Future ISICR Meetings

2008 Meeting
Joint ISICR/ICS
Montreal, Canada
Oct. 12-16
www.cytokines2008.org

2009 Meeting
Joint ISICR/ICS/SLB
Lisbon, Portugal

2010 Meeting
Joint ISICR/IC
Chicago, Illinois

ISICR WWW Site

www.ISICR.org

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ISICR

INTERNATIONAL SOCIETY FOR INTERFERON AND CYTOKINE RESEARCH

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ISICR 2008 Seymour and Vivian Milstein Award Winner

Dr. Giorgio Trinchieri
Director, Cancer and Inflammation Program
Chief, Laboratory of Experimental Immunology
Center for Cancer Research
National Cancer Institute
Frederick, Maryland 21702-1201

Giorgio Trinchieri received his medical degree from the University of Torino, Italy, in 1973. He was a member of the Basel Institute for Immunology (Basel, Switzerland) and an investigator at the Swiss Institute for Experimental Cancer Research (Epalanges sur Lausanne, Switzerland). From 1979 to 1999 he was at Wistar Institute in Philadelphia and became Hilary Koprowski Professor and Chairman of the Immunology Program; he was also Wistar Professor of Medicine at the University of Pennsylvania. He was then director of the Schering Plough Laboratory for Immunological Research in Dardilly, France, and an NIH Fogarty Scholar at the Laboratory for Parasitic Diseases, NIAID, before becoming director of the Cancer and Inflammation Program (CIP) and chief of the Laboratory of Experimental Immunology at NCI in August 2006. As CIP director, he oversees the operations of two major NCI intramural laboratories, the Laboratory of Experimental Immunology and the Laboratory of Molecular Immunoregulation. These two laboratories constitute the major immunologic component of the CCR's inflammation and cancer initiative, which spans the NCI's campuses in Frederick and Bethesda and seeks to partner NCI's expertise in inflammation and immunology with its cutting-edge cancer etiology and carcinogenesis program. He has been interested for many years in the interplay between inflammation/innate resistance and adaptive immunity, and in the role of pro-inflammatory cytokines and interferons in the regulation of hematopoiesis, innate resistance and immunity. In 1989, his group at the Wistar Institute discovered Interleukin-12, and he has spent many years characterizing the molecular mechanisms of IL-12

(continued on page 2)

*(**Milstein Award**, cont. from page 1)*

production and action, and the role of this molecule in tumour immunity, infections and autoimmunity. His main focus of research is now the role of inflammation, innate resistance, and immunity in carcinogenesis, cancer progression, and prevention or destruction of cancer.

2008 ISICR Honorary Member Awards

George John Galasso, Ph.D., Sc.D.

George Galasso was born in New York City. He graduated from Cardinal Hayes High School in 1950 and Manhattan College in 1954. He then served in the US Army 1954-56 and subsequently went to the University of North Carolina where he obtained a Ph.D. degree in Microbiology in 1960. Following a Post-Doctoral fellowship he became Research Assistant Professor at the UNC Medical School.

In 1964 he became Associate Professor of Microbiology in the University of Virginia Medical School where he did research and taught Virology. In 1968 he was accepted into the Grants Associates Program of the NIH a highly selective training program of the NIH for Health Science Administrators and began his career as an administrator of scientific programs. In 1969 he was asked to initiate an Antiviral Research Program for the National Institute of Allergy and Infectious Diseases. This was a period of skepticism for the efficacy of antiviral agents to combat viral diseases. The initial goal was to determine whether interferon had a role in treating disease and to determine whether chemical agents could be used to treat viral diseases. Due to his efforts adenine-arabinoside was shown effective against herpes encephalitis, the first time an antiviral agent was successfully used to treat an ongoing serious viral disease. This showed that antiviral agents could indeed prove effective and paved the way for other antiviral agents. He also was a leader in interferon clinical trials and was instrumental in showing the role of interferon in zoster, laryngeal papilloma and the efficacy of interferon in hepatitis and contributed to its use against cancer. His efforts in this field of antiviral research have been internationally recognized. He served on the US-USSR science exchange program and headed delegations to the then USSR in the 70s. He served as Director of the World Health Organization (WHO) Collaborating Center for Interferon Reference and Research, and as a member on the WHO Expert Panel on Viral Diseases (Interferon and Antivirals). He was made an honorary faculty member of the Hubei Medical School, Wuhan, Peoples Republic of China where he participated in a virology course for representatives of all the provinces of China in 1983.

In 1973 he became Chief of the Infectious Diseases Branch with responsibility for development of vaccines and antivirals to combat all infectious diseases. In 1983 he became Associate Director for Extramural Programs of the NIH, with responsibility for NIH policies involving grants and contracts. He developed the first set of Conflict of Interest Rules in conjunction with the other research agencies of the government. He is the author of nearly 100 scientific articles and reviews. He has served on the editorial board of several scientific journals and as Review Editor for Antiviral Research. He edited several texts notably four editions of, *Antiviral Agents and Viral Diseases of Man, Practical Diagnosis of Viral Infections and Practical Guidelines in Antiviral Therapy*.

He was active in the early stages of the formation of the Interferon Research Society and the founder of the International Society for Antiviral Research (ISAR) in 1985 and was active in this Society even after retirement.

*(**Honorary Awards**, cont. from page 2)*

Dr. Paula Pitha-Rowe

Professor
The Sidney Kimmel Comprehensive Cancer Center
Johns Hopkins School of Medicine
Baltimore, MD 21231

Paula Pitha-Rowe has been interested in the effects viral infection on the expression of cellular genes and in the novel approaches to modulation of the antiviral and anti-inflammatory responses. She received her PhD from the Academy of Sciences, Prague, Czech Republic and did postgraduate training in the Institute of Organic Chemistry and Biochemistry, Prague and at the Johns Hopkins University School of Medicine. The overall goal of her group is to understand the molecular mechanisms that govern the innate immune response to infectious agents. There is growing evidence which indicates that the inflammatory response plays an important role in autoimmune disease and triggers carcinogenesis. Thus, identification of the critical cellular elements involved in the innate immune recognition of infectious pathogens may provide a lead to our understanding of the role of inflammatory responses in cancer and provide a new therapeutic target.
The long-term goal of Dr. Pitha-Rowe's research is to understand the cellular responses to infection and the relationship between viral pathogenicity and oncogenicity. Over the past several years, her group has identified three novel transcription factors, IRF-3, IRF-5, and IRF-7, and has shown that these factors serve as direct transporters of virus-mediated signaling. The research advances of her group have revealed a critical role of these factors in expression of the early inflammatory genes in infected cells. Her laboratory, together with other laboratories, has shown that these factors are also activated in response to bacterial infection upon binding of ligands to Toll receptors. In their recent studies, her group has shown that over-expression of IRF-5 in B cells induces p21waf and proapoptotic genes in a p53-independent manner. Altogether, these data indicate that the genes of the IRF family play a critical role in the differentiation and maturation of lymphoid cells, apoptosis and activation of early inflammatory cytokines. Whether IRFs play a role in the innate response to HIV-1 infection also is being examined. The importance of IRFs in innate immunity has been recently demonstrated by the observation that many viruses target their function as a part of viral mimicry. The Kaposi's sarcoma virus (KSHV) that is associated with Kaposi's sarcoma and B cell lymphoma encodes four IRF homologues that associate with cellular IRFs and modulate their function, as well as the functions of other cellular proteins. Recent data from Dr. Pitha-Rowe's group show that one of these KSHV-encoded IRFs-vIRF-3-is a nuclear protein that associates with the c-myc suppressor and thus may contribute to KSHV/AIDS-associated tumorigenicity.

Clinical Research
The recent findings of Dr. Pitha-Rowe's laboratory have potential clinical application. First, Dr. Pitha-Rowe has shown that IRFs can serve as an effective adjuvant of DNA-stimulated immune responses to a DNA-encoded viral antigen. These adjuvants will be eventually tested in clinical settings, both for viral infection (HIV-1, HCV) and tumor vaccines. Second, she has shown that IRF-5 has proapoptotic activity that is independent of p53. Thus, IRF-5 expression may increase sensitivity of p53-defective tumors to proapoptotic drugs. Finally, she has recently shown that an IFN-induced protein (ISG) is an effective inhibitor of HIV-1 replication and enhances the virus-mediated antiviral response.

(Adapted from the Johns Hopkins University School of Medicine and the Sidney Kimmel Cancer Center websites).

Seymour & Vivian Milstein Young Investigator Award Winners

Dr Toby Lawrence
Senior Lecturer, MRC New Investigator
Centre for Cancer & Inflammation
Institute of Cancer and CRUK Clinical Centre
Barts and The London School of Medicine and Dentistry
Charterhouse Square
London EC1M 6BQ, UK

Dr. Lawrence gained his PhD in experimental pathology from the University of London at the William Harvey Research Institute under the mentorship of the late Prof Derek Willoughby during which time he developed research interests in endogenous anti-inflammatory mechanisms and the resolution of inflammation. During his early post-doctoral studies Dr. Lawrence identified an important role for the NFkappaB pathway in the resolution of acute inflammation. With the award of post-doctoral fellowships from the Arthritis Research Campaign and the Wellcome Trust Dr. Lawrence continued his studies at the University of California San Diego, in the Laboratory of Signal Transduction and Gene Regulation under Prof Michael Karin, where he gained training in the use of molecular genetics to study the role of cell signaling pathways in the regulation of inflammation and immunity. These studies focused on the role of the IkappaB kinase (IKK) in the biology of inflammation; using tissue specific gene targeting to establish the specific role of IKK in the inflammatory response. In 2004 he returned to London as a Lecturer at Imperial College London in the Kennedy Institute of Rheumatology. Dr. Lawrence joined the Institute of Cancer in 2006 as a Senior Lecturer and established the Inflammation Biology group in the Centre for Cancer & Inflammation. The major research focus of the group is to understand the fundamental mechanisms by which inflammation promotes cancer. With particular reference to the role of stromal and inflammatory cells in carcinogenesis

Dr. Tao Lu
Project Staff
Cleveland Clinic Foundation
Cleveland, OH USA

Dr. Tao Lu received her Ph.D. degree in Molecular Cellular Biology from the Medical University of Ohio at Toledo, Ohio, USA, in 2001. Since then, she has been working with Dr. George Stark at the Cleveland Clinic Foundation. Her current position is Project Staff in the Dept. of Molecular Genetics. Dr. Lu's major research interest is NFkB, cancer cell signaling and cytokines. In the Stark lab, she has demonstrated that constitutive activation of NFkB in cancer is almost always caused by the constitutive secretion of one or more factors that activate NFkB in an autocrine fashion from outside the cell. She found that TGF-beta activates NFkB in a subset of tumors and mutant cell lines, and that the basis of increased secretion is an increased steady state level of the mRNA. In addition she found that TGF-beta activates NFkB by recruiting the IL-1 receptor and, conversely, that IL-1 activates SMADs by recruiting the TGF-beta receptor. This unusual crosstalk is argued to be especially important in the immediate vicinity of tumors or at sites of inflammation, where the concentrations of TGF-beta or IL-1 are likely to be high. Her current work focuses on how NFkB may be regulated by negative feedback mechanisms and she has employed a novel method in which retroviral vectors are used to insert strong promoters more or less randomly into the genomes of mammalian cells as a method for identifying genes that regulate NFkB.

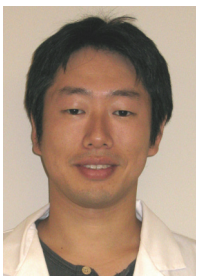
Dr. Lu is a recipient of 2 "Innovator Awards" from the Cleveland Clinic Foundation and is a past recipient of an ISICR Travel Award.

*(**Young Investigators Awards**, cont. from page 4)*

Dr. Cesar Munoz-Fontela

Postdoctoral Fellow
Mt. Sinai School of Medicine
New York, NY USA

Dr. Cesar Munoz-Fontela obtained his Ph. D. in viral oncology under the supervision of Dr. Carmen Rivas, at the Complutense University of Madrid (Spain). His research was focused on understanding the mechanisms by which human herpesvirus 8 (HHV-8/KSHV) counteracted the host immune response and its relationship with cancer development. In KSHV-dependent lymphomas only a small subset of latent viral genes are expressed, suggesting that the transforming potential of the virus relies on the expression of such genes. His work revealed that one of these KSHV latent genes, K10.5, encoded a multifunctional protein, LANA2/v-IRF3, with the ability to target several cellular pathways such as p53, the double-stranded RNA-dependent protein kinase (PKR), the Rb family of pocket proteins, 14-3-3 and FOXO3a. Furthermore, he discovered that LANA2/v-IRF3 is the only Interferon (IFN)-antagonist protein known so far with the ability to directly bind tubulin and prevent taxol-dependent microtubule stabilization. These findings, helped to explain why patients undergoing KSHV-derived lymphomas fail to respond to taxol chemotherapy. As part of his graduate training, he was also involved in research related to the antiviral activity of IFN-inducible tumor suppressor genes. In collaboration with Dr. Manuel Serrano (CNIO, Spain) he and his laboratory established that mice that harbor an extra copy of the p53 gene are more protected from infection with VSV and Vaccinia virus due to an enhanced apoptotic response. Moreover, in collaboration with Dr. Serrano and Dr. Mariano Esteban (CNB, Spain), his laboratory demonstrated for the first time that ARF, another IFN-inducible tumor suppressor, had a strong antiviral effect due to its ability to directly bind to Nucleophosmin. By doing so, ARF prevents nucleophosmin-dependent inactivation of PKR. He joined Dr. Stuart Aaronson's lab at Mount Sinai School of Medicine (New York) in 2006 as a post-doctoral fellow, where he is investigating the role of tumor suppressor genes on IFN-dependent antiviral immunity. In collaboration with Dr. Adolfo Garcia-Sastre (MSSM, New York), he discovered that IRF9 is a novel p53 direct transcriptional target. Through direct up-regulation of IRF9, p53 enhances IFN-dependent activation of antiviral genes in response to viral infection. Moreover, p53 participates in a positive feed back loop between IFN signaling and production, helping to establish an IFN-dependent antiviral state in bystander cells undergoing viral infection. His ongoing projects also indicate that tumor suppressor genes, such as p53 and Rb, are frequent targets not only for oncoproteins from DNA viruses, but also for IFN-antagonist proteins from RNA viruses such as Influenza NS1 or Ebola VP35. These findings further point to the role of tumor suppressor genes in innate antiviral immunity.



Dr. Takeshi Saito

Senior Fellow
Department of Immunology
University of Washington
School of Medicine
Seattle, WA USA

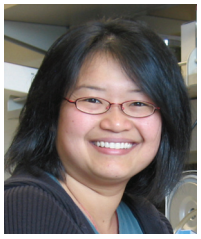
Dr. Takeshi Saito received his medical and scientific training at training at Showa University, School of Medicine, Tokyo (2004). His Ph.D. training was conducted in the Division of Internal Medicine, Department of Gastroenterology under Prof. Keiji Mitamura, and focused on defining the viral genetic determinants conferring resistance or sensitivity of hepatitis C virus to interferon therapy. After completing his doctoral studies, he received further training as a clinical hepatologist/general gastroenterologist, and practiced medicine under the mentorship of Professor Michio Imawari, during which time he became interested in the basic mechanisms of liver diseases. Dr. Saito has pursued this interest during his post-doctoral training in the laboratory of Dr. Michael Gale Jr. where he is currently a Senior Fellow in the Department of Immunology at the University of Washington School of Medicine. Dr. Saito's career goal is to become a physician-scientist and conduct translational research of liver-related diseases. While in Dr. Gale's laboratory, Dr. Saito has been working to understand how host innate

*(**Young Investigators Awards**, cont. from page 5)*

immune receptors sense HCV infection and initiate the innate immune response that involves type I interferons and proinflammatory cytokines that regulate hepatic immunity. Dr. Saito's research has revealed how hepatitis C virus triggers innate immunity through processes of pathogen recognition receptor (PRR) sensing of pathogen associated molecular patterns (PAMPs) during infection. His work has defined 1) Retinoic acid inducible gene I (RIG-I) is the major PRR that senses HCV RNA in the host cell; 2) the C-terminal repressor domain of RIG-I as the off/on switch of innate immunity against hepatitis C virus and other RNA viruses; 3) the specific RNA PAMP signature recognized by RIG-I and encoded in the HCV genome; 5) Conserved PAMP motifs within the genome of RNA viruses that trigger RIG-I-dependent signaling of innate immunity. Dr. Saito's studies have shown that antiviral immunity is triggered by sequence-specific RNA recognition by RIG-I, and provides novel insights into the immune-stimulatory processes induced by the RIG-I/PAMP interaction. His current interest is to apply his research toward improving adjuvant and vaccine designs to drive protective immune responses against pathogenic viruses.

Christina Fleischmann Award Winner

Special thanks to the Fleischmann Foundation for the continuing support of this award



Dr. Yueh-Ming (Ming) Loo
Acting Instructor
Dept. of Immunology
University of Washington
Seattle, WA USA
Email: looy@u.washington.edu

Dr. Yueh-Ming Loo is an Acting Instructor in the Department of Immunology at the University of Washington School of Medicine. Her research in the laboratory of Dr. Michael Gale Jr. is focused on understanding the mechanisms by which RNA viruses trigger and control innate immune signaling through the cellular RIG-I/IPS-1 pathway. A major focus of her studies to define the role of IPS-1 and the nature of the IPS-1 signalosome that mediates interferon production and innate immunity during RNA virus infection.

Ming received her Ph.D. in Microbiology and Immunology from the State University of New York at Buffalo where her training with Dr. Thomas Melendy focused on virus-host interactions required for papillomavirus DNA replication. She pursued her post-doctoral training with Dr. Michael Gale, Jr. at the University of Texas Southwestern Medical Center at Dallas where she independently identified IPS-1, otherwise known as Cardif, VISA or MAVS as a novel adaptor required for signaling interferon production by the RIG-I-like helicases RIG-I and MDA5 during RNA virus infection. Her studies showed that Hepatitis C virus (HCV), a human pathogen of global public health concern expresses a protease NS3/4A that specifically targets and cleaves IPS-1 to release it from the outer membrane of the mitochondria. HCV is thus able to efficiently abrogate interferon production and evade the host innate immune response to establish chronic infections. Confocal microscopy and biochemical analyses of patient liver biopsies showed that NS3/4A cleavage of IPS-1 occurs in vivo in the human patient, and can vary from patient to patient such that those who exhibit little or partial IPS-1 cleavage were correspondingly able to mount a more robust innate immune response during infection, thus promoting viral clearance. NS3/4A-specific protease inhibitors not only rescued IPS-1 from cleavage, but further restored interferon production and the innate antiviral response in infected cells, thus providing strong evidence identifying IPS-1 as a potential therapeutic target for HCV infection. Additionally, she has shown that IPS-1 is essential for establishing innate immunity to many RNA viruses that are also significant human pathogens. The viruses, which were derived from different virus families and whose RNA genomes harbor distinct PAMP features were shown to differentially engage RIG-I and MDA5 to trigger innate immunity. In addition, her collaborative studies have shown that RIG-I regulates cell permissiveness to HCV infection, and that RIG-I signaling is regulated by protein antagonists encoded by the Ebola virus and influenza viruses. Overall,

*(**Fleischmann Award Winner**, cont. from page 5)*

her research has revealed IPS-1 as an essential signaling adaptor of the RIG-I pathway regulated by HCV infection in vivo, and has revealed distinct mechanisms by which different RNA viruses trigger and control RIG-I signaling.

Dr. Loo has co-authored several scientific publications and commentaries, and has published a book chapter describing the mechanisms by which RNA viruses regulate host innate immune defenses. She is a member of the International Society for Interferon and Cytokine Research and the American Society for Virology, and serves as an ad-hoc manuscript reviewer for various scientific journals.

Seymour & Vivian Milstein Travel Award Winners

Through the generosity of the Seymour and Vivian Milstein Foundation, the ISICR is pleased to designate almost $50,000 for the 2008 Travel Awards. Winners come from more than 10 countries, clearly demonstrating the international focus of our society.

Sabrina Brzostek
Daniel Burke
Arindam Chakrabarti
Mounira Chelbi-Alix
Hui-Chen Chen
Venugopalan Cheriyath
George Christophi
Ahmet Civas
Silvia Correia
Ana Costa-Pereira
Brian Doehle
Beihua Dong
Jennifer Drahos
Eugene Friedman
Carole Galligan
Ana Gamero
Yunfei Gao
FuiGoon Goh
Jennifer Gommerman
Geetanjali Gupta
Deborah Hodge
Wei-Chun Huang-Fu
Katharine Irvine
Brendan Jenkins
Danlin Jia
Sven Klaschik
Thomas Krausgruber
Malathi Krishnamurthy
Helle Kristiansen
Thomas Kuri
Virginia Maina
Atsuko Masumi
Zora Melkova
Eliane Meurs
Reem Mohammed
Markus Mordstein
Anna Överby
Zulema Antonia Percario
Courtney Plumlee
Maya Poffenberger
Zoran Popmihajlov
Chiara Porta
Tracy Putoczki
M R Sandhya Rani
Giovanna Romeo
Shamith Samarajiwa
Ayca Sayi
Emmanuel Thomas
Scott Thomson
Chafia Touil-Boukoffa
Deborah Vestal
Angela Walker
Ajay Wanchu
Wei-Bei Wang
Joanna Wegrzyn
Christine White
Mumtaz Yaseen
Ying Zheng

The ISICR wishes to express its deepest appreciation to the Milstein Family for their continuous support of the Society. The ISICR is honored by the generosity of the Milstein Family in being able to recognize the scientists whose work has been instrumental in understanding the role of interferons in the host response to infectious disease and cancer, through the Milstein Award and Milstein Young Investigator Awards. Furthermore, the Milstein Travel Awards provide an important mechanism for enabling the future generation of scientists to attend and participate in the Society, thus ensuring a continuum of scientific excellence in interferon and cytokine research.

ISICR OFFICERS for 2009-2011

Secretary 2009-2011 - Tom Hamilton
Treasurer 2009-2011 - Robert Friedman

NEW ISICR MEMBERS

We welcome all the new members to the ISICR and encourage their participation in the annual meeting as well as ISICR committees and initiatives.

Ergin Ayaslioglu
Kirikkale Univ, Ankara, Turkey

Christine Brostjan
Medical Univ of Vienna, Vienna, Austria

Tilmann Buerckstuemmer
Rsch Ctr for Molecular Medicine, Vienna, Austria

Saurabh Chattopadhyay
Cleveland Clinic, Cleveland, OH

Ting Chen
Natl Taiwan Univ College, Taipei, Taiwan

Silvia Correia
Inst Gulbenkian Ciencia, Oeras, Portugal

Tyler Curiel
Univ Texas Health Sci Ctr, San Antonio, TX

Angela Drew
Univ of Cincinnati, Cincinnati, OH

Jarmila Eajicova
Charles Univ, Prague, Czech Republic

Mona El Gamal
Rsch Inst Univ EL Minufiya, Cairo, Egypt

Volker Fensterl
Cleveland Clinic, Cleveland, OH

Jamie Flammer
Weill Cornell Med Col, New York, NY

Yunfei Gao
Univ of Toronto, Toronto, Canada

Fui Goh
Imperial College, London, UK

Tom Imamichi
National Cancer Institute-Frederick, Frederick, MD

Madhulika Jupelli
Univ of Texas - San Antonio, TX

*(**New Members**, cont. from page 8)*

Sven Klaschik
National Cancer Institute-Frederick, Frederick, MD

Shyam Kottilil
National Institute of Allergy & Infectious Disease, Bethesda, MD

Thomas Krausgruber
Imperial College London, UK

Vijay Kumar
Panjab Univ, Chandigarh, India

Alain Lamarre
INRS-Institut Armand-Frappier, Univ of Quebec, Quebec, Canada

Evert Lamme
Merck Serono International SA, Geneva, Switzerland

Ahmed Lasfar
New Jersey Medical Sch - UMDNJ, Newark, NJ

Vito Lauta
Univ of Bari Med Sch, Bari, Italy

Wen Li
Univ of Sydney, Sydney, Australia

Vincent Lombardi
Whittemore Peterson Inst, Reno, NV

Sanna Makela
National Public Hlth Inst, Helsinki, Finland

Douglas McCarthy
Univ of Toronto, Toronto, Canada

Giuliana Medrano
Arkansas State Univ, Jonesboro, AR

Herwig Moll
Medical Univ, Vienna, Austria

Angel Morrow
National Institute of Allergy & Infectious Disease, Bethesda, MD

Cesar Munoz-Fontela
Mount Sinai Sch of Med, New York, NY

Maya Poffenberger
Univ of British Columbia, Victoria, British Columbia, Canada

Malek Ahmadi Pour
Institut Pasteur, Paris, France

Tracy Putoczki
Ludwig Inst for Cancer Rsch, Melbourne, Victoria, Australia

Mahboob Qureshi
Touro Univ Nevada, Henderson, NV

Westley Reeves
Univ of Florida, Gainesville, FL

Takeshi Saito
Univ of Washington, Seattle WA

Sergey Smirnov
New Jersey Medical Sch - UMDNJ, Newark, NJ

Esther Tarrab
INRS-Institut Armand-Frappier, Univ of Quebec, Quebec, Canada

*(**New Members**, cont. from page 9)*

Nick Underhill-Day
Univ of Birmingham, Birmingham, UK

Hong Wang
Nomell Chemical Senses Ctr, Philadelphia, PA

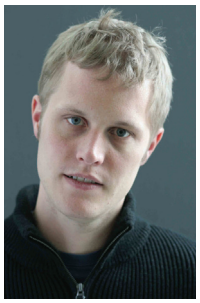
Hui Xiao
Cleveland Clinic Foundation, Cleveland, OH

Yingxin Xu
United BioSource Corp, Concord, MA

Angela Walker
Univ of Missouri-Columbia, Columbia, MO

Thomas Wynn
National Institute of Allergy & Infectious Disease, Bethesda, MD

New Member Minibios



Tilmann Bürckstümmer, Ph.D.
Senior Postdoctoral Fellow
CeMM, Research Center for Molecular Medicine
of the Austrian Academy of Sciences
Vienna, Austria

Tilmann Bürckstümmer obtained his PhD in biochemistry from the Robert-Koch-Institut in Berlin, Germany. His work there was focused on understanding the interplay between Hepatitis C virus and the host. Since 2005, Tilmann is a postdoctoral fellow in the laboratory of Giulio Superti-Furga where he developed a method to analyze protein-protein interaction in mammalian cells. He has recently applied this method to characterize the molecular machine associated with TANK-binding kinase 1 (TBK1), work that lead to identification of DDX3 as an effector of TBK1. His current research is focused at understanding the pathway that triggers interferon-production at different levels (cytoplasmic nucleic sensors, signaling intermediates, transcription factors) to provide a more comprehensive understanding of the underlying signaling events.

Christine Brostjan, Ph.D.
Associate Professor
Medical University of Vienna
Department of Surgery
Research Laboratories AKH 8G9.13
Vienna General Hospital
Waehringer Guertel 18-20
A-1090 Vienna, Austria

Christine Brostjan, Associate Professor for Vascular Biology at the Medical University of Vienna, has placed her scientific focus on endothelial cell biology. Starting in the field of xenotransplantation and immunosuppression (PhD work at the Deaconess Hospital, Harvard Medical School) she then moved to the area of tumor angiogenesis and established her research group at the Vienna General Hospital. More recent work has led her to investigate the endothelial response to interferon and TLR ligands.

Silvia Correia
Infection and Immunity Lab
Instituto Gulbenkian Ciencia
Oeiras, Portugal

Silvia Correia obtained her undergraduate and master degrees from Leicester University and Sheffield University, UK, respectively. She has since returned to Portugal where she is now finishing her PhD degree on the identification of novel viral genes that modulate interferon (IFN) responses. So far she has identified two unassigned genes: one from African swine fever virus that inhibits the induction of IFN expression and the other from murine-gammaherpesvirus-68 that inhibits both the induction of IFN expression and its signaling through the receptors.

*(**New Members Mini Bios**, cont. from page 10)*

Professor Alain Lamarre, Ph.D.

Immunovirology Laboratory
INRS-Institut Armand-Frappier
Institut national de la recherche scientifique
531, boulevard des Prairies
Laval (QC) Canada H7V 1B7

After the completion of his Ph.D. in Immunovirology at the Institut Armand-Frappier, Dr. Lamarre received postdoctoral training with Nobel laureate Rolf M. Zinkernagel at Zurich's Institute of Experimental Immunology in Switzerland. He then returned to Canada to head the Immunovirology Laboratory of the Institut Armand-Frappier, INRS, Université du Québec where he holds the Jeanne and J.-Louis Lévesque Chair in Immunovirology of the J.-Louis Lévesque Foundation. The main interest of his laboratory is to better understand the mechanisms that influence the establishment of viral persistence both in murine infection models and in human diseases. His group is particularly interested in studying the influence of B cell repertoire diversity on the induction of T cell responses against Lymphocytic choriomeningitis and Hepatitis C viruses. Another major focus of his laboratory aims at defining the mechanisms responsible for the late appearance of neutralizing antibodies in chronic viral infections. He also plays an important role in a number of international collaborations aimed at developing innovative vaccines against chronic viral infections and cancer based on the immunomodulatory properties of plant virus-like particles.

The ISICR newsletter - 15 years old

Howard Young

The ISICR newsletter is now in its 15th year of publication. I hope that members find the newsletter to be a useful benefit of society membership. It is the goal of the Associate Editors and myself to have the newsletter be something you read and pass on to others (hopefully enticing them to join the ISICR) and if printed, kept as a reference (at least for the recipes) rather than tossing it into the recycling bin. We have tried hard not to take ourselves too seriously and hope to continue to include items that might make you laugh or at least crack a smile (can you think of another scientific newsletter that consistently contains weird humor and cartoons?). Having created/edited newsletters for my college fraternity and when I worked at Life Technologies, I decided to start the ISICR newsletter in 1994. Chris Czarniecki was my first co-editor and I received strong encouragement for this effort from Sid Pestka. For a few years we published 4 times/year but that got to be more than I could handle so we now publish 3 times/year (Spring, at the time of the ISICR Annual Meeting and after the meeting or whenever the committee chairs get me their meeting minutes). Some of the features in the current issue originated back in the first and second years of publication (Clinical Trials, WWW, Biotech Briefs, Reviews of Interest) while other features (e.g. New Member Minibios) came along later. Not everything has worked, as only a few members use the newsletter to advertise open positions, the attempt to use the newsletter to help members search for collaborators on certain projects or unique reagents never caught on, no one submitted helpful techniques relevant to interferon/cytokine biology (probably because they want to publish them in a journal that they can list on their CV) and spontaneous submissions of items to include are rare. However, we are always open to new ideas and features that will enhance the value of every issue (including money in each issue was suggested but ruled out when we determined that Monopoly $ could not be used to pay for pipette tips or recombinant cytokines). As Editor-in-Chief I have been

(***Newsletter**, cont. from page 11*)

assisted by many Associate Editors throughout the 15 years of publication and I am grateful for all their efforts. Currently, Hannah Nguyen and Thomas Tan have worked with me since 1999 (Hannah) and 2001 (Thomas) and the contributions of both of these dedicated individuals are critical for the success of each and every issue. In fact without their help, I would probably have even less hair on the top of my head (I know people are thinking that he doesn't have any hair on the top of his head so how could he have less. Consider that one of the mysteries of life).

As always, I invite all ISICR members to contribute material and/or ideas for any newsletter issue and would welcome anyone who would like to become an Associate Editor. The pay isn't great but it looks good on your CV. All back issues are on the ISICR website so they are available at any time in order to see what we have done over the years. Remember, we can be bribed with chocolate so don't hesitate to send us material that highlights any of your recent achievements.

With the continued support of the ISICR, we'll keep marching on. While I may not be around 15 years from now, hopefully the ISICR and the newsletter will still be going strong.

THE ISICR NEWSLETTER

VOLUME 1 NUMBER 1

We are happy to launch the ISICR newsletter. We anticipate publishing 4 times/year and providing useful information to the ISICR membership. There are a number of categories in which we would welcome input from the membership. These include:

1. Listing of active clinical trials utilizing interferons/cytokines
2. Recent approvals of interferons/cytokines for clinical use or diagnostic testing
3. Teaching articles: For example, an article on in situ staining of interferons/cytokines, with a list of antibodies that work in the procedure, would be most welcome.
4. Positions open/Positions wanted
5. Request for reagents/request for help- Having trouble finding a cDNA clone or an antibody or having problems with a procedure? We will list your request with your phone/fax/e-mail address.
6. Business briefs- bullet summaries of recent business actions which relate to the interferon/cytokine field.
7. Meeting announcements
8. ISICR member notes (Promotions, position changes, awards, etc)
9. Humor

We welcome your ideas and suggestions for this newsletter. Please contact us with your thoughts. Information for the next newsletter should be sent to either editor by November 1, 1994.

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Happy 15th year Anniversary to the ISICR Newsletter!


Hannah Nguyen

In commemoration of 15 years of what I think has become a very resourceful magazine and a symbol of what I believe is a united and happy research society, here are 15 bits of trivia related to our newsletter:

1. How the newsletter started: as quoted by Howard Young, "…15 years ago I suggested to Sid Pestka that we should have a newsletter and he replied that I should start one."

2. Number of pages of first issue: 4 (1st page displayed for your entertainment just above this article with the complete issue available on the ISICR website under Newsletter Archives)

3. Average number of pages of last 3 issues: 32

4. Average number of issues published per year: 4 up to 1998, 3 since then

5. Number of issues published since the newsletter's inception: 45

6. Year the newsletter converted to color print: 1999

7. Past and current Associate Editors: Christine Czarniecki, Gerald Sonnenfeld, Bratko Filipic, Pat Fitzgerald-Bocarsly, Paul Drew, Hannah Nguyen, Venky Ramakrishna, Seng-Lai (Thomas) Tan.

8. ISICR Cheerleading traits of newsletter: Award winner listings, interviews of award winners, meeting announcements and minutes, ISICR member news updates, welcoming new ISICR members

9. Topics which have stuck through thick and thin since the newsletter's inception: clinical trial listings, positions open, corporate sponsor acknowledgments, biotech briefs, meeting announcements, lab-related interferon and cytokine resources and reagents, ICISR member news updates, humor

(***15th Anniversary**, cont. from page 12*)

10. More recently added topics: ISICR President's message, special topic interviews of ISICR members, new member minibios, tourist info for meeting venues, ISICR Slide Repository

11. Number of Reviews of Interest listed since the newsletter's inception: 583

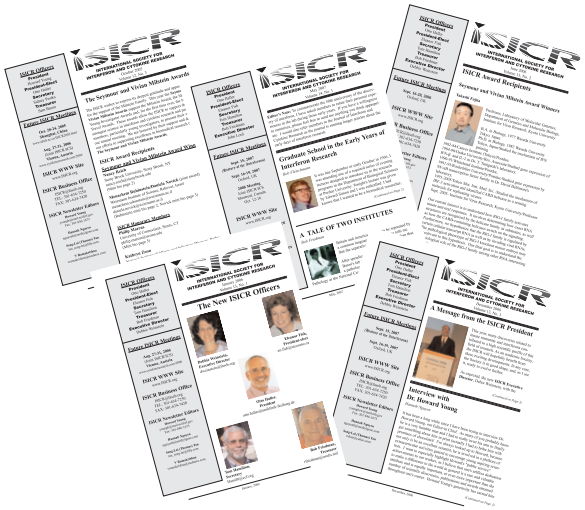
12. Number of Resource Web sites listed since the newsletter's inception: 424

13. Number of chocolate-related blurbs listed since the newsletter's inception: 10 (V2.4, 4.3, 4.4, 5.2, 7.1, 8.3, 10.2, 11.1, 12.3, 13.1)

14. Number of recipes included in the newsletter: 10 (V2.4, 3.2, 3.4. 4.3, 7.2, 8.1, 9.1, 11.1, 11.3, 13.2)

15. Most important common denominator for all issues: Howard Young, who has contributed by a long shot way more than the rest of us to this newsletter. Without him we would not be enjoying such top-quality reading material for this many years. I say we send him (at least) 15 boxes of chocolates.

Thank-you Howard!!!!

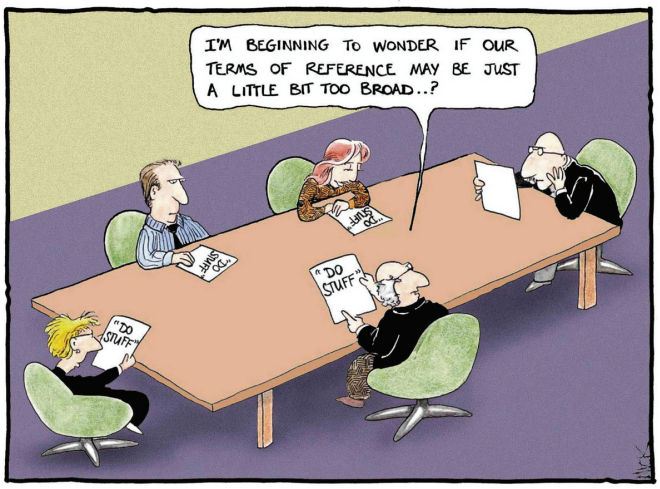


THE ISICR SLIDE REPOSITORY

Ever see a slide in a talk that you wish you could use for your own presentation? Well now this may be possible through the ISICR Slide Repository. Members can now go in and post slides that they have developed or download slides that others have provided to the membership. **OVER 470 SLIDES ARE NOW AVAILABLE!!!!!!** For this member only feature, you need to have your member number so if you are not sure what that is, please contact the membership office. We urge members to upload general slides that other members can use for lectures, classes, seminars, etc. Slides are not to be changed without permission from the donor and all copyright permissions must be obtained. The repository now has a useful search capability that allows you to find slides on a particular topic. If you have trouble uploading or downloading slides, please contact Howard Young at youngh ow@mail.nih.gov.

PLEASE CONSIDER CONTRIBUTING YOUR SLIDES. The success of this initiative depends upon you, the membership!!!!

Lab Meetings



Cartoon by Nick D Kim, nearingzero.net. Used by permission. This image is available in the ISICR slide repository.

Availability of Interferon and Cytokine Reference Reagents, Antisera, and Standards

Sidney E. Grossberg, Chairman, ISICR Standards Committee

Investigators, pharmaceutical manufacturers, and commercial laboratories should be aware of the new National Institutes of Health repository for cytokine reference reagents, antisera, and some World Health Organization (WHO) International Standards, for which new request procedures, explained below, are now required. Such materials are essential for the standardization of cytokine products, as well as in bioassays for conversion of Laboratory Units to International Units, neutralization assays, establishment of relative sensitivity of bioassays, immunologic identification of cytokines, among other uses. As before, the National Institute for Biological Standards and Control (NIBSC), www.nibsc.ac.uk (South Mimms, Hertfordshire, UK) remains an excellent source for cytokine reagents and WHO International Reference Preparations

For many years the U.S. National Institute of Allergy and Infectious Diseases (NIAID) had maintained a repository for interferon standards, antisera, and some WHO International Reference Preparations that had been prepared for the NIH. These had been maintained by contract with different companies, e.g., KamTek. More than a year ago, such materials were transferred along with many other types of biological materials to BEI Resources (Biodefense and Emerging Infections Resources Repository), which was established to control under a single authority the distribution of all reference biological materials, including those agents that might be used for criminal, terrorist, or other nefarious purposes, in addition to antisera and other benign reference reagents, such as interferons. BEI Resources has contracted with the American Type Culture Collection (ATCC) for the distribution of these materials, but approval to receive materials must first be obtained by application to BEI Resources.

General information and instructions for application are available at www.beiresources.org (previously listed in the ISICR Standards Committee meeting minutes as www.bioresources.com). You and your institution must first apply to BEI Resources to obtain approval before you will be able to obtain materials of any sort. Although we have been told that their application procedure has been improved, it may still be slow. You should obtain and complete the required forms listed at their registration page, http://beiresources.com/registration/index.cfm. The Material Transfer Agreement (MTA) that your institution must file with the application expressly forbids sharing materials outside the institution. The Indemnification clause about liability stated in the standard form MTA can be modified, but you or your institutional official may need to negotiate modifications in the language if it conflicts with your institutional rules. In principle, this should be resolved without too much difficulty for Biocontainment Level 1 class of materials, to which interferons and antisera belong. Once the approval process has been completed, which may take a month or more, the ordering process, which can be accomplished through the internet, is relatively straightforward and efficient. A person at the repository who has been helpful in the past is Patrick Bodishbaugh, contact@bioresources.org, phone 1-800-359-7370. The NIH Project Officer, Susan Peacock, can be contacted if necessary at peacocksusan@niaid.nig.gov, 1-301-451-5093.

A Product Information Sheet taken from the NIAID catalog describing the reference reagent is supposed to be linked to the web site product page, but may be incomplete. In the past, an NIH Reference Reagent Note accompanied the reference material, standard, or antiserum that had been prepared for the NIH. Such Reference Notes provided valuable scientific information about reference reagent preparation, purification, characterization, and assignment of potency along with supporting data. BEI Resources staff have indicated their intention to supplement Product Information Sheets with information available from the Reference Reagent Notes.

Dr. Connie Young of BEI Resources has graciously provided the following table for the information of the ISICR membership.

NIAID Research Resources for Interferon Research

BEI Number	Product	NIAID Number	On Web	Add info/ paper	Date of Ref. Note
NR-3072	Freeze-Dried Human Anti-Human Interferon Alpha Antibody Reference	G037-501-572	Yes	No. 44	Feb-95
NR-3073	Freeze-Dried Human Anti-Human Interferon Beta Antibody Reference	G038-501-572	Yes	No. 45	Feb-95
NR-3074	Freeze-Dried Rabbit Reference Interferon	G019-902-528	Yes	No. 10A	Dec-73 revised Jun-80
NR-3076	Freeze-Dried Reference Murine Interferon Alpha	Ga02-901-511	Yes	No. 40	Mar-87
NR-3077	Freeze-Dried Reference Human Interferon Alpha (Namalwa/Sendai)	Ga23-901-532	Yes	No. 30	Jan-84
NR-3078	Freeze-Dried Reference Human Interferon Alpha (Leukocyte/Sendai)	Ga23-902-530	Yes	No. 29	Jan-84
NR-3079	Freeze-Dried Reference Murine Interferon Beta	Gb02-902-511	Yes	No. 41	Mar-87
NR-3080	Freeze-Dried Reference Human Interferon Beta	Gb23-902-531	Yes	No. 35	Mar-87
NR-3081	Freeze-Dried Reference Murine Interferon Gamma	Gg02-901-533	Yes	No. 42	Mar-87
NR-3082	Freeze-Dried Reference Murine Interferon Alpha/Beta	Gu02-901-511	Yes	No. 39	Mar-87
NR-3083	Freeze-Dried Reference Human Recombinant Alpha 2 Interferon	Gxa01-901-535	Yes	No. 31	Jan-84
NR-3085	Freeze-Dried Reference Human Recombinant Interferon Beta/ser	Gxb02-901-535	Yes	No. 37	Mar-87
NR-3086	Freeze-Dried Human Interferon Gamma Reference	Gxg01-902-535	Yes	No. 43	Feb-95
NR-3087	Sheep Antiserum to Mouse L-Cell Interferon	G024-501-568	Yes	No. 19	Aug-80
NR-3088	Control Antiserum (Sheep) to Mouse L-Cell Interferon	G025-501-568	Yes	No. 20	Aug-80
NR-3089	Sheep Antiserum to Human Leukocyte Interferon	G026-501-568	Yes	No. 22-R	Mar-81 revised Sep-95
NR-3090	Control Antiserum (Sheep) to Human Leukocyte Interferon	G027-501-568	Yes	No. 23	Mar-81
NR-3091	Sheep Antiserum to Human Fibroblast Interferon	G028-501-568	Yes	No. 24	Mar-81
NR-3092	Control Antiserum (Sheep) to Human Fibroblast Interferon	G029-501-568	Yes	No. 25	Mar-81
NR-3093	Calf Antiserum to Human Lymphoblastoid Interferon Alpha	G030-501-553	Yes	None	
NR-3094	Rabbit Antiserum to Mouse Gamma Interferon	G032-501-565	Yes	No. 32	Aug-84
NR-3095	Control Antiserum to Mouse Gamma Interferon	G033-501-565	Yes	No. 33	Aug-84
NR-3096	Rabbit Antiserum to Human Gamma Interferon	G034-501-565	Yes	No. 34	Aug-84
NR-4283	Control Antiserum (Calf) to Human Lymphoblastoid Interferon Alpha	G031-501-553	Yes	None	

Clinical Trials

Hannah Nguyen

More information on this list can be obtained at http://clinicaltrials.gov [CT], http://www.centerwatch.com/search.asp [CW], or http://clinicalstudies.info.nih.gov [CCNIH].

Safety and Efficacy of Different Doses of MK7009 Administered With Pegylated-Interferon and Ribavirin. Contact: Toll Free Number 1-888-577-8839. Locations: Louisiana, United States, 70808; Laboratoires Merck Sharp & Dohme - Chibret, Paris Cedex 8, France, 75114; and Merck Sharp & Dohme (New Zealand) Ltd., Auckland, New Zealand. Study Director: Medical Monitor, Merck. Study ID Numbers: 2007_658, MK7009-007

BRAVO Study: Laquinimod Double Blind Placebo Controlled Study in RRMS Patients With a Rater Blinded Reference Arm of Interferon β-1a (Avonex®). Contacts: Dan Bar-Zohar 972-9-8631970 and David Kormann, 215-293-6309. 149 Study Locations. Principal Investigators: Douglas L. Vollmer, St. Joseph's Hospital & Medical Center, and Per S Sorensen, Copenhagen Trial Unit, Center for Clinical Intervention Research. Study ID Numbers: MS-LAQ-302.

Octreotide and Interferon Alfa-2b or Bevacizumab in Treating Patients With Metastatic or Locally Advanced, High-Risk Neuroendocrine Tumors. 247 Study Locations. Study Chair: James Yao, MD, M.D. Anderson Cancer Center. Study ID Numbers: CDR0000579151, SWOG-S0518

Phase II Study of Anti-Ganglioside GD3 Mouse/Human Chimeric Antibody KW2871 Combined With High Dose Interferon-alpha2b in Patients With Metastatic Cutaneous Melanoma. Locations in Pennsylvania and Illinois, USA. Study Chair: John Kirkwood, MD, University of Pittsburgh. Study ID Numbers: LUD2007-001, UPCI07-023, UCH15689B

Fatigue and IL-1 Blockade (Anakinra) in Primary Sjøgrens Syndrome. Contact: Roald Omdal, MD, PhD, 47-5151-8000, omro@sus.no. Location: Stavanger University Hospital, Stavanger, Norway, N-4068. Study ID Numbers: P REK NORD 60/2007, 2007-000475-41 (EudraCT), 17772 (NSD), 07/03834-7 (SLK).

Topical IL-1Ralpha Treatment of Posterior Blepharitis. Contact: Leila Smaga, 617-573-4439, Leila_Smaga@meei.harvard.edu. Location: Massachusetts Eye and Ear Infirmary, Boston, Massachusetts, United States, 02114. Principal Investigator: Reza Dana, MD, MPH, MSc. Study ID Numbers: 07-07-047.

A Phase I Study of De-Immunized DI-Leu16-IL2 Immunocytokine after Ritixumab in Patients With B-Cell Non-Hodgkin's Lymphoma. Location: City of Hope Comprehensive Cancer Center, Duarte, California, United States, 91010-3000. Contact: Phyllis Broene, 800-826-4673, PBroene@coh.org. Principal Investigator: Ryotaro Nakamura, MD, Beckman Research Institute. Study ID Numbers: CDR0000598679, CHNMC-03131, EMD-CHNMC-03131.

Combination Study of SB-485232 (Interleukin 18) and Doxil For Advanced Stage Epithelial Ovarian Cancer. Locations in California and Pennsylvania, USA. Study Director: GSK Clinical Trials, MD, GlaxoSmithKline. Study ID Numbers: ILI108621.

A Safety Study of the JAK2 inhibitor XL019 in Adults With Myelofibrosis. Locations and Contacts in 5 US States. Study ID Numbers: XL019-001.

Laboratory-Treated Autologous Lymphocytes, Aldesleukin, and GM-CSF in Treating Patients With Recurrent, Refractory, or Metastatic Non-Small Cell Lung Cancer. Location: Roger Williams Medical Center, Providence, Rhode Island, United States, 02908-4735. Contact: Abby Maizel, MD, PhD, 401-456-2662. Study Chair: Abby Maizel, MD, PhD, Roger Williams Medical Center. Study ID Numbers: CDR0000577502, RWMC-RWH-07-349-32.

*(**Clinical Trials**, cont. from page 16)*

Placebo-Controlled, Dose-Escalation Study of the Safety of IMO-2125 (synthetic DNA-based agonist of Toll-like receptor 9) in Hepatitis C-Infected Patients. Contact: Katherine Berezny, 919-668-8453, katherine.berezny@duke.edu. Locations in 4 US States and Puerto Rico. Study Director: Alice Bexon, MD, Idera Pharmaceuticals. Study ID Numbers: IMO-2125-001.

Tumor Necrosis Factor (TNF)-α Blockade for Psoriatic Arthritis. Contact: Lai-Shan Tam, MD, (852)2632-3173, lstam@cuhk.edu.hk. Location: Prince of Wales Hospital, Hong Kong, China. Principal Investigator: Edmund K Li, MD, Chinese University of Hong Kong. Study ID Numbers: PSA-2006-002.

Reviews of Interest

Arend WP, Palmer G, Gabay C. IL-1, IL-18, and IL-33 families of cytokines. *Immunological Rev*. 223: 20-38, 2008.

Bettelli E, Korn T, Oukka M, Kuchroo VK. Induction and effector functions of T(H)17 cells. *Nature* 453:1051-1057, 2008.

Cawthorn WP, Sethi JK. TNF-alpha and adipocyte biology. *FEBS Lett*. 582: 117-131, 2008.

Choubey D, Panchanathan R. Interferon-inducible Ifi200-family genes in systemic lupus erythematosus. *Immunol. Lett*. 119: 32-41, 2008.

Constantinescu SN, Girardot M, Placquet C. Mining for JAK-STAT mutations in cancer. *Trends in Biochemical Sci*. 33: 122-131, 2008.

Filippi CM, von Herrath MG. IL-10 and the resolution of infections. *J. of Pathology*. 214: 224-230, 2008.

Hennighausen L, Robinson GW. Interpretation of cytokine signaling through the transcription factors STAT5A and STAT5B. *Genes & Development* 22: 711-721, 2008.

Kaisho T, Tanaka T. Turning NF-κB and IRFs on and off in DC. *Trends in Immuno*l. 29: 329-336, 2008.

Leonard WJ, Zeng R, Spolski R. Interleukin 21: a cytokine/cytokine receptor system that has come of age. *J. Leukoc. Biol*. 84: 348-356, 2008.

Levine SJ. Molecular Mechanisms of Soluble Cytokine Receptor Generation Stewart J. *J. Biol. Chem*. 283: 14177-14181, 2008.

Mikkola ML. TNF superfamily in skin appendage development. *Cytokine & Growth Factor Rev*. 19: 219-230, 2008.

Moustakas A, Heldin C-H. Dynamic control of TGF-beta signaling and its links to the cytoskeleton. *FEBS Lett*. 582: 2051-2065, 2008.

Panopoulos AD, Watowich, SS. Granulocyte colony-stimulating factor: Molecular mechanisms of action during steady state and 'emergency' hematopoiesis. *Cytokine* 42: 277-288, 2008.

Rowell E, Merkenschlager M, Wilson CB. Long-range regulation of cytokine gene expression *Curr. Op. in Immunol*. 20: 272-280, 2008.

Sadler AJ, Williams BRG. Interferon-inducible antiviral effectors. *Nature Rev. Immunol*. 8: 559-568, 2008.

Free Job Postings for ISICR members!!!

Have an open position in your lab? Take advantage of the free job posting site on the ISICR website. Just go to http://www.isicr.org/pages/jobfinder/ to post your open position. Postings are free to ISICR members and ISICR corporate sponsors.

Current Posting

A Postdoctoral Position is available immediately to investigate molecular mechanisms of cytokine-induced apoptosis in cancer cells. The research is focused on understanding the functional role of STAT transcription factors in the activation of novel cellular death pathways, inflammation and cancer. The candidate must be a recent Ph.D. with a strong background in molecular and cellular and/or immunology. Highest priority will be given to applicants who have expertise in gel electrophoresis, qPCR methodology, fluorescence microscopy and flow cytometry. Experience in working with animal models is desirable. Applicants must be highly motivated and have a solid publication record in a relevant field. Examples of our work can be found in the following references:

oScarzello A.J. et al. (2007) "A Mutation in the SH2 domain of STAT2 prolongs tyrosine phosphorylation of STAT1 and promotes type I IFN-induced apoptosis" Mol Biol Cell 18, 2455-62 (PMID: 17442890)

o Gamero AM, Potla R, Sakamoto S, Baker DP, Abraham R, Larner AC. (2006) Type I interferons activate apoptosis in a Jurkat cell variant by caspase-dependent and independent mechanisms. Cell Signal. 18,1299-308 (PMID: 16337360)

To apply, please electronically submit a detailed curriculum vitae, a statement of research objectives and contact information for three references to: Dr. Ana M. Gamero, Assistant Professor, Department of Biochemistry, Temple University School of Medicine, 3440 North Broad Street, Philadelphia, PA 19140, email to: gameroa@temple.edu.

Biotech News

Clips from the *Daily Drug News*
Hannah Nguyen

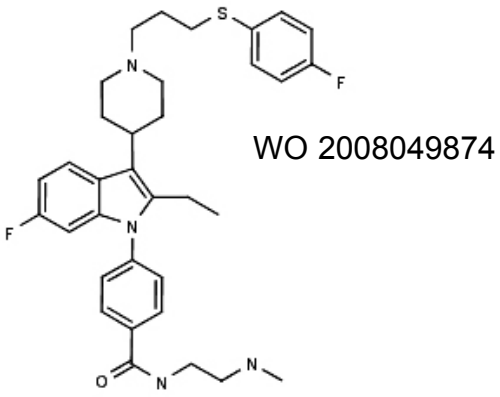
[July 09, 2008] Anadys receives clearance in the Netherlands for phase I clinical trial of ANA-773. Anadys Pharmaceuticals has received clearance to initiate a phase I clinical trial of ANA-773, a toll-like receptor-7 (TLR7) agonist for the treatment of chronic hepatitis C (HCV), under a clinical trial application in the Netherlands. Following initial dosing in healthy volunteers, this trial will explore every-other-day dosing over 28 days in HCV patients. The trial will be conducted under a two-part protocol. Part A of the study will include both single and multiple doses of ANA-773. Successive cohorts of volunteers will receive ascending doses of the drug. The primary objectives of part A are to assess safety and tolerability. In part B of the study, HCV patients will receive ANA-773 every other day for 28 days. The primary objectives of part B are to assess safety, tolerability and viral load decline. The starting dose level will be selected based on safety, tolerability and immune responses seen in part A. The study design will allow initial dosing in HCV patients at a dose that will have demonstrated a desired magnitude of immune stimulation in the healthy subjects, and that dosing in patients will begin prior to completion of dose escalation in part A of the study. Approximately 40 healthy volunteers and 24 patients will be enrolled. Dosing will begin in healthy volunteers within the next few weeks and in HCV patients early in the fourth quarter of 2008 (Anadys Pharmaceuticals News Release).

[July 10, 2008] FDA accepts Hemispherx Biopharma's Ampligen NDA for review.
The FDA has accepted for review Hemispherx Biopharma's NDA for Ampligen(R) (atvogen), an experimental toll-like receptor (TLR) therapy to treat chronic fatigue syndrome (CFS), originally submitted in October 2007. The Ampligen(R) NDA includes more than 15 years of efficacy/safety data on its potential use to treat CFS consisting of the

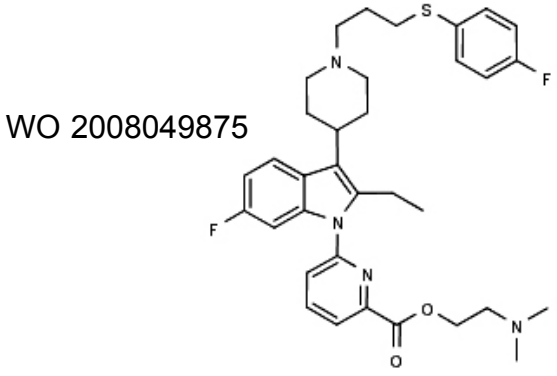
(***Biotech News**, cont. from page 18*)

the dosing of more than 1,200 clinical trial subjects with approximately 90,000 doses administered in various studies (including non-CFS) (Hemispherx News Release).

[July 17, 2008] Novel nti-inflammatory agents claimed in recent patent literature. Boehringer Ingelheim has announced the development of two series of chemokine CCR3 receptor modulators that are claimed for use in the therapeutic intervention of inflammatory disorders, including rheumatoid arthritis, atherosclerosis, asthma and allergic conditions and chronic obstructive pulmonary disease (WO 2008049874 and WO 2008049875).



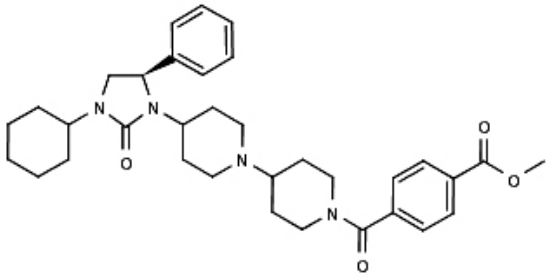
WO 2008049874



WO 2008049875

[July 21, 2008] EMEA grants orphan medicinal product designation to ProtAffin's PA-401. ProtAffin has received orphan medicinal product designation in the European Union from the EMEA for its lead product PA-401 (recombinant human CXCL8 mutant) for the prevention of delayed graft function after solid organ transplantation. PA-401 is ProtAffin's lead antiinflammatory product and is a modified form of the human chemokine IL-8 which acts as a potent, targeted antiinflammatory protein preventing the infiltration of neutrophils in the first days following solid organ transplantation (ProtAffin News Release).

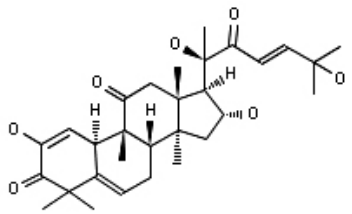
[July 22, 2008] New treatment option for HIV infection disclosed in recent Genzyme patent. Research undertaken at Genzyme has resulted in the development of a series of compounds that act as chemokine CCR5 receptor antagonists and are expected to be of use in combating HIV infection. Additional applications include inflammatory conditions and autoimmune diseases (WO 2008070758).



[July 24, 2008] JSI-124 enhances activity of cisplatin against resistant head and neck carcinoma. Treatment with cisplatin of head and neck carcinoma is often limited due to resistance. Researchers at the University of Michigan have studied a combination of cisplatin with a novel JAK/STAT3 inhibitor, JSI-124 (cucurbitacin I, elatericin B), in order to improve current therapy and to understand mechanisms of resistance. JSI-124 alone at 178.3 nM inhibited tumor growth by 50% in the UM-SCC-PT head and neck squamous cell carcinoma cell line. Additionally, JSI-124 (50 nM) or cisplatin (10 mcM) alone was associated with 29% and 27% of cell viability loss, respectively, while combination of both compounds caused loss of cell viability of greater than 50%. JSI-124 induced apoptosis, decreased phosphorylated STAT3 and reduced STAT3 mRNA, but not EGFR or Bcl-XL expression, in these cells, while cisplatin increased EGFR, STAT3, BCL-XL and survivin mRNA expression. The results indicate that inhibition of the EGFR pathway by a STAT3 inhibitor such as JSI-124 may enhance the efficacy of cisplatin in

(***Biotech News**, cont. from page 19*)

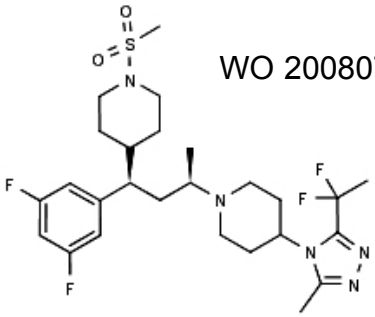
head and neck cancers (Brenner, C.J. et al 7th Conf Head Neck Cancer (July 19-23, San Francisco) 2008, Abst S370).



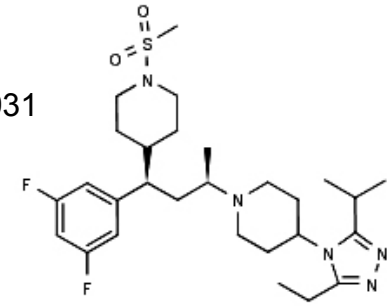
[July 29, 2008] Nventa reports final data from HspE7 phase I cervical dysplasia trial. Nventa Biopharmaceuticals has completed analysis of immunological data from all four cohorts of its phase I clinical trial for HspE7 (HspE7/Poly-ICLC) and has identified a dose regimen of 500 mcg of HspE7 and 1,000-2,000 mcg of Poly-ICLC, a Toll-like receptor 3 (TLR3) adjuvant, for subsequent phase II trials. The purpose of the phase I trial was to determine the safety, tolerability and immunogenicity of HspE7 plus escalating doses of adjuvant (50, 500, 1,000 and 2,000 mcg of Poly-ICLC). All dose regimens were found to be safe and well tolerated. Immunogenicity analysis demonstrated that the adjuvant potently enhanced HPV16 E7-specific T-cell responses in subjects who demonstrated no or low responses at baseline. In the first cohort (500 mcg of HspE7 and 50 mcg of Poly-ICLC), which was designed to establish a baseline for the study, there was limited HPV16 E7-specific T-cell responses. In cohort 2 (500 mcg of HspE7 and 500 mcg of Poly-ICLC), three out of four patients showed HPV16 E7-specific T-cell responses. In the third cohort (500 mcg of HspE7 and 1,000 mcg of Poly-ICLC), HPV16 E7-specific T-cell responses were elicited in all four subjects and all of these T-cell responses represented significant changes from baseline, indicating that the responses were a direct result of treatment with HspE7. In the trial's fourth and final cohort (500 mcg of HspE7 and 2,000 mcg of Poly-ICLC), two of five patients had significant increases in HPV16 E7-specific T-cells from baseline while the remaining three patients maintained high levels of HPV16 E7-specific T-cells that were already present at baseline. The absolute levels of HPV16 E7-specific T-cells in patients in the fourth cohort were similar to levels observed in the third cohort (Nventa Biopharmaceuticals News Release).

[July 29, 2008] Novel treatment options for autoimmune diseases reported by AstraZeneca. Scientists at AstraZeneca have patented two series of compounds that function as modulators of chemokine CCR5 receptors for use in the therapeutic intervention of autoimmune disorders, inflammatory conditions, notably rheumatoid arthritis, transplant rejection and HIV infection (WO 2008071927 and WO 2008071931).

WO 2008071927

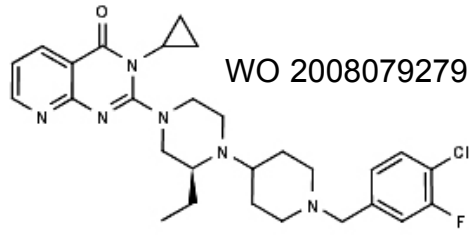


WO 2008071931



[July 29, 2008] Recent patents disclose novel anti-inflammatory agents. Schering-Plough and Pharmacopeia have jointly divulged a novel series of heterocyclic compounds that act as chemokine CXCR3 receptor antagonists. Their use in the treatment of inflammatory and immunological disorders is claimed. These include rheumatoid arthritis, inflammatory bowel disease, graft rejection, multiple sclerosis, psoriasis, ophthalmic inflammation and dry eyes (WO 2008079279).

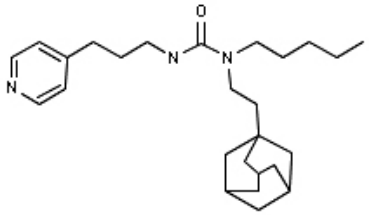
(***Biotech News**, cont. from page 20*)



[July 31, 2008] FDA advisory committee recommends approval of Chugai and Roche's Actemra in RA. The Arthritis Advisory Committee of the FDA has recommended approval of Chugai and Roche's Actemra(R) (tocilizumab), a humanized anti-human IL-6 (interleukin-6) receptor monoclonal antibody, as a treatment for rheumatoid arthritis (RA). The companies submitted a biologics license application (BLA) to the FDA seeking approval to market the antibody in late 2007 (Chugai News Release).

[August 06, 2008] Heplisav phase III study meets primary endpoint. Topline immunogenicity results from a phase III trial comparing Heplisav(TM), an investigational hepatitis B virus (HBV) vaccine, to the currently marketed HBV vaccine Engerix-B(R), showed that the study met its primary endpoint. The PHAST (Phase 3 HeplisAv Short-regimen Trial) study evaluated a two-dose regimen of Heplisav(TM) administered at 0 and 1 month compared to a three-dose regimen of Engerix-B(R) administered at 0, 1 and 6 months. The primary endpoint was the proportion of subjects who developed protective antibodies to hepatitis B after administration. In PHAST, 95.1% of subjects who received two doses of Heplisav(TM) developed protective antibodies to hepatitis B when measured at 12 weeks versus 81.1% of subjects who received three doses of Engerix-B(R) when measured at 28 weeks. The multicenter study evaluated 2,427 subjects aged 11-55 years in Canada and Germany. Results of additional analyses from this trial will be presented in the future. Heplisav(TM) is being jointly developed by Dynavax Technologies and Merck & Co. for use in adults and in patients with end-stage renal disease. The FDA previously placed a clinical hold on the two INDs for Heplisav(TM) that is still in effect. In issuing the clinical hold, the FDA requested a review of clinical and preclinical safety data for Heplisav(TM). Additionally, the FDA requested all available information about a single case of Wegener's granulomatosis reported in this phase III trial. Heplisav(TM) is based on Dynavax's proprietary immunostimulatory sequence (ISS) that specifically targets Toll-like receptor 9 (TLR9) to stimulate an innate immune response. Heplisav(TM) combines ISS with HBV surface antigen (HBsAg) and is designed to enhance the speed of protection (Dynavax Technologies News Release).

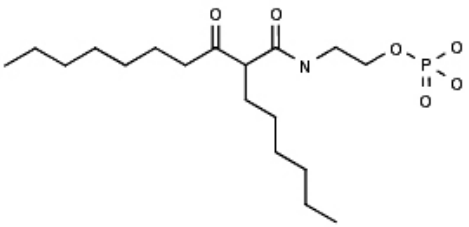
[August 11, 2008] Mechanism of action discovered for the novel TNF-alpha production inhibitor SA-13353. Recent experiments in rats have led to the discovery of the mechanism of action of a novel tumor necrosis factor (TNF)-alpha production inhibitor from Santen, SA-13353. In binding assays, the compound showed affinity for transient receptor potential vanilloid-1 (TRPV-1) (Ki = 35.8 nM; IC50 = 191 nM) over other kinases, phosphatases or proteases. In vitro, SA-13353 (0.03-1 mcM) induced calcitonin gene-related peptide release in a rat dorsal ganglion neuron culture, which was inhibited by pretreatment with the TRPV-1 inhibitor capsazepine. This effect was also observed in rats, where SA-13353 inhibited LPS-induced TNF-alpha production, although it was reversed by sensory denervation or when rats were pretreated with capsazepine. Similar results were obtained in mice. On the other hand, therapeutic activity of oral SA-13353 was studied in a model of collagen-induced arthritis in rats showing dose-dependent inhibition of hindpaw swelling and significant decrease in joint destruction. These results suggest that SA-13353's antiarthritic effect via inhibition of TNF-alpha release was mediated through by the TRPV-1 receptor in capsaicin-sensitive afferent neurons (Murai, M. et al. Eur J Pharmacol 2008, 588(2-3): 309). SA-13353 is currently being evaluated in phase II clinical trials.



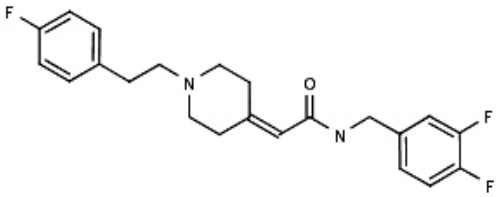
(***Biotech News**, cont. from page 21*)

[August 13, 2008] MyD88 deficiency associated with rare pyogenic bacterial infections in children. An international study has identified a new rare infectious disease affecting children and that involves defects in the innate immune system. Scientists analyzed nine children suffering from recurrent pyogenic bacterial infections caused by Streptococcus pneumoniae, Staphylococcus aureus and Pseudomonas aeruginosa, but that were otherwise healthy and resistant to most common bacteria, virus and parasitic infections. Children did not react to the infection with an inflammatory response showing no fever or leukocytosis. Genetic analysis revealed complete deficiency of MyD88, a key downstream adapter protein required for Toll-like receptor (TLR) and interleukin-1 receptor (IL-1R) signaling. These results suggest that MyD88-dependent TLR and IL-1R signaling are specifically required to provide protective immunity against some pyogenic bacteria but not for most natural infections (von Bernuth, H. et al. Science 2008, 321(5889): 691; Institut d'Investigacions Biomediques August Pi i Sunyer News Release).

[August 26, 2008] NeoPharm reveals new agents for atopic dermatitis in recent patent. Researchers at NeoPharm have claimed the use of a series of ceramide derivatives for treating atopic dermatitis. Exemplified compounds such as K6PC-9P are capable of augmenting Th1 cytokine and interleukin-2 production and diminishing TH2 cytokine and interleukin 4 levels (WO 2008078965).



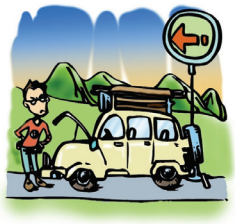
[August 28, 2008] Recent Novartis patent describes new agents for respiratory disorders. Novartis scientists have imparted a novel series of piperidin-acetamide derivatives that act as chemokine CCR3 receptor antagonists with selectivity over alpha1-adrenoceptors. They are described as being of particular use in treating respiratory disorders such as asthma, among other inflammatory and allergic diseases, as a result of their ability to suppress eosinophil infiltration and activation (WO 2008092844).



Zen thoughts to ponder

1. Do not walk behind me, for I may not lead. Do not walk ahead of me, for I may not follow. Do not walk beside me either. Just pretty much leave me the hell alone.

2. The journey of a thousand miles begins with a broken fan belt and leaky tire.



3. It's always darkest before dawn. So if you're going to steal your neighbor's newspaper, that's the time to do it.

4. Don't be irreplaceable. If you can't be replaced, you can't be promoted.

5. Always remember that you're unique. Just like everyone else.

6. Never test the depth of the water with both feet.

7. If you think nobody cares if you're alive, try missing a couple of car payments.

8. Before you criticize someone, you should walk a mile in their shoes. That way, when you criticize them, you're a mile away and you have their shoes.

9. If at first you don't succeed, sky diving is probably not for you.

10. Give a man a fish and he will eat for a day. Teach him how to fish, and he will sit in a boat and drink beer all day.

11. If you lend someone $20 and never see that person again, it was probably a wise investment.

12. If you tell the truth, you don't have to remember anything.

13. Some days you're the bug; some days you're the windshield.

14. Everyone seems normal until you get to know them.

15. The quickest way to double your money is to fold it in half and put it back in your pocket.

16. A closed mouth gathers no foot.

17. Duct tape is like 'The Force'. It has a light side and a dark side, and it holds the universe together.

18. For the male members of the ISICR: There are two theories to arguing with women. Neither one works.

19. Experience is something you don't get until just after you need it.

20. Never, under any circumstances, take a sleeping pill and a laxative on the same night.

WWW

Biorating
www.biorating.com

Biorating.com features a free public repository of user ratings and primary data for commercial antibodies across a range of applications. Researchers benefit from access to independent evaluation by experienced bench-level scientists, thereby reducing the amount of time and money spent searching for quality application-specific antibodies. A core set of top academic laboratories routinely screen antibodies submitted for evaluation, and the wider community of users are invited to post their data and rankings. With easy to navigate, continuously updated information from scientific peers, Biorating supports your research with road-tested practical information customized to your needs. Our contributors rate what is in the vial, so you can spend more time thinking out of the box!

Biosolutions
http://bioisolutions.blogspot.com/

Biosolutions is a repository of Biological Information. Biological Informations are provided via animations, lectures and videos. The aim of site is create a knowledge base among the student community

COSMIC, the Catalogue of Somatic Mutations in Cancer
http://www.sanger.ac.uk/cosmic

COSMIC (Catalogue of Somatic Mutations in Cancer;) is a comprehensive resource that aims to curate the world's literature on somatic mutations in known cancer genes. The catalog includes full and up-to-date curation of mutation data in over 60 well known point-mutated genes, together with novel gene fusion products expressed across genome rearrangement breakpoints, and all of the somatic mutation data from candidate gene screens at the UK's Cancer Genome Project.

CytoSVM
http://bioinf.xmu.edu.cn/software/cytosvm/cytosvm.php

CytoSVM is a Support Vector Machine (SVM)-based server especially constructed to classify the putative cytokine-receptor interactions in basis of their primary sequences. The putative cytokine-receptor interactions have been screened against the whole genomes of human and mouse.

Dendritic cell antigen animation
http://www.ab-direct.com/literature/multimedia-397.html

Dendritic cells capture foreign material, process it and pass it to naïve T cells to initiate immune responses. Cytotoxic T cells then seek out and destroy cells expressing the antigen, while helper T cells stimulate B cells to produce antibodies. Whether derived from myeloid or lymphoid lineages, mature dendritic cells carry out their function through interactions with major histocompatibility complex (MHC) molecules. There are two types of MHC: class I and class II and, therefore, two pathways of dendritic cell antigen presentation. The animation provides a simple overview of each pathway.

Gene2Mesh
http://portal.ncibi.org/gateway/gene2mesh.html

Gene2MeSH is an automated annotation tool that associates Medical Subject Heading (MeSH) terms with genes using the National Library of Medicine's PubMed literature database. The significance of association between genes and MeSH terms is evaluated using Fisher's exact test and displayed in an interface in order of significance score. Users may search by gene name or MeSH term and view or download results via the web interface. Gene2MeSH also provides relevant links to protein interactions in MiMI as well as reference links to Entrez, the MeSH browser, and PubMed.

WWW (continued)

Gene Quantification

www.Gene-Quantification.info

The Gene Quantification page describes and summarises all technical aspects involved in quantitative gene expression analysis using real-time qPCR & qRT-PCR. It presents a lot of new and innovative applications, chemistries, methods, algorithms, cyclers, kits, dyes, analysis methods, events, and services involved. Commercial and academic institutions can present their qPCR tools right here on our qPCR platform.

Immune Attack

http://fas.org/immuneattack/

The Federation of American Scientists (FAS) presents Immune Attack™, an educational video game that introduces basic concepts of human immunology to high school and entry-level college students. Designed as a supplemental learning tool, Immune Attack aims to excite students about the subject, while also illuminating general principles and detailed concepts of immunology.

Michigan Molecular Interactions

http://mimi.ncibi.org

Protein interaction data exists in a number of repositories. Each repository has its own data format, molecule identifier and supplementary information. Michigan Molecular Interactions (MiMI) assists scientists searching through this overwhelming amount of protein interaction data. MiMI gathers data from well-known protein interaction databases and deep-merges the information. Utilizing an identity function, molecules that may have different identifiers but represent the same real-world object are merged. Thus, MiMI allows the users to retrieve information from many different databases at once, highlighting complementary and contradictory information. To help scientists judge the usefulness of a piece of data, MiMI tracks the provenance of all data. Finally, a simple yet powerful user interface aids users in their queries, and frees them from the onerous task of knowing the data format or learning a query language. MiMI allows scientists to query all data, whether corroborative or contradictory, and specify which sources to utilize. MiMI is part of the National Center for Integrative Biomedical Informatics (NCIBI).

Molecular Visualizations of DNA

http://www.youtube.com/watch?v=4PKjF7OumYo

Pathway Central

http://www.sabiosciences.com/pathwaycentral.php

We have built Pathway Central- a pathway study resource for you to find pathway reviews and presentation-ready pathway maps. You can browse the relevant pathways based on your research areas. Simply click on one of the ten research areas listed below, and the associated individual pathways will be listed. Or you can easily search the pathways of your interest with key words. Alternatively, you can view the complete list of pathways in the Pathway Central.
Practical Flow Cytometry

InVitrogen provides online access to Howard Shapiro's Practical Flow Cytometry at

http://www.invitrogen.com/site/us/en/home/Products-and-Services/Applications/Cell-and-Tissue-Analysis/Flow-Cytometry/FC-Misc/Thank-You-for-Registering.reg.us.html (Note that you do have to fill out a short information request form.)

Vadlo

http://www.vadlo.com/

Vadlo is brought to you by two biology scientists who wish to make it easier to locate biology research related information on the web. Vadlo search engine caters to all branches of life sciences. This beta offering allows users to search within five categories: Protocols, Online Tools, Seminars, Databases and Software.

WWW (continued)

Protocols category will let you search for methods, techniques, assays, procedures, reagent recipes, plasmid maps, etc. Online Tools will cater calculators, servers, prediction tools, sequence alignment and manipulation tools, primer design etc. Seminars are essentially powerpoint files for presentations, lectures and talks. Databases will take you to, well, databases, resources, compilations, lists etc. It is here that you can also search for your favorite genes and proteins. Software category is for bioinformatics experts who are looking for codes, scripts, algorithms, executables, downloadable programs and collaborations. Please realize this is beta, or pre-beta, if you prefer. Vadlo index is expanding everyday and your favorite link/s will be indexed pretty soon.

We can not stress enough the importance of using peer-reviewed journal articles and textbooks when designing experiments or making reagents. Your first experiment should always be based on such authentic and complete sources. However, the material generously shared on the web by many groups with expertise in the field is important too. Such Lab protocols can provide you with quick reference, tips and tricks and li'l secrets - minor modifications that may make your life easy and can save a lot of time and resources. So, use Vadlo with wisdom. We wish you the best!

Virtual Cell's Educational Animations

http://vcell.ndsu.nodak.edu/animations/

In addition to Virtual Cell's online game modules, animations have been developed to introduce students to new concepts. By walking through the still images and movies included for each topic, viewers can easily choose between either studying a specific step from one of the processes or taking a more immersive look at the process in it's entirety. In order to better serve all levels of educational interest, each topic is being offered with a choice between two approaches:

-- **FIRST LOOK** - An introductory level explanation of each topic and its animation. Intended for students in a general biology class at the freshman college level.

-- **ADVANCED LOOK** - An in-depth look at the information covered by each animation. Intended to be of use for advanced biology students from the baccalaureate to graduate level.

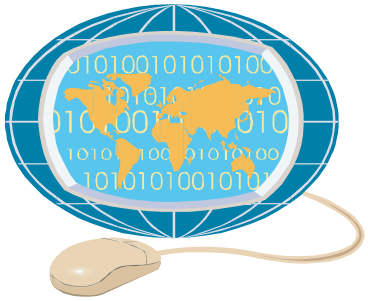
A Web Atlas of Cellular Structures Using Light and Confocal Microscopy

http://www.itg.uiuc.edu/technology/atlas/

This web site displays a series of light and confocal micrographs illustrating a variety of subcellular structures and organelles. We hope to provide a useful educational resource for people interested in cytology who do not have access to advanced imaging technologies or cell biological expertise.

We have employed a cultured epithelial cell line, CV1-monkey kidney cells, for these experiments and stained for such organelles as the Golgi apparatus, the endoplasmic reticulum, nucleus, mitochondria, and several cytoskeletal elements. Protocols for each staining as well as a description of the microscopes utilized are provided.

Confocal and fluorescent microscopy images help to reveal the intriguing structures of the cell. We hope also to demonstrate the high quality imaging techniques available for such visualization. This project has been made possible with the support of the Beckman Institute of Advanced Science and Technology's Imaging Technology Group at the University of Illinois at Champaign-Urbana.
Sheela Konda, Steve Rogers and Daniel E. Weber



SnapShot: Cytokines I

Cristina M. Tato and Daniel J. Cua
Schering-Plough Biopharma (Formerly DNAX Research), Palo Alto, CA 94304, USA

Cell

Cytokine	Receptor	Source	Targets	Major Function	Disease Association
Interleukin (IL)-1α; IL-1β	IL1RI and IL1R-AcP	Macrophages, many others	Macrophages, thymocytes, CNS, others	Inflammatory; promotes activation, costimulation, and secretion of cytokines and other acute-phase proteins; pyrogenic	↑= inflammatory bone resorption; gout; promotes Th17 response
IL-1ra (antagonist)	Soluble decoy receptor: IL1RII and IL1R-AcP			IL-1ra and the soluble decoy receptor complex inhibit IL-1-mediated inflammatory responses	
IL-2	IL2Rα, IL2Rβ, and IL2Rγ	T cells	T, B, NK cells, and macrophages	Proliferation; enhancement of cytotoxicity, IFNγ secretion, and antibody production	↓= lymphoproliferative disease and susceptibility to autoimmune disease; reduced Treg development. ↑= reduced Th17 development.
IL-3	IL3Rα and βc	T cells, mast cells, eosinophils	Hematopoietic progenitors, macrophages, mast cells	Differentiation and survival of lymphoid and myeloid compartment	
IL-4	IL4Rα and IL2Rγ or IL4Rα and IL13R	T cells, mast cells	T cells, B cells, macrophages, monocytes	Proliferation; differentiation of Th2; promotes IgG and IgE production; inhibits cell-mediated immunity and Th17 development	↓= susceptibility to extracellular pathogens and decreased response to allergens. ↑= allergic asthma.
IL-5	IL5Rα and βc	Th2 cells	Eosinophils, B cells	Proliferation and activation; hallmark of Th2 effector cells	↓= eosinophil and B-1 cell deficiency. ↑= allergic asthma.
IL-6	IL6Rα and gp130	Macrophages, T cells, fibroblasts, and others	Wide variety of cells: B cells, T cells, thymocytes, myeloid cells, osteoclasts	Inflammatory and costimulatory action; induces proliferation and differentiation; synergizes with TGFβ to drive Th17	↓= deficient innate immunity and acute-phase responses, lymphopenia
IL-7	IL7Rα and IL2Rγ	Thymic stromal cells, bone marrow, and spleen	B cells, T cells, thymocytes	Homeostasis, differentiation, and survival	↓= severe combined immune deficiency (SCID)
IL-9	IL9R and IL2Rγ	T cells (Th2)	T cells, mast cells, neutrophils, epithelial cells	Proliferation; promotes Th2 cytokine secretion	
IL-10	IL10R1 and IL10R2	Differentiated T helper cells, Tregs, B cells, dendritic cells, others	Macrophages, T cells, dendritic cells, B cells	Immune suppression; decreases antigen presentation and MHC class II expression of dendritic cells; down-regulates pathogenic Th1, Th2, and Th17 responses	↓= immune pathology due to uncontrolled inflammation. ↑= inhibits sterile immunity to some pathogens.
IL-11	IL11Rα and gp130	Stromal cells	Hematopoietic stem cells, B cells, megakaryocytes	Proliferation	↑= exacerbates airway diseases
IL-12 (p35 + p40)	IL12Rβ1 and IL12Rβ2	Macrophages, dendritic cells, B cells, neutrophils	T cells, NK cells	Differentiation and proliferation; promotes Th1 and cytotoxicity	↓= impaired Th1 responses and increased susceptibility to intracellular pathogens
IL-13	IL13R and IL4Rα	T cells	B cells, macrophages, others	Goblet cell activation in lung and gut; proliferation and promotion of IgE production; regulation of cell-mediated immunity	↓= impaired Th2 responses to extracellular pathogens and allergens. ↑= exacerbates airway diseases.
IL-14	Not defined	T cells	B cells	Promotion of B cell growth	
IL-15	IL15Rα, IL2Rβ, and IL2Rγ	Broad expression in hematopoietic cells	T cells, NK cells, epithelial cells, others	Proliferation and survival; cytokine production	↓= deficiency in NK cells and defective generation of memory T cells
IL-16	Not defined	T cells, eosinophils, mast cells	$CD4^+$ T cells	Recruitment of $CD4^+$ T cells	

See online version for abbreviations and references.

SnapShot: Cytokines II

Cristina M. Tato and Daniel J. Cua
Schering-Plough Biopharma (Formerly DNAX Research), Palo Alto, CA 94304, USA

Cell

Cytokine	Receptor	Source	Targets	Major Function	Disease Association
Interleukin (IL)-17A	IL17RA or IL17RC	Th17 cells and others	Mucosal tissues, epithelial and endothelial cells	Proinflammatory; protective immunity in lung; tight junction integrity; promotes mobilization of neutrophils and cytokine production by epithelial cells; promotes angiogenesis	↓= susceptibility to extracellular pathogens ↑= exacerbates organ-specific autoimmune inflammation
IL-17B, IL-17C, IL-17D, IL-17E (see IL-25)		Intestine and pancreas (17B); thymus and spleen (17C); T cells, smooth muscle cells, epithelial cells (17D)			
IL-17F	IL17RA or IL17RC	Th17 cells	Mucosal tissues, epithelial and endothelial cells	Similar function as IL-17A but with 2 logs lower receptor affinity	Not well defined. ↑ = increases neutrophil recruitment at high concentration.
IL-18	IL18R and IL18-R-AcP	Macrophages, others	Th1 cells, NK cells, B cells	Proinflammatory; induction of IFNγ	↓ = impairs Th1 responses
IL-19	IL20R1 and IL20R2	Monocytes, others	Keratinocytes, other tissues	Proinflammatory	↑ = psoriasis
IL-20	IL20R1 or IL22R1 and IL20R2	Monocytes, others	Keratinocytes, other tissues	Proinflammatory	↑ = psoriasis
IL-21	IL21R and IL2Rγ	Differentiated T helper cells (Th2 and Th17 subsets)	T cells, B cells, NK cells, dendritic cells	Proliferation of T cells; promotes differentiation of B cells and NK cytotoxicity	
IL-22	IL22R1 and IL10R2; IL22BP	Th1 and Th17 cells, NK cells	Fibroblasts, epithelial cells	Inflammatory, antimicrobial	↑ = psoriasis
IL-23 (p19 + p40)	IL23R and IL12Rβ1	Macrophages and dendritic cells	T cells	Inflammatory; promotes proliferation of Th17 cells	↑ = susceptibility to extracellular pathogens. ↓ = exacerbates organ-specific autoimmune inflammation.
IL-24	IL20R1, IL22R1, IL20R2, other. Intracellular?	Monocytes, $CD4^+$ T cells	Keratinocytes		↑ = antitumor effects
IL-25	IL17RB	Th2 cells, mast cells	Non-B, non-T, $cKit^+$, $FcεR^-$ cells	Promotes Th2 differentiation and proliferation	↓ = impairs Th2 responses to extracellular pathogens such as worms
IL-26	IL22R1 and IL10R2	Activated T cells			
IL-27 (p28 + EBI3)	WSX-1 and gp130	Activated dendritic cells	T cells, others	Induction of early Th1 differentiation by stimulating expression of the Tbet transcription factor; Inhibition of effector Th17 cell responses by inducing STAT-1-dependent blockade of IL-17 production	↓ = immune pathology due to uncontrolled inflammatory response
IL-28A/B/IL29 (IFNλ family)	IL28R1 and IL10R2	Activated subsets of dendritic cells?		May promote antiviral responses	
IL-30 (p28 subunit of IL-27)					
IL-31	IL31Rα and OSM-Rβ	Activated T cells	Myeloid progenitors, lung epithelial cells, keratinocytes	Proinflammatory	↑ = atopic dermatitis; allergic asthma
IL-32				Induces proinflammatory cytokine production	
IL-33	ST2 and IL1R-AcP	Macrophages, dendritic cells	Mast cells, Th2 cells	Costimulation, promotes Th2 cytokine production	↑ = atopic dermatitis, allergic asthma
IL-35 (p35 + EBI3)		Tregs	Effector T cells	Immune suppression	

 See online version for abbreviations and references.

SnapShot: Cytokines III

Cristina M. Tato and Daniel J. Cua
Schering-Plough Biopharma (Formerly DNAX Research), Palo Alto, CA 94304, USA

Cell

Cytokine	Receptor	Source	Targets	Major Function	Disease Association
TNFα	Murine: TNFR,p55; TNFR,p75 Human: TNFR,p60; TNFR,p80	Macrophages, monocytes, T cells, others	Neutrophils, macrophages, monocytes, endothelial cells	Inflammatory; promotes activation and production of acute-phase proteins	↓ = disregulated fever; increased susceptibility to bacterial infection; enhanced resistance to LPS-induced septic shock ↑ = exacerbation of arthritis and colitis
LTα	Murine: TNFR,p55; TNFR,p75 Human: TNFR,p60; TNFR,p80	T cells, B cells	Many cell types	Promotes activation and cytotoxicity; development of lymph nodes and Peyer's patches	↓ = defective response to bacterial pathogens; absence of peripheral lymph nodes and Peyer's patches
LTβ	LTβR	T cells, B cells	Myeloid cells, other cell types	Peripheral lymph node development; proinflammatory	↓ = increased susceptibility to bacterial infection; absence of lymph nodes and Peyer's patches ↑ = ectopic lymph node formation
LIGHT[a]	LTβR, DcR3, HVEM	Activated T cells, monocytes, DCs	B cells, NK cells, DCs, other tissue	Costimulatory; promotes CTL activity	↓ = defective CD8 T cell costimulation
TWEAK	Fn14	Monocytes, macrophages, endothelial	Tissue progenitors, epithelial, endothelial	Proinflammatory; promotes cell growth for tissue repair and remodeling	
APRIL	TACI, BAFF-R, BCMA	Macrophages, DCs	B cell subsets	Promotes T cell-independent responses; B cell homeostasis and differentiation	↓ = impaired class switching to IgA
BAFF (BlyS)	TACI, BAFF-R, BCMA	Macrophages, DCs, astrocytes	B cells	B cell maturation and survival	↓ = B cell lymphopenia; defective humoral immunity ↑ = SLE-like syndrome
TL1A	DcR3, DR3	Macrophages, endothelial cells	Activated T cells	Promotes proliferation and cytokine production	
GITRL[a]	GITR	DCs, macrophages, B cells, others	T regulatory cells, activated T cells	Costimulatory	
OX40L[a]	OX40	Activated T cells, B cells, DCs, monocytes	T cells, B cells, DCs	Costimulatory; activation and migration of monocytes	↓ = impaired humoral responses
CD40L[a] (CD154)	CD40	T cells, monocytes, macrophages, others	B cells, APCs	Costimulatory; promotes T cell-dependent responses; B cell differentiation and class switching	↓ = defective antibody responses and germinal center formation; hyper-IgM syndrome ↑ = SLE-like syndrome
FASL[a]	FAS, DcR3	Activated T cells, B cells, and NK cells	APCs, many other cell types	Regulatory; proapoptotic	↓ = lymphoproliferative disease and systemic autoimmunity
CD27L[a] (CD70)	CD27	Activated T cells, B cells, DCs, monocytes	T cells, activated B cells	Costimulatory	
CD30L[a] (CD153)	CD30	Neutrophils, B cells, macrophages, activated T cells	T cells, B cells	Costimulatory; promotes proliferation and cytokine production	Viral CD30 blocks Th1 response
4-1BBL[a]	41BB	Activated T cells, B cells, DCs, monocytes, macrophages	Activated T cells, B cells, DCs	Costimulatory; promotes activation and migration of monocytes	
TRAIL[a]	TRAIL-R1 (DR4), R2 (DR5), R3 (DcR1), and R4 (DcR2)	Activated NK cells, T cells	Many cell types	Costimulatory; promotes NK cell functions; proapoptotic	↓ = defective NK-mediated antitumor response ↑ = enhanced responsiveness to autoantigens
RANK Ligand[a] (TRANCE)	RANK receptor or osteoprotegrin	T cells and osteoblasts	Osteoclasts, many cell types	Costimulatory; promotes osteoclastogenesis and cytokine production	↓ = osteopetrosis ↑ = osteoporosis

[a]Known to exhibit bidirectional signaling via the ligand as well as the receptor.

See online version for abbreviations and references.

SnapShot: Cytokines IV

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Cell

Cytokine	Receptor	Source	Targets	Major Function	Disease Association
FLT3 Ligand	Receptor tyrosine kinases	Diverse tissue	DCs, other myeloid cells	Differentiation and proliferation; synergizes with stem cell factor	↓ = impaired hematopoietic stem cell repopulation and B cell precursors
G-CSF	GCSFR dimer	Macrophages, fibroblasts, other tissues	Committed progenitors	Differentiation and activation of granulocytes	↓ = neutropenia
GM-CSF	GM-CSFRα, βc	T cells, macrophages, fibroblasts, others	Macrophages, granulocytes, dendritic cells, and progenitors	Inflammatory; induction of activation; differentiation, growth, and survival	↓ = affects alveolar function
IFNα/β/ω	IFNαR1, IFNαR2	Macrophages, fibroblasts, plasmacytoid DCs, others	NK cells, many others	Promotes resistance to viral pathogens; promotes increased expression of MHC class I	↓ = impaired antiviral responses
IFNγ	IFNγR1, IFNγR2	Th1 cells, NK cells, CD8 T cells	Macrophages, NK cells, T cells, others	Promotes activation of APCs and cell-mediated immunity; increased MHC class II expression	↓ = susceptibility to intracellular pathogens
LIF	LIFR, gp130	Macrophages, T cells, fibroblasts, uterus, others	Embryonic stem cells, hematopoietic cells, others	Cell survival	↓ = deficient hematopoietic progenitor cells; defective blastocyst implantation
M-CSF	Receptor tyrosine kinases	Monocytes, fibroblasts, others	Committed myeloid progenitors	Differentiation; proliferation and survival	↓ = monocyte deficiency; osteopetrosis
MIF	CD74 trimer, CD44	Macrophages, T cells	Macrophages	Cell migration, DTH response	↓ = susceptibility to Gram-negative bacteria
OSM	LIFR or OSM-Rβ, gp130	Macrophages, fibroblasts, others	Myeloid cells, embryonic stem cells, T cells, others	Differentiation; induction of immune response (early)	
Stem Cell Factor	Receptor tyrosine kinases	Bone marrow	Stem cells, mast cells	Activation and growth	↓ = impaired hematopoietic stem cell proliferation and melanocyte production
TGFβ 1,2,3	TGFβR type I, type II, and type III	T cells, DCs, macrophages, others	All leukocyte populations	Regulatory; inhibits growth and activation; Treg maintenance; synergizes with IL-6 to promote Th17	↓ = increased susceptibility to autoimmune disorders ↑ = fibrotic diseases
TSLP Ligand	TSLPR, IL7Rα	Skin, lung, and gut	DCs and other myeloid cells	Promotes Th2 development (human); B cell development (mouse)	↑ = atopic diseases

SnapShot: Cytokines I-IV

Cytokines I highlights the first 16 interleukins, which were named in the order of their discovery. Many of the interleukins listed form homodimeric compounds and use the γc and/or βc chains in their receptors. (Cell *132*, p. 324)

Cytokines II describes the remainder of the numerically named interleukins, many of which were discovered during the last decade. Due to the complexity of the heterodimeric ligands and receptors used by some of these factors, they present a unique challenge in the elucidation of their function during health and disease. (Cell *132*, p. 500)

Cytokines III reviews the TNF family. Two important features of this group include the homotrimeric motif for both the ligands and the receptors and the capacity for bi-directional signaling. Targeting this family of inflammatory modulators has revolutionized the treatment of autoimmune disorders such as rheumatoid arthritis. (Cell *132*, p. 900)

Cytokines IV includes many of the factors discovered prior to the current scheme for interleukin nomenclature. For historical reasons, the original names are still in use. Note that many of these factors share receptors with other interleukins, such as IL-7Rα and gp130, which are used by TSLP and IL-6, respectively. (Cell *132*, p. 1062)

1062 Cell *132*, March 21, 2008 ©2008 Elsevier Inc. DOI 10.1016/j.cell.2008.02.024

See online version for abbreviations and references.

Tato CM, Cua DJ. SnapShot: Cytokines IV. Cell. 2008 Mar 21;132(6):1062.e1-2. Copyright Elsevier. Reproduced with permission.

YOU KNOW YOU ARE LIVING IN 2008 when...

1. You accidentally enter your PIN on the microwave.

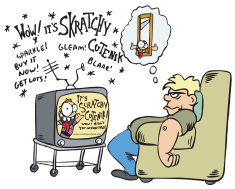
2. You haven't played solitaire with real cards in years.

3. You have a list of 15 phone numbers to reach your family of three.

4. You e-mail the person who works at the desk next to you.

5. Your reason for not staying in touch with friends and family is that they don't have e-mail addresses.

6. You pull up in your own driveway and use your cell phone to see if anyone is home to help you carry in the groceries.



7. Every commercial on television has a web site at the bottom of the screen.

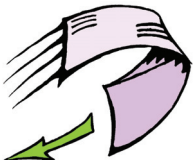
8. Leaving the house without your cell phone, which you didn't even have the first 20 or 30 (or 60) years of your life, is now a cause for panic and you turn around to go and get it.

10. You get up in the morning and go online before getting your coffee.

11. You start tilting your head sideways to smile. :)

12. You're reading this and nodding and laughing.

13. Even worse, you know exactly to whom you are going to forward this message.



14. You are too busy to notice there was no #9 on this list.

15. You actually scrolled back up to check that there wasn't a #9 on this list.

Do you know Montréal?

Researched by Thomas Tan (with input from Montrealers Nahum Sonenberg and Hannah Nguyen)

Montréal has been hailed as North America's most Eurocentric city with a great deal of charm and a myriad of activities. With numerous restaurants of all kinds, public gardens, museums and art galleries, shopping malls and charming terraces, Montréal is an ideal venue for the 7th Joint Meeting of the International Society for Interferon and Cytokine Research and the International Cytokine Society "Cytokines 2008". For the adventurous scientist in you, here are our recommendations for top picks and sweet deals as well as off-the-beaten paths.

1) Take Metro to Place d' Armes or Champ de Mars and walk towards the river to Old Montréal (http://vieux.montreal.qc.ca/). This is THE tourist spot-lots of souvenir shops, variety of restaurants, historic and European-influenced buildings, cobble-stone streets, horse buggy rides, and the Vieux-Port, its original trade port. This section of the city is quite walkable and some of the houses there date back over 300 years and are among the oldest in North America.

2) Montréal has fantastic museums. Museum of Fine Arts (http://www.macm.org/fr/expositions/49.html), where one of the Cytokine meeting's social programs will be held, is considered to be the city's most prominent museum. Museum of Contemporary Art (http://www.macm.org/fr) is the only museum in Canada devoted exclusively to contemporary art. But our favorite is the Museum of Archaeology and History (http://www.pacmuseum.qc.ca/). One well-kept secret is the great observatory that is at the top of the tower and is free with the price of admission - it provides a wonderful view of Montreal and the water. Finally, located in downtown Montreal, the McCord Museum of Canadian History (http://www.mccord-museum.qc.ca/en/) is a short walk from Queen Elizabeth Fairmont. Associated with McGill University, the McCord Museum of Canadian History showcases collections of scores of 19th- and 20th-century benefactors.

3) Also walkable from Queen Elizabeth Fairmont is the Mary Queen of the World Cathedral-Basilica. This Cathedral is a scaled-down homage to St. Peter's Basilica in Rome, covering less than a quarter of the area of its Roman inspiration. Most impressive is the 249-ft high dome, about a third of the size of the original. The statues standing on the roofline represent patron saints of the region, providing a local touch. A must-see is the dazzling combination of blue and gold and fascinating details at Notre Dame Basilica (http://www.basiliquenddm.org/), which is also part of the Cytokine meeting's social programs.

Photo from Wikipedia

4) Need a miracle for your research experiment to work so you could nail your PhD thesis, or to overcome your fear of public speaking so you could nail your talk at the conference? Head to St. Joseph's Oratory (http://www.saint-joseph.org), another very tourist-popular spot. Visitors come from over the world and miraculously get healed here-even the wheelchair-bound climbed the 100 steps to be healed.

5) On an NIH budget? Your cheapest bet: Mont-Royal (pronounced mawn-row-yal in French) (http://www.lemontroyal.qc.ca/en_index2.html). Located in the middle of the city, at the top of this big hill, lies Chalet du Mont-Royal, a beautiful park to relax (once you get there), and offers a spectacular view of the city and the St. Lawrence. There are buses that take you to the top but we think it's more fun and more rewarding if you climb all the way up

(***Do You Know**, cont. from page 32*)

by yourself. You know what they say-the harder you had to work for something, the more you would reap and enjoy the benefits of your hard work! Or, if you're up for it, join our crazy Associated Editor, Thomas Tan, who will attempt to retrace the course for the annual Mont-Royal Summit Quest 100Km (http://www.geocities.com/pnd97/FA50.html#100K).

View from the top. Photo by VirtualTourist Member lotta29

6) Now that you've worked out an appetite, try one of Montréal's best kept secret-Schwartz's smoked meat on the Main (http://www.schwartzsdeli.com/). Meat's not your thing? Chu Chai on 4088, rue St-Denis corner Duluth E serves delicious gourmet duck, beef and shrimp...all made completely from SOY!

7) A walk up Saint-Denis Street & Saint-Laurent Boulevard is a must for shopping enthusiasts but of interest to anyone who wants to see Montreal at its hippest and most happening, the area in and around Saint-Denis Street & Saint-Laurent Boulevard (not too far from Parc Jean Mance) boasts hundreds of great restaurants, boutiques, and shops, populated by hundreds of well dressed beautiful people. St Catherine Street is another tourist popular shopping area and very close to the conference venue. The street is also home to Christ Church Cathedral, the only church in Canada that sits atop a shopping mall.

8) Escape from the crowds on a three-hour bike adventure around the city led by a professional tour guide (note: tour must be booked at least 48 hours in advance of your travel date at http://affilate.unaira.com/Inventory/details.php?ProductID=122122#orderForm). Bonus: After the tour, you can keep your bicycle to use for the rest of the day to continue exploring at your leisure.

9) Escape from the metropolis all together by biking, paddling, or taking a boat tour along the Lachine Canal National Historic Site (**www.pc.gc.ca/canallachine**), which offers a 14-Km bicycle and pedestrian pathway, many picnic areas and open green spaces.

10) For the nature lover, the Laurentides (www.laurentians.com) is only about 45 min north of Montréal and offers a wide array of outdoor sports to choose from, ranging from hiking to canoeing, camping, fishing, cross-country skiing, snowshoeing, and more.

Some Cool Facts about Montréal

1) Wish you were in Beijing for this year's Olympics? Well, Montréal was the host of the 1976 Olympic Games. Its Olympic Park (http://www.rio.gouv.qc.ca/index.jsp?locale=en) includes the controversial Olympic Stadium, which was home of the Montreal Expos, the **Botanical Garden**, the **Montreal Tower Observatory**, and the **Biodome**.

2) Montréal has the world's largest underground pedestrian network, named "**The Underground City**" (very close to the conference venue). There are 20 miles of brightly lit connecting passageways and hubs below the core of the city where visitors can shop, dine and sightsee. The underground provides access to over 3000 boutiques, theaters, office buildings, cinemas, attractions, universities, hotels, museums, shopping malls and metro stations. Some 500,000 people use the underground city every day, especially to escape the traffic and/or Montréal's harsh winter or hot summer.

3) Montréal is an internationally acclaimed gastronomy. From the city's famous smoke meat to the exquisite plates of fine culinary art, Montréal offers

(***Cool Facts**, cont. from page 33*)

diversified types of cuisine in the widest variety of settings and is now a member of the **World Network of Gourmet Cities**.

4) First city to receive the **geotourism accreditation** from the National Geographic Society for the wealth of its heritage and for the high quality of services offered to visitors.

5) The city is the birthplace of **Miguel Duhamel**, arguably the most successful motorcycle racer Canada has ever produced, and it has hosted a **Formula One** race for 21 consecutive years, a feat no city in the U.S. has been able to pull off.

6) **Notre-Dame** was the first Gothic Revival style church to be built in Canada. The **Notre-Dame-des-Neiges Cemetery** is affiliated with Notre-Dame Basilica. This cemetery, the largest in Canada and the third largest in North America, is located on Mount Royal.

7) The four floral emblems on the **Flag of Montréal** represent the four main European ethnic groups that initially settled the city: French, English, Irish and Scottish.

8) Montrealers pronounce it "Muntreal", not "Mahntreal" and they greet everyone, from lifelong bosom friends to some one they' just met once a few years ago, with a two-cheek kiss.

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