

President's Invitation

Dear Colleague,

It is with great pleasure that we extend a warm invitation to participate in the 14th International Conference of the Inflammation Research Association. The 2006 Conference will be held October 15-19, 2006 in a new location, the beautiful Hyatt Regency Chesapeake Bay Golf Resort, Spa and Marina, located on the scenic shore of the Choptank River in Cambridge, Maryland. In keeping with the theme of previous IRA Conferences, the location was selected to facilitate close scientific and social interaction between attendees, with abundant opportunities to catch up with old friends and to make many new ones.

The program will commence at 3 pm on Sunday, October 15th with a special session, Patient Perspective on Inflammatory Disease. Three patients suffering from inflammatory diseases will share their personal viewpoints on their specific disease and therapies. This is intended to focus the meeting on the fact that although many important advances in treatment have been made in recent years, there is still a significant remaining unmet medical need that impacts the quality of life of millions of people.



Peter Libby, M.D.

The scientific program commences that same evening with a Plenary Lecture from the world-renowned scientist, Peter Libby, M.D. (Harvard Medical School), discussing the role of "Inflammation in Cardiovascular Disease." It is a great honor to have Professor Libby

join us as his career has focused on the translation of findings from the bench to the bedside, a topic that has been an area of focus for the IRA throughout its history.

Our Main Symposia, featuring invited speakers, will include Cardiovascular Inflammation; Fibrosis and the Resolution of Chronic Inflammation; New Strategies for Lead Optimization of Novel Anti-inflammatory Agents and a Symposium sponsored by the Society for Leukocyte Biology: Leukocytes and the Inflammatory Response. The final Symposium, New Drugs: Targeting Inflammation in Disease closes the Conference on the morning of October 19th and will feature exciting new clinical data.

In addition to the Main Symposia we will have four Mini-symposia with speakers selected from the submitted abstracts. Poster sessions will round out the general scientific sessions (poster presenters are automatically entered into a competition for the poster with the "greatest therapeutic potential," generously supported by a grant from Cerep).

The Conference will also feature opportunities for scientists early in their careers. The Van Arman Scholarship Competition provides the opportunity for five young scientists to attend the meeting at the expense of the IRA and to compete for a first prize of \$2,000. In addition, two events are being planned to allow investigators new to inflammation research to network with IRA members and gain advice on career development. These sessions are open to any meeting attendee who feels he/she could benefit.

On behalf of the organizing committee, I look forward to welcoming you to the Conference and to sharing great science and social events in a wonderful location.

Sincerely yours,
Richard J. Griffiths, Ph.D.
President, Inflammation Research Association

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C. Gordon Van Arman Scholarship Competition

Always a highlight of the Conference, the Inflammation Research Association sponsors a competition encouraging excellence and quality in inflammation research in young scientists. Contestants must be candidates for advanced degrees: M.S., Ph.D., M.D., D.O., D.D.S., D.V.M, or first year post-doctoral fellows. Through this award, the IRA hopes to develop an interest in and a commitment to both exploratory and applied research in inflammation.

The competition and awards are in recognition of the late C. Gordon Van Arman, who had a long and distinguished research career as an industrial scientist, publishing over 100 scientific papers. The development of diphenoxylate, disopyramide, sulindac, and diflunisal can be directly attributed to his work. In addition, in 1970 Dr. Van Arman along with Edward L. Takesue, Marvin E. Rosenthale and Mary Lee Graeme founded the Inflammation Research Association as an informal forum for scientists to exchange research ideas.

Prior to the 14th International Conference, contestants submitted mini-papers and the Scholarship Committee selected the five finalists. Criteria for selection as a finalist include originality of research, presentation and interpretation of data, and methods and techniques. The Finalists will receive financial support to attend the Conference and participate in poster and oral presentations to the Scholarship Committee. Oral presentations are open to all meeting attendees and the Awards will be presented at the Conference Banquet on Wednesday, October 18, 2006.

The five finalists in the 2006 C. Gordon Van Arman Scholarship Award competition are:

- **Therapeutic Antagonism of IL-13 Receptors Abrogated Chronic Fungal Asthma** Esther S. Choi, University of Michigan, Department of Pathology
- **Inhibitory- κ B Kinase- α is Required for Antigen-Driven Inflammation** Ella Johnson, Centre for Biochemical Pharmacology, William Harvey Research Institute
- **Blocking LFA-1 Activation with Lovastatin Prevents Allo-immune Responses in Mouse GVHD** Dan Li, Department of Blood and Marrow Transplantation, The University of Texas M. D. Anderson Cancer Center
- **The Use of Conformational Peptide Probes to Aid in the Identification of Novel PPAR Selective Modulators** Niharika B. Mettu, Department of Pharmacology and Cancer Biology, Duke University Medical Center
- **P2X7 Receptor-Induced Secretion of IL-1 β and**

Inflammasome Proteins is Mediated by Exocytosis of Secretory Lysosome Yan Qu, Department of Pharmacology, School of Medicine, Case Western Reserve University

Eight distinguished judges hold the honor of selecting the winner. The judges are: Jim Trzaskos, Chair (Bristol-Myers Squibb), Laurent Audoly (MedImmune), Richard Carlson (Emeritus), Bruce Jaffee (Novartis), Claudia Kasserra (Schering-Plough), Ken Kilgore (GlaxoSmithKline), Caralee Schaefer (Berlex) and John Somerville (Bristol-Myers Squibb).

For information please contact the Chairperson for the Scholarship Committee:

Jim Trzaskos, Ph.D., james.trzaskos@bms.com.

2006 CEREP Bioscience Poster Award

“The Most Therapeutic Potential”

CEREP, the preclinical discovery partner for over 400 companies, is sponsoring the CEREP Bioscience Poster Award at the 14th International Conference. CEREP thus continues an important tradition of the Inflammation Research Association conferences, as the competition has been part of each meeting since the 9th International Conference when three representatives of the Inflammation Research Association Board formed the judging panel and selected three posters judged to describe work with the most therapeutic potential.

The competition, most recently known as the GE Healthcare Scholarship Competition and previously as the Amersham Biosciences Poster Competition, is open to authors of posters presented at the 14th International Conference. Posters detailing previously unpublished work are eligible, excluding those presented by the C. Gordon Van Arman Award Finalists. Authors selected for Mini-Symposia presentations at the Conference may also prepare a poster for entry into the competition. Andy Glasebrook (Lilly) will chair the judging team that will review the posters at the Cambridge, Maryland meeting and announce the three winners at the Conference Banquet on October 18th. The judges will select the top three posters representing research with the most therapeutic potential and cash prizes of \$2,500, \$1,000 and \$500 will be awarded.

International Meetings Announcements

October 15 - 20, 2006 Les Diablerets, Switzerland	2006 Gordon Research Conference NOX Family NADPH Oxidases	www.grc.org/programs/2006/nox.htm
November 9 - 11, 2006 San Antonio, Texas, USA	Society for Leukocyte Biology/International Endotoxin and Innate Immunity Society, Joint Meeting	www.leukocytebiology.org www.ieiis.org/sanantonio2006.html
November 9 - 16, 2006 Philadelphia, PA, USA	American College of Allergy, Asthma & Immunology, 2006 Annual Scientific Meeting	www.acaai.org
November 11 - 15, 2006 Washington, DC, USA	American College of Rheumatology, 70th Annual Scientific Meeting	www.rheumatology.org
November 30 - December 2, 2006 Winnipeg, Manitoba	Canadian Arthritis Network 2006 Annual Scientific Conference	www.arthritisnetwork.ca
December 7 - 10, 2006 Prague, Czech Republic	OsteoArthritis, International World Congress	www.oarsi.org
January 26, 2007 Paris, France	Oxidative Stress and Inflammation	www.gremi.asso.fr/meeting_stress_oxydant/ukSO/
February 23 - 27, 2007 San Diego, California, USA	American Academy of Allergy, Asthma & Immunology, 63rd Annual Meeting	www.aaaai.org
June 13 - 16, 2007 Barcelona, Spain	EULAR: European League Against Rheumatism 2007 Annual Meeting	www.eular.org
June 16-20, 2007 Copenhagen, Denmark	Inflammation, 8th World Congress	www.inflammation2007.dk
August 12 - 17, 2007 Rio de Janeiro, Brazil	Immunology, 13th International Congress	www.immunorio2007.org.br
November 7 - 11, 2007 Boston, MA, USA	American College of Rheumatology, 71st Annual Scientific Meeting	www.rheumatology.org
November 14 - 17, 2007 Dallas, Texas, USA	American College of Allergy, Asthma & Immunology, 2007 Annual Scientific Meeting	www.acaai.org
December 6 - 9, 2007 Miami Beach, FL USA	OsteoArthritis, International World Congress	www.oarsi.org

8th World Congress on Inflammation

Copenhagen 16-20 June 2007
www.inflammation2007.dk

The series of Inflammation World Congresses, as organized by the International Association of Inflammation Societies (IAIS) was initiated in Vienna in 1993, and the meetings have since been held every second year. In line with the previous meetings - Vienna 1993, Brighton 1995, Tokyo 1997, Paris 1999, Edinburgh 2001, Vancouver 2003 and Melbourne 2005 - Copenhagen 2007 will provide an excellent opportunity to interact with your colleagues from academia, clinical centers and industry, while enjoying an exciting scientific program.

Along with Plenary Lectures, Symposia, Focus Groups and an

exciting program of Poster Topics, a full social program has been planned around the many spectacular sites and venues of Copenhagen.



Congress Awards

- THE IAIS YOUNG INVESTIGATOR AWARD 2007
- IAIS WOMEN IN INFLAMMATION SCIENCE AWARD
- THE BEST POSTER AWARD

Call for Papers

You are invited to submit an abstract describing an original work within the field of Inflammation to present at the Congress, either orally or as a poster. For further information on submission deadlines and requirements visit www.inflammation2007.dk



Reflections of a President...



Richard Griffiths

I have been involved with the Inflammation Research Association since attending the 1988 International Conference in White Haven, PA. Back then I never thought I would have the privilege to serve as President of the organization, however, on October 7th, 2004, at the conference dinner on the last night of the 12th International Conference Steve Stimpson handed me the official seal of office (a very fine clock). The past two years have passed quickly and it is difficult to believe we are on the eve of the 14th

International Conference (superstition prevented a 13th International!!).

It has been a great honor and pleasure to serve as President and the experience was made more enjoyable by the efforts of an extremely enthusiastic team of officers, board members and liaisons. It is remarkable that in an age when free time is at a premium, the organization continues to flourish based largely on the efforts of extremely busy people who give of their time to make things happen.

This term's outstanding scientific program (page 5) featured a wide variety of topics and aspects of inflammatory mechanisms and diseases. I would like to express my thanks to the organizers of these meetings for their efforts.

I would like to thank our Vice-President, Loran Killar for her excellent job in co-ordinating the scientific program for the 2004-2006 term. I also thank Bill Selig, our Treasurer, for managing the finances and installing a new system streamlining our bookkeeping that will make the lives of future treasurers a lot easier. I thank Amy Roshak and Arpita Maiti, who over the course of two years split the responsibility of Secretary, for improving the IRA website and for finally getting us to the point where all communications with our members are now electronic. The election process for the new board took place in the spring of 2006 and I welcome Lisa Schopf, Brydon Bennett, Tineke Meijers, Bruce Tomczuk and Lisa Olson to our 2006-2008 Board. Due to a health related issue Loran will unfortunately not be able to take on the role of President and Bill Selig will serve in that role. All Loran's friends wish her well and a speedy recovery.

I would also like to thank the outgoing board members David Becherer (GSK), Shripad Bhagwat (Ambit), Joan Chapdelaine (Calvert Preclinical) and Amy Roshak (GSK) for their many

contributions to the organization and I hope that they will continue to participate in the years to come.

After visits to the beautiful Sagamore Hotel in 2002 and 2004 we have relocated the International Conference from the shores of Lake George, NY to the shore of the Chesapeake Bay. The board is excited about this new location and asks your feedback on the facility as planning for the 15th International Conference begins. Identifying and planning the logistics for a meeting at a new site adds to the already substantial workload for the team that is responsible for this task. My heartfelt thanks and gratitude go to Rich McLaughlin, Deborah Wolff, Joan Chapdelaine and especially Marcia Bliven for once again thinking through every step of the process to ensure success.

Finally, I wish the new board and its leaders best wishes for the next two years and look forward to seeing the direction in which they take this unique organization. It has been a privilege to be President and I look forward to maintaining the many friendships I have made and attending many more meetings in the future.

Richard Griffiths
President, Inflammation Research Association

OFFICERS AND DIRECTORS FOR THE 2006-2008 TERM OF THE IRA

Officers

President	Loran Killar, Ph.D., Pfizer (MI)
Vice President	William M. Selig, Ph.D., NitroMed (MA)
Secretary	Arpita Maiti, Ph.D., Angiotech Pharm. (BC)
Treasurer	John E. Somerville, Ph.D., Bristol Myers Squibb (NJ)

Directors

Laurent Audoly, Ph.D., MedImmune (MD)
Brydon Bennett, Ph.D., Celgene (CA)
Andrew Glasebrook, Ph.D., Lilly (IN)
Tineke Meijers, Ph.D., TNO (ON)
James L. Mobley, Ph.D., Pfizer (MI)
Lisa Olson, Ph.D., Abbott (MA)
Lisa Schopf, Ph.D., Abbott (MA)
Joel E. Tucker, Ph.D., Amgen (WA)
Bruce Tomczuk, Ph.D., J&J (PA)

Past President	Richard Griffiths, Ph.D., Pfizer (CT)
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Focused Topic Meetings

IRA Scientific Program 2004 - 2006

- Oct. 4-7, 2004 **12th International Conference of the IRA** Bolton Landing, NY
- Mar. 11, 2005 **Emerging Biologic Therapies for Autoimmune Diseases** Palo Alto, CA
Organized by: Anthony Manning (Roche) and Andrew Chan (Genentech)
- Aug. 20-24, 2005 **IAIS 7th World Congress on Inflammation**
IRA-Sponsored Symposium: Chronic Obstructive Pulmonary Disease
Melbourne, Australia
Organized by: Deborah Slipetz (Merck-Frosst) and Laurent Audoly (Merck-Frosst)
- Oct. 7, 2005 **The Inflammatory Basis of Pulmonary Fibrosis** Cambridge, MA
Organized by: Claire Brown (Genzyme), Jim Ellis (Nitromed), Bill Selig (Nitromed) and Lisa Schopf (Abbott)
- Nov. 4, 2005 **Acne as an Inflammatory Disease**
New York City, NY
Organized by: Donald Collins (Estee Lauder Companies)
- Dec. 5-6, 2005 **Nitric Oxide 2005: 25 years Beyond EDRF** Boston, MA
Organized by: Jim Ellis, Gordon Letts and Bill Selig (Nitromed)
- Mar. 17, 2006 **The Macrophage Lineage as a Mediator of Remodeling Diseases**
New Brunswick, NJ
Organized by: Bruce Tomczuk and Carl Mantey (J&J)
- May 2-3, 2004 **Innate Immunity and Inflammation**
Blacksburg, VA
Organized by: Liwu Li (Virginia Tech) and Andrew Glasebrook (Lilly)
- Oct. 15-19, 2006 **14th International Conference of the IRA** Cambridge, MD

The Inflammation Research Association encourages half-day through one and one-half day Scientific Meetings. A few years ago, the majority of these meetings were half-day sessions at the New York Academy of Science. We now enjoy full day meetings at sites around the country. Exciting, interesting topics are being planned, so be sure to visit the IRA website, www.inflammationresearch.org for up-to date information.

Contact Bill Selig (wselig@nitromed.com) with your ideas for topics, potential presentations, and relevant speakers and see www.inflammationresearch.org/meet-howto.html for suggestions and guidelines as to initiating a Focused Topic Meeting.

Report on the IRA Focused Topic Meeting, The Macrophage Lineage as a Mediator of Remodeling Diseases

March 17, 2006, East Brunswick, NJ.

The belief that an understanding of the macrophage lineage will lead to improved therapeutics attracted over 110 industry and academic scientists to discuss the diverse roles of macrophages in pathogenesis and repair.

Macrophages as mediators of tissue pathogenesis:

E. Richard Stanley, Ph.D, (Dept. Developmental & Molecular Biology, Albert Einstein College of Medicine, New York, NY) described two genetic models of macrophage mediated pathogenesis. Mice bearing a hypomorphic mutation in macrophage actin-associated tyrosine phosphorylated protein (MAYP, also known as proline serine threonine-interacting protein 2 (PSTPIP2)) developed an autoinflammatory disease of the dermis and digits resembling psoriatic arthritis. Lesions occurred in the absence of lymphocytes, featured numerous macrophages, and could be prevented by specific depletion of macrophages with clodronate-liposomes. Mutant macrophages secrete elevated levels of pro-inflammatory cytokines, but how loss of MAYP leads to pathogenesis is unknown. In a second model, transgenic expression of CSF-1 from the CSF-1 receptor (CSF-1R) promoter led to elevated levels of inflammatory cytokines, leading to wasting, osteoporosis and joint disease.

David Nikolic-Paterson, Ph.D. (Dept. Nephrology, Monash Medical Centre, Clayton, Australia) used adoptive transfer to demonstrate that macrophages can injure the kidney. Renal damage in anti-glomerular basement membrane (GBM) nephritis was prevented when leukocytes were depleted with cyclophosphamide, but injury was restored by adoptive transfer of macrophages. Proteinuria and mesangial cell proliferation was proportional to the number of macrophages transferred. In a streptozotocin-



induced mouse model of type I diabetes, macrophages accumulated in the glomerulus and tubulointerstitium. Accumulation was dependent on MCP-1 because glomerular and, in particular, tubular macrophage numbers were reduced in MCP-1-deficient mice, and MCP-1-deficient mice were resistant to albuminuria and fibrosis. Overall, the data supported a role for macrophages in human renal diseases where renal macrophage numbers are often elevated and can predict disease progression.

Jeremy S. Duffield, M.D., Ph.D. (Renal Division, Brigham & Women's Hospital, Harvard University, Boston, MA) demonstrated that macrophages activate hepatic and renal fibrosis. Chronic administration of carbon tetrachloride caused hepatic fibrosis in mice. Male to female bone marrow chimeras confirmed a bone marrow origin for a subpopulation of dendritiform macrophages that were abundant during scar formation. Specific ablation of this population led to secondary apoptosis of myofibroblasts and reduced fibrosis. Similarly, selective ablation of macrophages during nephrotoxic serum-induced glomerulonephritis reduced proteinuria along with crescent formation, tubule epithelial apoptosis, myofibroblasts, and collagen-deposition.

Teresa Tetley, Ph.D. (National Heart and Lung Institute, Imperial College, London, U.K.) reported how macrophages are a major source of proteases during the pathogenesis of congestive obstructive pulmonary disease (COPD). Macrophages are the predominant leukocytes in airways, and increase many-fold in patients with COPD. Elastolytic activity is upregulated in alveolar macrophages isolated from COPD patients and several macrophage-derived metallo- and cysteine proteases have elastolytic activity. Further, metalloproteases inactivate α 1-antitrypsin thereby increasing neutrophil elastase activity. Cigarette smoke impacts the interaction of macrophages with alveolar type I (ATI) and type II (ATII) epithelial cells and can induce apoptosis. Macrophages from COPD patients were less able to clear apoptotic cells, and failure of macrophages to clear apoptotic cells may contribute to emphysema. Bacterial lipopolysaccharide (LPS) present in cigarette smoke extracts may influence the interaction between macrophages and ATI and ATII cells through toll-like receptor signaling.

Myeloid colony stimulating factors in pathogenesis:

John Hamilton, Ph.D., DSc. (Department of Medicine, University of Melbourne, Melbourne, Australia) proposed that myeloid growth factors (GM-CSF, CSF-1 and G-CSF) can be viewed as proinflammatory cytokines, acting locally and possibly systemically, by increasing myeloid cell numbers and their activation state – the so-called “CSF network” hypothesis. GM-CSF gene deficiency in mice resulted in gene dose-dependent resistance to disease in models of arthritis and multiple sclerosis. A mechanism for disease resistance in GM-CSF-deficient mice was suggested by the reduced macrophage and neutrophil exudation in a mBSA-induced peritonitis model. Neutralizing antibodies to GM-CSF reversed ongoing collagen-induced arthritis and blocked LPS-induced pulmonary neutrophilia in wild-type mice. The observation that

recombinant GM-CSF activated disease flairs in RA patients lends further support for GM-CSF as a pro-inflammatory cytokine in human disease.

Tripathi B. Rajavashisth, Ph.D. (Division of Endocrinology, Metabolism and Molecular Medicine, UCLA, Los Angeles, CA) described a pathogenic role for CSF-1 in atherosclerosis. Oxidized-LDL (Ox-LDL) activates endothelial expression of CSF-1 that importantly contributes to the development of atherosclerotic plaques by increasing the influx and survival of macrophages into the intima and differentiation to foam cells. Loss of CSF-1 function due to the osteopetrotic (Csf1op) mutation markedly reduced atherosclerosis in apolipoprotein (apo) E or low-density lipoprotein (LDL) receptor-deficient mice. CSF-1 exists in at least three isoforms, a secreted glycoprotein, a secreted and/or extracellular matrix-associated proteoglycan, and a membrane spanning isoform with cell-surface biological activity. A balance between circulating and cell/tissue-associated CSF-1 isoforms was hypothesized to mediate vascular homeostasis. Transgenes were designed to selectively express each isoform of CSF-1 on an apoE; Csf1op/op genetic background. Only the proteoglycan form fully restored lesion formation. The cell surface and secreted glycoprotein forms restored the number of circulating monocytes, but the mice remained partly resistant to fatty lesion formation. The data suggested distinct functions for specific CSF-1 isoforms in homeostatic and pathologic processes. Targeting specific forms may have therapeutic value in cardiovascular disease.

The macrophage as a mediator of metabolic disease:

Anthony Ferrante, Jr., M.D., Ph.D. (Department of Medicine, Columbia University, New York, NY) explored the hypothesis that metabolic imbalances in adipose recruit macrophages and activate macrophage expression of TNF α and other cytokines that mediate systemic complications associated with obesity. Stromal cells isolated from adipose expressed TNF α , and clodronate-mediated macrophage deletion identified macrophages as the source of TNF α . Reduced adipose TNF α in Tnf α -null bone marrow chimeras confirmed that TNF α originated from bone marrow-derived cells. A transwell system demonstrated that adipocytes express factors that induce macrophage TNF α and MCP-1. In turn, macrophages produced factors that modulated adipocyte expression profiles in a manner that mimics changes in adipose tissue gene expression in obese mice. CCR2 is the receptor for the monocyte recruitment factor, MCP-1, and CCR2 gene deficient C57B6 mice developed a reduced level of adiposity on a high fat diet. Significantly, insulin sensitivity was improved and the rise in fasted glucose and insulin levels was less severe in CCR2-deficient mice. These beneficial effects were also achieved in wild-type obese mice dosed with INCB3344, a potent CCR2 antagonist (Incyte). The results suggested that macrophages recruited to adipose may activate insulin resistance. However, because CCR2 may function on other cell types, further studies are needed to identify conclusively the roles of macrophages in CCR2-dependent metabolic regulation.

The macrophage lineage as mediator of tissue repair:

Greer Murphy, M.D., Ph.D. (Stanford University School of Medicine, Palo Alto, CA) reported that microglia may protect brain tissue from injury. In Alzheimer's disease (AD) amyloid plaques become enveloped by activated microglia (brain resident macrophages) expressing IL-1 and other mediators. The toxic effects of these mediators in pure neuron cultures led to the hypothesis that microglia promote AD pathogenesis. However, studies with hippocampal slice explant cultures called into question a pathogenic role for microglia in AD. Hippocampal slice cultures contained viable neurons, astrocytes, and microglia with authentic tissue cytoarchitecture. Addition of CSF-1 together with amyloid- β peptide activated microglial expression of IL-1, but did not activate neuronal cell death in explant cultures. Neuronal cell death could be induced with the excitotoxin, NMDA, but CSF-1 blocked NMDA-induced neuron death. CSF-1R expression was markedly increased on microglia in PDAPP and Tg2576 transgenic mouse models of AD. To investigate consequences of CSF-1R expression, mouse BV2 immortalized microglial cells were transfected with CSF-1R gene (10-100-fold overexpression). When added to rat hippocampal slice cultures, BV2-CSF-1R cells integrated into the three-dimensional tissue and protected explants from NMDA neurotoxicity. Neuroprotection was blocked completely with a CSF-1 hammerhead ribozyme. Thus, rather than contributing to neuronal death, microglia afforded protection. Additionally, microglia can engulf amyloid- β oligomers and fibrils. Microglia cleared plaque present on AD brain slices, and CSF-1R expression enhanced and a CSF-1 ribozyme inhibited clearance. A greater understanding of the role of microglia in plaque clearance and neuroprotection may lead to novel strategies to reverse plaque formation in humans.

Jeremy S. Duffield, M.D., Ph.D. (Harvard University, Boston, MA) reported that large, round macrophages were numerically predominant during the resolution of carbon tetrachloride-induced liver fibrosis. Specific depletion of macrophages during resolution reduced the resorption of collagen and elastin from scars and prevented loss of myofibroblasts. The large, round macrophages expressed MMP13 and gpNMB enabling efficient proteolysis and phagocytosis. Male to female bone marrow chimeras identified this subpopulation as "resident" tissue-derived. A similar population of large, round, tissue-derived phagocytes arose during the resolution of ischemia/reperfusion-injury of the kidney, and was shown to mediate clearance of the protein casts. The data suggested that a subpopulation of resident-derived macrophages with high phagocytic capacity mediate tissue repair and oppose functions of bone marrow-derived macrophages during injury.

Conclusions:

An exciting day of discussion reaffirmed the critical contribution of macrophages during tissue pathogenesis and repair. A greater understanding of subpopulations and environmental queues favoring repair vs injury will surely lead to novel therapeutic approaches in a great variety of diseases.

Contributed by Carl Manthey (JJPRD) and Bruce Tomczuk (JJPRD)

Report on the IRA Focused Topic Meeting, The Blue Ridge Regional Conference on Innate Immunity and Inflammation

May 4-5, 2006, Blacksburg, VA.

The Blue Ridge Regional Conference on Innate Immunity and Inflammation was held May 4-5, 2006 on the campus of Virginia Tech in Blacksburg, Virginia. Organized by Drs. Liwu Li (Virginia Tech), Andy Glasebrook (Lilly) and Jim Mobley (Pfizer), the conference brought together and fostered close collaboration among local academic, pharmaceutical and clinical researchers interested in the emerging field of innate immunity and inflammation.

46 registrants submitted 16 abstracts on molecular signaling, cellular subsets and translational research in innate immunity. Dr. Richard Griffiths (Pfizer), President of the Inflammation Research Association, opened the meeting by welcoming the attendees and explaining the role of the Inflammation Research Association and its sponsorship of these regional Focused Topic Meetings.

Dr. Lewis Lanier (UCSF), incoming President of the American Association of Immunologists gave the keynote presentation on NK Receptors Regulating Innate and Adaptive Immunity. Other invited speakers included Dr. Sabine Ehrt (Cornell University), who discussed Host Immune Response to Mycobacterium tuberculosis; Dr. Jill Suttles (University of Louisville), who spoke on CD40 Signaling and CD40:TLR Cross-talk in Inflammation; and Dr. Liwu Li (Virginia Tech), who presented research into Regulation of Innate Immunity and its Connection with Human Diseases. The day and a half conference included 14 oral presentations of selected abstracts and late-breaking submissions, punctuated by break periods during which there was a lively exchange of ideas and "what ifs."

The breathtaking natural vista of the surrounding New River Valley provided a great backdrop for the event, and the meeting facilities at The Inn at Virginia Tech and Skelton Conference Center were exceptional. The local hospitality, scenery, and close access to many outstanding groups in inflammation research supplemented the excellent presentations and discussion which will certainly make possible future regional or international meetings exploring innate immunity and inflammation.

Submitted by:
Andrew Glasebrook
(Lilly) and Liwu Li
(Virginia Tech)





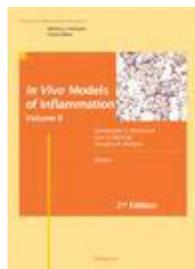
Company News and People on the Move

Richard Dyer (former IRA President), has retired from Pfizer and is now Vice President of Development and Scientific Affairs at TCH Pharmaceuticals, Inc. in Ann Arbor, MI. Dick can be contacted by e-mail at Richard.Dyer@TCHPharmaceuticals.com and by telephone using (734) 649-1654.

Dick Carlson (former IRA and IAIS officer) has formally retired and relocated to Overland Park, Kansas to visit with his son for a while. He plans to settle in Lindsborg, Kansas and work his family's 160 acres of wheat and sorghum fields. Dick hopes to learn farming and may also try to raise a few head of cattle and sheep. Dick says he has always enjoyed the IRA and IAIS Meetings ("good science, fellowship and the "hospitality suite" after hours"). He has been an active member of the IRA since its inception and has held various positions (including secretary) for many years. Dick was the driving force behind the young investigator awards and has given huge amounts of his time to the development and encouragement of young scientists. He was a key contributor to the formation of the IAIS and acted as secretary during its early years. Dick has long been a strong supporter of both organizations and an active organizer, most recently at the 2003 Vancouver Meeting. Dick will be truly missed and promises to leave his new contact information on the IRA website directory when settled.

Lisa Marshall (former IRA President) joined Johnson and Johnson Pharmaceuticals in July 2005 as a Senior Director in the Compound Development Team and is responsible for two Phase I programs targeting Pulmonary Inflammatory Diseases. Lisa was previously with GSK and can be contacted using lmarsha8@prdus.jnj.com.

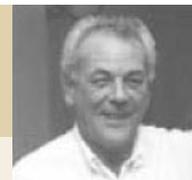
Jim Ellis reports that he has joined Surface Logix (Boston, MA) as Vice President, Pharmacology and Translational Medicine, where he will lead multiple discovery programs on cardiovascular, metabolic, inflammatory and fibrotic diseases. Previously, Jim was Executive Project Director and Chief Biology Advisor at NitroMed. Jim's email address is jellis@surfacelogix.com.



Lisa Marshall (JJPRD), **Doug Morgan** (Biogen Idec) and **Christopher Stevenson** (Novartis Institute of Biomedical Research) are the co-editors of [In Vivo Models of Inflammation, Volume 2](#), published by Birkhäuser and scheduled for release in November 2006.

[In Vivo Models of Inflammation](#) describes current animal model systems used to emulate inflammatory diseases. The second edition acts as a complement to the first edition by updating the standard models that are utilized through explanations of new models in emerging areas of inflammation research.

John Gareth Bowen (1947 – 2005)



On a beautifully sunny day last November 2nd, colleagues and friends gathered at St. Helen's Church in Plungar, Leicestershire, England to celebrate and remember the life of Gareth Bowen. Sadly, Gareth passed away as a result of pancreatic cancer. Gareth was a marvelous person with a great sense of humor. He was a true gentleman who was always ready to help fellow scientists without reservation. Gareth served for many years in different capacities for BIRA, including Chairperson, and was instrumental in the writing and implementation of the by-laws for the IAIS. He was a founding member of the IAIS and, until his death, an active officer. It was only recently that the IAIS awarded him a plaque for his numerous contributions. His leadership was always evident and his willingness to be involved with the planning and running of domestic and international scientific meetings earned him close friends from all parts of the world. Karaoke time at the IRA International Conference will never be quite the same again without Gareth's wonderful baritone voice to elevate the level of competition. He will be sadly missed by everyone.

Gordon Letts
IAIS President

Richard Griffiths
IRA President

The Newsletter

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