J. Clegg, Randy J. Seeley, Matthias H. Tschöp, Christopher L. Karp.

PP2-057 HIGH CIRCULATING LEVELS OF FREE INTERLEUKIN-18 IN PATEINTS WITH ACTIVE SLE IN THE PRESENCE OF ELEVATED LEVELS OF INTERLEUKIN-18 BINDING PROTEIN. Daniela Novick, Daniel Elbirt, Galit Miller, Charles A Dinarello, Menachem Rubinstein and Zev M Sthoeger.

*Travel Award Recipient

PP2-058 INDUCTION OF REGULATORY MONOCYTES: A NOVEL MECHANISM OF GLUCOCORTICOIDS TO BLOCK AUTOIMMUNITY. Georg Varga, Jan Ehrchen, Nadine Nippe, Athanasios Tsianakas, Matthias Ross, Andreas Luegering, Tilmann Spieker, Kirsten Roebrock, Ralph Lippe, Klaus Tenbrock, Pieter J. M. Leenen, Johannes Roth, and Cord Sunderkoetter.

PP2-059 IMMUNE REGULATION IN CD8+ T CELL-MEDIATED PULMONARY DISEASE. Milena J. Tosiek, Achim D. Gruber, Marcus Gereke, Dunja Bruder.

PP2-060* CROSS-TALK BETWEEN FIBROBLASTS AND ENDOTHELIAL CELLS INFLUENCES THE RECRUITMENT AND RETENTION OF LYMPHOCYTES IN A CO-CULTURE MODEL OF INFLAMMATION. Helen M. McGettrick, Andrew Filer, Christopher D. Buckley, G. Ed Rainger and Gerard B. Nash.

PP2-061 THE EFFECT OF ALPHA-1-ANTITRYPSIN ON DENDRITIC CELL MATURATION AND MIGRATION: POSSIBLE MECHANISM FOR ALLOGRAFT TOLERANCE. Mark Mizrahi, Eyal Ozeri, Galit Shahaf, Keren Bellacen, Hadas Moser, David Ohayon, Charles A Dinarello and Eli C Lewis.

PP2-062 PROTECTION OF ISLET ALLOGRAFTS BY IN VIVO INTRODUCTION OF AN EXTRACHROMOSOMAL PLASMID EXPRESSING HUMAN ALPHA-1-ANTITRYPSIN. Galit Shahaf, Hadas Moser, Keren Bellacen, Mark Mizrahi and Eli C Lewis.

PP2-063 ISLET ALLOGRAFT SURVIVAL AND ANTI-INFLAMMATORY CYTOKINE PROFILE ARE PROVIDED BY CIRCULATING TRANSGENIC LUNG-SPECIFIC ALPHA-1-ANTITRYPSIN. Galit Shahaf, Nathaniel DeFelice, Chad Ficek, Frida Friedman, Charles A Dinarello and Eli C Lewis.

PP2-064 HIGH ANTITUMOR ACTIVITY OF RLI, AN IL15-IL15RALPHA FUSION PROTEIN, IN METASTATIC MELANOMA AND COLORECTAL CANCER. Anne Bessard, Véronique Solé, Grégory Bouchaud, Agnès Quéméner, Yannick Jacques.

PP2-065 SERPINB9 EXPRESSION IN RENAL TUBULAR EPITHELIAL CELLS IS INDUCED BY TOLL-LIKE RECEPTOR 3. Kirstin M. Heutinck, Jorien Kassies, Ineke J.M. ten Berge, Jörg Hamann, Ajda T. Rowshani.

PP2-066* OPPOSITE EFFECTS OF INTERFERON REGULATORY FACTORS 3 AND 7 ON DIFFERENTIAL MODIFICATION OF HISTONE H3 ASSOCIATED TO HUMAN INTERFERON-A GENE PROMOTERS. Pierre Génin, John Hiscott and Ahmet Civas.

PP2-067 DIRECT INTERACTION OF RECEPTOR FOR ADVANCED GLYCATION END PRODUCTS WITH CPGA AND COMPLEMENT COMPONENT C3A AUGMENTS INTERFERON ALPHA IN HUMAN PBMCS. Xin Li, Jun Kuai, Kristina Cunningham, Benfang Ruan, Lori Fitz, Karl Nocka, Aaron Winkler, Janet Paulsen, Debbie Pittman, Lih Ling Lin, David Winkler.

PP2-068 IRAK-M IS A CENTRAL REGULATOR OF THE OPPOSITE EFFECTS OF ACUTE AND CHRONIC ALCOHOL EXPOSURE ON LPS-INDUCED INFLAMMATION IN HUMAN MONOCYTES. Pranoti Mandrekar, Shashi Bala, Donna Catalano, Karen Kodys, Gyongyi Szabo.

PP2-070 DIVERGENT EFFECTS OF EARLY NEUTRALIZATION VS. SUSTAINED SUPPRESSION OF ENDOTHELIAL CELL GROWTH FACTOR, ANGOIPOIETIN (ANG)-2, IN HEMORRHAGE PRIMING FOR ACUTE LUNG INJURY (ALI) FOLLOWING SUBSEQUENT SEPTIC CHALLENGE IN MICE. Joanne Lomas-Neira, Fabienne Venet, Chun-Shiang Chung, Rajan Thakkar, Alfred Ayala.

PP2-071* ABERRANT PD-L1 EXPRESSION CONTRIBUTES TO SUSCEPTIBILITY TO AUTOIMMUNE MYOCARDITIS FOLLOWING COXSACKIEVIRUS INFECTION. Maya C. Poffenberger and Marc S. Horwitz.

PP2-072 ACTIVATION OF THE RIG-I SIGNALING PATHWAY INHIBITS HIV-1 REPLICATION. Mayra Solis, Peyman Nakhaei and John Hiscott.

PP2-073 ANTI-TNFa THERAPY MODULATES THE IL-33/ST2 AXIS IN INFLAMMATORY BOWEL DISEASE. Luca Pastorelli, Sharon B.Hoang, Rekha R. Garg, Luisa Spina, Claudio Fiocchi, Maurizio Vecchi, and Theresa T.Pizarro.

PP2-074 NATTECTIN A FISH C-TYPE LECTIN LICENSES MACROPHAGES TO DIFFERENTIATE INTO CELLS EXHIBITING TYPICAL DENDRITIC CELLS FUNCTION. Tania C Saraiva, Lidiane Z Grund, Evilin N Komegae, Douglas Boletini-Santos, Anderson D Ramos, Katia Conceição, Noemia M Orii, Carla Lima, Monica Lopes-Ferreira.

PP2-075 THE COUNTER-BALANCE OF LONG-TERM EXPANSION OF MEMORY AND REGULATORY CELLS SUSTAINS CHRONIC ASTHMA IN A MURINE MODEL. Milena F Kabbara, Lidiane Z Grund, Evilin N Komegae, Douglas Boletini-Santos, Monica Lopes-Ferreira, Carla Lima.

PP2-076 IMMMUNOMODULATION BY QUERCETIN AND INTERFERON-b IN MULTIPLE SCLEROSIS PATIENTS. Kailash Chadha, Zohara Sternberg, PhD. Alicia Lieberman, Allison Drake, Bianca Weinstock-Guttman, Frederick Munschauer.

PP2-077 INHIBITION OF MURINE ΓΔ LYMPHOCYTE EXPANSION AND EFFECTOR FUNCTIONS BY REGULATORY AB T-CELLS THROUGH GITR-MEDIATED CELL-CELL INTERACTIONS. Natacha Gonçalves Sousa, Julie C. Ribot, Ana deBarros, Daniel V. Correia, Íris Caramalho, Bruno Silva-Santos.

PP2-078* IL-23 INDUCED SIGNALING AND IL-23 RECEPTOR EXPRESSION IN HUMAN CD4 T CELLS. Nor Fazila Che Mat, Christina Guzzo, and Katrina Gee.

PP2-079* INTERLEUKIN-27 INDUCES A PRO-INFLAMMATORY CYTOKINE AND CHEMOKINE PROFILE IN RESTING HUMAN MONOCYTES. Christina Guzzo, Nor Fazila Binti Che Mat, and Katrina Gee.

PP2-080 INVESTIGATION OF CXCL12-INDUCED T-LYMPHOCYTE MIGRATION BY PROTEOMICS ANALYSIS OF ISOLATED PSEUDOPODIA. Dustin N.D. Lippert and John A. Wilkins.

PP2-081* THE ROLE OF beta-GLUCAN RECEPTOR DECTIN-1 IN PHAGOCYTOSIS AND TNF-ALPHA PRODUCTION BY MACROPHAGES. Seon-A Jang, Sulkyoung Park, Kyung-Suk Kim, Haemi Joo, Suhkneung Pyo, Kwang-Hee Yang, Eun-Hwa Sohn.

PP2-082 CHARACTERIZATION OF MUCOSAL AND SYSTEMIC IMMUNE RESPONSES ELICITED BY INTERLEUKIN CYTOKINES AS MUCOSAL ADJUVANT AGAINST INFLUENZA VIRUS. Hiroyuki Kayamuro.

PP2-083 ARYL HYDROCARBON RECEPTOR REGULATES LIPOPOLYSACCHARIDE-INDUCED INFLAMMATORY RESPONSES. Akihiro Kimura, Tetsuji Naka, Taisuke Nakahama, Ichino Chinen, Kazuya Masuda and Tadamitsu Kishimoto.

PP2-084 IN VITRO INDUCTION OF CLASS SWITCH RECOMBINATION TO IgG1 IS FAVORED BY STIMULATION VIA BCR, CD40, TLR9 AND BAFF. Gabriela Lopez-Herrera, Ulrich Salzer, Hermann Eibel, Bodo Grimbacher.

PP2-086 STAT FAMILY OF TRANSCRIPTION FACTORS IN THE MODULATION OF THE RESPONSE TO SUPERANTIGENS. Eder Mateus, Georgina Civit, Joan Manils, Consol Benaiges, Esther Moga, Candido Juarez.

PP2-087 MILD ELECTRICAL STIMULATION SUPPRESSES INTERLEUKIN-2 EXPRESSION IN JURKAT T CELLS. Yuichiro Shimauchi, Saori Morino, Shuichiro Yano, Mary Ann Suico, Tsuyoshi Shuto, Hirofumi Kai.

PP2-088 ANTI-INFLAMMATORY COMPOUNDS FROM MEDICINAL HERBS CAPABLE OF MODULATING CYTOKINES EXPRESSION IN HUMAN PRIMARY BLOOD MACROPHAGES. Cindy LH Yang, Liangjie Wang, Terry CT Or , Stanley CC Chik, James CB Li and Allan SY Lau.

PP2-089 APPLICATION OF A HIGH SENSITIVITY EVIDENCE BIOCHIP ARRAY TO THE MULTIPLEXED MEASUREMENT OF CYTOKINES IN SALIVA. Maria L. Rodriguez, Robert I. McConnell, Frances M. Kelly, Stephen P. Fitzgerald.

PP2-090 MAST CELLS ARE REQUIRED FOR INTERLEUKIN-18 DEPENDENT MUCOSAL IMMUNE RESPONSES. Yasuhiro Abe, Hiroyuki Kayamuro, Yasuo Yoshioka, Shu-hei Arita, Haruhiko Kamada, Tetsuya Nomura, Norio Itoh, Tomoaki Yoshikawa, Kazuya Nagano, Shin-ich Tsunoda, Yasuo Tsutsumi.

PP2-091 CREATION OF LYSINE-DEFICIENT MUTANT LYMPHOTOXIN-a WITH RECEPTOR SELECTIVITY BY USING PHAGE DISPLAY. Yasuo Yoshioka, Hikaru Watanabe, Tomohiro Morishige, Xinglei Yao, Shinji Ikemizu, Chioko Nagao, Shandar Ahmad, Kenji Mizuguchi, Yasuo Tsutsumi, Yohei Mukai, Naoki Okada, Shinsaku Nakagawa.

PP2-092 ANTIMICROBIAL CATHELICIDIN PEPTIDE CAP11 SUPPRESSES THE PRODUCTION AND RELEASE OF SEPTIC MEDIATORS IN ENDOTOXIN SHOCK MICE. Taisuke Murakami, Hiroshi Tamura, Isao Nagaoka.

PP2-093 NOVEL ANTIBODIES FOR ASSAYING MÜLLERIAN INHIBITORY SUBSTANCE. Ischenko A., Trofimov A., Petrov A., Rodin S., Zhakhov A., Simbirtsev A.

PP2-094* STIMULATED PLASMACYTOID DENDRITIC CELLS ACTIVATE NATURAL KILLER CELLS VIA SECRETED FACTORS ALONE. Shaheed A. Abdulhaqq, Costin Tomescu, Luis J. Montaner.

PP2-095 PRIMING FOR TH2 DIFFERENTIATION BY IL-2-MEDIATED INDUCTION OF IL-4 RECEPTOR A CHAIN EXPRESSION. Wei Liao, Dustin E. Schones, Jangsuk Oh, Yongzhi Cui, Kairong Cui, Tae-Young Roh, Keji Zhao, and Warren J. Leonard.

PP2-096 SWINE MIXED CONTINUOUS FLOW BACTERIAL CULTURE INDUCES IL-1BETA, IFN GAMMA, AND IL-18 PRODUCTION IN SPLENOCYTES DERIVED FROM NEONATAL SWINE. Kenneth J. Genovese, Haiqi He, David J. Nisbet, Roger B. Harvey.

PP2-097 MACROPHAGES REPRESENT THE PRIMARY INJURY-RESPONSIVE ANTIGEN PRESENTING CELL TYPE. Kimiko Ikeda, Goro Tajima, Fionnuala O'Leary, Marc Hanschen, Adam Delisle, James Lederer.

PP2-098 EVALUATION OF THE EFFECT OF RICIN INTOXICATION ON SERUM CYTOKINE LEVELS IN MICE. Hardeep S. Bhogal, Lori J. McLaws, L.M. Negrych, and John W. Cherwonogrodzky.

LB-13 MODULATION OF DENDRITIC CELL ACTIVATION BY CHEMOKINES AND CELLULAR INJURY. Faris Q. Alenzi, Mohammed W. Al-Rabia,Badi Q. Alenazi, Iman El-tounsi, Shamweel Ahmad, Essam H. Matter and Richard Wyse.

LB-14 THE EFFECT OF TACROLIMUS ON ALTERNATE T-CELL ACTIVATION PATHWAYS. Asha A. Kulkarni-Almeida, Bindu Hegde, Prabha Misra, Periyasamy Giridharan, Amit Khanna, Muralidhara Padigaru and Arun Balakrishnan.

LB-15 ACTIVATION OF A MIR-9/NF-KB REGULATORY LOOP IN HUMAN MONOCYTES AND NEUTROPHILS EXPOSED TO PROINFLAMMATORY SIGNALS. Flavia Bazzoni, Laura Mori, Marzia Rossato, Marco Fabbri, Daniele Gaudiosi, Massimiliano Mirolo, Nicola Tamassia, Alberto antovani, Marco A. Cassatella, and Massimo Locati.

LB-16 POTENTIAL MECHANISM OF IMMUNE REGULATION VIA THE LINK ARYL HYDROCARBON RECEPTOR (AHR) AND INDOLEAMINE 2,3-DIOXYGENASE (IDO) IN MURINE DENDRITIC CELLS. Nam T. Nguyen, Akihiro Kimura, Tadamitsu Kishimoto.

LB-17 RECOGNITION VERSUS ADAPTIVE UPREGULATION AND DEGRADATION OF CC CHEMOKINES BY THE CHEMOKINE DECOY RECEPTOR D6 ARE DETERMINED BY THEIR N-TERMINAL SEQUENCE. B Savino, EM Borroni, N Machado Torres, P Proost , S Struyf, A Mortier, A Mantovani, M Locati, R Bonecchi.

LB-18 TLR SIGNALING INCREASES IMMUNOGENICITY OF RETROVIRAL HIV-1 VACCINE CANDIDATE. Lars Toft, Martin Tolstrup, Ole S. Søgaard, Jesper Melchjorsen, Lars Østergaard, Shervin Bahrami, Finn S. Pedersen and Mogens Duch.

LB-19 ROLE OF TIR8/SIGIRR, A NEGATIVE REGULATOR OF IL-1/TLR SIGNALING, IN THE PULMONARY IMMUNE RESPONSE TO PSEUDOMONAS AERUGINOSA INFECTION. Véliz T., Moalli F., Paroni M., Polentarutti N., Anselmo A., Riva F., Mantero S., Mantovani A., Garlanda C.

B Immunoregulation I

CBSS1-4* A CRITICAL FUNCTION OF TGF-BETA IN THE GENERATION OF ADAPTIVE AND NATURAL CD4+FOXP3+ REGULATORY T CELLS. WanJun Chen.

*Travel Award Recipient

CBSS1-5 DUAL FUNCTION FOR A VISION-RELATED MOLECULE: RETINOIC ACID IN THE EYE MAY CONTRIBUTE TO OCULAR IMMUNE PRIVILEGE BY INDUCING T REGULATORY CELLS. Ru Zhou, Rachel R Caspi.

A Immunoregulation II

CBSS5-1 IMMEDIATE MEDIATORS OF THE INFLAMMATORY RESPONSE ARE POISED FOR RAPID GENE ACTIVATION THROUGH RNA POLYMERASE STALLING. Megan Kennedy, Sergei Nechaev, Daniel A. Gilchrist, Ginger W. Muse, Yurii Chinenov, Karen Adelman Inez Rogatsky.

CBSS5-2 LOX-1 AS NATURAL IFN-α MEDIATED SIGNAL FOR APOPTOTIC CELL UPTAKE AND ANTIGEN PRESENTATION IN DENDRITIC CELLS. Stefania Parlato, Giulia Romagnoli, Francesca Spadaro, Irene Canini, Paolo Sirabella, Paola Borghi, Carlo Ramoni, Ilaria Filesi, Silvia Biocca, Lucia Gabriele and Filippo Belardelli.

CBSS5-3* NOVEL GENE EXPRESSION PATTERNS IN IFN-GAMMA 3'UNTRANSLATED REGION AU-RICH ELEMENT-DELETED MICE. Deborah L. Hodge, Cyril Berthet, Jeff Subleski, Vincenzo Coppola, Matthew Buschman, Catherine Razook, Howard A. Young.

CBSS5-4 THE IL-27 P28 SUBUNIT BINDS CLF TO FORM A CYTOKINE REGULATING NK AND T CELL ACTIVITIES REQUIRING IL-6R FOR SIGNALING. Sandrine Crabé, Angélique Guay-Giroux, Aurélie Tormo, Dorothée Duluc, Rami Lissilaa, Florence Guilhot, Ulrick Mavoungou-Bigouagou, Fouad Lefouili, Isabelle Cognet, Walter Ferlin, Greg Elson, Pascale Jeannin and Jean-François Gauchat.

CBSS5-5* THE TYPE I INTERFERON (IFN)-α MEDIATES A MORE SEVERE NEUROLOGICAL DISEASE IN THE ABSENCE OF THE CANONICAL SIGNALING MOLECULE INTERFERON REGULATORY FACTOR (IRF) 9. Markus J. Hofer, Wen Li and Iain L. Campbell.

CBSS5-6 UNC93 HOMOLOG B1 REGULATES THE BALANCE OF TOLL-LIKE RECEPTOR 7 AND TOLL-LIKE RECEPTOR 9 RESPONSES RECIPROCALLY IN DENDRITIC CELLS. Ryutaro Fukui, Shin-ichiroh Saitoh, Fumi Matsumoto, Hiroko Kozuka-Hata, Masaaki Oyama, Koichi Tabeta, Bruce Beutler, and Kensuke Miyake.

CBSS5-7 IDENTIFICATION OF A NOVEL ANTIGEN PRESENTING CELL POPULATION MODULATING ANTI-INFLUENZA TYPE-2 IMMUNITY. Jae-Kwang Yoo, Carole Galligan, Daniel Burke, Carl Virtanen, Eleanor N. Fish.

B Inflammation & Pathogenesis

PP2-099 LYMPHOTOXIN: FROM INFLAMMATION TO LYMPHOID ORGANS AND BACK. Nancy H. Ruddle.

PP2-100 INTERLEUKIN 15 IN ACUTE PANCREATITIS. Chooklin S., Bihalskyy I., Lyba M.

PP2-101 PREDICTIVE VALUE OF INTERLEUKIN-6 AND TRANSFORMING GROWTH FACTOR-BETA1 IN IDENTYFYING PATIENTS WITH A FIRST RESTENOSIS, RECURRENT RESTENOSIS OR A HISTORY OF RESTENOSIS. Bartosz Hudzik, Janusz Szkodzinski, Wojciech Romanowski, Krzysztof Wilczek, Rafal Wojnar, Andrzej Lekston, Lech Polonski, Barbara Zubelewicz-Szkodzinska.

PP2-102 EFFECT OF CRUDE EXTRACT OF ANISAKIS SIMPLEX LARVAE ON ACTIVATION OF HUMAN EOSINOPHILS. Solah Park.

PP2-103 A NOVEL C-TYPE LECTIN PROTEIN FROM THE Thalasshopryne maculosa VENOMOUS FISH WITH INFLAMMATORY-INDUCING ACTIVITY IN MICE. Douglas Boletini-Santos, Katia Conceição, Ines Sosa-Rosales, Mônica Lopes-Ferreira, Carla Lima.

PP2-104 INTERLEUKIN-10 INHIBITS EXPRESSION OF GENES INDUCED BY WILD-TYPE NEISSERIA MENINGITIDIS IN HUMAN MONOCYTES. Unni Gopinathan, Reidun Øvstebø, Ole Kristoffer Olstad, Peter Kierulf, Petter Brandtzaeg and Jens Petter Berg.

PP2-105 MICRORNA PATTERNS IN HUMAN MONOCYTES INDUCED BY WILDTYPE VERSUS LPS DEFICIENT NEISSERIA MENINGITIDES - RELATIONSHIP BETWEEN MICRORNA AND LPS SENSITIVE GENES. Reidun Øvstebø, Kari Bente Foss Haug, Carola Henriksson, Hans Christian Dalsbotten Aass, Anne Marie Siebke Trøseid, Ole Kristoffer Olstad, Peter Kierulf, Petter Brandtzæg, Jens Petter Berg.

PP2-106* SECONDARY NECROSIS OF APOPTOTIC NEUTROPHILS INDUCED BY THE HUMAN CATHELICIDIN LL-37 IS NOT PROINFLAMMATORY TO PHAGOCYTOSING MACROPHAGES. Hsin-Ni Li, Peter G. Barlow, Johan Bylund, Annie Mackellar, Åse Björstad, James Conlon, Pieter S. Hiemstra, Chris Haslett, Mohini Gray, A. John Simpson, Adriano G. Rossi and Donald J. Davidson.

PP2-107 ABSENCE OF IFN-GAMMA ACCELERATES THROMBUS RESOLUTION THROUGH ENHANCED MMP-9 AND VEGF EXPRESSION. Toshikazu Kondo, Mizuho Nosaka, Yuko Ishida, Akihiko Kimura, Yumi Kuninaka, Masanori Inui, and Naofumi Mukaida.

PP2-108 CCL5-CCR5 AXIS MEDIATES SKIN WOUND HEALING BY PROMOTING ENDOTHELIAL PROGENITOR CELL ACCUMULARION. Yuko Ishida, Akihiko Kimura, Masanori Inui, Yumi Kuninaka, Kouji Matsushima, Naofumi Mukaida, Toshikazu Kondo.

PP2-109 USING LUMINEX (xMAP) TECHNOLOGY TO ASSAY FOR INFLAMMATORY CYTOKINES IN CELL CULTURE SUPERNATANTS FROM DAUDI AND OVCAR-3 CELLS TREATED WITH INTERFERON. Joseph Bekisz, Hana Schmeisser, David Stephany and Kathryn Zoon.

PP2-110* PROSTAGLANDIN E2 (PGE2) INDUCES IL-6 IN HUMAN ORBITAL FIBROBLASTS THROUGH A CREB-DEPENDENT MECHANISM. Nupur Raychaudhuri and Terry J. Smith.

PP2-111 INVOLVEMENT OF TSUKUSHI IN THE PATHOGENESIS OF DSS-INDUCED COLITIS IN MICE: POTENTIAL ROLE OF TSUKUSHI IN INFLAMMATORY RESPONSES. Kenji Watanabe, Shogo Shimasaki, Tsuyoshi Shuto, Mary Ann Suico, Tomoaki Koga, Takashi Sato, Kunimasa Ohta and Hirofumi Kai.

PP2-112 INDUCTION OF NEUTROPHIL DEGRANULATION BY S100A9 VIA A MAPK-DEPENDENT MECHANISM. Jean-Christophe Simard, Denis Girard, and Philippe A. Tessier.

PP2-113 MYD88 GENE KNOCKOUT INHIBITS THE DEVELOPMENT OF LUPUS-LIKE DISEASE IN NZB/W F1 MICE. Tomoharu Ohkawara Minoru Fujimoto Tetsuji Naka.

PP2-114 THE FUNCTION OF IL-17-PRODUCING CELLS IN INFLAMMATORY DISEASE. Aoi Akitsu.

PP2-115* THE EFFECT OF VITAMIN C ON STRESS-INDUCED CHANGES IN HEART. Hyemin Kim, Jae Seung Kang, Hyung Gun Maeng, Se Yeon Bae, Na Eun Lee, Joo Myoung Kong, and Wang Jae Lee.

PP2-116 CITRULLINATION OF CXCL12 AFFECTS ITS INFLAMMATORY AND ANTI-HIV-1 ACTIVITY. Sofie Struyf, Samuel Noppen, Tamara Loos, Anneleen Mortier, Mieke Gouwy, Hannelien Verbeke, Karel Geboes, Dominique Schols, Jo Van Damme, and Paul Proost.

PP2-117* THE ROLE OF CM1 ON THE MONOCYTE OF RHEUMATOID ARTHRITIS VIA THE INDUCTION OF INFLAMMATORY CYTOKINES, TNF-α, IL-1 α/β, IFN-γ, AND PGE2. Seyeon Bae, Jae Seung Kang, Hye Min Kim, Hyung Gun Maeng, Joo Myoung Kong, Na Eun Lee and Wang Jae Lee.

PP2-118* METRONIDAZOLE-INDUCED PERTURBATIONS OF THE INTESTINAL MICROBIOTA INCREASE HOST SUSCEPTIBILITY TO CITROBACTER RODENTIUM-INDUCED COLITIS. Marta Wlodarska and B. Brett Finlay.

PP2-119 CONSTITUTIVELY ACTIVE STAT3 TRIGGERS THE DEVELOPMENT OF AUTOIMMUNE MYOCARDITIS. Annalisa Camporeale, Francesca Marino, Alessia Brero, Roberto Chiarle, Ole Jensen, Renzo Levi, Valeria Poli.

PP2-120 DIFFERENTIAL ROLE T HELPER 1, T HELPER 2 AND CD4+CD25+ REGULATORY T CELLS IN THE DEVELOPMENT OF COLLATERAL VESSELS FOLLOWING INDUCED ACUTE ISCHEMIA. Laura Pontecorvo, Eugenio Stabile, Leopoldo Laricchia-Robbio, Giuseppe Rosano, Andrea la Sala.

PP2-121 IDENTIFICATION OF GENES INVOLVED IN UNCONVENTIONAL PROTEIN SECRETION PATHWAYS. Helena Raquel, Catarina Moita, Nir Hacohen, Luis Moita.

PP2-122 ROLES OF THE PUTATIVE EPIGENETIC REGULATOR TET1 IN INNATE IMMUNE RESPONSES. Ana Neves-Costa, Luís Moita.

PP2-123 ROLE OF ADAM17 AND IL-6 TRANSSIGNALING IN INFLAMMATORY BOWEL DISEASE. Nina Adam, Jürgen Scheller, Philip Rosenstiel, Christian Sina, Olga Gavrilova, Stefan Rose-John, Athena Chalaris.

PP2-124 SUPEROXIDE-INDEPENDENT KYNURENINE SYNTHESIS IN HUMAN CHRONIC GRANULOMATOUS DISEASE: A 'RADICAL' DIFFERENCE BETWEEN MICE AND HUMANS. Kol A. Zarember, Suk See De Ravin, Debra Long-Priel, John I. Gallin, Douglas Kuhns, Harry L. Malech.

PP2-125 MECHANISMS OF NEUTROPHILS AND T-LYMPHOCYTE ACCUMULATION DURING EXPERIMENTAL PLEURAL INFECTION INDUCED BY MYCOBACTERIUM BOVIS BCG. Souza MC, Candea ALP, Menezes de Lima Junior O, Penido C, Costa MF, Henriques MG.

PP2-126* EGR-1 MEDIATED TNFa PRODUCTION IS ASSOCIATED WITH HEPATOPROTECTION AFTER ACUTE CARBON TETRACHLORIDE EXPOSURE IN MICE. Michele T. Pritchard, Jessica I. Cohen, Sanjoy Roychowdhury and Laura E. Nagy.

PP2-127 SYSTEMS BIOLOGY APPROACHES TO UNDERSTAND SEPSIS. Olga M. Pena, Christopher D. Fjell, Disha Raj, David Lynn, Robert Hancock.

PP2-128 DECREASED WHOLE BLOOD CYTOKINE PRODUCTION DURING A PHASE I TRIAL OF THE HISTONE DEACETYLASE INHIBITOR ITF2357. Tiziano Oldoni, Antonio Furlan, M. Valmen Monzani, Charles A. Dinarello.

PP2-129 ROLE OF LEUKAEMIA INHIBITORY FACTOR IN INFLAMMATORY ARTHRITIS – PRO- OR ANTI- INFLAMMATORY? Aradhana Upadhyay and Jalal A. Jazayeri.

LB-20 PRESENCE OF FOXP3HIIL-17+ T REGULATORY CELLS CONTRIBUTE TO GENDER DISPARITY OBSERVED IN A SPONTANEOUS MODEL OF CROHN'S DISEASE-LIKE ILEITIS. Rekha R. Garg, Luca Pastorelli, Theresa T. Pizarro.

LB-21* TNF CORRELATED WITH NUMBER OF INFAMMED CELLS IN RADICULAR CYSTS. Jurisic V, Jurisic M.

LB-22 EFFECTOR FUNCTIONS OF IL-17 IN COLLAGEN-INDUCED ARTHRITIS AND POTENT INHIBITION BY IFN-GAMMA. Hilde Kelchtermans, Evelien Schurgers, Lies Geboes, Tania Mitera, Jo Van Damme, Jacques Van Snick, Catherine Uyttenhove, Patrick Matthys.

LB-23 MODULATION OF INFLAMMATORY CYTOKINES DURING FOLLOW-UP OF PATIENTS UNDERGOING CARDIAC SURGERY. V. Dadorante, R. Rabossi, E. Toniato, A.L. Di Mele, D. D'Ettorre, M. Romagnoli, V. Villani, G. Draicchio, S. Martinotti, and G. Di Giammarco.

LB-24 IMMUNOLOGY CHANGES IN PATIENTS WITH SEBORRHOIC DERMATITIS. Polesko I.V., Butov Y.S., Malinovskya V.V.

LB-25 INVOLVEMENT OF HVEM IN OBESITY-INDUCED INFLAMMATORY RESPONSES. Ha-Jung Kim, Chu-Sook Kim, Teruo Kawada, and Rina-Yu.

A Inflammation and Cancer

SLBAW1-A C-MYC TRIGGERS MACROPHAGE ALTERNATIVE ACTIVATION AND CONTROLS MACROPHAGE ACTIVITY AND SURVIVAL IN TUMOUR. Pello OM, De Pizzol M, Mirolo M, Mantovani A, Locati M.

CBSS2-3 GRIM-19: A NOVEL GROWTH REGULATOR THAT INHIBITS STAT3 AND BEYOND. Dhan V. Kalvakolanu, Shreram C. Nallar, Peng Sun, Sudhakar Kalakonda.

CBSS2-4* IL-11 MEDIATED STAT3 ACTIVATION IN INFLAMMATION AND CANCER. Tracy Putoczki, Stefan Thiem, Andrew Jarnicki, Brent Mckenzie, Stefan Rose-John, Matthias Ernst.

CBSS2-5 ORIGIN, PHENOTYPE AND FUNCTION OF MONOCYTE/MACROPHAGE SUBSETS IN DISTINCT MAMMARY TUMOR MICROENVIRONMENTS. Kiavash Movahedi, Damya Laoui, Conny Gysemans, Geert Stangé, Jan Van den Bossche, Danny Pipeleers, Patrick De Baetselier, Jo A. Van Ginderachter.

*Travel Award Recipient

CBSS2-6 INTERFERON-ALPHA BOOSTS ANTI-TUMOR IMMUNITY THROUGH EFFECTS ON T CELLS AND DENDRITIC CELLS AND AUGMENTS THE CLINICAL EFFICACY OF REGULATORY T CELL DEPLETION. Shawna Wall, Suzanne Thibodeaux, Benjamin Daniel, Duane Jeansonne, Xiuhua Sun, Sara Ludwig, Dakshayani Lomada, Mark Kious, Tyler J. Curiel.

PP1-009* A TRANSGENIC ANIMAL MODEL FOR THE INVESTIGATION OF THE ROLE OF TYPE I INTERFERONS IN T LYMPHOCYTE BIOLOGY. Nadia Kavrochorianou, Maria Evangelidou, Michael Tovey, George Thyphronitis, Sylva Haralambous.

PP1-010 LOSS OF INOS EXPRESSION IN THE MOUSE RENAL CELL CARCINOMA RENCA CELL LINE IS MEDIATED BY MIR-146A AND CONFERS RESISTANCE TO MACROPHAGE-INDUCED APOPTOSIS. Michal A. Rahat, Christina Perske, Sharon Sheffy, Bernhard Hemmerlein, and Nitza Lahat.

PP1-011 ROLE OF INTERFERON-ACTIVATED MACROPHAGES IN ERADICATION OF HUMAN TUMOR CELLS BY INNATE IMMUNITY. Samuel Baron, Julie Horowitz, Joyce Poast, Angel Morrow, Samuel Fey, Joel Finbloom, Hana Schmeisser, Joseph Bekisz, and Kathryn Zoon.

PP1-012* CYTOKINE PROFILE OF SUCCESSFUL CANCER IMMUNOSURVEILLANCE MEDIATED BY TUMOR-SPECIFIC CD4+ T CELLS. Ole Audun Werner Haabeth, Kristina Berg Lorvik, Bjarne Bogen, and Alexandre Corthay.

PP1-013 INDUCTION OF CCL13 EXPRESSION IN SYNOVIAL FIBROBLASTS UNDERLINES A SIGNIFICANT ROLE OF ONCOSTATIN M IN RHEUMATOID ARTHRITIS. C. Hintzen, S. Quaiser, T. Pap, H. Hermanns.

PP1-014 CCL3-CCR5 AXIS REGULATES INTRATUMORAL ACCUMULATION OF LEUKOCYTES AND FIBROBLASTS, AND PROMOTES ANGIOGENESIS IN MURINE LUNG METASTASIS PROCESS. Yu Wu, Ying-Yi Li, Tomohisa Baba and Naofumi Mukaida.

PP1-015 COMPLEMENT C1Q SIGNALS DANCING OF TUMOR SUPPRESSOR WWOX/WOX1 ON CELL SURFACE FOR APOPTOSIS. Nan-Shan Chang.

PP1-016 SPECIFIC GENETIC ALTERATIONS IN THE GENE FOR IL-15 CAN ENHANCE ITS TRANSLATION EFFICIENCY IN CELLS THAT SHOW TRANSLATIONAL DOWN-REGULATION. Wu TG, Grewe CF, Idossa DW, DeWall MR, and Fleischmann WR Jr.

PP1-017 IFN-BETA PRO-APOPTOTIC AND ANTIPROLIFERATIVE ACTIVITY IS SUPERIOR TO IFN-ALPHA IN ADULT T-CELL LEUKEMIA: EX VIVO RESPONSE PREDICTS SURVIVAL. Johan Van Weyenbergh, Ricardo Khouri, Daniele Decanine, Kristof Theys, Koen Deforche, Aline Clara Silva, Lourdes Farre, Achilea Bittencourt, Anne-Mieke Vandamme.

PP1-018* UNCONTROLLED HERPES SIMPLEX VIRUS-1 REPLICATION DUE TO TYPE I IFN RECEPTOR DEFICIENCY RESULTS IN SELECTIVE DESTRUCTION OF LYMPHATIC VESSELS AND INHIBITION OF ANTIGEN PRESENTATION. Todd R. Wuest, Daniel J. J. Carr.

PP1-019 THE WILD TYPE RNASE L BUT NOT MUTANT LABORATORY AND NATURAL VARIANTS SUPPRESSES THE RNA BINDING PROTEIN, HUR: EFFECTS ON CYTOKINE 3'UTR. Latifa Al-Haj, Wijdan Al-Ahamdi, Maha Al-Ghamdi, Maher Al-Saif, Edward Hitti, Robert H. Silverman, and Khalid S. A. Khabar.

PP1-020* DISRUPTION OF EPIDERMAL SPECIFIC STAT3 EXPRESSION AND DELAYED SKIN TUMOR DEVELOPMENT IN HPV8 TRANSGENIC MICE. Marco De Andrea, Massimo Rittà, Manuela Landini, Cinzia Borgogna, Michele Mondini, Manuela Baccarini, Herbert Pfister, Gian Paolo Marcuzzi, Marisa Gariglio, Santo Landolfo.

PP1-022 CHARACTERIZATION OF GENE-TARGETED MURINE EMBRYONIC STEM CELLS EXPRESSING A STAT3-YFP ALLELE. Anne Schmitt, Andrea Küster, Rebekka Schneider, Martin Zenke, Valeria Poli, Gerhard Müller-Newen.

PP1-023 INTESTINAL INFLAMMATION IS COORDINATED BY THE METALLOPROTEASE ADAM17. Stefan Rose-John, Athena Chalaris, Nina Adam, Christian Sina, Philip Rosenstiel, Karina Reiss, Joanna Cichy, and Jürgen Scheller.

PP1-024 HIGH-DENSITY LIPOPROTEINS INHIBIT INFLAMMATORY PATHWAYS IN HUMAN MONOCYTES ACTIVATED UPON CHRONIC INFLAMMATORY CONDITIONS BY CELLULAR CONTACT WITH STIMULATED T CELLS. Danielle Burger, Lyssia Gruaz, and Jean-Michel Dayer.

PP1-025* INFECTION OF ENDOTHELIAL CELLS WITH KAPOSI'S SARCOMA-ASSOCIATED HERPESVIRUS SELECTIVELY INHIBITS RECRUITMENT OF FLOWING NEUTROPHILS IN AN INFLAMMATORY MODEL. Lynn M. Butler, Rachel L. Wheat, Hannah C. Jeffery, Gerard B. Nash and David J. Blackbourn.

PP1-026 REGULATION OF INNATE IMMUNITY AND INFLAMMATION BY THE MITOTIC KINASE PLK1 THROUGH INHIBITION OF IKKj ACTIVITY. Stéphanie Dabo, Malek Ahmadi Pour, Damien Vitour, Olivera Grubisha, Myriam Vilasco, Pierre-Olivier Vidalain, Yves Jacob, Frédéric Tangy, John Hiscott and Eliane F. Meurs.

PP1-027* MECHANISMS OF IRF5-MEDIATED APOPTOTIC CELL SIGNALING AND TUMOR SUPPRESSION. Guodong Hu, Lisong Yang, Justyna Korczeniewska and Betsy J. Barnes.

PP1-028* ULTRAVIOLET B-INDUCED ACTIVATION OF MELANOCYTES IS MEDIATED THROUGH INTERFERON-GAMMA SECRETED BY MACROPHAGES. M. Raza Zaidi, Edward De Fabo, Sean Davis, Cari Graff-Cherry, Teresa Hawley, Lionel Feigenbaum, Elaine Fuchs, Thomas Hornyak, Heinz Arnheiter, Giorgio Trinchieri, Frances Noonan, Paul Meltzer, Glenn Merlino.

PP1-029 MIGRATION AND INTERLEUKIN-8 RELEASE OF GRANULOCYTES BY SOLUBLE AND NANOVESICLE-ASSOCIATED CD30 FROM HODGKIN LYMPHOMA CELLS. Hinrich P. Hansen, Vijaya Lakshmi Simhadri, Andreas Engert and Elke Pogge von Strandmann.

PP1-030 PREDICTION OF OVERALL SURVIVAL THROUGH PRE-OPERATIVE BLOOD PLASMA OF COLORECTAL CANCER PATIENTS. Kazuko Uno1, Kiyotaka Okuno, Katsumi Yagi, Junji Hamuro.

PP1-032 THE EXPRESSION OF HIGH MOBILITY GROUP BOX 1 PROTEIN AND ITS RECEPTOR RAGE IN BREAST AND COLON CARCINOMAS. Nora Kostova, Stanislava Zlateva, Iva Ugrinova and Evdokia Pasheva.

PP1-033 EXPRESSION OF mRNA LEPTIN AND ITS RECEPTOR IN COLORECTAL CANCER. M.Stachowicz, Barbara Zubelewicz-Szkodziñska, Urszula Mazurek, Ewa Nowakowska-Zajdel, Ma³gorzata Muc-Wierzgoñ, Teresa Kokot .

PP1-034* NEUROPROTECTION BY ERYTHROPOIETIN AGAINST TAXANE INDUCED PERIPHERAL NEUROPATHY. Ilaria Cervellini,Carla Porretta-Serapiglia, Ezia Bello, Roberta Frapolli, Norberto Oggioni, Annalisa Canta, Cristina Meregalli, Raffaella Lombardi, Pietro Ghezzi, Giuseppe Lauria, Maurizio D'Incalci, Guido Cavaletti, Roberto Bianchi.

PP1-035 HYPOXIA-INDUCIBLE FACTOR 1á IS UPREGULATED BY ONCOSTATIN M VIA JAK/STAT3 AND THE MEK/ERK1/2 PATHWAY. Stefan Vollmer, Valérie Kappler, Jakub Kaczor, Daniela Flügel, Catherine Rolvering, Nobuyuki Kato, Thomas Kietzmann, Iris Behrmann, Claude Haan.

PP1-036* ROLE OF THE IKK-RELATED KINASES IN THE INDUCTION OF HIF-1ALPHA DURING HYPOXIA. Simon-Pierre Gravel, Darren E. Richard, Marc J. Servant.

PP1-037 SOCS1 HYPERMETHYLATION INCREASES TNF-ALPHA INDUCED APOPTOSIS IN A HUMAN HEPATOMA CELL LINE. Mehdi Yeganeh, Sheela Ramanathan, Chantal Leblanc and Subburaj Ilangumaran.

PP1-038 A CARCINOGENIC HETEROCYCLIC AMINE, 2-AMINO-1-METHYL-6-PHENYLIMIDAZOL[4,5-b]PYRIDINE (PhIP), ATTENUATES LIPOTEICHOIC ACID-STIMULATED TNF-a EXPRESSION. Jintaek Im, Hyung Shim Choi, Sun Kyung Kim, Sang Su Woo, Young Hee Ryu, Seok-Seong Kang, Cheol-Heui Yun, Seung Hyun Han.

PP1-039 FREQUENT EXPRESSION OF TRAIL-R2 IN HUMAN BREAST TUMORS REVEALED BY ANTIBODY PROTEOMICS TECHNOLOGY. Kazuya Nagano, Takuya Yamashita, Takayuki Okamura1, Takanobu Watanabe, Souichiro Kanasaki, Yasuhiro Abe, Haruhiko Kamada, Shin-ichi Tsunoda and Yasuo Tsutsumi.

PP1-041 CANCER HAZARD OF CARBON NANOTUBES: SIZE/SHAPE-DEPENDENT INDUCTION OF DNA DAMAGE AND INFLAMMATION. Kohei Yamashita, Yasuo Yoshioka, Hiroyuki Kayamuro, Tokuyuki Yoshida, Kazuma Higashisaka, Yasuhiro Abe1, Tomoaki Yoshikawa, Norio Itoh, Shin-ichi Tsunoda, Yasuo Tsutsumi.

PP1-042* THE ROLE OF CCL5/RANTES IN REGULATING NUTRIENT RECEPTOR TRAFFIKING, METABOLISM AND PROTEIN EXPRESSION IN ACTIVATED T CELLS. Olivia Chan, Thomas Murooka, Eleanor Fish.

PP1-043 TRANSGENIC MICE EXPRESSING HUMAN HERPESVIRUS 8 ENCODED VIRAL INTERLEUKIN-6 REVEAL FEATURES OF MULTICENTRIC CASTLEMAN'S DISEASE. Jan Suthaus, Christiane Stuhlman-Laiesz, Wolfram Klapper, Jürgen Scheller and Stefan Rose-John.

PP2-028 ANTIMICROBIAL CATHELICIDIN PEPTIDE CAP11 SUPPRESSES HMGB1 (HIGH MOBILITY GROUP BOX-1) RELEASE FROM LIPOPOLYSACCHARIDE-STIMULATED MONONUCLEAR PHAGOCYTES VIA THE PREVENTION OF NECROTIC CELL DEATH. Isao Nagaoka, Kentaro Shibusawa, Taisuke Murakami and Hiroshi Tamura.

LB-26 EPITHELIAL-MESENCHYMAL INTERACTION IN CANCER: THE ROLE OF CHEMOKINES. Barbora Dvorankova, Karel Smetana, Pavol Szabo, Vit Hajduch, Zdenek Cada, Hynek Strnad, Michal Kolar.

LB- 40 HEME OXYGENASE-1 PROTECTS AGAINST SEVERE SEPSIS VIA INHIBITION OF HEME-MEDIATED SENSITIZATION TO CELL DEATH AND RELEASE OF HMGB1. Rasmus Larsen, László Tokaji, Raffaella Gozzelino, Dolores Bonaparte, Moises M. Cavalcante, Angelo Chora, Silvia Cardoso, Gabriela Silva and Miguel P. Soares.

LB-45 SYSTEMS BIOLOGY APPROACH DEFINES THE MOLECULAR PHENOTYPE OF HUMAN TUMOR ASSOCIATED MONOCYTES/MACROPHAGE IN RENAL CELL CARCINOMA Manesh Chittezhath, Manprit Kaur Dhillon, Brendon Ang, Revathi Kamaraj, Henry Yang, Rajeev Singh, Alvin S.C. Wong and Subhra K Biswas

B Macrophages and Chronic Inflammation

CIS5-3 MACROPHAGE EFFECTOR FUNCTION IN ANTI-FILARIAL NEMATODE IMMUNITY IS INDEPENDENT OF ARGINASE 1, RELMA AND YM-1. Stephen Jenkins and Judith Allen.

CIS5-4 STABILIN-1 –MULTIFUNCTIONAL RECEPTOR LINKING ENDOCYTOSIS AND SECRETION IN MACROPHAGES. Julia Kzhyshkowska, Alexei Gratchev, Liis Krusell, Srinivas Mamidi, Vladimir Riabov, Jingjing Zhang, Gail Workman, E. Helene Sage, Sergij Goerdt.

CIS5-5 EXPRESSION OF THE INHIBITORY CD200 RECEPTOR IS ASSOCIATED WITH ALTERNATIVE MACROPHAGE ACTIVATION. Nathalie Koning, Marco van Eijk, Walter Pouwels, Michael S.M. Brouwer, David Voehringer, Inge Huitinga, Robert M. Hoek, Geert Raes, and Jörg Hamann.

CIS5-6 CONTROL OF RSV-INDUCED LUNG INJURY BY ALTERNATIVELY ACTIVATED MACROPHAGES IS IL-4RALPHA-, TLR4-, AND IFN-BETA-DEPENDENT. Jorge C. Blanco, Kari A. Shirley, Lioubov M. Pletneva, Adam C. Puche, Achsah D. Keegan, Gregory A. Prince, Stefanie N. Vogel.

PP2-186* THE ROLE OF ACTIVATING TRANSCRIPTION FACTOR 3 IN STRESS-INDUCED MACROPHAGE APOPTOSIS AND SENESCENCE. Kyung-Ho Kim, Eunhwa Sohn, Dong Kwon Rhee, Suhkneung Pyo.

PP2-187* APOPTOSIS RESISTANCE IN HIV-EXPOSED MONOCYTES IS MODULATED BY INTERFERON GAMMA AND CCR5-INDUCIBLE p53 EXPRESSION. Bethsebah N. Gekonge and Luis J. Montaner.

PP2-189 THE INFLAMMASOME-MEDIATED CASPASE-1 ACTIVATION CONTROLS ADIPOCYTE DIFFERENTIATION AND INSULIN SENSITIVITY. Rinke Stienstra, Leo A.B. Joosten, Tim Koenen, Jos W.M. van der Meer, Cees J. Tack, Thirumala Kanneganti, Mihai G. Netea.

*Travel Award Recipient

PP2-190 A FUNCTIONAL ROLE FOR DEATH RECEPTOR-3 IN ARTHRITIS. Anwen S. Williams, Melanie J. Bull, Zarabeth Mecklenburgh, Claudia J. Calder, Jason P. Twohig, Carole Elford, Bronwen Evans, Aymen Al Shamkhani , Eddie Wang.

PP2-191 VEGF AS A REGULATOR OF MACROPHAGE-INDUCED INFLAMMATION. Melissa L. Petreaca, Manuela Martins-Green.

PP2-192 INDUCING REGULATORY MACROPHAGES THROUGH DISSECTING THE SPECTRUM OF MACROPHAGE ACTIVATION KINETICALLY AND GENETICALLY. M Sohel Mia, Roham Parsa and Robert A. Harris.

PP2-193 DIFFERENTIATION, ACTIVATION AND FUNCTION OF CD11b+Ly6C+ TNF/iNOS-PRODUCING DENDRITIC CELLS DURING PARASITIC INFECTION. Tom Bosschaerts, Martin Guilliams, Benoit Stijlemans, Yannick Morias, Daniel Engel, Frank Tacke, Bucala Richard, Thierry VandenDriessche, Marinee K. Chuah, Patrick De Baetselier, Alain Beschin.

PP2-194 NATIVE LOW-DENSITY LIPOPROTEIN UPTAKE BY MACROPHAGE COLONY-STIMULATING FACTOR DIFFERENTIATED MACROPHAGES IS MEDIATED BY MACROPINOCYTOSIS AND MICROPINOCYTOSIS. Howard S. Kruth, Joshua J. Anzinger, Janet Chang, Francisco J. Leyva, Bum-Chan Park, and Lois E. Greene.

PP2-195* CONTRIBUTION OF HEPATIC MACROPHAGES TO ETHANOL-INDUCED APOPTOSIS OF NON-PARENCHYMAL CELLS IN MOUSE LIVER. Jessica I. Cohen, Sanjoy Roychowdhury, Megan R. McMullen, Abram B. Stavitsky, and Laura E. Nagy.

LB-27 MACROPHAGE RESPONSES TO INTERLEUKIN-17 ARE REGULATED BY LOCATION AND INFLAMMATION. Jobert G. Barin, Farhan Quader, G. Christian Baldeviano, Monica V. Talor, Ping Chen, Dongfang Zheng, Daniela Cihákóvá, Noel R. Rose.

LB-28 GLUCOCORTICOIDS INDUCE COORDINATED EXPRESSION OF MS4A GENES IN HUMAN MACROPHAGES. Maria De Pizzol, Oscar M Pello, Giovanna Mantovani, Massimiliano Mirolo, Alberto Mantovani and Massimo Locati.

A Neutrophil Biology

SLBAW1-B GRANULE EXOCYTOSIS CONTRIBUTES TO TNF-ALPHA AND PAF-INDUCED PRIMING IN HUMAN NEUTROPHILS. Silvia M. Uriarte, Madhavi J. Rane, Gregory C. Luerman, Junyi Le, Richard A. Ward and Kenneth R. McLeish.

CBSS6-2 INTER-KINGDOM SIGNALLING: A QUORUM-SENSING MOLECULE OF PSEUDOMONAS AERUGINOSA ACTIVATES HUMAN POLYMORPHONUCLEAR NEUTROPHILS (PMN). Gertrud Maria Hänsch, Ursula Obst.

CBSS6-3 FUNCTIONAL COOPERATION BETWEEN Fc GAMMA RIIa AND Fc GAMMA RIIIb ON HUMAN NEUTROPHILS. Louis Marois, Guillaume Paré, Myriam Vaillancourt, Emmanuelle Rollet-Labelle and Paul H. Naccache.

CBSS6-4 A CRITICAL ROLE OF NITRIC OXIDE IN THE RESOLUTION OF INFLAMMATION. Yoshiro Kobayashi, Takehiko Shibata, and Kisaburo Nagata.

CBSS6-5 LEISHMANIA PROMASTIGOTES INDUCE THE FORMATION OF NEUTROPHIL EXTRACELLULAR TRAPS. Christelle Gabriel, Denis Girard, W.Robert McMaster, Albert Descoteaux.

PP1-154 NEUTROPHIL MECHANOSENSING. Jonathan S. Reichner, Patrick W. Oakes, Dipan C. Patel, Nicole A. Morin, Daniel P. Zitterbart, Ben Fabry, and Jay X. Tang.

PP1-155 FIBRINOGEN AND NEUTROPHIL RECRUITMENT. Vanda Vitorino de Almeida, H. S. Rosário and C. Saldanha.

PP1-157* SCAVENGER RECEPTOR MARCO DAMPENS ACUTE INFLAMMATION AND REDUCES SURVIVAL IN A MOUSE MODEL OF INFLUENZA INFECTION: A HARMFUL EFFECT OF MACROPHAGE TIDINESS? Sanjukta Ghosh, Lester Kobzik.

PP1-159 HEAT SHOCK PROTEINS 70 IN SURGICAL STRESS: THORACOTOMY VS HERNIORRHAPHY. R.Ramos, E. Dulin, MJ Sánchez, MC Guisasola.

PP1-160 IN VITRO AND IN VIVO PRO-INFLAMMATORY ACTIVITIES OF TITANIUM DIOXIDE (TIO2) NANOPARTICLES. David M Garcês Gonçalves, Denis Girard.

PP1-161 EXPRESSION OF CD99 AND CD47 AS PREDICTIVE MARKERS OF NEUTROPHIL TRANSENDOTHELIAL MIGRATION. Susan Kennedy, Adnan Raza, Belinda Maher, Alfred E. Wood, R.William G. Watson.

PP1-162 MONOSODIUM URATE CRYSTALS INHIBIT THE PHOSPHATASE ACTIVITY OF SHP-1 IN HUMAN NEUTROPHILS. Oana Popa-Nita, Sophie Proulx, Emmanuelle Rollet-Labelle, Paul H. Naccache.

PP1-163 ACTIVATION OF CIRCULATING NEUTROPHILS VIA THEIR Fc GAMMA RECEPTORS : POSSIBLE INVOLVEMENT IN RHEUMATOID ARTHRITIS. Emmanuelle Rollet-Labelle, Louis Marois, Arpita Chakravarti, Patrice E. Poubelle, Paul H. Naccache.

PP1-164 HUMAN SOLUBLE CXCR2 IN HEALTH AND DISEASE. Sviatlana Akalovich, Kanstantsin Katlinski, Yuliya Katlinskaya, Tatyana Doroshenko, Nikolai N. Voitenok.

PP1-165* ENZYME-LINKED IMMUNOASSAYS DIFFERENTIALLY RECOGNIZING GLYCOSYLATED AND DEGLYCOSYLATED FORMS OF SOLUBLE HUMAN CXCR2. Yuliya Katlinskaya, Sviatlana Akalovich, Kanstantsin Katlinski, Nikolai N. Voitenok.

PP1-166 REGULATION OF Fc GAMMA RIIa FUNCTIONS BY SRC HOMOLOGY 2-CONTAINING INOSITOL 5-PHOSPHATASE 1 ON HUMAN NEUTROPHILS. Myriam Vaillancourt, Louis Marois, Emmanuelle Rollet-Labelle, Paul H. Naccache.

PP1-167 EXOCYTOSIS OF SERINE PROTEASES AND MODULATION OF NEUTROPHIL INFLAMMATORY RESPONSE IN THE PRESENCE OF LEISHMANIA INFANTUM. Cláudia Marques, Armanda Rodrigues, Gabriela Santos-Gomes.

PP1-169 DISPARATE ROLES OF INTRACELLULAR AND EXTRACELLULAR NUCLEAR FACTOR ERYTHROID 2 (NF-E2) IN REGULATION OF NEUTROPHIL APOPTOSIS. Paul Johnson, Shunying Jin, Silvia Uriarte, Gregory C. Luerman, Alex B. Lentsch, Madhavi J. Rane.

*Travel Award Recipient

PP1-170 IMPACT OF THE PHOSPHATIDYLINOSITOL 3-KINASE (PI3K) PATHWAY ON CYTOKINE GENERATION BY HUMAN NEUTROPHILS. Carl F. Fortin and Patrick P. McDonald.

B New T-Helper Subsets

PP1-108 NOVEL gd T CELL SUBSETS WITH DISTINCT FUNCTIONS IN IMMUNITY TO INFECTION AND TUMOURS. A. deBarros, J. Ribot, M. Chaves-Ferreira, F. d¡¦Orey, D. J. Pang, J. F. Neves, J. Borst, A. C. Hayday, D. J. Pennington, B. Silva-Santos.

PP1-109 PRO-INFLAMMATORY TH17 CYTOKINES STIMULATE THE EXPRESSION OF NOVEL IL-1 CYTOKINES IN VITRO AND IN VIVO : IMPLICATIONS IN PSORIASIS PATHOGENESIS. Yijun Carrier, Hak-Ling Ma, Lee Napierata, Clayton Small, Margot O'Toole, Deborah A. Young, Lynette A. Fouser, Cheryl Nickerson Nutter, Mary Collins, Kyri Dunussi-Joannopoulos, Quintus G. Medley.

PP1-110 IL-6-INDEPENDENT ARTHRITIS AND TH17 DIFFERENTIATION IN MICE. Satoshi Ikeda.

PP1-111 REGULATION OF IL-4R SIGNALING IN TH17 CELLS. Laura A. Cooney, Sujata Sarkar, David A. Fox.

PP1-112 IL-6 IS REQUIRED FOR THE GENERATION OF T FOLLICULAR HELPER CD4 T CELLS DURING TOXOPLASMA GONDII INFECTION. Jonathan Silver, Jason Stumhofer and Christopher Hunter.

PP1-113 IN VITRO GENERATED TH17 CELLS MAINTAIN CYTOKINE PROFILE IN WILD TYPE BUT NOT IN LYMPHOPENIC HOSTS IN EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS. Monica Leung.

A Pathogen Manipulation of Cytokine Responses

CIS3-3 INHIBITION OF TYPE I INTERFERON TRANSCRIPTION BY IRF3/7 SUMOYLATION,. Keiko Ozato, Tsung-Hsien Chang, Toru Kubota, Mayumi Matsuoka, Mike Bray, Steven Jones. .

CIS3-4 INTERFERON AND INFLUENZA VIRUSES: THE YIN AND YANG OF SURVIVAL. Danlin Jia, Renee Chan, Malik Peiris, John Nicholls, Eleanor N. Fish.

CIS3-5 MICROBIAL IMMUNE EVASION THROUGH EXPLOITATION OF MACROPHAGE PATTERN-RECOGNITION RECEPTORS. George Hajishengallis, Shuang Liang, Min Wang, and Kathy Triantafilou.

CIS3-6 A NOVEL FUNCTION OF THE CROHN'S DISEASE–ASSOCIATED NOD2 MUTANT 1007FS IN THE REGULATION OF HUMAN IL10 GENE TRANSCRIPTION. Xiaojing Ma, Eiichiro Noguchi, Yoichiro Homma, Xiaoyan Kang, Mihai G Netea.

CIS3-7 V PROTEIN-MEDIATED BLOCK OF MX TRANSCRIPTION IS ESSENTIAL FOR MORBILLIVIRUS VIRULENCE. Nicholas Svitek, Roberto Cattaneo, and Veronika von Messling.

CIS3-8 C1q ENHANCES PHAGOCYTOSIS OF MYCOBACTERIUM AVIUM THROUGH A PERTUSSIS TOXIN SENSITIVE PATHWAY. Kristen Ploetze, Michael Kuelbs, and Suzanne S. Bohlson.

PP1-072 HIV-1 TAT-INDUCED SUPPRESSOR OF CYTOKINE SIGNALING 3 INHIBITS INTERFERON-β SIGNALING IN MACROPHAGES: IMPLICATIONS FOR HIV-ASSOCIATED DEMENTIA. Lisa Nowoslawski Akhtar, Hongwei Qin, Janice E. Clements, and Etty N. Benveniste.

PP1-073* ROLE OF MYELOID RELATED PROTEINS 8/14 IN THE INNATE IMMUNE CONTROL OF LEISHMANIASIS. Irazú Contreras, Marina T. Shio, Philippe A. Tessier and Martin Olivier.

PP1-074 INHIBITION OF TYPE III INTERFERON ACTIVITY BY POXVIRUS IMMUNOMODULATORY PROTEINS. Prasanthi Bandi, Nicole E. Pagliaccetti, Michael D. Robek.

PP1-075* MOUSE STAT2 IS A DENGUE VIRUS HOST RESTRICTION FACTOR. Joseph Ashour, Maudry Laurent-Rolle, Courtney Ray Plumlee, Dabeiba Bernal, Ana Fernandez-Sesma, Christian Schindler, Adolfo García-Sastre.

PP1-076 IMMUNOSUPPRESSIVE IL-10 PRODUCTION BY NATURAL KILLER CELLS IS ELICITED BY SYSTEMIC BUT NOT LOCAL INFECTIONS. Georgia Perona-Wright, Rajat Madan, Katja Mohrs, Frank M. Szaba, Lawrence W. Kummer, Christopher L. Karp, Stephen T. Smiley, Lawrence L. Johnson, and Markus Mohrs.

PP1-077 INCREASED IL-7 AVAILABILITY COULD OVERCOME THE ANTI-PROLIFERATIVE CAPACITY OF TYPE-I IFN IN NAÏVE CD8 T CELLS DURING HIV INFECTION. Christopher Wilhelm, Michael Proschan, Friesen Travis, Bishop Hague, Gregg Roby, Catherine Rehm, Clifford Lane and Marta Catalfamo.

PP1-078 MECHANISMS UNDERLYING HIV-1 SUPPRESSION OF IFNƒ× SIGNALING PATHWAYS: A ROLE FOR TRANSACTIVATOR TAT IN THE INDUCTION OF SOCS-2 IN PRIMARY HUMAN BLOOD MONOCYTES. James C.B. Li, Sherman M. Cheng, Howard C.H. Yim, Davy CW Lee, and Allan S.Y. Lau.

PP1-079 THE ROLE OF CCR5 IN VACCINIA VIRUS PATHOGENESIS. Ramtin, Rahbar & Eleanor N. Fish.

PP1-080 INDUCTION OF INFLAMMATORY AND Th1 CYTOKINES IN PIGS INOCULATED WITH THE LOW VIRULENT ASFV/NH/P68 (NHV). S. Gil, N. Sepúlveda, M.F. Potier, A. Leitão, V. Michaud, E. Albina, and C. Martins.

PP1-081 INDUCTION OF INTERLEUKIN-8 EXPRESSION BY HUMAN CYTOMEGALOVIRUS UL76 PROTEIN. H. Costa1, R. Nascimento, J. Sinclair, RME Parkhouse.

PP1-082 INTERLEUKIN-1 TYPE I RECEPTOR SIGNALING TRIGGERS THE INFLAMMATORY RESPONSE IN COXSACKIEVIRUS B3 INFECTION. Fabienne Rehren, Oliver Dittrich-Breiholz, Michael Kracht, Albert Heim.

PP1-083 ABERRANT TYPE I IFN PATHWAY RESPONSE TO VIRAL INFECTION IN CHRONIC FATIGUE SYNDROME (CFS). Judy A. Mikovits, Kathryn S. Hagen, Daniel L. Peterson, Michael Dean, and Vincent C. Lombardi.

PP1-084 PARASITE KILLING BY AN EFFECTOR MONOCYTE SUBSET DURING MURINE CUTANEOUS LEISHMANIASIS. Ricardo Goncalves, Xia Zhang, and David M. Mosser.

*Travel Award Recipient

PP1-085 AGING EXACERBATES CYTOKINE RESPONSE AFTER PULMONARY INFECTION. Elizabeth J. Kovacs, Cory Deburghgraeve, Eva L. Murdoch, Vanessa Nomellini, and Jessica Palmer.

PP1-086 REGULATION OF HUMAN ENDOGENOUS RETROVIRUSES OF THE W FAMILY BY TYPE III INTERFERON ë2 AND HIV IN ASTROCYTES. Alessandra Mei, Giuseppe Mameli, Caterina Serra, Luciana Poddighe, Elena Uleri and Antonina Dolei.

PP1-087 CYTOKINES AND CHEMOKINES EXPRESSION IN AVIAN CELLS INFECTED WITH SALMONELLA ENTERICA. Ahmed M Setta, Michael A Jones and Paul A Barrow.

PP1-088* CASPASE-12 DEFICIENCY RESULTS IN HYPERINFLAMMATORY RESPONSES TO LETHAL MALARIA. Jenny Miu, Maya Saleh, Mary M. Stevenson.

PP1-089 5'-TRI-PHOSPHORYLATED ADENOVIRUS VIRAL ACCESSORY RNAs POTENTLY INDUCE INTERFERON IN A PROTEIN KINASE R-DEPENDANT MANNER. Ronald G. Jubin, Diane Vy, Doranelly Koltchev, Sidney Pestka.

PP1-090 HTLV-1 EVADES TYPE I IFN SIGNALING BY INDUCING THE SUPPRESSOR OF CYTOKINE SIGNALING : « SOCS-1 ». Stephanie Oliere, Meztli Arguello, Thi Lien-Anh Nguyen, Edouardo Hernandez, Agnes Lezin and John Hiscott.

PP1-091* PLASMACYTOID DENDRITIC CELL INTERACTION WITH HIV-INFECTED T CELLS: LIVE CELL NIBBLING VS CELL-CELL FUSION. Evan S. Jacobs, Thaddeus C. George, Sukhwinder Singh, Richard Wnek, Paul Fischer, Shila K. Nordone, Gregg A. Dean, and Patricia Fitzgerald-Bocarsly.

PP1-093 HEPATITIS C VIRUS NS3/4A PROTEIN DECREASES INTERFERON-ALFA PRODUCTION IN HEK293 CELLS. Maarit Sillanpää , Pasi Kaukinen , Krister Melén and Ilkka Julkunen.

PP1-094* HEPATITIS B VIRUS POLYMERASE INTERFERES WITH INTERFERON-â INDUCTION. Shiyan Yu, Jieliang Chen, Min Wu, Hui Chen, Zhenghong Yuan.

PP1-095 HIV TAT MODULATION OF IFN-g-INDUCED EXPRESSION OF AUTOPHAGY-ASSOCIATED GENES IN PRIMARY HUMAN BLOOD MACROPHAGES. K.Y. Au, James C.B. Li, Howard C.H. Yim, J.W.Fang, Allan S.Y. Lau.

PP1-096 MYCOBACTERIUM TUBERCULOSIS INDUCES A BYSTANDER EFFECT ON DC DIFFERENTIATION THROUGH THE RELEASE OF IL-10. Maria Elena Remoli, Elena Giacomini, Elisa Petruccioli, Valerie Gafa, Martina Severa, Elisabetta Iona, Maria Cristina Gagliardi, Richard Pine, Robero Nisini, Eliana Marina Coccia.

PP1-097 DENGUE VIRUS CORE PROTEIN MEDIATES REDUCTION OF ANTIVIRAL ACTIVITY IN HUH7 CELLS. Corey A. Balinsky, Hana Schmeisser and Kathryn C. Zoon.

PP1-099* THE ROLE OF IMMUNOGLOBULIN E ANTIBODIES IN PROTECTION AGAINST PLASMODIUM FALCIPARUM. Reem K. Mohamed, Muntaser E. Ibrahim, Ibrahim M. Elhassan.

PP1-100* A ROLE FOR STAT3 IN IL-10 DOWNREGULATION OF IFN-ã-INDUCED MHC CLASS II MOLECULE EXPRESSION ON PRIMARY HUMAN BLOOD MACROPHAGES. Lally L.Y. Chan, Benny K.W. Cheung, Allan S.Y. Lau.

PP1-101 IL-1 FAMILY MEMBER 7 IS A FUNDAMENTAL INHIBITOR OF INNATE IMMUNITY. Marcel F Nold, Claudia A Nold-Petry, Jarod A Zepp, Philip Bufler and Charles A Dinarello.

PP1-102 HBsAg SELECTIVELY INHIBITED IL-12 PRODUCTION BY INTERFERING WITH TLR2-INDUCED SIGNALING PATHWAY. Yunwen Hu, Zhiao Chen, Yuming Chen, Zhenghong Yuan.

PP1-103 INDUCTION OF HELMINTH-SPECIFIC IL-17 BY DENDRITIC CELLS EXPOSED SIMULTANEOUSLY TO SCHISTOSOME AND BACTERIAL ANTIGENS. Rachel J. Lundie, Georgia Perona-Wright, Stephen J. Jenkins and Andrew S. MacDonald.

PP1-104* A NOVEL SOLUBLE PROTEIN WHICH AFFECTS CELL SUSCEPTIBILITY TO A BROAD RANGE OF VIRAL INFECTIONS. Erin Rogers, Raymond Alverez, Anna Vyakarnam, Eleanor Fish.

LB-43 HEME SENSITIZATION TO TNF-MEDIATED PROGRAMMED CELL DEATH DICTATES THE OUTCOME OF PLASMODIUM INFECTION IN MICE. R. Gozzelino, E. Seixas, Â. Chora, A. Ferreira, G. Silva, R. Larsen, S. Rebelo, C. Penido, RN. Smith, A. Coutinho & MP. Soares.

B Pattern Recognition Receptors & Inflammation

PP1-044* INHIBITION BY PROXY: LPS-DRIVEN B CELL RESPONSES IN RP105-DEFICIENT MICE. Jessica L. Allen, Senad Divanovic, David Rawlings, Fred Finkelman, Christopher L. Karp.

PP1-045 CYSLT1 EXPRESSION AND FUNCTION ARE DOWNREGULATED DURING DENDRITIC CELL MATURATION WITH ZYMOSAN: ROLE OF IL-10 AND PROSTAGLANDINS. Maryse Thivierge, Jana Stankova, and Marek Rola-Pleszczynski.

PP1-046 ORTHOGONAL SCREENS FOR INNATE IMMUNE SENSORS. Tilmann Burckstummer, Christoph Baumann, Stephan Bluml, Evelyn Dixit, Gerhard Durnberger, Cristina Melinte, Melanie Planyavsky, Martin Bilban, Jacques Colinge, Keiryn L Bennett, Giulio Superti-Furga.

PP1-047 ASSOCIATION BETWEEN MYCOBACTERIUM LEPRAE INFECTION AND NOD-LIKE RECEPTOR PROTEINS (NLR) SIGNAL PATHWAY. Tae Jin Kang, Geum Seon Lee, Se-Kon Kim, Gue-Tae Chae.

PP1-048 IL-17A SYNERGISTICALLY INCREASES TOLL-LIKE RECEPTOR2/4 -DEPENDENT IL-8 EXPRESSION IN HUMAN CYSTIC FIBROSIS BRONCHIAL EPITHELIAL CELLS. Tsuyoshi Shuto, Shota Mizunoe, Shingo Suzuki, Mary Ann Suico, Tomoaki Koga, Takashi Sato, Dieter C. Gruenert and Hirofumi Kai.

PP1-049 LIPOPOLYSACCHARIDE DECREASES SIGIRR GENE EXPRESSION POSSIBLY BY SUPPRESSING SP1 VIA TLR4-P38 MAP KINASE PATHWAY. Keiko Ueno-Shuto, Tsuyoshi Shuto, Kosuke Kato, Hiromichi Sakai, Tomomi Ono, Mary Ann Suico, Yuji Uchida, Naofumi Tokutomi and Hirofumi Kai.

PP1-050 CURCUMIN DECREASES TOLL-LIKE RECEPTOR 2 GENE EXPRESSION AND FUNCTION IN HUMAN MONOCYTIC THP-1 AND NEUTROPHILIC HL-60 CELLS. Tomomi Ono, Tsuyoshi Shuto, Yuko Ohira, Mary Ann Suico, Tomoaki Koga, Takashi Sato, Keizo Sato and Hirofumi Kai.

*Travel Award Recipient

PP1-051 THE TLR-INDEPENDENT DNA RECOGNITION PATHWAY IN MURINE MACROPHAGES: LIGAND FEATURES AND MOLECULAR SIGNATURE. Evren Karayel, Tilmann Bürckstümmer, Martin Bilban, Gerhard Dürnberger, Stefan Weitzer, Javier Martinez and Giulio Superti-Furga.

PP1-052 TRIL IS A NOVEL MODULATOR OF TLR4 WITH ENRICHED EXPRESSION IN THE BRAIN. Thaddeus Carlson, Susan Carpenter, Ying Gao, Karen Percival, Scott H. Schelling, Amha G. Hewet, Wen Kuang, Padma Reddy, Debra G. Goodwin, Cheryl Nickerson-Nutter, Mayra Senices, Deborah Young, Leila Bradley, Lih-Ling Lin, James D. Clark, Ai.

PP1-053 CREATION OF A MUTANT IFN-α8 WITH ENHANCED ANTI-HCV ACTIVITY USING THE PHAGE DISPLAY TECHNIQUE. Tomoyuki Kawara , Yasuhiro Abe , Haruhiko Kamada , Shin-ichi Tsunoda , Masuo Kondoh , Yasuo Tsutsumi and Kiyohito Yagi.

PP1-054* HUMAN DEAD-BOX PROTEIN 3 ENHANCES PATTERN-RECOGNITION RECEPTOR -INDUCED IFN-BETA PRODUCTION AND IS A TARGET FOR VIRAL MANIPULATION. Martina Schroeder, Marcin Baran, Andrew Bowie.

PP1-055 A TLR9-AGONIST ADJUVANT INDUCES CELLULAR MEMORY IN RESPONSE TO PNEUMOCOCCAL CONJUGATE VACCINE IN HIV-INFECTED ADULTS. Rasmus Offersen, Ole S. Søgaard, Jesper Melchjorsen, Martin Tolstrup, Lars Østergaard.

PP1-056 NOD-LIKE RECEPTORS - PHAGOLYSOSOMAL COMPARTMENTS AND PAMP RECOGNITION. Andrea Rittger, Jenny Schröder, Andreas Till, Philip Rosenstiel, Paul Saftig.

PP1-058 CPG OLIGODEOXYNUCLEOTIDE AND DOUBLE-STRANDED RNA (POLY I:C) SYNERGIZE THE EXPRESSION OF INOS, PROINFLAMMATORY CYTOKINE, AND CHEMOKINE GENES IN CHICKEN PERIPHERAL BLOOD MONOCYTES. Haiqi He, Kathryn M. MacKinnon, Kenneth J. Genovese, and Michael H. Kogut.

PP1-059 CASPASE-1 IS PROTECTIVE DURING TRAUMA AND HEMORRHAGIC SHOCK IN MICE. Christoph L Menzel, Qian Sun, Hans-Christoph Pape, Timothy R Billiar, Melanie J Scott.

LB-29 HUMAN MILK CONTAINS AND MODULATES THE EXPRESSION OF THE SOLUBLE PATTERN RECOGNITION RECEPTOR PTX3. S. Jaillon, C. Beauvillain, P. Jeannin, C. Garlanda1, B. Bottazzi, P. Descamps, A. Mantovani, Y. Delneste.

LB-30 CONSTITUTIVE PHOSPHORYLATION OF TBK1 AND ENHANCED TLR3-DEPENDENT IFN-b PRODUCTION IN THE ABSENCE OF SHIP-1. Joan Ní Gabhann, Nadia Ben Larbi, Rowan Higgs, Kiva Brennan, Jacqueline E. Damen, Gerald Krystal and Caroline A. Jefferies.

LB-31 EXPRESSION PATTERNS OF HUMAN INTERFERON-ALPHA AND INTERFERON-LAMBDA SUBTYPES BY MONOCYTES, DENDRITIC CELLS AND B CELLS. Viraj P. Mane, Srikant Bykadi, Maria Navarro, Ronald Jubin and Ronald L. Rabin.

A Recent Advances

SLBAW1-C HEIGHTENED ACTIVATION OF PLASMACYTOID DENDRITIC CELLS AND INCREASED NK ACTIVITY IN HIV-1 EXPOSED, UNINFECTED INTRA-VENOUS DRUG USERS. Costin Tomescu, Shaheed A. Abdulhaqq, David S. Metzger, Angela Kapalko, Karam C. Mounzer, Vernon C. Maino, Luis J. Montaner.

SLBAW2-A A DIFFUSION BARRIER IN THE PLASMA MEMBRANE DURING THE CLOSURE STAGE OF MACROPINOCYTOSIS. Timothy P. Welliver, Joel A. Swanson.

CSS2-1* THYMIC CD70-CD27 SIGNALS PROMOTE THE DIFFERENTIATION OF AB AND GD T CELL SUBSETS. Julie C. Ribot, J. Coquet, A. Barros, V. Peperzak, D. J. Pang, J. F. Neves, A. C. Hayday, D. J. Pennington, J. Borst, B. Silva-Santos.

CSS2-2 CHARACTERIZATION OF A NEW POPULATION OF CD4+ INNATE SPLEEN CELLS THAT PRODUCE IL-22 DURING INFLAMMATORY PROCESSES. Laure Dumoutier, Ciriana Orabona, Magali de Heusch and Jean-Christophe Renauld.

CSS2-3 CASPASE-8 REGULATES CELLULAR RESPONSE TO PATTERN RECOGNITION RECEPTORS AND PREVENTS SPONTANEOUS TRIGGERING OF CHRONIC INFLAMMATION BY THEIR ENDOGENOUS ACTIVATORS. Andrew Kovalenko, Akhil Rajput, Jin-Chul Kim, Tae-Bong Kang, Konstantin Bogdanov, Oliver Dittrich-Breiholz, Michael Kracht, Ori Brenner and David WallachCaspase-8 regulates cellular response to pattern recognition receptors and prevents spontaneous trigge.

CSS2-4 TGFβ1 SIGNALS TIAF1 SELF-ASSOCIATION, AMYLOID SUPERINDUCTION AND APOPTOSIS. Nan-Shan Chang.

CSS2-5 GENETIC VARIANTS AND DISEASE-ASSOCIATED FACTORS CONTRIBUTE TO IRF-5 EXPRESSION IN PRIMARY BLOOD CELLS OF SLE PATIENTS. Betsy J. Barnes.

CSS2-6 ACUTE T CELL LEUKEMIA: AN IN VIVO STUGGLE BETWEEN HTLV-1-PRODUCTION AND TYPE 1 INTERFERON PRODUCTION. Francis Ruscetti, Cari Petrow-Sadowski, John Janik, Ying Huang, Daniel Bertolette, John Morris, Thomas Waldmann, and Kathryn Jones.

CSS2-7* T CELL RECEPTOR AGONISTS AND TUMOR BIOMARKERS FOR GAMMA-DELTA T-CELL-BASED IMMUNOTHERAPY OF LYMPHOMAS AND LEUKEMIAS. Daniel V. Correia, Anita Q. Gomes, F. d'Orey, Ana R. Grosso, Telma Lança, Bruno A. Cardoso, Cristina Ferreira, João T. Barata, and Bruno Silva-Santos.

CSS2-8 INTRACELLULAR INHIBITORS OF CYSTEINE CATHEPSINS IN ACTIVATED MACROPHAGES. Katarina Maher, Špela Konjar, Slavko Ceru, Matthew Bogyo, Eva Cerovnik, Boris Turk and Nataša Kopitar-Jerala.

PP1-115 APPLICABILITY OF NITRIC OXIDE PLATFORM TO SCREENING THE CYTOKINE-INDUCING ACTIVITY OF DRUGS. Zdenìk Zídek, Antonín Holý, Eva Kmoníèková.

PP1-116 ACTIVATION OF IFN-g SECRETION BY Ca2+-ATPase INHIBITORS THAPSIGARGIN AND TRILOBOLIDE. Eva Kmonícková, Juraj Harmatha, Karel Vokác, Petra Kostecká, Zdenek Zídek.

PP1-119 THE EVALUATION OF IMMUNOLOGICAL SAFETY OF THE COMBIHIVVAC CANDIDATE VACCINE AGANST HIV-1. S.G. Gamaley, G.M. Sysoeva, E.D. Danilenko, V.I. Masycheva, L.I. Karpenko.

*Travel Award Recipient

PP1-120 TNFR1-SELECTIVITY IMPROVEMENT OF MUTANT TNF WITH ANTAGONISTIC ACTIVITY. Tetsuya Nomura, Yasuhiro Abe, Masaki Inoue, Yasuo Yoshioka, Hiroyuki Kayamuro, Yohei Mukai, Madoka Taniai, Tsunetaka Ohta, Shinsaku Nakagawa, Haruhiko Kamada, Shin-ichi Tsunoda and Yasuo Tsutsumi.

PP1-121 DISSECTING INTERACTIONS BETWEEN INTERLEUKIN-1ALPHA AND HISTONE ACETYLTRANSFERASE COMPLEXES BY IN VIVO PROTEIN-TAGGING AND IMMUNOPRECIPITATION TECHNIQUES. Blanka Vicenova, Miroslava Buryskova, Ladislav Burysek, Josef Novak, Martin Pospisek.

PP1-122 ALPHA-ELEOSTEARIC ACID INDUCES AUTOPHAGY-DEPENDENT CELL DEATH THROUGH TARGETING AKT/MTOR AND ERK1/2 SIGNALING PATHWAYS. Jeong-Min Eom, Min-Ji Seo, Seung-Hyun Han, Chong-su Cho, Cheol-Heui Yun .

PP1-125 PHAGOCYTE-DERIVED REACTIVE OXYGEN SPECIES AS SUPPRESSORS OF INFLAMMATION. Kelly L. Brown, Karin Christenson, Martina Sundqvist, Anna Karlsson, Claes Dahlgren, Johan Bylund.

PP1-126* SPECIES-INDEPENDENT BIOASSAY FOR QUANTIFICATION OF ANTIVIRAL TYPE-I INTERFERONS BASED ON LUCIFERASE-EXPRESSING RIFT VALLEY FEVER VIRUS. Thomas Kuri, Matthias Habjan, Friedemann Weber.

PP1-127 A HUMAN IL10 BAC TRANSGENE REVEALS TISSUE-SPECIFIC CONTROL OF IL-10 EXPRESSION: IMPLICATIONS ON DISEASE OUTCOMES. Jay H. Bream, Dilini Ranatunga, Christian M. Hedrich, Fengying Wang, Daniel W. McVicar, Nathan Nowak, Trupti Joshi, Lionel Feigenbaum, Lindsay R. Grant, Simona Stäger.

PP1-128* POLYMORPHISMS OF INOSITOL HEXAKISPHOSPHATE KINASE 2 IN HEAD AND NECK SQUAMOUS CELL CARCINOMA. Bei H. Morrison, Eric Lamarre, Joseph Scharpf, Robert R. Lorenz and Daniel J. Lindner.

PP1-129* OVEREXPRESSION OF DOWN SYNDROME CANDIDATE REGION 1 RESULTS IN DEFECTIVE T CELL DEVELOPMENT AND FUNCTION. Katherine R Martin, Bernadette Scott, Melanie A Pritchard.

PP1-130 IL-23, IL-17RA AND IL-17A INHIBITION IN AN INDUCIBLE PSORIATIC-LIKE MOUSE MODEL. Jennifer E. Towne, Huyen Dinh, Yu Zhang, Keith L. Bailey, Joel E. Tocker and John E. Sims.

PP1-131 INFLAMMASOME COMPONENTS COORDINATE MACROPHAGE AUTOPHAGY AND PYROPTOSIS. Jean-Francois Dubuisson, Brenda Byrne, Jenny Persson, Russell Vance and Michele Swanson.

PP1-132 EOTAXIN-1 REGULATES ANTIFILARIAL IMMUNITY VIA EOSINOPHIL ACTIVATION. Katrin Gentil, Christian S Lentz, Ajit Kamath, Sabine Specht, Achim Hoerauf.

PP1-134 IL-4 PRODUCING CD4+ T CELLS IN REACTIVE LYMPH NODES DURING HELMINTH INFECTION ARE T FOLLICULAR HELPER CELLS. Irah L. King and Markus Mohrs.

PP1-135* CLEARANCE OF APOPTOTIC CELLS IS IMMUNOSUPPRESSIVE IN TYPE-1 AND TYPE-2 MACROPHAGES. Marieke A. Hoeve, Sandra Franz, Ian Dransfield.

PP1-137 IL-4 TRIGGERS ALTERNATIVE ACTIVATION OF DENDRITIC CELLS. Peter C. Cook, Lucy H. Jones, Alex T. Phythian-Adams, Rachel J. Lundie, Judi E. Allen and Andrew S. MacDonald.

PP1-139 INTERFERON-l IS FUNCTIONALLY AN INTERFERON BUT STRUCTURALLY RELATED TO THE IL-10 FAMILY. Ole J Hamming, Susanne Vends, Benjamin J Willson, Jens CB Madsen, Georges Lutfalla, Jean-Pierre Levraud & Rune Hartmann.

PP1-140 IMPAIRED CHEMOTAXIS OF DENDRITIC CELLS IN PATIENTS WITH MYOCARDIAL INFARCTION. Pawel Wolkow, Jacek Godlewski, Anna Gebska, Marek Jawien, Rafal Olszanecki, Jacek Jawien, Krzysztof Zmudka, Ryszard Korbut.

PP1-141 IFN-ALPHA IMPROVES VGAMMA9VDELTA2 T-CELLS RESPONSE TO SYNTHETIC PHOSPHOANTIGENS IN HCV-INFECTED PATIENTS, SUGGESTING COMBINED IMMUNOTHERAPY STRATEGIES. Eleonora Cimini, Cecile Bonnafous, Eleonora Lalle, Helene Sicard, Concetta Castilletti, Gianpiero D'Offizi, Maria R. Capobianchi, Federico Martini, Chiara Agrati.

PP1-144 XOMA 052, A REGULATORY MONOCLONAL ANTIBODY TARGETING IL-1 BETA, REDUCES BIOMARKERS OF CARDIOVASCULAR RISK IN ANIMAL MODELS. Vin Bhaskar, Alexander Owyang, Steve Lee, Lisa Gross, Johnny Yin, Alan Solinger, Hany Zayed, Seema Kantak, Pat Scannon, Stephen K. Doberstein.

PP1-146* MICRORNA EXPRESSION IN MYELOID DIFFERENTIATION. Megha Rajasekhar, Jeff Holst and John E.J. Rasko.

PP1-148* EFFECTS OF INTERCELLULAR COMMUNICATION BETWEEN T HELPER CELLS ON TH1/TH2 DIFFERENTIATION. Shlomit Reich-Zeligr, Yaron E Antebi, and Nir Friedman.

PP1-149 IN VITRO ANTIPROLIFERATIVE EFFECTS OF INTERFERONS IN COMBINATION WITH THE PROTEASOME INHIBITOR BORTEZOMIB. Doranelly Koltchev, Sidney Pestka and Ronald G. Jubin.

PP1-150 THE NOVEL AND SELECTIVE JAK3 INHIBITOR WYE-152038 IS HIGHLY EFFICACIOUS IN A MOUSE MODEL OF RHEUMATOID ARTHRITIS. Katherine J. Seidl, Tsung H. Lin, Martin Hegen, Elizabeth Quadros, Cheryl L. Nickerson-Nutter, Kenneth C. Appell, Andrew G. Cole, Yuefei Shao, Steve Tam, Michael Ohlmeyer, Bojing Wang, Debra G. Goodwin, Earl F. Kimble, Jorge Quintero, Min Ga.

PP1-151 A CRITICAL REAPPRAISAL OF THE A226V MUTATION IN CHIKUNGUNYA OUTBREAKS: POSSIBLE ROLE IN INCREASED PATHOGENESIS? Licia Bordi, Concetta Castilletti, Eleonora Lalle, Silvia Meschi, Marina Selleri, Roberta Chiappini, Daniele Lapa, Antonino Di Caro, Maria R. Capobianchi.

PP1-152* MODULATION OF NITRIC OXIDE PRODUCTION BY TH1/TH2/TH17/TREG CYTOKINES IN CULTURE OF ECHINOCOCCUS GRANULOSUS PROTOSCOLECES: POSSIBLE PRESENCE OF PARASITIC iNOS. Manel Amri, Dalila Mezioug, Samia Bouaziz, Sofiane-Zaki Abdi and Chafia Touil-Boukoffa.

LB-32 ULTRA-SENSITIVE CYTOKINE QUANTIFICATION. Michael Adler, Mark Spengler, Sven Schulz, Andreas Jonas; Jan Detmers.

B Sensing of Fungal and Parasitic Infection

CIS7-3 VITAMIN A DERIVED RETINOIC ACID SIGNALING MEDIATES INTESTINAL IMMUNE HOMEOSTASIS AND IMMUNITY. Jason A. Hall, Cheng-Ming Sun, Guillaume Oldenhove, Elizabeth Wohlfert, B50 Breeder Techs, Robin Kastenmeyer, Yasmine Belkaid.

CIS7-4 THE INDUCTION OF IL-10 BY FUNGI IN DENDRITIC CELLS DEPENDS ON CREB ACTIVATION BY THE COACTIVATORS CBP AND TORC2 AND AUTOCRINE PGE2. Sánchez Crespo, M., Alvarez, Y., Municio, C., Alonso, S., Fernández, N.

CIS7-5 TH17/IL-17 RECEPTOR SIGNALING AND NOT TH1 CELLS ARE ESSENTIAL FOR MUCOSAL HOST DEFENSE AGAINST ORAL CANDIDIASIS. Sarah L. Gaffen, Heather R. Conti, Fang Shen, Namrata Nayyar, Eileen Stocum, Jianing Sun, Matthew J. Lindemann, Allen Ho, J. Hoda Hai, Patricia Masso-Welch, Mira Edgerton.

CIS7-6 THE ROLES OF C-TYPE LECTINS IN THE HOST DEFENSE AGAINST FUNGAL INFECTION. Shinobu Saijo, Satoshi Ikeda, Aoi Akitsu, Noriyuki Fujikado and Yoichiro Iwakura.

PP1-153 TH17 LYMPHOCYTES ARE EXPANDED IN MULTIPLE SCLEROSIS AND ARE INHIBITED BY INTERFERONS. Francesco Novelli, Laura Conti, Daniela Boselli, Simona Rolla, Marinella Clerico, Giulia Contessa, and Luca Durelli.

PP2-130 CASPASE-1, BUT NOT ASC OR NLRP3 INFLAMMASOME COMPONENTS, MEDIATES IL-1BETA ACTIVATION AND ANTIFUNGAL DEFENSE IN DISSEMINATED CANDIDIASIS. Frank van de Veerdonk, Leo A.B. Joosten, Sanne Smeekens, Jos W.M. van der Meer, Thirumala Kanneganti, Mihai G. Netea.

LB-33 ROLE OF COMPLEMENT AND FC-GAMMA-R IN THE PROTECTIVE ACTIVITY OF THE LONG PENTRAXIN PTX3 AGAINST ASPERGILLUS FUMIGATUS. Federica Moalli, Andrea Doni, Livija Deban, Teresa Zelante, Silvia Zagarella, Barbara Bottazzi, Luigina Romani, Alberto Mantovani, Cecilia Garlanda.

LB-34* COMPLEMENT C3 PLAYS AN ESSENTIAL ROLE IN THE CONTROL OF OPPORTUNISTIC FUNGAL INFECTIONS. Mohlopheni J. Marakalala, S. Vicky Tsoni, Ann M. Kerrigan, Naren Srinivasan, Maureen Duffield, Philip R. Taylor, Marina Botto, Chad Steele and Gordon D. Brown.

A Signaling Session

PP2-131 TRANSLATIONAL CONTROL AS THE BASIS OF THE DIFFERENTIAL ANTIPROLIFERATIVE POTENCY OF IFNa2 and IFNb. Ignacio Moraga, Gilles Uzé and Sandra Pellegrini.

PP2-132 GLATIRAMER ACETATE INDUCES SECRETED IL-1RA PRODUCTION IN HUMAN MONOCYTES THROUGH PI3Kä/MEK/GSK3 PATHWAY(S). Rakel Carpintero, Karim J Brandt, Lyssia Gruaz, Patrice Lalive, Danielle Burger.

PP2-133 DESIGNER INTERFERONS REVEAL THE RELATION BETWEEN RECEPTOR BINDING AND DIFFERENTIAL BIOLOGICAL ACTIVITY. Gideon Schreiber, Daniel Harari, Eyal Kalie and Diego A. Jaitin.

PP2-134 HIGH RESOLUTION STRUCTURE OF THE COMPLEX OF INTERFERON-L1 WITH ITS RECEPTOR INTERFERON-LR1. Eugenia Magracheva, Wei Li, Sergei Kotenko, Alex Wlodawer and Alexander Zdanov.

PP2-135 LIVE AND IN COLOR – SINGLE CELL DYNAMICS OF IFN INDUCTION AND ACTION. Ulfert Rand, Johannes Schwerk, Julia E. Pulverer, Maren Freund, Hansjörg Hauser, Mario Köster.

PP2-136 THE IFN-INDUCED GTPASE, MGBP-2, INHIBITS TNF-a INDUCTION OF MATRIX METALLOPROTEINASE-9 (MMP-9) BY INHIBITING ACTIVATION OF NF-KB AND RAC 1. Angela F. Messmer-Blust, Sujata Balasubramanian, Chuan H. Yang, Meiyun Fan, Jill A. Trendel, Lawrence M. Pfeffer, and Deborah J. Vestal.

PP2-138 IRF8 IS PRESENT IN MICROGLIA IN THE CNS AND MODULATES THE RESPONSE OF THESE CELLS TO IFN-g. Sally L. Carter, Tomohiko Kanno, Keiko Ozato and Iain L. Campbell.

PP2-139* MODULATION OF TLR4 MEDIATED INFLAMMATION BY DEREGULATED GP130-STAT SIGNALLING. C.J. Greenhill, M. Najdovska, P. Hertzog, A. Mansell, B.J. Jenkins.

PP2-140* INJURY INDUCES DIFFERENTIAL SIGNALING BY CD4+ T-REGULATORY VERSUS NON-REGULATORY T CELLS. Marc Hanschen, Goro Tajima, Fionnuala O'Leary, Kimiko Ikeda, James A. Lederer.

PP2-141 TUMOR SUPPRESSOR WWOX/WOX1 CROSS TALKS WITH MEK KINASE AND IκBα DURING T CELL ACTIVATION. Hsin-Ping Lin, Shenq-Shyang Huang, and Nan-Shan Chang.

PP2-142 RNA ACTIVATORS OF THE STRESS KINASE PKR ARE ABUNDANT IN 5'-DOMAINS OF INFLAMMATORY CYTOKINE AND CHEMOKINE mRNA. Raymond Kaempfer, Smadar Cohen-Chalamish, Tami Bar Moshe, Yona Banai, Lise Sarah Namer and Dalia Hillman.

PP2-143* FUNCTIONAL REDUNDANCY OF SOCS3. H Kiu, AW Roberts, WS Alexander.

PP2-144* STAT1 EXPRESSION REPRESSES THE EXPRESSION OF MITOCHONDRIAL ENCODED RNAS. Magdalena Szelag, Ramesh Potla, Jennifer Sisler, Hossein Hamed, Paul Dent, Andrew C. Larner.

PP2-145* BCL-3 ATTENUATES LPS-INDUCED MACROPHAGE TNF ALPHA PRODUCTION THROUGH INTERACTION WITH NF-KB P50 HOMODIMERS AFTER ACUTE ALCOHOL EXPOSURE. Shashi Bala, Alex Tang, and Gyongyi Szabo1.

PP2-146 A NATURAL ANTISENSE TRANSCRIPT OF INTERFERON-α1 GENE REGULATES VIRAL INFECTION-MEDIATED MRNA EXPRESSION LEVEL AND FURTHER INDUCTION OF INTERFERON-α1 PROTEIN. Tominori Kimura, Jiang Shiwen, Mikio Nishizawa and Masao Nishikawa.

PP2-147 INVESTIGATION OF THE ROLE OF TRANS-SIGNALLING BY THE SOLUBLE INTERFERON RECEPTOR IN TYPE I INTERFERON-REGULATED RESPONSES. Niamh E. Mangan, Shamith A. Samarajiwa and Paul J. Hertzog.

PP2-149 RIBOSOMAL PROTEIN GENE PROMOTER-BASED SYSTEM FOR SELECTIVE ASSESSMENT OF POST-TRANSCRIPTIONAL REGULATION OF CYTOKINE GENES. Edward Hitti, Suhad Al-Yehya, Maher Al-Saif, Peer Mohideen, Lina Omar, and Khalid S. A. Khabar.

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PP2-150* PML POSITIVELY REGULATE INTERFERON GAMMA SIGNALING. Jamila El Bougrini, Laurent Dianoux and Mounira K. Chelbi-Alix.

PP2-151 IKKj AS AN OPERATOR OF VIRUS-INDUCED SIGNALING. Benjamin tenOever.

PP2-152 CYTOHESIN-1 REGULATES OXIDATIVE BURST AND DEGRANULATION IN NEUTROPHILS. Mohammed-Amine El Azreq, Valérie Garceau, Danielle Harbour, Christophe Pivot-Pajot and Sylvain G. Bourgoin.

PP2-153 FORCED HOMO- AND HETERODIMERIZATION OF ALL GP130-TYPE COMPLEXES LEADS TO CONSTITUTIVE LIGAND INDEPENDENT SIGNALING, AND CYTOKINE INDEPENDENT GROWTH. Jan Suthaus, Anna Tillmann, Inken Lorenzen, Elena Bulanova, Stefan Rose-John and Jürgen Scheller.

PP2-154 A ROLE FOR JANUS KINASES IN CHEMORESISTANCE. Catarina R. do Carmo, Michael J. Seckl and Ana P. Costa-Pereira.

PP2-155* DEFINING WEST NILE VIRUS AGONISTS OF THE RIG-I AND MDA5 SIGNALING PATHWAYS. Sudha Pandit, Lisa Injaian, Jennifer German, Katherine Pankow, Brenda L. Fredericksen.

PP2-156* STAT3 NEGATIVELY REGULATES ANTIVIRAL RESPONSES THROUGH SUPPRESSION OF TLR AND TYPE I IFN-MEDIATED RESPONSES. Wei-Bei Wang, Hao-Kang Deng, David E. Levy and Chien-Kuo Lee.

PP2-157 POLO-LIKE KINASE 1 (PLK1) REGULATES IFN INDUCTION BY MAVS. Damien Vitour, Stéphanie Dabo, Malek Ahmadi Pour, Myriam Vilasco, Pierre-Olivier Vidalain, Yves Jacob, Mariana Mezel-Lemoine, Suzanne Paz, Meztli Arguello, Rongtuan Lin, Frédéric Tangy, John Hiscott and Eliane F. Meurs.

PP2-158 LOSS OF T-CELL IL-6R EXPRESSION DURING INFLAMMATION: IL-6 TRANS-SIGNALING REGULATES T CELL TRAFFICKING AND EFFECTOR CHARACTERISTICS. Gareth W. Jones, Rachel M. McLoughlin, Victoria J. Hammond, Clare R. Parker, John D. Williams, Raj Malhotra, Jürgen Scheller, Stefan Rose-John, Nicholas Topley and Simon A. Jones.

PP2-159 IL-7 ACCELERATES HUMAN T-CELL LEUKEMIA PROGRESSION IN VIVO. Ana Silva, Ben Seddon and Joao T. Barata.

PP2-160 CELLULAR DYNAMICS OF THE STAT3 TRANSCRIPTION FACTOR. Velasco Cimica, Janaki Iyer, and Nancy C. Reich.

PP2-161* IFN-MEDIATED REGULATION OF PROTEIN TRANSLATION AND ITS ROLE IN AN ANTIVIRAL IMMUNE RESPONSE. Daniel Burke, Nahum Sonenberg, Leonidas C. Platanias, Eleanor N. Fish.

PP2-162* THE E3 UBIQUITIN LIGASE TRIAD3A NEGATIVELY REGULATES THE RIG-I/MAVS SIGNALING PATHWAY BY TARGETING TRAF3 FOR DEGRADATION. Peyman Nakhaei, Thibault Mesplede, Mayra Solis, Qiang Sun, Tiejun Zhao, Long Yang, Tsung-Hsien Chuang, Carl F. Ware, Rongtuan Lin , John Hiscott.

PP2-163 ACTIVATION OF ID3 EXPRESSION BY TGFβ IN PRIMARY HUMAN MACROPHAGE REVEALS INVOLVEMENT OF SMAD1/5 MEDIATED SIGNALING. Dinara Nurgazieva, Ming Wen, Julia Kzhyshkowska, Sergij Goerdt, Alexei Gratchev.

PP2-164* DISCOVERING AND CIRCUMVENTING PITFALLS ANALYZING FLUORESCENCE RESONANCE ENERGY TRANSFER BETWEEN PROTEINS IN CELLS: APPLICATION TO IFN RECEPTORS. Christopher D. Krause, Lara S. Izotova, Barbara Schwartz, Chinkuei Kuo, Gina DiGioia, and Sidney Pestka.

PP2-165 STING IS AN ENDOPLASMIC RETICULUM ADAPTOR THAT FACILITATES INNATE IMMUNE SIGNALING. Hiroki Ishikawa and Glen N Barber.

PP2-166* CHARACTERIZATION OF PUTATIVE STAT2 PHOSPHORYLATION SITES THAT REGULATE THE CELLULAR RESPONSES TO TYPE I INTERFERONS. Håkan C. Steen and Ana M. Gamero.

PP2-167 LOW TOXICITY SIGNALING BY MONOPHOSPHORYL LIPID A. Thomas C. Mitchell, Caglar Cekic, Carolyn R. Casella, and Chelsea A. Eaves.

PP2-168* A MATHEMATICAL MODEL PREDICTS THE KINETICS OF P58IPK TOPOLOGY IN RESPONSE TO INFLUENZA VIRUS INFECTION. Alan G. Goodman, Bertrand C. W. Tanner, Wendy E. Thomas, Michael G. Katze.

PP2-169 DISPARITY IN PTPN1 AND PTPN2 MODULATION OF IL-6 PROMOTER ACTIVATION BY PLATELET-ACTIVATING FACTOR. Geneviève Hamel-Côté, Steeve Veronneau, Simon Rollin, Marek Rola-Pleszczynski and Jana Stankova.

PP2-170 BIOCHEMICAL MONITORING OF THE EARLY ENDOCYTIC TRAFFIC OF THE TYPE I INTERFERON RECEPTOR. Béatrice Payelle-Brogard and Sandra Pellegrini.

PP2-171 ADIPONECTIN DECREASES EXPRESSION OF TLR4 AND MYD-88 INDEPENDENT SIGNAL TRANSDUCTION IN RAW 264.7 MACROPHAGES. Palash Mandal, Megan R. McMullen, Pil-Hoon Park, Thierry Roger and Laura E. Nagy.

PP2-172* MECHANISMS CONTROLLING DIFFERENTIATION AND SELF-RENEWAL OF IL-3 DEPENDENT ER-HOXB8 MYELOID PROGENITORS. Bin Wen, Jinglong Chen, Jane Olsen, Shamaruh Mirza, and Ian G Young.

PP2-173 ACT1, A NOVEL U-BOX E3 UBIQUITIN LIGASE FOR IL-17R-MEDIATED SIGNALLING. Caini Liu, Wen Qian, Youcun Qian, Natalia V. Giltiay, Yi Lu, Shadi Swaidani, Saurav Misra, Li Deng, Zhijian J. Chen, and Xiaoxia Li.

PP2-174* FUNCTIONAL ROLE OF CD157 IN MONOCYTE MIGRATION. Rossella Parrotta, Nicola Lo Buono, Paola Bovino, Simona Morone, Mariama El Baroudi, Erika Ortolan, Fabio Malavasi and Ada Funaro.

PP2-175 ADAM17-MEDIATED SHEDDING OF THE IL-6R INDUCES CLEAVAGE OF THE MEMBRANE STUB BY γ-SECRETASE. Athena Chalaris, Jessica Gewiese, Lina Fleig, Alex Schneede, Stefan Rose-John, Jürgen Scheller.

PP2-176 HEXAMER, TETRAMER OR BOTH: THE IL6-IL-6R-GP130 COMPLEX. Inken Lorenzen, Markus Perbandt, Georg Wätzig, Stefan Rose-John, Rolf Hilgenfeld, Joachim Grötzinger.

*Travel Award Recipient

PP2-177 UNRAVELING VIRAL INTERLEUKIN-6 BINDING TO GP130 AND ACTIVATION OF STAT-SIGNALING PATHWAYS INDEPENDENT OF INTERLEUKIN-6. Nina Adam, Björn Rabe, Joachim Grötzinger, Stefan Rose-John, Jürgen Scheller.

PP2-178* STAT5A NUCLEAR TRAFFICKING. Ha Youn Shin.

PP2-179* SOCS3 BLOCKS BREAST TUMOR KINASE ACTIVATION OF STAT3. Yiwei Gao and Nancy C. Reich.

PP2-180 TOLL-LIKE RECEPTOR LIGAND-INDUCED SYNERGISTIC INTERFERON GENE EXPRESSION IN HUMAN MONOCYTE-DERIVED DENDRITIC CELLS. Sanna M. Mäkelä, Pamela Österlund, Taija E. Pietilä, and Ilkka Julkunen.

PP2-181 DIFFERENTIAL TYROSINE PHOSPHORYLATION OF THE STAT5A TRANCRIPTION FACTOR BY ONCOGENIC KINASES. Nicolas Chatain, Michael Vogt, Stefan N. Constantinescu, Marc Kerenyi, Matthias Mayerhofer, Richard Moriggl, Veronika Sexl, Gerhard Müller-Newen.

PP2-182 THE ROLE OF THE N-TERMINAL DOMAIN IN DIMERIZATION AND NUCLEOCYTOPLASMIC SHUTTLING OF LATENT STAT3. Michael Vogt, Tamas Domoszlai, Dzina Kleshchanok, Swen Lehmann, Walter Richtering, Gerhard Müller-Newen.

PP2-183 DIRECTED IMMOBILIZATION OF FLUORESCENTLY LABELLED CYTOKINES FOR THE ANALYSIS OF THEIR SIGNAL TRANSDUCTION BY CONFOCAL MICROSCOPY. Tobias Recker, Daniel Haamann, Doris Klee, Stefan Barth, Gerhard Müller-Newen.

PP2-184 TOLL-LIKE RECEPTOR 3 TRIGGERING RENDERS RENAL EPITHELIAL CELLS SENSITIVE TO FAS-INDUCED APOPTOSIS. Kirstin M. Heutinck, Jorien Kassies, Eric Eldering, Ajda T. Rowshani, Ineke J.M. ten Berge, Jörg Hamann.

PP2-185* MITOCHONDRIAL DYNAMICS AND INNATE ANTIVIRAL RESPONSES REGULATED BY RIG-I-LIKE RECEPTOR. Kazuhide Onoguchi, Mitsutoshi Yoneyama, and Takashi Fujita.

LB-35 ACTIN TURNOVER AND MICROTUBLES POLYMERIZATION ARE REQUIRED FOR LIGAND-DEPENDENT D6 UPREGULATION AND SCAVENGING. Elena M Borroni, Benedetta Savino, Massimiliano Mirolo, Nina P Machado Torres, Achille Anselmo, Chiara Buracchi, Alberto Mantovani, Massimo Locati, Raffaella Bonecchi.

LB-36 DOK-1 AND DOK-2 EXPRESSION INFLUENCES T CELL DEVELOPMENT. Besin Gilles, Saba Ingrid, Jananji Silvana and Duplay Pascale.

LB-37 THE IMPACT OF DEFECTIVE GP130 SIGNALING ON THE GLIAL RESPONSE TO INTERLEUKIN-6 TRANSSIGNALING. Ricardo F. Frausto, Jürgen Scheller, Stefan Rose-John, Brendan J. Jenkins, Matthias Ernst, Iain L. Campbell.

LB-38 THE TRANSCRIPTION FACTOR CREB AS A DIFFERENTIATOR OF MK-2 INHIBITION FROM MSK1/2 INHIBITION. Julia Guzova, Gabriel Mbalaviele, Joseph Monahan and John Schindler.

LB-39 THE FUNCTION OF MAPK ACTIVATED PROTEIN KINASES IN REGULATING PKR ACTIVITY. Christina Luig, John Hiscott, Stephan Ludwig.

*Travel Award Recipient

B Signaling Session I

CBSS4-3* ESSENTIAL REGULATORY ROLE OF NOX2 IN RIG-I-MEDIATED INNATE IMMUNE RESPONSES. Nathalie Grandvaux, Anton Soucy-Faulkner, Karin Fink, Alexis Martel, Loubna Jouan, Daniel Lamarre, and Esperance Mukawera.

CBSS4-4* REGULATION OF NFKB BY NSD1/FBXL11-DEPENDENT REVERSIBLE LYSINE METHYLATION OF P65. Tao Lu, Mark W. Jackson, Benlian Wang, Maojing Yang, Mark R. Chance, Masaru Miyagi, Andrei V. Gudkov, and George R. Stark.

CBSS4-5 TRAF3 RECRUITMENT TO Sec16A AND p115 REVEALS A NEW ROLE FOR THE ER-TO-GOLGI TRANSPORT COMPARTMENTS IN INNATE IMMUNITY. Jean-François Clément, Lisa D'Ambrosio, Gregory Emery, Anne-Claude Gingras, Sylvain Meloche and Marc J Servant.

CBSS4-6 TANK IS A NEGATIVE REGULATOR OF TLR SIGNALING AND CRITICAL FOR PREVENTING AUTOIMMUNE NEPHRITIS. Osamu Takeuchi, Tatsukata Kawagoe and Shizuo Akira.

LB-44 ROLE OF HYDROGEN PEROXIDE ON NF- κB ACTIVATION: FROM INDUCER TO MODULATOR Virgínia Oliveira-Marques*; Luísa Cyrne, H. Susana Marinho, Fernando Antunes

A Signaling Session II

CBSS8-3* DEREGULATED ACTIVATION OF CYTOKINE SIGNALING BY INTERLEUKIN-6 (IL-6) IN THE PATHOGENESIS OF EMPHYSEMA. Saleela M Ruwanpura, Jessica Jones, Louise McLeod, Alistair Miller, Philip Bardin, Gary Anderson and Brendan J Jenkins.

CBSS8-4 IL-22, A TH17 CYTOKINE, INDUCES A SYSTEMIC ACUTE PHASE RESPONSE. Spencer C. Liang, Debra Pittman, Yijun Carrier, Debra G. Goodwin, Kathleen Shields, Andre-Jean Lambert, Scott H. Schelling, Quint Medley, Hak-Ling Ma, Mary Collins, Cheryl Nickerson-Nutter, Kyriaki Dunussi-Joannopoulos, Lynette A. Fouser.

CBSS8-5 INTERLEUKIN-6 INDUCES TRANSLOCATION OF THE ADAPTER PROTEIN GAB1 BY MAPK-DEPENDENT PHOSPHORYLATION OF GAB1 ON SERINE 552. Alexandra Wolf, René Eulenfeld, Stephan M. Feller, Fred Schaper.

CBSS8-6 ROLE OF SMALL RNAS GENERATED BY RNASE L IN SIGNALING INNATE IMMUNITY AGAINST HEPATITIS C VIRUS. Robert H. Silverman, Takeshi Saito, Michael Gale Jr, and Krishnamurthy Malathi.

Late Breaking Abstracts

Allergy and Mast Cells

LB-01

ROLE OF AIRWAY EPITHELIUM IN ENGULFING APOPTOTIC EOSINOPHILS

Faris Q. Alenzi, Ph.D.

Objective: This study aims to investigate which recognition pathways are important in engulfing apoptotic eosinophils. Methods: Here, two epithelial cell were selected namely (large airway bronchial epithelial cells) LAECs and A549. The inhibition assay was examined by resting and dexamethasone-stimulated epithelial cells. Confocal microscopy confirmed the engulfment of apoptotic eosinophils. Results: Macrophages and LAECs recognized and phagocytosed apoptotic eosinophils. Dexamethasone and IL-1 increased the capacity of LAECs to engulf apoptotic eosinophils. More interestingly, inhibiting monoclonal antibodies (Mabs) abolished the uptake of apoptotic cells by LAECs. Conclusion: On the basis of the above findings, the LAECs is capable of recognizing and engulfing apoptotic eosinophils and that enhanced by interlukin-1 (IL-1β) and dexamethasone.

Anti-Tumor Immunity

LB-02

IL-12 INHIBITS DIRECTLY THE GROWTH OF HUMAN PRIMARY ACUTE MYELOID LEUKEMIA CELLS

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Acute myeloid leukemia (AML) is characterized by the rapid proliferation of malignant cells which accumulate in the bone marrow. In pediatric patients with AML, the 5-year survival rates range from 40% to 50%, pointing to the urgent need for novel therapeutic approaches. In this study, we have investigated i) the expression and function of IL-12R in AML cells, ii) the direct anti-tumor activity of the cytokine on AML cells in vitro and in vivo and iii) the mechanisms involved. IL-12R expression in four human AML cell lines and in neoplastic cells from 14 AML patients was studied by flow cytometry. Primary AML cells and cell lines were treated with hrIL-12 and tested for apoptosis (propidium iodide/AnnexinV double staining and flow cytometric analysis), proliferation (Ki67 staining and flow cytometric analysis) and angiogenesis (CAM assay). Angiogenic genes modulated by IL-12 in primary AML cells were studied by PCR array technique. SCID-NOD mice were injected intra-peritoneum with the U937 AML cell line and treated with hrIL-12 or medium. Tumor masses from SCID-NOD mice were explanted two weeks after U937 inoculation and analyzed by immunohistochemistry, flow cytometry and PCR array. Neoplastic cells isolated from AML patients and AML cell lines express constitutively the heterodimeric IL-12R. IL-12 treatment of both primary AML cells and cell lines inhibited significantly proliferation and angiogenesis in vitro while unaffecting apoptosis. The inhibition of angiogenesis was related to down-regulation of a wide panel of pro-angiogenic genes including CCL2, VEGF-D, VEGFR2 and neuropilin 2. T umors formed by U937 cells in SCID/NOD mice were significantly smaller following IL-12 vs PBS treatment due to inhibition of angiogenesis and induction of apoptosis. This study demonstrates for the first time that IL-12 inhibits directly the growth of human primary AML cells and may provide a rational basis for the development of a clinical trial.

Biological Therapeutics

LB-03

ADALIMUMAB IN SEVERE ACUTE SCIATICA. A RANDOMIZED,DOUBLE BLIND, PLACEBO CONTROLLED CLINICAL TRIAL

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Objective: Based on several experimental results and on a preliminary study, a trial was undertaken to assess the efficacy of adalimumab, a TNF-α inhibitor, in patients with radicular pain related to lumbar disc herniation.
Methods: The multicenter, randomized controlled trial was conducted between May 2005 and December 2007 in Switzerland. Patients with acute (< 12 weeks) and severe (Oswestry Disability index (ODI) > 50) radicular leg pain and imaging-confirmed lumbar disc herniation were randomized to receive as adjuvant therapy, either 2 subcutaneous injections of adalimumab 40 mg at 7 days interval or matching placebo . The primary outcome was leg pain, recorded every day for ten days and at 6-weeks and 6-months on a visual analogue scale (0 to 100), and analysed using longitudinal models for repeated measures analysis . In case of surgery, last observation before intervention was carried forward. (ClinicalTrials. gov number, NCT00470509). Results: 265 patients were screened, 61 enrolled (adalimumab= 31), four lost

to follow-up. Over time, leg pain decreased significantly more in the adalimumab than in the placebo group (p<0.001), but the effect size was relatively small (13.8 (CI95% -11.5 – 39.0) at 6 months). Less surgical discectomies were performed in the adalimumab group (6 versus 13, p=0.04). Conclusion: The addition of adalimumab to the treatment of patients suffering from acute and severe sciatica resulted in a significant decrease in leg pain and significantly less surgical procedures.

LB-04

APG2305 - A NEW ORALLY-ACTIVE SELECTIVE PEPTIDIC ANTAGONIST OF IL-23R

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Introduction: Interleukin (IL)-23 has emerged as a key player in IBD, psoriasis, and multiple sclerosis. IL-23 belongs to the IL-12 family of cytokines. IL-23 acts on the IL-23 receptor (IL-23R), predominantly present on TH17 cells required for the induction and maintenance of chronic inflammation. IL-23R signaling via STAT-3 enhances the maintenance of the TH17 population characterized by the production of IL-17. Two injectable monoclonal antibodies available commercially or in development are competitive inhibitors of both IL-12 and IL-23. There is a need to develop specific non-competitive inhibitors of IL23R to avoid the possible deleterious effects on immune surveillance of inhibiting IL-12. Additionally, IL-23R inhibitors that could be administered orally would improve patient compliance. Objectives: To design small, selective, orally active, peptidic, non-competitive IL-23R antagonists. Method: Using our Module-X™ platform, we have designed small d-peptides that reproduce hinge regions of IL-23R and have evaluated their activities in vitro and in vivo in models of inflammation both by systemic and oral administration. Results: APG2305 (1 kDa), an 8-amino acid D-peptide, did not displace bound [125I]-IL-23. [125I]-APG2305 demonstrated specific and selective binding to IL-23R with an IC50 of displacement and a KD both in the nanomolar or sub-nanomolar range. APG2305 inhibited IL-23-induced STAT3 phosphorylation and IL-17 generation in mouse splenocytes with nanomolar or better IC50's and did not inhibit IL-12-induced STAT4 phosphorylation. Intraperitoneal and oral administration of APG2305 inhibited inflammatory effects in mouse models of PMA-induced dermatitis, experimental autoimmune encephalomyelitis (EAE) and collagen-induced arthritis (CIA). Two truncations of APG2305 also showed in vitro and in vivo efficacy. Conclusion: We hereby describe the discovery of an orally active, potent and specific small peptide antagonist of IL-23R.

LB-05

TNR1-14 AND TNR1-23 - NEW POTENT PEPTIDIC ORALLY ACTIVE ANTAGONISTS OF TNFR1

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Introduction: Tumor necrosis factor (TNF), a cytokine involved in acute and chronic inflammatory pathologies, is known to induce the expression of other pro-inflammatory mediators such as IL-6 and IL-1. There are several specific TNF antagonists commercially available. Even though these antagonists are effective, they are administered by injection and competitively inhibit TNFα. There is a need to develop specific oral inhibitors of TNF receptors to improve patient compliance. Objectives: To design peptidic allosteric inhibitors targeting flexible and allosteric regions of the TNFR1 receptor. These inhibitors would disturb specific inter- or intramolecular protein-protein interactions and selectively alter certain biological effects but preserve others. These small peptides may also be amenable for oral administration if they are potent, protected from degradation, and an adequate amount can traverse the paracellular pores in the gut. Methods: Using the Module-X™ platform we designed small d-peptides from regions of TNFR1. Peptides were evaluated in vitro and in vivo in models of inflammation after systemic and oral administration. Results: Two peptides, called TNR1-14 and 1-23, demonstrated 50 to 60% inhibition of IL-6 synthesis induced by TNFα (sub-nanomolar IC50). Additionally, the binding of each peptide was determined to be allosteric as demonstrated by the saturable effects of increasing amounts of TNFα on the EC50 of each peptide in Schild plots. In vivo, TNR1-14 and 1-23 were efficient in preventing inflammatory edema in a model of PMA-induced dermatitis and in inhibiting LPS or TNF-induced hypotension in rats when systemically or orally administrated. Conclusion: We hereby describe the discovery of potent, orally active, small peptides antagonists (Allosteramers™) of TNFR1 which exhibit allosteric properties and efficacy in vitro and in vivo in models of inflammation after systemic and oral administration.

Chronic Inflammatory Disease

LB-06

A NOVEL IMMUNE ORALLY ACTIVE MODULATOR ISOXAZOLINE INHIBITS INFLAMMATORY CYTOKINE PRODUCTION FROM TH-17 CELLS

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Appropriate immune responses against pathogens are crucial for controlling infection, tumor development and inflammatory disorders. CD4+ T helper (Th) cells are vital in modulating the proper innate and adaptive immune responses, which are mediated by an increase of cytokine production. Differentiated Th subsets, Th1, Th2 and Th17 cells, are distinguished via the production of the cytokines IFN-©, IL-4, and IL-17, respectively. Given the strong association between excessive Th17 activity and human disease, the identification of new therapeutic approaches that target Th17 cells is important. In the present study, primary monocytes/macrophages, a well-defined immune cells commonly used for the study of inflammation, were studied to determine the molecular mechanism underlying the inhibitory effect of immune modulator compound isoxazoline, such orally active isoxazole compounds have diverse activities and are being explored for their effects in inflammatory disease, allergy and cardiovascular diseases, and in LPS-induced inflammatory responses and infection. The mechanism of action of these compounds is not clearly understood. We report that isoxazoline directly inhibits the maturation and activation of macrophages and dendritic cells, and this inhibition includes the suppression of LPS-induced IL-1β and IL-23 production. Next, we show that isoxazoline also inhibits the generation Th17 cells *in vitro*, suggesting both cell autonomous and non-cell autonomous mechanisms to inhibit the development of Th17 cells. Therefore, isoxazoline should be further studied for targeting inflammatory immune disorders caused by excessive activation of Th17-mediated immune responses.

LB-07

CROSS-TALK BETWEEN IFN γ AND HEDGEHOG SIGNALING RESTORES ADIPOGENESIS IN 3T3-L1 CELLS.

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Obesity is an important risk factor for type 2 diabetes and cardiovascular disease. Hedgehog (Hh) signaling has been shown recently to play an important role in inhibiting fat formation. Interferon gamma (Ifn γ), a cytokine synthesized and secreted by T lymphocytes which production is increased in obesity,has been shown to inhibit the differentiation of adipocytes and play a role in development of insulin resistance. In this study we aimed to clarify the potential interplay between Hh signaling and Ifnγ in adipocytes. Luciferase activity was measured in hedgehog reporter cell lines Shh-light II. Relative mRNA levels of selected genes were analyzed by real-time PCR. Morphology of 3T3-L1 cells was judged by Oil-Red-O (OrO) staining. A two-tailed Student's t-testwas performed to test for statistical differences. Luciferase activity of the Hh-target geneGli1 was induced >20 fold by synthetic hedgehog pathway activator (SAG) in Shh-light II cells. This induction was strongly inhibited by simultaneous Ifnγ treatment (up to 7 fold reduction, $p<0.01$). Next, 3T3-L1 cells were induced to form adipocytes and stimulated during 10 days of differentiation with 10 ng/ml of SAG and/or 50 ng/ml Ifnγ. Co-stimulation (SAG plus Ifnγ) of 3T3-L1 cells blunted the SAG-induced increase of Hh-pathway target genes (Gli1, Gli2, Ptch1, Ptch2 and Hhip; up to 60 fold inhibition, $p<0.001$). Interestingly, combined treatment (SAG + Ifnγ) also efficiently abrogated inhibition of markers of adipocyte differentiation mediated by each of these stimuli and reverted RNA levels back up to 65% of those of untreated cells (SAG vs. SAG+Ifnγ, $p<0.001$). This was associated with the reappearance of typical morphological changes associated with normal adipocyte differentiation (lipid droplets) that were completely blocked by SAG and partially inhibited by Ifnγ. We concluded that co-activation of Hh and Ifnγ signaling cascades in 3T3-L1 cells allows them to re-enter the full differentiation program.

LB-08

CYTOKINES MODULATION OF INTERFERON GAMMA-1B THERAPY IN IDIOPATHIC PULMONARY FIBROSIS (USUAL INTERSTITIAL PNEUMONIA)

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Idiopathic pulmonary fibrosis is a rare pulmonary disease. Its histopathologic variant, Usual Interstitial Pneumonia (UIP), seems to have a specific pathogenesis that makes it resistant to conventional therapy (steroids and immunosuppressant) determining poor prognosis. Some previous studies demonstrated a potential therapeutic role of interferon gamma 1B that attends in Th1/Th2 balance, important mechanism in pulmonary fibrogenesis. Unfortunately this therapy, in a large study, proves to be inefficacy in a significant part of patients even if, some clinical observations show a good response in a subgroup of patients. Aim of study is to identify patients clinically responding to IFN-γ and correlate response to cytokine modulation. We monitored nine patients with UIP (with histologic confirmation) treated with interferon gamma 1B (IFNγ1B) trough complete respiratory evaluation (functional study, blood gases exchange, dyspnea degree, exercise endurance) and cytokines levels (IL-1, IL-2, IL-4, IL-5, IL-6, IL-8, IL-10, IL-10, TNFα and IFN-γ). Under citofluorimetric evaluation we found that at least inflammatory cytokines (IL-2, IL-6) are downregulated for some extent on patients showing an IFN-γ mediated therapeutic response. Perhaps the level of response with regard to the interstitial inflammatory disease progression vary a lot among individuals and show significant differences in terms of relapse or reacutization of the fibrotic process.

IFN-stimulated genes

LB-09

VIRUS INDUCED IRF-1 MEDIATES INTERFERON-INDEPENDENT ANTIVIRAL EFFECTS THROUGH THE INDUCTION OF VIPERIN

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IFN induction and function initiated by viral infection is frequently antagonized by viral proteins. Experiments with cells and mice that cannot respond to type I IFNs indicate that further IFN-independent antiviral mechanisms exist. Viperin is an interferon stimulated gene that is induced by type I IFNs. We found that viperin is also induced by Vesicular Stomatitis Virus and Newcastle Disease Virus infection in cells that lack the IFN-α receptor chain ($IFNAR^{-/-}$). Further experiments indicate an IFN-independent mechanism for viperin induction. Subsequent analysis revealed that IRF-1 directly induced the expression of viperin in an IFN-independent manner through two conserved interferon regulatory factor-elements (IRF-Es) of the viperin promoter. We showed that STAT1 is essential for virus induced viperin expression but is not directly involved in the induction. Since IRF-1 is strongly induced by viral infection and IRF-1 KO mice exhibit increased susceptibility to VSV infection we conclude that IRF-1 replaces the IFN system, especially when viruses evade immunity by the inhibition of the IFN system.

Immunopathogenesis

LB-10

INNATE RECOGNITION OF HERPES SIMPLEX VIRUS BY HUMAN PRIMARY MACROPHAGES IS MEDIATED BY INTRACELLULAR PATTERN RECOGNITION RECEPTORS

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Recognition of pathogens is essential for the development of innate and adaptive immunity. In mice Toll-like receptor (TLR) and non-TLR pathways have been shown to recognize herpes simplex virus (HSV). Here, we describe how herpes simplex virus (HSV) is recognized by human primary macrophages; a cell type important for direct anti-viral actions as well as orchestration of the immune response against HSV infection. In human macrophages, a number of inflammatory cytokines (including TNF-alpha, IL-6 and CCL3), IFNs (IFN-beta and IFN-lambda) and IFN-stimulated genes (CXCL9 and CXCL10) were upregulated early after infection. In murine macrophages, reports have shown TLR2- and TLR9-mediated recognition of HSV. In contrast, we observed TLR2- and TLR9-independent cytokine production in the human macrophages. Furthermore, we found that early virus-induced cytokine responses and virus-activated intracellular signalling (IRF3, NF-kappaB and MAPK pathways) were dependen t on virus entry and virus replication. Together, our results

suggest an intracellular recognition pathway which is dependent on accumulation of viral replication intermediates, like double-stranded RNA, in human macrophages.

LB-11

TOPICAL DELIVERY OF NOVEL DRUG FORMULATION "VIFERON®, GEL FOR LOCAL TREATMENT" IN GENITAL HERPES GUINEA PIG MODEL.

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The topical delivery of novel interferon - contained drug "Viferon®, gels for local treatment (40000 IU/ml)" was evaluated in the genital herpes simplex virus guinea pig model. The MS-strain of HSV-2 was utilized for intravaginal inoculation of guinea pigs with approximately 1.2*10(5) plaque forming units. The guinea pigs were treated "Viferon, gel" on the external genital skin three tames daily for five days beginning 48 h post viral inoculation. Control group animal were treated recombinant interferon alpha-2 aqueous solution in the same concentration (40000 IU/ml) or placebo (2% aqueous solution carmellosesodium) Application of "Viferon®, gel" caused reduction of lesion scores on four day treatment, whereas application of interferon formulation as solution or placebo was ineffective. This finding that "Viferon®, gel" posses antiherpesviral activity, relatedness with drug formulation and drug compounds. In control group animal treatment of aqueous solution recombinant interferon alpha-2 or 2% aqueous solution carmellosesodium was ineffective.

LB-12

TRANSCRIPTIONAL REGULATION OF T CELL DIFFERENTIATION DURING CHRONIC VIRAL INFECTION

E. John Wherry.

T cell exhaustion is common during chronic infections and can prevent optimal immunity. We have demonstrated that exhausted CD8+ T cells are subject to complex layers of negative regulation due to co-expression of multiple inhibitory receptors. Exhausted CD8+ T cells expressed up to 7 inhibitory receptors. Co-expression of multiple distinct inhibitory receptors correlated with greater T cell exhaustion and more severe infection. Although these and other studies have demonstrated the importance of inhibitory receptors and other pathways in T cell exhaustion, the underlying transcriptional mechanisms are unknown. We have recently defined a role for the transcription factor Blimp-1 in CD8+ T cell exhaustion during chronic viral infection. Blimp-1 repressed key aspects of normal memory CD8+ T cell differentiation and promoted high expression of inhibitory receptors during chronic infection. These cardinal features of CD8+ T cell exhaustion were corrected by conditionally deleting Blimp-1. Although high expression of Blimp-1 fostered aspects of CD8+ T cell exhaustion, haploinsufficiency indicated that moderate Blimp-1 expression sustained some effector function during chronic viral infection. Thus, we identify Blimp-1 as a transcriptional regulator of CD8+ T cell exhaustion during chronic viral infection and propose that Blimp-1 acts as a transcriptional rheostat balancing effector function and T cell exhaustion.

Immunoregulation

LB-13

MODULATION OF DENDRITIC CELL ACTIVATION BY CHEMOKINES AND CELLULAR INJURY

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Background and Objective Dendritic cells (DC) are professional antigen presenting cells expressing MHC class II, derived from a common marrow precursor. They are motile, diffused and have a spidery shape with many long cytoplasmic processes. The aim of this project was to test the hypothesis that cellular injury induces the activation and functional maturation of DC. Materials and Methods To test the effects of injury on DC activation, immature DCs were used as substrate for DC activation assays. They were obtained from their precursor in peripheral blood mononuclear cells (PBMNCs) by culturing them GM-CSF and IL-4. Expression of surface B7 was measured by immunofluorescence and flow cytometry. β- chemokines were used as potential injury mediators, including: RANTES, MIP-1α, MIP-1β, MCP-1,-2,-3 and -4, as well as other inflammatory cytokines such as TNF-α and IL -1. They were screened on immature DCs to examine whether or not they modulate B7-1 and B7-2. A model of cellular injury was established to investigate whether the injured parenchymal cells deliver signals to initiate DC activation or upregulation of B7-1/B7-2 by release of soluble mediators. H2O2 was used as an injury mediator to injure renal tubular epithelial cells (RTECs). Results RANTES, MIP-1α and MIP-1β upregulated B7-1. MCP-1,-2,-3 and -4 downregulated the expression of HLA-DR greatly. Furthermore, MCP-1,-2,-3 and -4 upregulated B7.2, while and -4 and MCP2 upregulated B7.1. We observed that immature DCs could not be readily stimulated with chemokines and pro-inflammatory cytokines IL-1 and TNF-α unless GM-CSF and IL-4 were used continuously. The supernatant of injured renal epithelial cells had an effect on DC activation. Conclusion These findings may explain the role of DCs as a link between the innate and the adaptive immune response, as well as being an active participant in determining the outcome of an antigen encounter.

LB-14

THE EFFECT OF TACROLIMUS ON ALTERNATE T-CELL ACTIVATION PATHWAYS

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The calcineurin inhibitor tacrolimus is an important therapeutic for treatment of patients with active rheumatoid arthritis (RA), especially in cases of resistance or intolerance to methotrexate or other disease-modifying antirheumatic drugs as well as in patients who are unresponsive to TNF-a antibody treatment. Tacrolimus (FK506) binds to FK506-binding protein (FKBP), forming a FK506–FKBP complex, which binds to and blocks calcineurin (CaN). The FK506–FKBP–CaN complex inhibits the activation of NF-ATc, thus preventing its entrance into the nucleus and further transcription of the IL-2 machinery. Although the drug FK506 inhibits T-cell activation, the mechanism of action of the drug responsible for the improvement in quality of life of rheumatoid arthritis (RA) patients is still uncharacterized. The T cell has a critical role in the pathogenesis of RA and recent developments

in the study of T cell activation have implicated a fine balance between the the Th1, Th2, Treg and Th17 cells for tilting the balance of immune crosstalk in the pathogenesis of RA. We investigated the spectrum of action of FK506 on T cell activation. We have shown in this study that FK506 is able to inhibit T cell activation mediated by NFAT as well as the TH17 paradigm. The drug FK506 inhibits activation of NFAT and hence transcription of all Th1 cytokines; i.e., IL-2, IFN-g, TNF-a. The compound moderately inhibits CD28 expression on Th1 cells and simultaneously, blocks the Th17 programmed cell induced production of IL-17 (IC50 < 0.03μM). It also inhibits expression of the related genes for the transcription factors; namely, ROR-g, STAT3, IL-17, IL-23, IL-7R, IL-23R and GROA. Our data for the first time provides evidence for the effect of tacrolimus on the inhibition of the Th17 cells and provides an explanation for the mechanism of action of the drug on alternate pathways of T cell activation implicated in the treatment of rheumatoid arthritis.

LB-15

ACTIVATION OF A miR-9/NF-kB REGULATORY LOOP IN HUMAN MONOCYTES AND NEUTROPHILS EXPOSED TO PROINFLAMMATORY SIGNALS.

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MicroRNAs are endogenous 21–23-nt noncoding RNAs that post-transcriptionally repress gene expression by binding in a sequence-specific manner to target mRNAs and impairing their translation and/or stability. miRNAs have been involved in a variety of biological processes, and recent evidence implicate miRNAs also in innate and acquired immunity, in the control of both differentiation and activation of distinct leukocyte subsets. To define their potential role in the control of inflammatory reactions, we characterized miRNAs regulated in human polymorphonuclear neutrophils (PMN) and monocytes in response to proinflammatory stimuli. In monocytes LPS was able to induce a set of miRNAs, including miR-187, miR-146b and the miR-99b/let-7e/miR-125a cluster. We also confirmed LPS induction of miR-132, miR146a and miR-155, in agreement with data obtained in human monocytic cell lines and murine macrophages. We identified miR-9 as the only miRNA up-regulated by LPS in both PMN and monocytes. miR-9 was also induced by TLR2 and TLR7/8 agonists and by the proinflammatory cytokines TNF- α and IL-1 β . In the human genome three distinct genes (C1orf61/CROC-4, BC036480, CR612213) encode three different miR-9 primary transcripts (pri-miR-9-1, pri-miR-9-2, pri-miR-9-3, respectively). Both in PMNs and in monocytes, LPS selectively induced miR-9-1. As demonstrated by selective inhibitors, LPS required an NF- κ B-dependent pathway to transactivate the promoter of the CROC-4 gene, which encoded miR9-1 in one of its intronic regions. Prediction algorithms identified NFKB1 as a potential miR-9 target, and experiments based on luciferase assay and on transfection of primary monocytes with miR-9-encoding vectors confirmed that miR-9 binds the 3'-untranslated region of NFKB1 and induces its degradation with time. In conclusion, evidence here reported candidate miRNAs as endogenous regulators of leukocyte activation induced by primary mediators during an inflammatory response. In particular, a regulatory circuit linking miR-9 and NF- κ B may be relevant to fine tune this key transcription factor during inflammation.

LB-16

POTENTIAL MECHANISM OF IMMUNE REGULATION VIA THE LINK ARYL HYDROCARBON RECEPTOR (AHR) AND INDOLEAMINE 2,3-DIOXYGENASE (IDO) IN MURINE DENDRITIC CELLS.

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IDO is a rate-limiting enzyme catalyzing tryptophan into kynurenine (Kyn) and other metabolites in several cell types including dendritic cells (DCs) by immune activation. IDO activity causes both innate and adaptive immune responses such as inhibition of T cell proliferation and apoptosis of Th1 cell. Recently, Ahr activation is known to induce immune regulation especially in T cells via exposure to Ahr agonist 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). In addition, both Ahr and IDO activation are critically responsible for development of regulatory T cell (Treg). Therefore, we hypothesize that there is a potential link between Ahr and IDO expression in DCs that can further regulate undifferentiated T cells. We investigated Ahr and IDO expression in bone marrow-derived dendritic cells (BMDCs) from wild-type (WT) and Ahr-KO C57BL/6J mice stimulated with LPS, CpG-ODN and TCDD for 24h. IDOmRNA was detected by RT-PCR. Ahr and IDO proteins were analyzed with Western blot. Assay of IDO activity was performed by measuring spectrophotometrically Kyn levels in culture supernatant. Cytokines in supernatant were measured by ELISA. We found that both LPS and CpG-ODN were able to induce Ahr in WT cells, separately. The treatment of only TCDD caused no effect on IDOmRNA expression in WT cells. However, treatment of LPS or CpG-ODN in combination with TCDD enhanced the IDOmRNA levels compared to treatment of two these compounds alone in WT cells. Interestingly, we showed that neither LPS nor CpG-ODN with or without TCDD induced expression of IDOmRNA in Ahr-KO cells. Consequently, Kyn levels were significantly reduced in Ahr-KO cells compared to WT cells. LPS and CpG-ODN up-regulated the IL-6 and TNF-alpha but down-regulated IFN-gamma production in Ahr-KO cells. Whether or not the link between Ahr and IDO and its consequence in DCs drives naïve T cell proliferation and differentiation is under investigation.

LB-17

RECONGNITION VERSUS ADAPTIVE UPREGULATION AND DEGRADATION OF CC CHEMOKINES BY THE CHEMOKINE DECOY RECEPTOR D6 ARE DETERMINED BY THEIR N-TERMINAL SEQUENCE

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The chemokine decoy receptor D6 controls inflammatory responses by selective recognition and degradation of most CCR1 to CCR5 agonistic ligands. CCL14 is a homeostatic chemokine present at high concentrations in the serum with a weak agonist activity on CCR1. Under inflammatory conditions, plasmin and UPA-mediated truncation of 8 amino acids generates the potent CCR1/CCR3/CCR5 isoform CCL14(9-74), which is further processed and inactivated by dipeptidyl peptidase IV/CD26 that generates CCL14(11-74). We report that D6 efficiently binds both CCL14 and its truncated isoforms. Like other D6 ligands, the biologically-active CCL14(9-74) induces adaptive upregulation of D6 expression on the cell membrane, and is rapidly

and efficiently degraded. In contrast, the D6-mediated degradation of the biologically-inactive isoforms CCL14(1-74) and CCL14(11-74) is very inefficient. Thus, D6 cooperates with CD26 in the negative regulation of CCL14 by the selective degradation of its biologically-active isoform. Analysis of a panel of CC chemokines and their truncated isoforms revealed that D6-mediated chemokine degradation does not correlate with binding affinity. Conversely degradation efficiency is positively correlated with D6 adaptive upregulation. Sequence analysis indicated that a proline residue in position 2 of D6 ligands is dispensable for binding but crucial for D6 adaptive upregulation and efficient degradation.

LB-18

TLR SIGNALING INCREASES IMMUNOGENICITY OF RETROVIRAL HIV-1 VACCINE CANDIDATE.

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Toll-like receptors (TLRs) recognize unspecific conserved microbial patterns and initiate adaptive immune responses by activating dendritic cells (DCs). Plasmacytoid dendritic cells (pDCs) have intracellular TLR7/8 and TLR9 receptors recognizing viral and bacterial nucleic acids, such as single stranded RNA and CpG DNA motifs, respectively. It has been reported that pDCs are most efficiently activated when simultaneously co-stimulated by both TLR7/8 and TLR9. Upon TLR stimulation, pDCs secrete interferon- α (IFN- α) that up regulates MHC-1 expression, leading to enhanced presentation of peptides derived from cytosolic proteins . We have studied effects of using the TLR9 agonist CpG ODN 1826 as vaccine adjuvant in a setting where Balb/c mice were vaccinated with non-replicating retroviral particles encoding an HIV-1 derived antigen. We show that TLR9 signaling increases cell-mediated immune responses against the HIV epitope. Another subset of dendritic cells, myeloid dendritic cells (mDCs) have TLR3, TLR4 and TLR7/8 receptors, recognizing double-stranded RNA, LPS and single stranded RNA. Upon TLR stimulation, mDCs secrete IL-12, a T cell stimulating factor. A beneficial effect of combining different TLR-agonists as vaccine adjuvants has been reported. We are currently investigating the effects of activating both pDCs and mDCs with combinations of TLR3-, TLR7/8- and TLR9-agonists when vaccinating Balb/c mice with retroviral particles.

LB-19

ROLE OF TIR8/SIGIRR, A NEGATIVE REGULATOR OF IL-1/TLR SIGNALING, IN THE PULMONARY IMMUNE RESPONSE TO PSEUDOMONAS AERUGINOSA INFECTION

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TIR8/SIGIRR isa member of the IL-1 receptor/Toll-like receptor (TLR/IL-1R) superfamily, expressed by epithelial cells and immature dendritic cells, which negativelyregulates the TLR/IL-1R signaling pathway leading to NF-kB activation and inflammatory responses. NF-kB, by regulating the transcription of genes for numerous inflammatory factors including IL-6 and CXCL1, is a central mediator of the lung epithelial cell response to the infection by Pseudomonas aeruginosa, a Gram-negative pathogen that can cause serious lung infection in immunocompromised individuals and cystic fibrosis patients. On the other side, uncontrolled cytokine release leads to pathological responses that inhibit bacterial clearance. To study the role of TIR8/SIGIRR in the pathogenesis of P. aeruginosain vivo,Tir8-deficient mice and wild-typemice were intratracheally inoculated with laboratory strain. The absence of TIR8/SIGIRR had a deleterious effect on the host in terms of mortalityand clearance of P. aeruginosa from the lung in an acute model of infection, as reflected by an increased number of colony-formingunits (cfu) ($P < 0.05$, Tir8-/- mice vs Tir8+/+mice). Increased susceptibility to P. aeruginosa was also associated to an exacerbated local productionof proinflammatory cytokines (IL-1β, TNFα, IL-6 and IL-23) and chemokines (KC, MIP-2, JE, IFNγ, MIP-1α, RANTES and EOTAXIN) in lungs and serum. The IL-1 receptor (IL-1R), which is negatively regulated by TIR8/SIGIRR, plays also an important role in the response to P. aeruginosa. A lack of IL-1R have a protective effect in the context of an acute infection with this pathogen. IL-1R deletionreverted the situation observed in TIR8-knockout mouse. Indeed, double deficient mice were found to have an increased resistance against Pseudomonas pneumonia, as reflected by an enhanced clearance of bacteria from the lungs, which was associated with reduced local cytokine and chemokine concentrations. These results suggest that TIR8/SIGIRR, plays a key role in modulating lung inflammation due to P. aeruginosa which, if uncontrolled, is responsible of inhibition of bacterial clearance and exacerbation of tissue pathology.

Inflammation & Pathogenesis

LB-20

PRESENCE OF FOXP3HIIL-17+ T REGULATORY CELLS CONTRIBUTE TO GENDER DISPARITY OBSERVED IN A SPONTANEOUS MODEL OF CROHN'S DISEASE-LIKE ILEITIS

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Previously, we demonstrated in a spontaneous murine model of Crohn's disease-like ileitis, i.e, SAMP1YitFc (SAMP) strain, the severity of chronic intestinal inflammation was modulated by estrogen receptor β-expressing FoxP3+ T regulatory cells (Tregs), which were increased and functionally more potent at downregulating pathogenic effector T cells in males (M) vs. females (F), and may account for the gender disparity in disease (F>M) observed in these mice. As emerging evidence suggests the existence of IL-17-producing FoxP3+ Tregs, the aim of the present study was to investigate the phenotypic expression of IL-17 on CD4+CD25+ cells in SAMP-M vs. -F mice. FACS analysis was performed on mesenteric lymph node (MLN) and spleen cells from SAMP vs. AKR (parental control) mice at puberty (6 wk) and during established disease (14 and 22 wks) for IL-17A and IL-17F; western blot analysis was used to confirm results. FACS results indicated significantly higher expression of IL-17F, but not IL-17A, on CD25+ splenocytes from 6-wk-old SAMP-F vs. -M (15.3 $\pm$3.43% vs. 5.0$\pm$1.90%, $p=0.034$); conversely, no differences were observed in age-matched AKR. However, significant differences were observed in 6-wk-old SAMP-M vs. AKR-M for FoxP3 (5.2$\pm$0.58%

vs. 0.7±0.08%, p=0.007) and IL-17F (4.2±0.55% vs. 1.1±0.75%, p=0.04). In MLN, FoxP3 expression was significantly greater in SAMP-F vs. AKR-F (5.0±0.58% vs. 0.36±0.10%, p=0.005), although IL-17F showed no significant differences. In mice with established disease (14 wk), no significant differences were observed in IL-17 expression in SAMP-M vs. –F, similar to age/gender-matched AKRs. Interestingly, gating of IL-17F on CD25+FoxP3hi population suggested the presence of FoxP3hiIL-17F+ on MLN, which was significantly increased in SAMP-F vs.-M (19.2±0.78% vs. 13±3.1%, p= 0.06); no significant difference was observed for FoxP3hi IL-17A+ cells. Western blot analysis confirmed these results. Taken together, these results suggest that differences in IL-17F, but not IL-17A, expression on CD4+CD25+ FoxP3+ Tregs may contribute to the severity of ileitis and the gender disparity observed in experimental Crohn's disease.

LB-21

TNF CORRELATED WITH NUMBER OF INFLAMED CELLS IN RADICULAR CYSTS

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TNF alpha is pleiotropnic cytokine included in different processes in immune system. We here estimated TNF-alpha concentration in 43 radicular cysts obtained from patients undergoing surgery, under local anaesthesia, and after aspiration of cystic fluid from non-ruptured cysts by enzyme-linked immunosorbent assay assay in respect of different clinical parameters as well as by histomorphometric analyses We correlated values of tumor necrosis factor-alpha (TNF-alpha) depending on the count of inflammatory cells with degree of vascularization in cystic fluid of radicular cysts. We was found significantly higher concentration of TNF-alpha is associated with smaller radicular cysts, higher protein concentration in cystic fluid as well as with higher presence of inflammatory cells, and increased degree of vascularization in peri-cystic tissues and cyst wall thickness. In addition we shown different cell types in tick and non tick cysts wall, determined by immunohistochemistry in peri-cystic tissues. We here shows that determination of TNF-alpha in cystic fluid simultaneously with other parameters can be an additional parameter for clinical diagnosis of inflamed cysts.

LB-22

EFFECTOR FUNCTIONS OF IL-17 IN COLLAGEN-INDUCED ARTHRITIS AND POTENT INHIBITION BY IFN-GAMMA

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Interleukin (IL)-17 is a pro-inflammatory cytokine in rheumatoid arthritis (RA) and in murine collagen-induced arthritis (CIA). Despite the exciting new knowledge about Th17 cells and IL-17, their mechanisms of action in the pathogenesis of arthritis are still unclear. In the present study we investigated the effector functions of IL-17 using the CIA model and using monoclonal neutralising anti-IL-17 antibody. As IFN-γ is counteracting the development of Th17 cells, we chose to induce CIA in IFN-γ receptor knockout (IFN-γ R KO) mice. An additional goal of this study was to verify whether IFN-γ, aside from its inhibitory activity on the production of IL-17, can influence the effector function of IL-17. Anti-IL-17 antibody inhibited development of CIA in IFN-γ R KO mice. In the joints of anti-IL-17-treated mice, neutrophil influx and bone destruction were absent. Treatment reduced the cellular response as well as the splenic expansion of CD11b+ cells, and systemic production of myelopoietic cytokines such as granulocyte macrophage colony-stimulating factor (GM-CSF), IL-6 and IL-12. IL-17 and TNF-α synergistically induced granulocyte chemotactic protein-2 (GCP-2), IL-6 and receptor activator of NF κB ligand (RANKL) in Mouse embryo fibroblasts (MEF). This induction was almost completely abrogated by IFN-γ in a STAT-1 (signal transducer and activator of transcription-1)-dependent way. In conclusion, our experiments underscore that IL-17 mediates its pro-inflammatory role in CIA mainly through stimulatory effects on granulopoiesis, neutrophil infiltration and bone destruction. Importantly, our data reveal an additional mechanism through which IFN- γ can attenuate some autoimmune diseases and autoimmune arthritis in particular. Apart from the inhibition of the production of IL-17, IFN-γ also abrogates some of the effector functions of IL-17. Thus, through its inhibition of the IL-17-induced production of IL-6, GCP-2 and RANKL, IFN- γ can profoundly limit granulopoiesis, mobilisation of neutrophils, and bone destruction, which are all important in joint inflammation.

LB-23

MODULATION OF INFLAMMATORY CYTOKINES DURING FOLLOW-UP OF PATIENTS UNDERGOING CARDIAC SURGERY

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Patients undergoing cardiac surgery may develop subliminal grades of chronic inflammation which modulate the level of seric cytokines. The purpose of the work was clinical and immunological estimation of level of a set of inflammatory cytokines (TNF-a, IL12p70, IL-10, IL-6, IL-1b,IL-8) detectable on patients undergoing cardiosurgical operations on the heart valves or other cardiac interventions . During follow-up after surgical treatment the level of cytokine vary significantly on all patients evaluated so far. In particular we detected an increase in the serum level of TNF-a, IL-10, IL-12p70 up to five times during patient recovery according to a time course profile. The precise role of each cytokine will be presented and discussed the correlation with the clinical outcome for all the patients enrolled in this study.

LB-24

IMMUNOLOGY CHANGES IN PATIENTS WITH SEBORRHOIC DERMATITIS.

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The purpose of our investigation was to study the immune status in patients with seborrhoic dermatitis (SD). 20 patients were investigated at the age of 14 to 65 years old: they included 8 women and 12 men. The immune status of patients SD was characterized by attributes of the expressed activation. Contents of IgG in blood was above the top border at 65 % of patients that testified to strengthened production of antibodies for a long time. The number of the activated cells circulating monocyties and young neutrophilies participating in the immune answer was increased. Spontaneous and induced production of active radicals of mature neutrophilies also has been

increased in 75 % and 60 %. Besides there was marked hyperplasia of NK-cells at 45 % of patients and strengthened expression of activation molecules HLA-DR on NK-cells at 60 % of patients. It is considered to be these shifts by characteristic attributes of a chronic infection. The ratio of subtypes of NK-cells at 90 % of patients SD has been changed in favour of NK-cells. Expression of molecules CD25 was increased at 90 % of the investigated patients, that is a characteristic attribute of an active phase of infectious-inflammatory process. The results of current investigation indicate the presence of chronic immune reaction in which the following immune-competent cells actively participate: phagocytes, NK-cells, CD4+ Ò-helper , CD8 + T-killers. Role of IgG and IgA antibodies also appears important. Therefore therapy of this disorder should combine both aethiological and pathogenetic approach.

LB-25

INVOLVEMENT OF HVEM IN OBESITY-INDUCED INFLAMMATORY RESPONSES.

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Obesity-induced chronic inflammation plays a pathogenic role in the development of obesity-related pathologies such as type II diabetes and atherosclerosis. HVEM/TNFRSF14, which is a receptor for LIGHT/TNFSF14 (lymphotoxins-related inducible ligand that competes for glycoprotein D binding to herpesvirus entry mediator on T cells), is a potent mediator of inflammatory responses and thus is implicated in various inflammatory pathologies. In this study, we investigated whether HVEM is associated with obesity-induced inflammatory responses and pathologies. HVEM-deficient mice and their wild-type control were fed a high-fat diet for 19 weeks and the obesity-induced inflammatory phenotypes and insulin resistance were determined. The HVEM-deficient obese mice fed a high-fat diet elicited the attenuation of body weight gain, adiposity, and glucose intolerance relative to the wild-type control mice. Expression levels of inflammatory cytokine/chemokine genes significantly decreased in the adipose tissue of the HVEM-deficient obese mice compared with those of the control. Our results indicate that HVEM-deficiency attenuates obesity-induced inflammatory responses and insulin resistance. HVEM may play a role in obesity-induced inflammation and pathologies such as insulin resistance.

Inflammation and Cancer

LB-26

EPITHELIAL-MESENCHYMAL INTERACTION IN CANCER: THE ROLE OF CHEMOKINES

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Epithelial-mesenchymal interaction (EMI) plays a key role in wound healing and cancer. In squamous cell carcinoma (SCC) tumour stroma originates from the mesenchyme and represents a microenviroment which influences the tumour and related cancer stem cells. The tumour itself originates from epithelial cells. Therefore, EMI influences the biology of tumour and so the patient survival. We prepared an in vitro model that enables us to study the epithelial-mesenchymal interaction. For this purpose we isolatedhuman dermal fibroblasts (HF) and interfolicular keratinocytes (HK) from the residual healthy skin and stromal fibroblasts from SCC of the root of tongue. We further used commercially available epithelial tumour cell line FaDu prepared from SCC of hypopharynx and spontaneously immortalized keratinocyte cell line HaCaT. We co-cultured dermal fibroblasts (HF) with various types of keratinocytes (HK, HaCaT or FaDu) in a transwell system in which the two cell populations were separated with a microporous membrane. We analysed the transcriptome of the fibroblasts using whole genome microarrays. Comparison of fibroblasts grown in the different co-cultures yielded remarkable differences in the transcriptomes. Among the genes most upregulated after co-cultivation with immortalized cell lines we observed chemokines IL-8 and CXCL-1, well known for their pro-inflammatory and oncogenic activity. The results were verified by ELISA test. The two proteins were significantly upregulated both at mRNA and protein level, together with their receptors. The obtained results indicate that the epithelial-mesenchymal interaction participates in the development of cancer-associated fibroblasts in changing their expression to support inflammation and tumorigenesis . This study was supported by Ministry of Education Youth and Sport of the Czech Republic, projects No. MSM0021620806 and NPVII 2B06106.

Macrophages and Chronic Inflammation

LB-27

MACROPHAGE RESPONSES TO INTERLEUKIN-17 ARE REGULATED BY LOCATION AND INFLAMMATION

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The purpose of this study was to examine how macrophage-lineage cells participate in T helper 17 (Th17)-mediated inflammatory responses. We were interested in how this response may be affected by compartmentalization, and the presence of an inflammatory microenvirionment. We isolated primary murine F4/80+ macrophages (Mϕ) from a broad variety of compartments including spleen, peripheral blood, peritoneum, gut, lung, liver, and CNS. We assayed the expression of the receptors IL17RA and IL17RC on these cells by flow cytometric methods, and observed the highest levels of IL17RA and IL17RC expression on Mϕ associated with mucosal surfaces, particularly gut and lung. Low levels of IL17 receptor expression were observed on Mϕ in lymphoid tissues, including spleen and peripheral blood. In order to examine the influence of inflammatory stimuli on the expression of IL17RA and IL17RC in vitro, bone marrow-derived macrophages, which express low levels of IL17RA and IL17RC were stimulated with combinations of TLR ligands, and proinflammatory cytokines. We observed coordinate expression of both IL17 receptor subunits controlled by TNFα and peptidoglycan signaling. Mice were immunized with CFA, in order to validate these effects in vivo with particular regard for their potential role in autoimmune disease pathophysiology. Following encounter with adjuvant, the expression

of both IL17RA and IL17RC was most dramatically increased on Mϕ in the liver. We further wished to determine biological consequences of IL17 signaling, and have begun to identify genes regulated by IL17 in macrophage-lineage cells. Together, these data indicate that macrophages participate in Th17-mediated immunity in a manner regulated by localization and inflamamtion. The TLR2 pathway, in conjunction with other proinflammatory signals, seems to enable the responsiveness of myeloid cells to IL17.

LB-28

GLUCOCORTICOIDS INDUCE COORDINATED EXPRESSION OF MS4A GENES IN HUMAN MACROPHAGES

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Macrophages are first-line cells of the innate immune system that provide immediate defence from foreign agents, contribute to inflammation resolution, and assist during the induction of adaptive immune response. Environment signals drive mononuclear phagocytes to exert specialized functions. IFNγ in concert with LPS induce "classically" activated macrophages (or M1 cells), which mediate resistance against intracellular parasites and tumors; in contrast, various forms of "non-classically" activated macrophages (or M2 cells) result from cell exposure to IL-4, TGF-β, immune complexes, IL-10 or glucocorticoids (GC), and support tissue repair. A comprehensive analysis of transcriptional profiles associated with human macrophage classic and alternative polarization led to the identification of a subset of MS4A proteins selectively expressed in M2 cells. MS4A is a poorly defined family of structurally-related cell-surface proteins spanning the membrane four times which include CD20, FcεRIβ and other 10 members of unknown function. Among these family members, our transcriptional profile analysis revealed a coordinated up-regulation of MS4A4A, MS4A6A, and MS4A7 in macrophages exposed to IL-4. Further analysis revealed that these transcripts are restricted to myeloid cells (monocytes, macrophages, and myeloid dendritic cells) and are up-regulated in vitro during macrophage activation by different M2-polarizing mediators, with GC being the most effective stimulus. To investigate the in vivo relevance of these results, we isolated circulating monocytes from Graves' syndrome patients before and after acute exposure to GC, and observed a significant increase in MS4A transcript levels (MS4A4A 20 fold, MS4A6A 5 fold and MS4A76 fold) after GC treatment, indicating that GC-dependent up-regulation occurs not only in vitro, but also in vivo. The biological functions of these MS4A proteins has not been defined yet, but our results on their regulated expression in myeloid cells by alternative polarizing agents suggest that they could be involved in resolution of inflammation.

Pattern Recognition Receptors & Inflammation

LB-29

HUMAN MILK CONTAINS AND MODULATES THE EXPRESSION OF THE SOLUBLE PATTERN RECOGNITION RECEPTOR PTX3.

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Innate immunity is the first line of defence against pathogens and plays a key role in the initiation, activation and orientation of adaptive immunity. The humoral arm of the innate immunity includes soluble pattern-recognition receptors (PRRs) such as collectins, ficolins, complement components and pentraxins. The prototypic long pentraxin PTX3 is rapidly produced and released by diverse cell types in response to proinflammatory signals. PTX3 binds selected microorganisms such as Aspergillus fumigatus and restores protective immunity against this pathogen in PTX3-/-mice. Neonates have an immature innate immune system and are more susceptible to bacterial infection than older children or adult. A beneficial effect of breast feeding on newborn health is highly demonstrated. This protective effect is mediated by nutrients, immunomodulatory mediators (IFNg, TNFa, or TGFb), innate immunity factors (soluble CD14, immunoglobulins, lactoferrin), and leukocytes contained in milk that can penetrate the newborn circulation. We thus hypothesized that milk may contain PTX3. We found high concentration of PTX3 in human colostrum (47.62 ± 13.5 ng/ml at day 1 post-delivery) compare to the one found in human serum (< 2 ng/ml). The presence of PTX3 in human colostrum seems to be due to the secretion of PTX3 by human mammary gland since we report the production of PTX3 by these cells. This PRR is also found in human milk cells (HMC), mainly in leukocytes, and penetrate into newborn tissus after suckling. Furthermore, human colostrum upregulated the PTX3 production by adult and neonate immunocompetent cells and we demonstrate that neonate mice present a deficit in their PTX3 production after LPS injection. Collectively, these data demonstrate that newborn have three distinct ways of PTX3 supplying by breast feeding: (i) soluble PTX3 in colostrum (ii) HMC that can secrete PTX3 upon stimulation in the specific tissue, (iii) an increase of PTX3 production by immune cells in the presence of colostrum. Thus, soluble or cell-derived PTX3 may participate to the beneficial role of breast feeding on the newborn health.

LB-30

CONSTITUTIVE PHOSPHORYLATION OF TBK1 AND ENHANCED TLR3-DEPENDENT IFN-β PRODUCTION IN THE ABSENCE OF SHIP-1

Joan Ní Gabhann, Nadia Ben Larbi, Rowan Higgs, Kiva Brennan, Jacqueline E. Damen, Gerald Krystal and Caroline A. Jefferies.

Autoimmune diseases such as systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA) result from a loss of tolerance to self-antigens and immune-mediated injury precipitated by the overproduction of type I interferon production and inflammatory cytokines. We have identified the inositol 5-phosphatase (SHIP-1) as a negative regulator of TLR3-induced type I IFN production. SHIP-1-deficient macrophages display enhanced TLR-induced IFN-β, and over-expression of SHIP-1 negatively regulates both TLR3 and TRIF-mediated IFN-β promoter activity, indicating that SHIP-1 negatively regulates TLR-induced IFN-β production. Further dissection of the IFN-β pathway implicates TBK1 as the target for SHIP-1. Critically, in the absence of SHIP-1, TBK1 appears to be hyper-phosphorylated both in unstimulated cells and following TLR3 stimulation. In addition, TBK1 appears to be constitutively associated with TRIF and TRAF3 in SHIP-1 deficient cells whereas in wild type cells this association is inducible following TLR3 stimulation. In support of a role for SHIP-1 in

regulating complex formation, confocal microscopy demonstrates that TBK1 distribution in the cell is significantly altered in SHIP-1-deficient cells, with more prominent vesicular staining observed compared to wild-type controls. Taken together, our results point to SHIP-1 being a critical negative regulator of IFN-β production downstream of TLR3 through the regulation of TBK1 localisation and activity.

LB-31

EXPRESSION PATTERNS OF HUMAN INTERFERON-ALPHA AND INTERFERON-LAMBDA SUBTYPES BY MONOCYTES, DENDRITIC CELLS AND B CELLS.

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Human interferon-alpha is a type I interferon (IFN) that comprises a family of thirteen highly homologous genes and twelve subtypes (two genes have an identical coding sequence). To determine unique roles for individual subtypes we employed two modifications of standard fluorescent-labeled probe technology --molecular beacons (MB) and locked-nucleic acids (LNA) to develop a qRT-PCR based assay highly sensitive and specific quantitative RT-PCR assay for each human IFN-alpha subtypes as well as the three subtypes of IFN-lambda. The PCR efficiencies of our twelve primer/probe sets mostly fall between 1.96 to 2.0 (2.0 represents perfect efficiency, i.e. doubling of template for every PCR cycle), sensitivity between 1-10 molecules of template per reaction, and a 10-cycle (1,000-fold) discrimination between target and non-target isoforms. We then determined expression patterns of IFN-alpha, IFN-lambda, IFN-beta and IFN-gamma in response to ligands of TLR 3, 4, and 9 and found variations in expression patterns among the cell types. These data indicate that expression patterns of Types I, II, and III IFN are both ligand and cell-type specific and suggest that individual subtypes and/or combinations of subtypes have unique roles in the innate immune response.
Recent Advances

LB-32

ULTRA-SENSITIVE CYTOKINE QUANTIFICATION

Michael Adler, Mark Spengler, Sven Schulz, Andreas Jonas; Jan Detmers.

Monitoring of low levels of cytokine requires appropriate detection technologies for different analytical challenges. By use of immunoassays with improved performance, an increase in matrix tolerance, dynamic quantification range and simultaneous recording of multiple target parameters is accessible. A comparison of applications for cytokine detection by ElectroChemiLuminescence ("ECL") and Immuno-PCR ("IPCR", "Imperacer")with Enzyme Linked Immuno Sorbent Assay ("ELISA") revealed the specific advantages of the enhanced immunoassay platforms. ECL is carried out by using special functional plate material, distinctively transition-metal labeled detection reagents and a matching instrument; IPCR utilizes a unique combination of standard ELISA protocol and efficient real-time PCR detection of antibody-DNA conjugates. While conventional ELISA is limited to a narrow detection window, the use of superior signal amplification technologies enabled target detection over 5+ orders of magnitude starting from the sub-pg/ml range. IPCR here excels with the highest sensitivity and linearity whereas ECL revealed superior signal intensities and the potential to measure up to 10 targets simultaneously in a single routine experiment ("Multiplex"). In contrast, ultra-sensitive IPCR is the key complementary technique for sample dilution strategies, thus allowing for circumvention of matrix background by simple addition of tailored buffers. In addition, this assay strategy also enables measurement of free vs. bound cytokines. The improved immunoassays are powerful tools for target identification and characterization in diagnostic, research and development applications as standard, increased and decreased levels of various cytokine targets are tested in a single experiment. A better robustness and sensitivity enables new straightforward strategies to work with biological matrices (Serum, cell culture media, etc.), thereby supporting demanding work in critical projects.

Sensing of Fungal and Parasitic Infection

LB-33

ROLE OF COMPLEMENT AND FC-GAMMA-R IN THE PROTECTIVE ACTIVITY OF THE LONG PENTRAXIN PTX3 AGAINST ASPERGILLUS FUMIGATUS

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PTX3 is a soluble pattern recognition molecule (PRM) playing a non-redundant role in resistance against Aspergillus fumigatus. We investigated the mechanisms underlying the PTX3-mediated opsonic activity and the involvement of complement, complement receptors and Fc γ receptors (Fc γ R), by in vitro and in vivo approaches. The PTX3 N-terminal domain was responsible of conidia recognition, but the full length molecule was necessary to have opsonic activity. PTX3 amplified conidia phagocytosis by human neutrophils in a complement-dependent manner, activated via the alternative pathway. Accordingly, in the presence of PTX3-opsonised conidia, CR3 activation, internalization, recruitment to the phagocytic cup and CR3-dependent phagocytosis were increased. Moreover, upon conidia opsonisation by PTX3, Fc γ RIIA/CD32 caused inside-out activation of CR3 and consequently phagocytosis of C3b-opsonised conidia. In vivo phagocytosis experiments performed with C1q- , C3- and Fc common γ chain-deficient mice and complement inhibitors supported in vitro data. Finally, the protective activity of recombinant PTX3 in aspergillosis was abolished in Fc common γ chain-deficient mice. The results reported here show that a complex interplay between complement activation and Fc γ R-mediated CR3 activation underlies the opsonic activity of PTX3. Thus, PTX3 is a fluid phase PRM whose opsonic activity is at the crossroad between complement, CR3- and Fc γ R-mediated recognition.

LB-34

COMPLEMENT C3 PLAYS AN ESSENTIAL ROLE IN THE CONTROL OF OPPORTUNISTIC FUNGAL INFECTIONS

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Fungal infections such as those caused by Candida albicans are an emerging problem resulting from modern medical interventions and the increasing prevalence of acquired immunodeficiency. The innate recognition of these pathogens is a crucial first step in the induction of protective anti-fungal immunity. Complement is thought to be one key component in this process, facilitating fungal recognition and inducing early inflammation, however, the roles of the individual complement components have not been examined extensively. We investigated the role of C3 in immunity to Candida albicans and C. glabrata in C3-sufficient and -deficient mice. In contrast to wild type mice, which were fully resistant to infection, C3-deficient mice were highly susceptible to infection with C. albicans and C. glabrata. Extensive characterization of the susceptibility of the C3-deficient mice, revealed that the absence of this complement component impaired fungal clearance but not inflammation. Interestingly, and in contrast, wild type mice were found to be more susceptible to infection with high doses of the non-pathogenic Saccharomyces cerevisiae, than were C3-deficient animals, and although this was found to be mouse strain dependent, these results suggest that complement can also contribute to pathology during fungal infections.

Signaling Session

LB-35

ACTIN TURNOVER AND MICROTUBLES POLYMERIZATION ARE REQUIRED FOR LIGAND-DEPENDENT D6 UPREGULATION AND SCAVENGING

Elena M Borroni, Benedetta Savino, Massimiliano Mirolo, Nina P Machado Torres, Achille Anselmo, Chiara Buracchi, Alberto Mantovani, Massimo Locati, Raffaella Bonecchi.

The decoy receptor D6 is a chemokine scavenger with a non-redundant role in the control of inflammatory processes. Though its signalling properties are still undefined, ligand engagement is known to induce rapid mobilization of D6 from recycling endosomes to cell surface and to improve its chemokine degradation efficiency. Internalization and recycling pathways of chemokine receptors are supported by cytoskeleton dynamics. In the present study, we show that in basal conditions D6 colocalized with microtubules but not actin filaments. Agents that disrupt (nocodazole) or stabilize (paclitaxel) microtubules did not affect receptor endocytosis. However, disruption of the recycling endosome by nocodazole increased D6 surface levels and this effect was reverted by dominant negative (DN)-Rab11, indicating that the microtubule network is required for the correct sorting of D6 to Rab11-positive slow-recycling endosomes. Agents that depolymerise (cytochalasin D) or stabilize (jasplakinolide) the actin cytoskeleton inhibited D6 constitutive endocytosis and increased receptor expression on cell surface. Interestingly, treatment with the actin depolymerising agent latrunculin A did not affect D6 internalization rate but still increased its membrane expression and colocalization with actin filaments. DN-Rab4 but not DN-Rab11 reverted latrunculin effect, suggesting that this depolymerising agent missorted D6 to Rab4-positive rapid recycling endosomes. After chemokine exposure, actin stress fibres rearranged in a thick ring of cortical actin below plasma membrane that strongly colocalized with the upregulated D6, similarly to the effect observed with latrunculin A. Nocodazole impaired D6 upregulation and scavenging,suggesting that intact microtubules are required to mobilize D6 from Rab11-positive slow-recycling endosomes. In conclusion, actin turnover sustains D6 constitutive endocytosis and its correct sorting to both rapid (Rab4) and slow (Rab11) recycling endosomes, and ligand-induced upregulation and scavenging require both actin and microtubules networks. Collectively, these results strongly suggest that regulation of cytoskeletal dynamics is required to modify D6 intracellular trafficking after chemokine engagement.

LB-36

DOK-1 AND DOK-2 EXPRESSION INFLUENCES T CELL DEVELOPMENT

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In T cells, two members of the Dok family, Dok-1 and Dok-2, are predominantly expressed. Several evidences suggest that Dok proteins act as negative regulator of T cell signaling. To clarify the role of Dok proteins in T cell development and T cell responses, we generated Dok-1 overexpressing transgenic mice. Overexpression of Dok-1 leads to reduced thymic cellularity due to a partial block at DN3 stage. Dok-1 overexpressing DN3 thymocytes express a rearranged TCRb. Altogether these results indicate that Dok-1 mediates negative signal through the pre-TCR. Although CD3, CD5 and CD69 expression on SP and DP thymocytes is normal, thymic selection at the DP stage leads to an accumulation of non-conventional CD8+ SP thymocytes. They express high levels of CD44 and CD122, low levels of CD24 and produce IFNγ when activated ex vivo. These CD8 T cells arise in RTOC reconstituted with DP thymocytes. Although some CD4+ T cells in the thymus and in the periphery exhibit also memory cell markers and rapid expression of cytokines, CD4+ T cells are less affected than CD8+ T cells by Dok-1 overexpression. In Dok-1 and Dok-2 deficient mice (DKO), no major defects in T cell development were detectable as documented by the analysis of cell surface markers. This altered lineage development of CD8+ T cells in the thymus is reminiscent to the innate-like CD8+ T cells that arise in Tec kinase (Itk-/- or Itk-/-Rlk-/-) deficient thymus. Thus, reduced T cell signaling due to Dok overexpression might lead to the development of cells that exhibit properties different from conventional T cells. We are currently analyzing the signaling pathways in DKO and Dok-1 overexpressing thymocytes to identify the mechanisms by which Dok proteins regulate T cell signaling and lineage development.

LB-37

THE IMPACT OF DEFECTIVE GP130 SIGNALING ON THE GLIAL RESPONSE TO INTERLEUKIN-6 TRANSSIGNALING.

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CNS production of IL-6 induces marked activation of astrocytes and microglia. This response is mediated by the gp130-STAT and gp130-SHP2 pathways, which are negatively regulated by SOCS3. We investigated the role of IL-6 signaling in the glial response to Hyper-IL6 or IL-6 using astrocytes and microglia from Y757F and

D STAT mice that were defective in gp130-SHP2 and gp130-STAT signaling, respectively. Compared with WT astrocytes, Y757F astrocytes had higher and more sustained activation of STAT1/3, while the levels of pY-SHP2, p-ERK and pS-Akt remained unchanged. In Y757F astrocytes SOCS3 protein production was delayed and peaked at 12 hours. In D STAT astrocytes there was weak activation of STAT1/3, while the levels of pY-SHP2, p-ERK and pS-Akt were higher and more prolonged compared with WT astrocytes. In contrast to WT and Y757F astrocytes, D STAT astrocytes showed weak and transient SOCS3 production. Irrespective of the astrocyte genotype, the kinetics of SOCS3 protein production showed a strong inverse correlation with STAT1/3 but not SHP2, ERK or Akt phosphorylation. The microglial response to Hyper-IL6 mirrored the response to IL-6. Compared with WT microglia and similar to Y757F astrocytes, Y757F microglia had stronger and protracted pY-STAT1/3 and unchanged pY-SHP2 and p-ERK, while pS-Akt was delayed. In contrast to Y757F astrocytes, the kinetics of SOCS3 production was similar in WT and Y757F microglia, while the levels were higher in the Y757F microglia. In D STAT microglia in contrast to D STAT astrocytes, there was no detectable activation of STAT1/3 or production of SOCS3, while pY-SHP2, p-ERK and pS-Akt were stronger and more sustained compared with WT microglia. In contrast to astrocytes, SOCS3 production in microglia showed a strong inverse correlation with all the phospho-proteins investigated. These results identify glial-specific differences in the execution of and responses to IL-6 transsignaling via the gp130-STAT versus gp130-SHP2 pathways.

LB-38

THE TRANSCRIPTION FACTOR CREB AS A DIFFERENTIATOR OF MK-2 INHIBITION FROM MSK1/2 INHIBITION.

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The mitogen activated protein kinase (MAPK) family of signal transduction enzymes, which includes p38, are activated in response to stress and inflammatory stimuli. Conventional p38 inhibitors block MK2 activation resulting in significant reduction of inflammation. Such inhibition of p38 activity can also lead to blockade of other p38 housekeeping functions due to the ability of p38 to phosphorylate additional substrates such as MSK1/2. In an attempt to circumvent these effects, we have designed p38-SSI (substrate selective inhibitor), which is selective for the p38-MK2 interaction while not affecting the phosphorylation of other p38 substrates. It is well established that MK-2 is the main kinase that phosphorylates HSP27, whereas MSK1, which is activated by p38 as well as by ERK, phosphorylate CREB, a bZIP transcription factor that activates target genes through cAMP response elements. To evaluate the feasibility of the p38-SSI concept, we developed CREB and HSP27 biochemical- and cell-based assays. Optimization experiments included selection of appropriate stimuli and cell types and time course studies. For example in IL1b-treated A549 cells, HeLa cells or HUVEC, we found that while the conventional p38 inhibitor inhibits the phosphorylation of both HSP27 and CREB, p38-SSI inhibited HSP27 phosphorylation without affecting CREB phosphorylation. These data indicate specific inhibition of the p38-MK2 but not p38-MSK interactions.

LB-39

THE FUNCTION OF MAPK ACTIVATED PROTEIN KINASES IN REGULATING PKR ACTIVITY

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Previously, it was shown that RNA viruses such as influenza- and reoviruses show enhanced replication efficiency in cells that are transformed with Sos, Ras or Raf. In these cells, even the influenza delNS1 virus is able to replicate and reveals oncolytic activity. At the molecular level, the growth advantage is due to an inhibition of the antiviral activity of the double-stranded RNA-activated protein kinase R (PKR). So far, the molecular link between the activated ERK pathway and inhibited PKR is elusive. Here, we present two downstream kinases of ERK, MAPKAPK2/3 (MK2, MK3) as interaction partners of a PKR regulating protein, p88. MK2/3 are able to form a complex with p88, p58 and PKR, which regulates negatively PKR- and eIF2a activity. Consistent with that, in MK2- and MK3-deficient cells influenza virus replication is strongly impaired. Furthermore, in MK2- and MK3-deficient cells virus protein synthesis is affected while viral mRNA remains unaffected. We conclude that MK2 and MK3 are a molecular link between an activated ERK and an inhibited PKR signaling pathway in Sos, Ras or Raf transformed cells.

Additional Abstracts

LB- 40

HEME OXYGENASE-1 PROTECTS AGAINST SEVERE SEPSIS VIA INHIBITION OF HEME-MEDIATED SENSITIZATION TO CELL DEATH AND RELEASE OF HMGB1.

Rasmus Larsen, László Tokaji, Raffaella Gozzelino, Dolores Bonaparte, Moises M. Cavalcante, Angelo Chora, Silvia Cardoso, Gabriela Silva and Miguel P. Soares.
Inflammation Laboratory, Instituto Gulbenkian de Ciência, Oeiras, Portugal.

Heme oxygenase-1 (Hmox1/HO-1) is a stress responsive gene that prevents the deleterious effects of inflammatory reactions by mechanisms that remain elusive. Severe polymicrobial sepsis, induced in mice by cecal ligation and puncture (CLP), led to HO-1 expression in infiltrating peritoneal leukocytes, kidney and liver. Mortality after CLP increased from 20% in wild type (Hmox1+/+) mice to 87% in HO-1 deficient (Hmox1-/-) mice. Hmox1-/- but not Hmox1+/+ mice developed end-stage multi-organ failure. Mortality of Hmox1-/- mice was associated with increased peritoneal leukocyte infiltration, but not with increased pro-inflammatory cytokine secretion or bacterial load in peritoneum, blood or organs. CLP induced a significant increase in cell-free hemoglobin, heme and free heme in Hmox1-/- relative to Hmox1+/+ mice. Furthermore, in wild type mice, administration of free heme resulted in 100% mortality to otherwise sub-lethal CLP. Free heme was found to sensitize primary hepatocytes to TNF, anti-Fas antibody, H2O2 or peroxynitrite mediated apoptosis. This cell death was associated with outward nuclear translocation and extra-cellular accumulation of the late-stage pro-inflammatory cytokine HMGB1. Accordingly, circulating and cytoplasmic HMGB1 was increased in Hmox1-/- relative to Hmox1+/+ mice following CLP. In conclusion, these data suggest that free hemoglobin and heme, released during severe sepsis, are important factors in the organ failure and death associated with severe, polymicrobial sepsis.

LB-41

*** Bonazinga Award Recipient**

THE MOLECULAR DETERMINANTS OF SEPSIS

Peter A. Ward, Department of Pathology, The University of Michigan Medical School, Ann Arbor, MI, USA

Our studies dealing with polymicrobial sepsis (cecal ligation and puncture, CLP) show robust activation of complement, with C5a generation and its interaction with its two receptors (C5aR, C5L2) to initiate a series of destructive events: 1.) Loss of innate immune functions of blood neutrophils (PMNs); 2.) A "cytokine storm" with unrestrained presence of high levels of proinflammatory cytokines; 3.) A C5a-dependent loss of catecholamine production due to apoptotic adrenal medullary cells; 4.) Lethality that can be related to generation of C5a and its interaction with C5a receptors; 5.) And, finally, impairment of cardiac function with loss of cardiac output and related changes. The adverse molecular events affecting cardiomyocyte function will be briefly presented.

LB-42

TLR3 SIGNALING IN SYNOVIOCYTES IS NEGATIVELY REGULATED BY EXTRACELLULAR ATP THROUGH INHIBITION OF NF-κB

Thusitha Gajanayake, Jakub Siednienko & Sinead Miggin
Immune Signaling Group, Institute of Immunology, Department of Biology, National University of Ireland Maynooth, Ireland

Overexpression of Toll-like receptor 3 (TLR3) in synoviocytes has recently been demonstrated in patients with rheumatoid arthritis (RA), though its regulation remains elusive. Extracellular adenosine triphosphate (ATP) is highly abundant in the synovial fluid of patients with RA and may regulate cellular responses, including TLR signaling. In this study, we hypothesized that extracellular ATP might regulate TLR3–induced signaling in human fibroblast-like synoviocytes (HFLS). We found that treatment of HFLS with the TLR3 ligand, poly(I:C), for 16 h induced RANTES/CCL5 and IL-6 secretion with higher level of RANTES and IL-6 were detected in RA cells (HFLS-RA) compared with normal cells (HFLS-N). Quantitative PCR analysis revealed that basal and poly(I:C)-induced TLR3 mRNA expression are higher in HFLS-RA compared with HFLS-N. Whilst treatment with ATP for 20 min dose-dependently suppressed TLR ligand-induced RANTES, IL-6 secretion remained unaffected. Also, ATP significantly induced IL-10 ($p<0.001$). An analogue of cAMP 8-Br-cAMP significantly reduced TLR-ligand induced RANTES secretion and induced IL-10 ($p<0.05$). Moreover, ATP inhibited poly(I:C)-induced NF-κB activation. Inhibition of NF-κB signaling by ATP may subsequently reduce RANTES secretion. Our data suggest that extracellular ATP appears to be a negative regulator of poly(I:C)-induced TLR3 signaling in RA. This study provides a novel therapeutic approach whereby TLR-induced immune responses may be modulated by cAMP in RA patients.

Kindly supported by SFI and HRB.

LB-43

HEME SENSITIZATION TO TNF-MEDIATED PROGRAMMED CELL DEATH DICTATES THE OUTCOME OF PLASMODIUM INFECTION IN MICE.

R. Gozzelino, E. Seixas, Â. Chora, A. Ferreira, G. Silva, R. Larsen, S. Rebelo, C. Penido, RN. Smith, A. Coutinho & MP. Soares. Instituto Gulbenkian de Ciência, Oeiras, Portugal.

Under homeostasis, heme acts as a prosthetic group in hemoproteins in which its reactivity is tightly controlled. However, under inflammatory conditions hemoproteins can be unfolded and degraded, releasing their non-covalently bound (free) heme that is cytotoxic, because the Fe atoms contained within its protoporphyrin IX ring are no longer controlled by the heme pockets of hemoproteins. We have previously shown that free heme released from hemoglobin (a hemoprotein) can dictate the lethal outcome of Plasmodium infection (malaria) in mice (Pamplona A. et al., Nat. Med. 2007, 13, 703). We now provide a molecular mechanism underlying that free heme sensitizes cells to undergo TNF-mediated programmed cell death, a cytotoxic effect that occurs independently of newly gene transcription and/or protein synthesis and relies on the unfettered generation of free radicals in response to TNF (Seixas E. et al., PNAS 2009, on line ahead of print). When exposed to free heme in vitro, hepatocytes respond to TNF by sustaining the activation of the c-jun N-terminal kinase (JNK), which leads to further accumulation of free radicals and to apoptosis, i.e. caspase-8 and -3 activation, DNA condensation. Inhibition of free radical accumulation by N-acetylcysteine (NAC) or Butylated hydroxyanisole (BHA), inhibition of JNK activation (pharmacologic) or JNK expression (shRNA) as well as inhibition of caspase -8 and 3 activation (pharmacologic) suppress the cytotoxic effects of free heme plus TNF in hepatocytes. Expression of the heme catabolyzing enzyme heme oxygenase-1 (HO-1) or the iron sequestering protein H-Ferritin in hepatocytes acts in an anti-oxidant manner to afford cytoprotection against heme plus TNF in vitro as well as in vivo, providing complete protection against those inflammatory diseases associated with hemolysis, namely Plasmodium infection in mice. In conclusion, this data reveals a novel mechanism via which free heme sensitizes hepatocytes to TNF-mediated programmed cell death, an effect countered by the expression of cytoprotective genes that prevent the cytotoxic effects of free heme.

LB-44

ROLE OF HYDROGEN PEROXIDE ON NF- κB ACTIVATION: FROM INDUCER TO MODULATOR

Virgínia Oliveira-Marques[1*], Luísa Cyrne[1], H. Susana Marinho[1], Fernando Antunes[1,2]
[1]Grupo de Bioquímica dos Oxidantes e Antioxidantes, Centro de Química e Bioquímica, Faculdade de Ciências da Universidade de Lisboa, Portugal
[2]Instituto Bento da Rocha Cabral, Lisboa, Portugal
[*]Current address: Inflammation Laboratory, Instituto Gulbenkian de Ciência, Oeiras, Portugal.

Hydrogen peroxide (H2O2) has been implicated in the regulation of the transcription factor NF-κB however both stimulatory and inhibitory effects are reported. The typical method used to expose cells to H2O2 is characterized by a single high dose of H2O2 in order to compensate for its rapid consumption by cells. The effective concentration of H2O2 is not known and the high dose often causes elevated oxidative damage that may mask any signaling role for H2O2. Here, we used a controlled and calibrated method - the steady-state titration - that is characterized by a continuous production of H2O2. A steady-state of H2O2 did not activate NF-κB, but significantly increased NF-κB activation when added simultaneously with TNF, in HeLa and MCF-7 epithelial cells. The degradation of the inhibitor of NF-κB - IκB-α - was sustained over time, leading to higher levels of nuclear NF-κB p65 proteins and increased expression of a set of pro-inflammatory (TNF, MCP-1, IL-8) and anti-inflammatory genes (heme oxygenase-1). Interestingly, the expression of other NF-κB-target genes remained unchanged. The transactivation potential of NF-κB is dependent on its affinity toward different κB sites in the promoter/enhancer region of target genes. In HeLa cells transfected with reporter plasmids containing different κB sequences, the lower

the apparent affinity of a κB site towards NF-κB, the higher the range of TNF concentrations where H2O2 up-regulated gene expression. Mathematical models indicate that upregulation of gene expression by H2O2 ceased when NF-κB fully occupied the κB sites. Overall, we propose a dual regulatory role for H2O2 during inflammation by simultaneously exacerbating inflammation through the production of higher levels of pro-inflammatory mediators and by attenuating possible adverse effects through induction of anti-inflammatory gene expression. It is predicted that genes with high-affinity sites remain insensitive to H2O2, whereas genes with lower-affinity sites are upregulated by H2O2.

LB-45

SYSTEMS BIOLOGY APPROACH DEFINES THE MOLECULAR PHENOTYPE OF HUMAN TUMOR ASSOCIATED MONOCYTES/ MACROPHAGE IN RENAL CELL CARCINOMA

Manesh Chittezhath[1], Manprit Kaur Dhillon[1], Brendon Ang[1], Revathi Kamaraj[2], Henry Yang[1], Rajeev Singh[3], Alvin S.C. Wong[2] and Subhra K Biswas[1]

[1]Singapore Immunology Network (SIgN), Agency for Science, Technology & Research (A*STAR), Singapore

[2]Departments of Pathology and

[3]Haematology-Oncology, National University Hospital, National University of Singapore, Singapore

Monocytes/macrophages constitute a major proportion of leukocyte infiltrates in solid tumors and mediate a variety of protumoral functions like angiogenesis, tumor cell proliferation, metastasis and immunosuppression. While several studies have documented the role of tumor associated monocytes/macrophages (TAM) in murine tumor models, investigations on human TAM remain sparse. Renal cell carcinoma (RCC) is one of the well-known urological cancers which are receptive to immunotherapy, but limited in clinical response due to its highly metastatic nature and immunosuppression. While adaptive immune response to this cancer has been well-studied, information on the role of monocytes/macrophages in human RCC is poorly investigated. The present study characterizes the repertoire of human TAM in RCC using extensive systems biology approach. Transcriptome analysis of monocytes from human RCC patients (RCC-Mo) revealed these cells to be in a transient inflammatory status, characterized by the upregulation of various cytokines, chemokines, growth factors and cell surface receptors. In conjunction, these cells showed higher expression of several C-type lectin/ phagocytosis-related receptors and protumoral genes like MMPs, VEGFA and CXCR4, reminiscent of the M2 macrophage phenotype. Surprisingly, these cells showed severely impaired inflammatory response when activated through the Toll-like receptor 4/Interleukin-1 receptor (TLR4/IL-1R) pathway. This was demonstrated by the drastic dowregulation of the inflammatory transcriptome and reduced expression of genes like TNFA, CCL3, IL-1B as well as IL-10 upon ex vivo stimulation. These results were validated by high throughput qPCR as well as an in vitro RCC tumor cell-human monocyte co-culture system. Signaling studies were performed to understand the molecular basis of the RCC-Mo phenotype. Using lentiviral/siRNA knockdown approach for key inflammatory pathway components, we demonstrate NF-κB activation to shaping the transient inflammatory status of RCC-Mo, under basal conditions. In contrast, the refractory nature of these monocytes to inflammatory stimuli was mediated by defective activation of transcription factors NF-κB and c-Jun. In conclusion, RCC-Mo under basal conditions show an inflammatory phenotype possibly linked with protumoral functions whereas upon ex vivo stimulation, they show a refractory state consistent with tumor-induced immunosuppression.

Late Breaking Author Index

Other Events

The following non-program events are also being held during the conference and are by invitation.

Saturday, October 17th

SLB Council Meeting
2:00 PM – 6:00 PM, Lisbon Marriot Hotel
SLB Council Dinner
7:00 PM – 10:00 PM, Offsite

Sunday, October 18th

SLB Council Meeting
8:30 AM – 12:30 PM, Pavilion 4: Room 1.01
ISICR Committee: Membership
8:30 AM – 10:00 AM, Pavilion 4: Room 1.03
ISICR Committee: Finance
8:30 AM – 10:00 AM, Pavilion 4: Room 1.04
ISICR Committee: Awards
8:30 AM – 10:00 AM, Pavilion 4: Room 1.05
Joint ISICR / ICS Meetings Committees
8:30 AM – 10:00 AM, Pavilion 4: Room 1.06
ISICR Committee: Nomenclature
10:30 AM – 12:00 PM, Pavilion 4: Room 1.03
ISICR Committee: Standards
10:30 AM – 12:30 PM, Pavilion 4: Room 1.04
ISICR Board of Directors
10:30 AM – 12:30 PM, Pavilion 4: Room 1.05
ISICR Committee: Publication
10:30 AM – 12:15 PM, Pavilion 4: Room 1.06
ICS Executive Committee Meeting
11:00 AM – 12:00 PM, Pavilion 4: Room 1.08
ICS Council Meeting
12:00 PM – 3:00 PM, Pavilion 4: Room 1.07

Monday, October 19th

Joint ISICR – ICS Board Meeting
6:00 AM – 7:30 AM, Lisbon Marriot Hotel
SLB Website Taskforce
12:00 PM – 1:30 PM, Pavilion 4: Room 1.03
PBL Interferon Source Luncheon
12:00 PM – 1:30 PM, Cafeteria: Private Space
SLB Publications Committee
12:00 PM – 1:30 PM, Pavilion 4: Room 1.01
PBL Interferon Source 20th Anniversary Celebration
7:30 PM – 11:30 PM, Offsite

Tuesday, October 20th

JLB Editorial Lunch
12:15 PM – 1:30 PM, Pavilion 4: Room 1.01
ISICR Editorial Committee
12:15 PM – 1:30 PM, Cafeteria: Private Space

Social Program

Opening Reception

Sunday, October 18th will mark the opening of the conference with the "Opening Reception" held in Pavilion 4 from 7:30 PM – 8:30 PM. Light Fare will be served and a cash bar will be available for refreshments. Please come and join your fellow attendees to celebrate the official opening of the program after the opening Keynote Lectures.

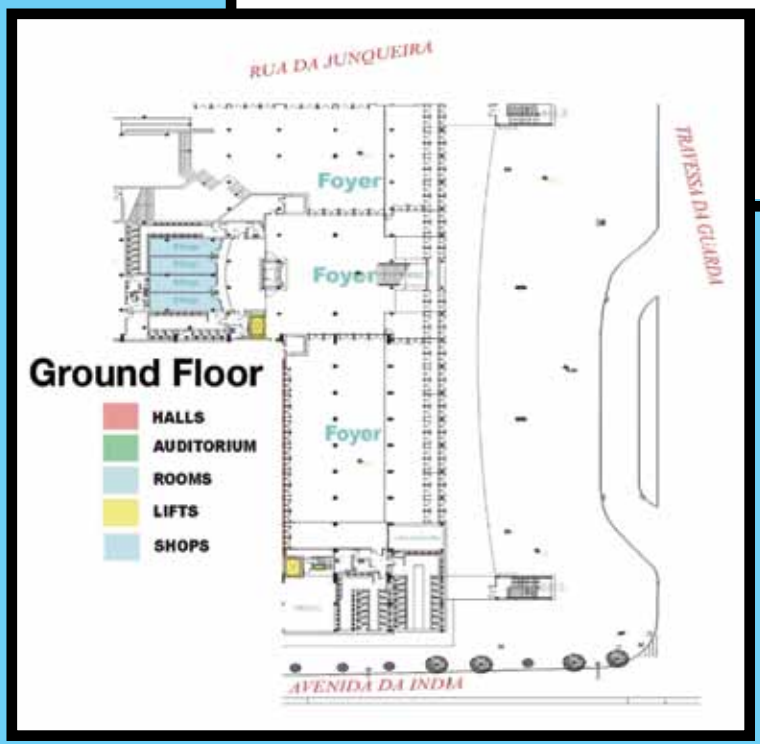
Student Mixer

Monday, October 19th will end with the Student Mixer sponsored in full by The Society for Leukocyte Biology. SLB truly values the participation of our more junior researchers and would like to foster further networking and community amongst our Student and Post-Doc attendees. Registered Students and Post-doc are welcome to join us at Trindade (instructions available at the registration area) for light fare and refreshments from 7:30 PM – 10:00 PM. While this event is free, you must visit the JLB Booth (4.18) to pick up a complimentary ticket.

Conference Banquet

Tuesday, October 20th will conclude with a celebration of the great gathering of the three societies at the National Agronomy Pavilion from 7:30 PM – 10:30 PM. Bus service from the conference hotels will be provided. Please see the conference bus schedule on page 67 for details on transportation for this event. A full dinner and entertainment will be provided along with a cash bar. Please don't miss this opportunity to celebrate the accomplishments of the three societies in organizing such a successful international meeting.

Conference Floor Plan



Conference Venue

The Tri-Society 2009 Conference is being held at the **Lisbon Congress Centre**. Built in 1989 and recently restyled and extended, the Lisbon Congress Centre is located in the historical and picturesque Belém quarter of the city that overlooks the River Tagus, and is just a few minutes from the city centre by any of a vast choice of modes of transport. The Congress Centre has a Cafeteria easily accessible near Pavilion 4 and is very close to many local eateries on the waterfront.

Local Hotels & Transportation

Airport
Aeroporto de Lisboa (about 4km northeast of the centre) is the major airport serving the Lisbon area.

There are two taxi stands within the perimeter of the airport, one at arrivals and the other at departures.

The fare on the taxi meter should read 2,50€ (daytime pick-up). Outside the city limits, city fares are charged per kilometer (km=0,42). 1,60€ is charged for the transportation of luggage or animals.

Before taking a taxi, inquire about the fare. An additional 20% is charged for services on Saturdays, Sundays and holidays and for nighttime service from 9pm to 6am.

TAP Portugal is the official carrier of the Cellular and Cytokine Interactions in Health and Disease Meeting, to be held in Lisbon, October 18-21, 2009 and is pleased to offer a discount to the participants who make their flight booking and buy their ticket exclusively through TAP Portugal's website, www.flytap.com.

Airport Transfer
• Carris buses
Listed below are the bus route numbers with the respective names of their 'end of the line' terminals. The airport stop is located mid-journey for these routes.

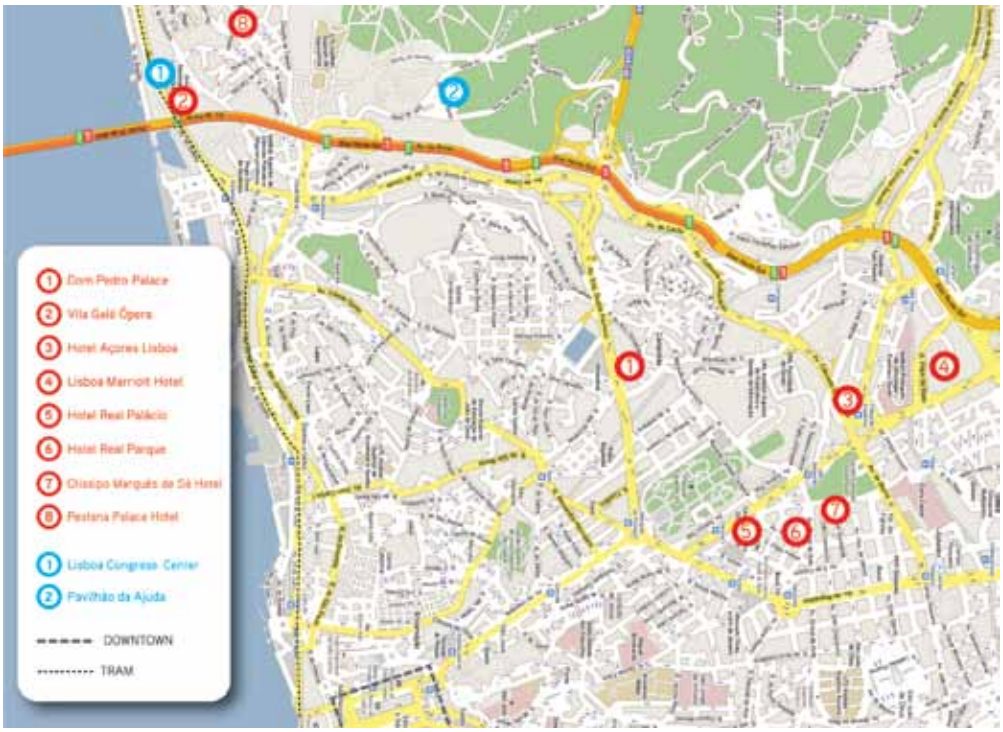
N.º 5 - Estação do Oriente / Aeroporto / Areeiro
N.º 22 - Portela / Aeroporto / Marquês de Pombal
N.º 44 - Moscavide / Aeroporto / Cais do Sodré
N.º 45 - Prior Velho / Aeroporto / Cais do Sodré
N.º 83 - Portela / Aeroporto / Amoreiras

The ticket may be purchased for 1.35€ from the driver as you board the bus.

• Aerobus (CARRIS Nº91)
Makes the run between Lisbon Airport and the city centre. Service begins at 07:45am and ends at 08:15pm. Buses pass every 20 minutes. The ticket may be purchased from the driver as you board the bus. Ticket for all-day travel: 3,50€

• Aeroshuttle (CARRIS Nº96)
Available everyday, every 30 minutes between 7AM and 11PM, the aeroshuttle connects Gare Oriente - Airport - Entrecampos - Sete Rios - Praça de Espanha.
Tickets (3,50€) can be purchased onboard and also in the Tourism Office at arrivals at the Aeroporto de Lisboa (public area).
Tel: +351 213 582 334. Website: www.carris.pt

• Rede Nacional de Expressos
This company operates throughout the country.
Tel: +351 707 223 344. Website: www.rede-expressos.pt



Trains
• Lisbon Metro - Although there is no direct connection to the airport, the nearest metro stations are 15 minutes away by bus (Gare do Oriente or Areeiro Stations).
Fare: 0,75€
Metropolitano de Lisboa Website: www.metrolisboa.pt

Local Public Transport
• Regular Fares: Adult fare (bus and métro): $2.75, 6 tickets: $12.00.
• The metro and bus Tourist Card: 1 day, $9.00; 3 days, $17.00.

* Note: fare rates and all information subject to change based on local services and availability.

Conference Bus Schedule

Morning Shuttle Service

Sunday, October 18th

Hotel Pick Up	Pick Up Time
Marriott (Society Council Meetings)	7:45 AM
General	11:30 AM, 1:30 PM
Pestana	11:30 AM, 1:30 PM
Real Parque	11:20 AM, 1:30 PM
Dom Pedro	11:30 AM, 1:40 PM
Real Palácio	11:30 AM, 1:30 PM
Olissippo M.Sa	11:30 AM, 1:30 PM
Açores Lisboa	11:30 AM, 1:30 PM

Monday, October 19th

Hotel Pick Up	Pick Up Time
Marriott	7:15 AM
Pestana	7:20 AM
Real Parque	7:20 AM
Dom Pedro	7:30 AM
Real Palácio	7:20 AM
Olissippo M.Sa	7:20 AM
Açores Lisboa	7:20 AM

Tuesday, October 20th

Hotel Pick Up	Pick Up Time
Marriott	7:30 AM
Pestana	7:30 AM
Real Parque	7:20 AM
Dom Pedro	7:30 AM
Real Palácio	7:30 AM
Olissippo M.Sa	7:30 AM
Açores Lisboa	7:30 AM

Wednesday, October 21st

Hotel Pick Up	Pick Up Time
Marriott	7:30 AM
Pestana	7:30 AM
Real Parque	7:20 AM
Dom Pedro	7:30 AM
Real Palácio	7:30 AM
Olissippo M.Sa	7:30 AM
Açores Lisboa	7:30 AM

Afternoon/Evening Shuttle Service

Sunday, October 18th

Congress Centre Pick Up To	Pick Up Time
Marriott	8:45 PM
Pestana	8:45 PM
Real Parque / D.Pedro	8:45 PM
Real Palácio	8:45 PM
Olissippo M.Sa	8:45 PM
Açores Lisboa	8:45 PM

Monday, October 19th

Congress Centre Pick Up To	Pick Up Time
Marriott	6:15 PM, 8:45 PM
Pestana	6:15 PM, 8:45 PM
Real Parque / D.Pedro	6:15 PM, 8:45 PM
Real Palácio	6:15 PM, 8:45 PM
Olissippo M.Sa	6:15 PM, 8:45 PM
Açores Lisboa	6:15 PM, 8:45 PM

Tuesday, October 20th

Congress Centre Pick Up To	Pick Up Time
Marriott	5:15 PM
Pestana	5:15 PM
Real Parque / D. Pedro	5:15 PM
Real Palácio	5:15 PM
Olissippo M.Sa	5:15 PM
Açores Lisboa	5:15 PM

Tuesday, October 20th

Banquet Transportation	Pick Up Time/Return Time
Marriott	7:00 PM / 10:30 PM
Pestana	7:00 PM / 10:30 PM
Real Parque / D. Pedro	7:00 PM / 10:30 PM
Real Palácio	7:00 PM / 10:30 PM
Olissippo M.Sa	7:00 PM / 10:30 PM
Açores Lisboa	7:00 PM / 10:30 PM

Wednesday, October 21st

Congress Centre Pick Up To	Pick Up Time
Marriott	6:30 PM
Pestana	6:30 PM
Real Parque / D.Pedro	6:30 PM
Real Palácio	6:30 PM
Olissippo M.Sa	6:30 PM
Açores Lisboa	6:30 PM

Touring Lisbon

ABOUT LISBOA

Lisboa´s location, spread over seven low hills, overlooking the river Tejo, once lured traders and settlers, and continues to be a stunning site.

Add to that the cultural diversity, a pleasantly temperate climate all year-round and a people that by longstanding tradition offer visitors a warm welcome.

Medieval Alfama is the charming and oldest part of the city with its maze-like streets, crowned by the impressive Castelo de São Jorge. The Baixa's commercial avenues lies just below. The elegant Chiado shopping area climbs away up another hill, next to Bairro Alto, home of much of the Lisbon nightlife.

The westernmost part of the city, Belém, was the birthplace of the Age of Discoveries and Parque das Nações (the 98 World Expo site) in the northeast side of the city is an area full of 21st century avant-garde architecture built on a most impressive river side site.

A VERY BRIEF HISTORY…

Lisboa dates back to pre-Roman times - legend has it that Ulysses founded the city, although it was more probably the Phoenicians. In its early years Lisbon was a constant battleground with Phoenicians, Greeks and Carthaginians taking turn to rule the city.

In 714 the powerful Moors arrived and, by fortifying the city, held out against Christian attacks for over 400 years. By 1147 the Moors' luck turned and the Christian Cruzaders recaptured Lisbon.

The 16th century was Portugal's short-lived golden era of sea exploration when riches were brought from across the oceans.

In the late 17th century the discovery of gold in Brazil saw Lisbon enjoy another luxurious period but this time it was cut short by the massive earthquake in 1755 which reduced the city to rubble.

In 1910 the monarchy fell and the first Portuguese Republic was proclaimed. Portugal's democratic phase lasted until 1926, when a military coup reduced Portugal to a period of totalitarian regime under the dictator António Salazar.

The costly colonial wars in 60`s and 70`s within African Portuguese territories, led to the “Carnation Revolution”, a nearly bloodless military coup on 25 April 1974. The new government instituted democratic reforms and granted independence to the African colonies in 1975. In 1986 Portugal became a full member of the European Union.

Tourism Resources:
The Visitors and Convention Bureau offers a wealth of information.

Turismo de Liboa\Visitors & Convention Bureau
Rua do Arsenal, 15
110-038 Lisboa
T: +351-210-312-700

Useful websites for planning your visit of the area:
www.visitlisboa.com
www.visitportugal.com

Notes

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IL-17F (h)	IL-6 (m, h)
IL-23 (m, h)	IL-8 (h)
TGFβ1 (m)	IL-10 (m, h)
	IL-12 (m, h)

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Schedule at a Glance

Sunday, October 18th

11:00 AM – 5:30 PM **Registration** – Ground Floor Foyer

12:30 PM – 2:15 PM **SLB Presidential Awards Presentations** – Auditorium 1

2:20 PM – 5:30 PM **Plenary Award Session** – Auditorium 1

4:30 PM – 8:30 PM **Exhibits** – Pavilion 4

5:30 PM – 7:30 PM **Joint Plenary Session 1:**
Opening Keynote Lectures – Auditorium 1

7:30 PM – 8:30 PM **Opening Reception** - Pavilion 4

Monday, October 19th

7:30 AM – 5:00 PM **Registration** – Ground Floor Foyer

8:30 AM – 6:30 PM **Exhibits** – Pavilion 4

8:00 AM – 9:30 AM **Joint Plenary Session 2:**
Pattern Recognition Receptors & Inflammation – Auditorium 1
Sponsored by PBL Interferon Source

9:30 AM – 10:00 AM **Coffee Break** – Pavilion 4 Sponsored by Invitrogen

10:00 AM – 12:00 PM **Concurrent Basic Science Symposia 1:**
Immunoregulation I – Auditorium 1

10:00 AM – 12:00 PM **Concurrent Basic Science Symposia 2:**
Inflammation and Cancer – Auditorium 2 Sponsored by Meso Scale Discovery

10:00 AM – 12:00 PM **Concurrent Immunopathogenesis Symposia 1:**
Immunopathogenesis I – Pavilion 5 A&B

12:00 PM – 1:30 PM **Lunch on your own**

1:30 PM – 3:00 PM **Joint Plenary Session 3:**
Anti-Tumor Immunity – Auditorium 1

3:00 PM – 3:30 PM **Coffee Break** – Pavilion 4 Sponsored by Amgen

3:30 PM – 6:00 PM **Concurrent Basic Science Symposia 3:**
Gene Activation – Auditorium 1

3:30 PM – 6:00 PM **Concurrent Immunopathogenesis Symposia 2:**
Immunopathogensis II – Auditorium 2 Sponsored by Celgene

3:30 PM – 6:00 PM **Concurrent Immunopathogenesis Symposia 3:**
Pathogen Manipulation of Cytokine Responses – Pavilion 5 A&B

6:00 PM – 8:30 PM **Focus Workshop: A Spotlight on: Interferon-lambda (IL-29)** – Pavilion 5 A&B Sponsored by Bristol-Myers Squibb

7:30 PM – 10:00 PM **Student/Post-Doc Mixer** – Trindade
Sponsored by The Society for Leukocyte Biology (SLB)

Tuesday, October 20th

8:00 AM – 5:00 PM **Registration** – Ground Floor Foyer

8:30 AM – 6:30 PM **Exhibits** – Pavilion 4

8:00 AM – 9:30 AM **Joint Plenary Session 4:**
New T-helper Subsets – Auditorium 1 Sponsored by Biogen Idec

9:30 AM – 10:00 AM **Coffee Break** – Pavilion 4 Sponsored by Biomonitor

10:00 AM – 12:00 PM **Concurrent Basic Science Symposia 4:**
Signaling Session I – Auditorium 1

10:00 AM – 12:05 PM **Concurrent Special Symposia 1: IFN in the Clinic:**
Immunotherapy of Multiple Sclerosis – Auditorium 2
Sponsored by Bayer Schering Pharma AG, Biogen Idec, Biomonitor, and Celgene

10:00 AM – 12:00 PM **Concurrent Special Symposia 2:**
Recent Advances – Pavilion 5 A&B

10:00 AM – 12:15 PM **Concurrent Basic Science Symposia 5:**
Immunoregulation II – Pavilion 5 C Sponsored by PBL Interferon Source

12:15 PM – 1:30 PM **Lunch On Your Own**

1:30 PM – 3:00 PM **Concurrent Basic Science Symposia 6:**
Neutrophil Biology – Auditorium 1

1:30 PM – 3:00 PM **Concurrent Basic Science Symposia 7:**
IFN-Stimulated Genes – Auditorium 2

1:30 PM – 3:00 PM **Concurrent Clinical Symposia:**
Biological Therapeutics – Pavilion 5 A&B

1:30 PM – 3:00 PM **Concurrent Immunopathogenesis Symposia 4:**
Inflammation & Pathogenesis – Pavilion 5 C

3:00 PM – 5:00 PM **Poster Session A** – Pavilion 4&5

7:30 PM – 10:30 PM **Conference Banquet**
National Agronomy Pavilion – Bus service provided from hotels

Wednesday, October 21st

8:00 AM – 2:30 PM **Registration** – Ground Floor Foyer

8:30 AM – 2:30 PM **Exhibits** – Pavilion 4

8:00 AM – 9:00 AM **Joint Plenary Session 5:**
The Macrophages in Health and Disease – Auditorium 1

9:00 AM – 9:30 AM **Coffee Break** – Pavilion 4
Sponsored by BD Biosciences

9:30 AM – 11:30 AM **Concurrent Immunopathogenesis Symposia 5:**
The Role of Tissue-Specific Macrophages in Chronic Disease Processes – Auditorium 1

9:30 AM –11:30 AM **Concurrent Basic Science Symposia 8:**
Signaling Session II – Auditorium 2

9:30 AM –11:30 AM **Concurrent Immunopathogenesis Symposia 6:**
Immunopathogensis III – Pavilion 5 A&B

11:30 AM – 12:30 PM **Lunch On Your Own –**
ISICR General Society Meeting – Auditorium 1
ICS General Society Meeting – Auditorium 2
SLB General Society Meeting – Pavilion 5 A&B

12:30 PM – 2:30 PM **Poster Session B** – Pavilion 4&5

2:30 PM – 4:30 PM **Concurrent Basic Science Symposia 9:**
Allergy and Mast Cells – Auditorium 1

2:30 PM – 4:30 PM **Concurrent Immunopathogenesis Symposia 7:**
Sensing of Fungal & Parasitic Infection and Host Response – Auditorium 2

2:30 PM – 4:30 PM **Concurrent Immunopathogenesis Symposia 8:**
Chronic Inflammatory Disease – Pavilion 5 A&B

4:45 PM – 6:15 PM **Joint Plenary Session 6:**
Closing Keynote Lectures – Auditorium 1