newsletter

Inflammation Research Association

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President's Welcome

Dear Colleague,

Thank you for your interest in and for being part of the Inflammation Research Association (IRA). This non-profit organization was founded over 36 years ago to "bring together scientists of all degree and experience levels" who are interested in inflammation research and promote open scientific discussion of novel therapeutics and the treatment of inflammatory disease. I emphasize that the IRA is essentially an all-volunteer organization that is heavily dependent on its membership to accomplish its goal.

Our recently elected Officers (John Somerville, Vice-President; Arpita Maiti, Secretary; Andrew Glasebrook, Treasurer) and Board of Directors (Laurent Audoly, Brydon Bennett, James Ellis, Richard Griffiths, Tineke Meijers, James Mobley, Lisa Olson, Lisa Schopf, Joel Tocker, and Bruce Tomczuk) are planning our regional focused topic meetings as well as the upcoming 15th International Conference (Autumn 2008). This group will focus and strategize on providing attendees with quality meetings that spotlight, in a continually changing world, the role of inflammation in disease. Please visit our website (www.inflammationresearch.org) for more information on these and future meetings.

The IRA recently held its very successful 14th International Conference at the Hyatt Regency Chesapeake Bay in Cambridge, Maryland (October 15-19, 2006). As in previous years, this meeting featured a mix of basic and applied research with ample opportunity to interact with scientists and clinicians from various sectors (academia, industry, and government), all in a relaxed environment. The meeting began with a special session, "Patient Perspective on Inflammatory Disease" during which three patients suffering from chronic inflammatory diseases described and shared their personal views of what it is like to live with their specific disease and therapy. This was a moving and inspirational session that set the tone for the rest of the meeting as many important advances in treatment have been made in recent years yet there is still a significant remaining unmet medical need that impacts the quality of life of millions of people.

The meeting also featured a "speed networking" session in which young investigators met with "seasoned" researchers from both academia and industry to gain insight into career paths and opportunities as well as the general direction of inflammation research. The outcome was very positive for all participants on both sides of the table!

During this upcoming term, your Board will further review and refine management of the organization and institute appropriate changes that will allow the IRA to flourish in a very competitive environment. We plan to continue using professional support for some

in this issue

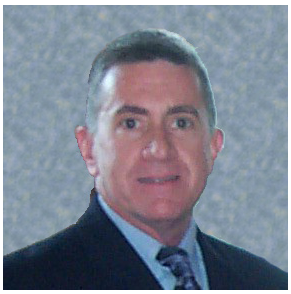
administrative functions allowing the Board to focus on meeting organization as well as fund raising to support this non-profit organization.

Our website is the primary portal that allows you to: 1) interact with other members by searching for their contact details and 2) receive up-to-date information about future IRA-sanctioned meetings and events. Remember that membership is free and it's only a matter of a few keystrokes to join. For existing members, we strongly encourage you to keep your contact information current since email is our sole means of communication.

The Inflammation Research Association is also a founding member of the International Association of Inflammation Societies (IAIS) which promotes inflammation research worldwide. The 8th World Congress of Inflammation was held June 16-20, 2007 in Copenhagen, Denmark and the IRA sponsored a symposium entitled "The Intersection of Cancer and Inflammation."

On behalf of the IRA Board, we look forward to your participation during the 2006-2008 term. Please feel free to drop us an email if you have questions, comments or want to participate at an organizational level in the IRA.

Respectfully,
William M. Selig
President, Inflammation Research Association



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International Meeting Announcements

September 16-19, 2007 Montreal, Canada	Bioactive Lipds in Cancer, Inflammation and Related Diseases 10th International Conference sponsored by Eicasonoid Research Fondation	www.bioactivelipidsconf.wayne.edu
September 17-20, 2007 Paris, France	IOIS: 9th Congress of the International Ocular Inflammation Society	www.iois-paris-2007.com
October 11-13, 2007 Cambridge, MA	40th Meeting of the Society for Leukocyte Biology	www.leukocytebiology.org
October 11-13, 2007 Halifax, Novia Scotia	Canadian Arthritis Network 2007 Annual Scientific Conference	www.arthritisnetwork.ca
October 14-19, 2007 Beijing, China	Frontiers in Gastrointestinal Cancer: Molecular Genetics, Inflammation, Early Detection and Therapy (T2). In Collaboration with and Cosponsored by Chinese Academy of Medical Sciences.	www.keystonesymposia.org
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
March 14-18, 2008 Philadelphia, PA	American Academy of Allergy, Asthma, & Immunology	www.aaaai.org
June 11-14, 2008 Paris, France	EULAR 2008: European League Against Rheumatism 2008 Annual Meeting	www.eular.org
August 17-22, 2008 Glasgow, United Kingdom	IASP: International Association for the Study of Pain® 12th World Congress on Pain®	www.iasp-pain.org
September 18-21, 2008 Rome, Italy	World Congress on Osteoarthritis	www.oarsi.org
October 24-29, 2008 San Fransisco, CA, USA	American College of Rheumatology 72nd Annual Meeting	www.rheumatology.org
November 7-12, 2008 Seattle, WA, USA	American College of Allergy, Asthma & Immunology	www.acaai.org
2009 Japan	Inflammation 9th World Congress	www.inflammation-iais.org
August 22-27, 2010 Kobe, Japan	14th International Congress of Immunology	www.ici2010.org

Focused Topic Meetings

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 planned so be sure to visit the IRA website, www.inflammationresearch.org for up-to-date information.

Contact Bill Selig (wselig@combinatorx.com) with your ideas for topics, 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/Amgen Pacific Northwest Meeting on "Pain, structure and new targets in osteoarthritis," April 23, 2007, Seattle, WA

Over 100 researchers, predominantly from the Seattle area but including contingents from the east coast, midwest and California attended this conference organized by Joel Tocker, Chris Gabel and Roy Black (all from Amgen) and held at the waterfront Bell Harbor Convention Center in Seattle.

Roy Black presented a brief introduction, noting the need to focus on pain as well as structure in the quest for OA therapeutic agents, as well as the need for biomarkers and new targets. James Henry (McMaster University) then described experiments employing the meniscal tear model in rats and suggesting that chronic pain in OA is neuropathic. He found altered sensitivity to neurotransmitters in spinal chord neurons, and alterations in proprioceptive dorsal horn neurons, e.g. the production of substance P. In a second talk on pain, Mark Chambers (Lilly) discussed the monoiodoacetate model of OA joint pain and its uses in preclinical drug discovery. Several agents have been found to provide benefit in this model, including calcitonin, a bisphosphonate, IL-1 receptor antagonist and estrogen. The estrogen effect was very rapid in onset, suggesting a CNS target.

The next two talks addressed how effects of candidate therapeutic agents can be monitored. David Eyre (U. of Washington) provided a succinct overview of collagens and their structure in cartilage, noting the aberrant appearance of type III collagen in OA. He estimated that the amount of type II collagen lost per day from a single knee should be detectable via a systemic biomarker, but presented data indicating that the CTXII neoepitope is too heterogenous in levels and in tissue source to be a reliable indicator of cartilage degradation. Jeff Evelhoch (Amgen) then reviewed the status of MRI as a tool in monitoring cartilage. He was optimistic about its use to measure both cartilage volume and proteoglycan content.

The final three talks related to the quest for new targets in OA. John Loughlin (U. of Oxford) has employed a genetic linkage approach, and he reported that the previously described linkage with alterations in the gene for a soluble, inhibitory Wnt receptor (FRZB) has held up in subsequent studies. He has also found linkages to an allele of the asporin gene that yields a form of the protein that sequesters TGFβ, and to the GDF5 locus (which encodes a TGFβ family member). Linda Sandell (Washington U.) reported on micro-array analyses with IL-1-β-stimulated chondrocytes, showing substantial upregulation of numerous chemokines, and the ability of TGFbeta to counter many of these effects of IL-1β. Finally, Karen Yates (Harvard Medical School) demonstrated that Wnts directly impact cartilage chondrocytes, some having catabolic effects and others stimulating anabolism. The sulfation of cell-surface proteoglycans appears to be required for Wnt signaling in these cells.

The conference concluded with a roundtable discussion led by Robin Poole (McGill University, emeritus) and Steve Bain (MDS Pharma). The merit of different pain models, evidence for the utility of the C2C neo-epitope in urine as a biomarker, and the importance of changes in chondrocyte phenotype were among the topics addressed.

Warm, sunny weather and excellent logistic support at the conference center contributed to an all-around successful meeting. The speakers departed on a high note following an excellent dinner at the Edgewater Hotel restaurant, which provides ideal views of Puget Sound, Mt. Rainier, and the Olympic Mountains.

Roy Black

14th International Conference of the IRA, Cambridge, MD, 15-19 October, 2006

The Hyatt Resort on the peaceful shore of Chesapeake Bay with a vista of reeds and waterfowl provided the setting for the 2006 International Conference, which was attended by about 300 participants. The rooms were comfortable and the pool warm, and walks or jogs along the shore seemed to be popular with the attendees.

A major focus in the first couple of days of the conference was on inflammation in atherosclerosis and the cardiovascular system. In the introductory plenary lecture, Peter Libby (Harvard University) described inflammatory processes involved at dif-

ferent stages of the atherosclerotic process. The adhesion molecule VCAM-1 is expressed on endothelium as a result of a high cholesterol diet and the chemokine, monocyte chemoattractant protein-1 (MCP-1) that acts on CCR-2, is frequently detectable in atherosclerotic plaques. Other chemokine receptors also contribute to the cellular response, including CXCR2 and CXCR4. M-CSF also appears to be a driving force to atherosclerosis, activating foam cells in the plaque. The cell surface molecule CD40 – usually considered an important T cell co-receptor - is expressed on endothelial and smooth muscle cells and macrophages in atheroma and anti-CD40 antibodies inhibit atheroma. It is now generally accepted that a major factor in myocardial infarction is the rupture of the fibrous cap of an unstable cardiac atheromatous plaque with the release of its highly thrombogenic necrotic contents. The stability of this fibrous cap is dependent on types I and III collagen. Inflammation within the plaque leads to interferon-γ production causing a reduction in collagen synthesis, production of matrix metalloproteinases MMP-1 and MMP-13 that degrade collagen, and the release by CD40-stimulated cells of the procoagulant tissue factor. A later lecture by Z. Galis (Lilly) drew attention to the role of locally generated reactive oxygen species in activating MMPs in atherosclerotic plaque. From a biomarker perspective, IL-6-stimulated C-reactive protein (CRP) levels in blood are predictive for mortality while soluble CD40 levels are predictive for future cardiovascular risk.

An excellent overview of the mechanisms controlling myofibroblast activity and fibrocyte infiltration was given by Anuk Das (Centocor). Fibrocytes enter from the circulation at sites of acute injury. These are CD43+, CD45+, Collagen-I+, CCR2+, CCR7+, CXCR4+ cells that are involved in all tissue repair responses. Inhibition of fibrocyte infiltration, as in CCR2 k.o. mice, provides protection against developing fibrosis. Myofibroblasts, expressing α-smooth muscle actin, are only associated with actively fibrosing sites and produce most of the matrix proteins at these sites. They are responsive to stimulation by TGFβ and PDGF and are upregulated in IPF. Both fibrocytes and myofibroblasts are considered to be targets for therapeutic regulation of fibroproliferative diseases.

In an excellent evening satellite symposium organized by the Society of Leukocyte Biology, some of the latest developments in the biology of specific leukocyte populations were presented. M.F. Smith (Charlottesville, VA) described the role of heparin sulphate proteoglycans (syndecans, SDCs) on macrophages as binding sites for Helicobacter pylori. SDC-1 and SDC-4 appear to be involved in the clearance of these bacteria. Eric Long (NIH) reviewed the mechanisms involved in the activation and inhibition of NK cells. Normally NK cells are maintained in an inhibited state by binding of their killer immunoglobulin receptors (KIR) – of which there is a whole family – to MHC-I molecules on the surface of "self" cells. "Non-self" cells are recognized by the absence or alteration of MHC-I molecules, resulting in activation. This activation process initially involves the adhesion molecule, LFA-1, which provides an early signal for membrane polarization and a second signal (e.g. CD16, NKG2D) is required for full activation.

One of the most exciting presentations was given by David M. Mosser (University of Maryland). He reviewed evidence for the heterogeneity of activated macrophages, which can be grouped into three different classes: Classically activated (ca) macrophages, generating NO and IL-12 in response to LPS/TNFα + IFNγ, alternatively activated (aa) macrophages, generating ornithine and urea in response to IL-4 and IL-13, and type II activated macrophages. Prior ligation of Fc receptors on ca-macrophages with immune complexes, followed by exposure to 0.5 ng LPS, converts these IL-12 producing cells to anti-inflammatory type II cells producing large amounts of IL-10! Similarly, in vivo, prior exposure to immune complexes protects mice from LPS toxicity. Under the same experimental procedure, IL-10-/- mice showed slightly prolonged survival (not significant) when compared with the lethal LPS dose only treated group. This switch from an IL-12 to an IL-10 generating phenotype occurs as a result of activation of Erk1/2 by the immune complexes, leading to histone phosphorylation which makes the transcription factors Sp-1 and Stat-3 accessible to expression by pro-inflammatory stimuli such as LPS. Compounds that induce IL-10 represent a novel class of anti-inflammatory compounds. Compounds that inhibit IL-10 production may improve immunity (vaccines).

A variety of novel anti-inflammatory compounds were presented, especially kinase inhibitors (MEK1/2, Jun, Syk, p38, Lck, Jak3) and some natural compounds. J.P. Gotteland (Serono) gave an update on c-Jun–N-terminal kinase inhibitor, AS602801, a drug candidate for the treatment of multiple sclerosis. N. Green (Wyeth Research) presented selective inhibitors of Tpl2 kinase and TNF-α production for the treatment of RA. Tpl2 (Cot/MAP3K8) is a serine/threonine kinase in the MAP3K family that is upstream of MEK in the ERK pathway. A clean Tpl2 inhibitor should selectively inhibit P-MEK formation. W.F. Westlin (Praecis Pharmaceuticals Inc.) described the disease-modifying activity of PPI-2458, an orally available inhibitor of methionine aminopeptidase type-2, in models of RA. PPI-2458 inhibits the growth of both endothelial cells, important in angiogenesis, and synovial fibroblasts that contribute to the formation of the erosive pannus responsible for the destructive effects in RA. It is an oral inhibitor of disease progression in the rat PG-PS (peptidoglycan polysaccharide) induced model of arthritis. Measurements of bone structural integrity using micro-computed tomography indicated dramatic protection and improvement in total bone volume and bone mineral density. Kevin Koch from Array BioPharma reported on ARRY-438162, a selective and potent inhibitor of MEK1/2 in clinical development for the treatment of inflammatory diseases. ARRY-438162 inhibits MEK1 and blocks downstream ERK1/ERK2 phosphorylation in cells. In

stimulated peripheral blood mononuclear cells, it inhibited TNF, IL-1 and IL-6 production. Efficacy in vivo was confirmed in collagen and adjuvant models of arthritis, as well as in IBD and acute pulmonary inflammation.

John Regan (Boehringer Ingelheim) gave a presentation on the development of dissociated glucocorticoid agonists. The goal was development of new GR agonists with reduced side-effects by increasing selectivity, by modifying the dosing regimen and through topical application. The compounds discovered were quinol-4-one derivatives of the A-ring of steroids, modified further on the D-ring with phenolic moieties to break the steroid structure and achieve bioavailability. Currently, the most promising compound is BI-115.

Two compounds in early stages of clinical development for the treatment of RA were presented. Rigel Pharmaceuticals is developing an oral inhibitor of Syk kinase, while a CCR-2 antagonist is an early clinical candidate in development by Merck.

The social program, always a highlight of the International Conferences, was disrupted by a sudden change in the weather, when rain forced a change in the Tuesday afternoon sports activities. Tineke Meijers rose admirably to the occasion, enlisting the hotel staff to organize a treasure hunt around the resort and hand out brochures and various odds and ends to the stream of competing teams that roamed the grounds. We were then introduced to the unusual team task of building a floatable boat from thick cardboard and tape within a limited time span. The Dutch Lions team – traditionally the team to beat in the (rained-off) volleyball contest – launched their rocket-shaped construction into the swimming pool only for the highly amused spectators to watch Tineke, a few minutes later, slowly roll off the "boat" and under the water! I and several other competitors suffered a very similar fate with our unstable "objects" and a most unlikely box-like construction was ultimately the one that survived a voyage across the pool and back.

The banquet on the final evening was well-attended, though the huge pieces of steak served to us defeated many with more modest appetites. Richard Griffiths duly handed over the Presidential responsibilities to Bill Selig and we all sent our wishes for a speedy recovery to Loran Kilar who had been expected to take over the position. The International Conference wouldn't be the same without the karaoke evening and Kevin Koch and Steve Stimpson were among the "regulars" to serenade us from the microphone, the over-attentive disk jockey frequently getting in on the act himself. While the absence of colorful figures such as the late Gareth Bowen was noted, it was encouraging to see a number of younger participants showing not only great singing enthusiasm, but also readiness to continue well after midnight! This bodes well for the future social program of the International Conferences.

Mike Parnham

2006 CEREP Bioscience Poster Award

CEREP, a preclinical discovery partner for over 400 companies, sponsored the CEREP Bioscience Poster Award at the 14th International Conference. The competition, most recently known as the GE Healthcare Scholarship Competition and previously as the Amersham Biosciences Poster Competition, was open to authors of all posters detailing previously unpublished work, excluding those presented by the C. Gordon Van Arman Award Finalists.

Andy Glasebrook (Lilly) chaired the judging team, and Dick Dyer (TCH Pharmaceuticals), Bernie Zeiher (Pfizer), Jim Mobley (Pfizer), and Zorina Galis (Lilly) had the honor of reviewing the posters and judging the competition at the Cambridge, Maryland Meeting. The three winners were announced at the banquet by Dr. Sam Paliwal of CEREP.

First prize of $2500, for the poster representing research with the most therapeutic potential was awarded to William F. Westlin and his colleagues from Praecis Pharmaceuticals for Poster A162 entitled: Disease-modifying activity in a model of rheumatoid arthritis with an orally available inhibitor of methionine aminopeptidase type-2, PPI-2458.

Second prize of $1000 went to the authors of Poster A129, authored by Patrice Lee et al, a team of scientists from Array Biopharma and Lovelace Respiratory Research Institute for their work entitled: Effects of a p38 MAP kinase inhibitor in an animal model of pulmonary inflammation via intratracheal or inhaled delivery.

Researchers representing Purdue University and Endocyte were awarded the Third prize ($500) for Poster A133: Selective targeting of drugs to activated macrophages: use in imaging and therapy of inflammatory diseases, as presented by Philip Low (Endocyte).

The IRA thanks CEREP for continuing this 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.

C. Gordon Van Arman Scholarship Winners

The C. Gordon Van Arman Award, always a highlight of the Conference and named in recognition of the late C. Gordon Van Arman encourages excellence and quality in inflammation research in young scientists. Through this award, the IRA hopes to develop an interest in and a commitment to both exploratory and applied research in inflammation.

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. 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 received financial support to attend the Conference and participated in a presentation and discussion of their mini-papers and posters to the Scholarship Committee. The final phase of the competition was a 15 minute oral presentation that was open to all meeting registrants and the Awards were presented at the Conference Banquet.

As always, the high quality of the entries made the scholarship committee's task of selecting a winner difficult.

The prizes were presented by the 2006 Van Arman Award Chairperson Jim Trzaskos (Bristol-Myers Squibb) at the conference banquet. The Scholarship Committee included 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), all of whom dedicated many hours both before and during the conference to reviewing the work of all entrants and then selecting the winners.

1st Prize: $2000

Yan Qu, of Case Western Reserve University, was awarded first prize for work entitled: P2X7 Receptor-Induced Secretion of IL-1β and Inflammasome Proteins is Mediated by Exocytosis of Secretory Lysosome.

2nd Prize: $1000

Second prize went to Niharika B. Mettu, Duke University Medical Center, for the research of: The Use of Conformational Peptide Probes to Aid in the Identification of Novel PPAR Selective Modulators.

3rd Prize: $750

Ella Johnson of the William Harvey Research Institute, received the third prize of $750 for: Inhibitory-κB Kinase-α is Required for Antigen-Driven Inflammation.

Honorable Mentions $500

Esther S. Choi, University of Michigan, and Dan Li, University of Texas M. D. Anderson Cancer Center, each received Honorable Mention Awards for their respective works: Therapeutic Antagonism of IL-3 Receptors Abrogated Chronic Fungal Asthma, and Blocking LFA-1 Activation with Lovastatin Prevents Allo-immune Responses in Mouse GVHD.

Company News and People on the Move

Bill Selig, IRA President, has joined CombinatoRx as Senior Director, Pharmacology & Toxicology. Previously, Bill was a Senior Project Director at Nitromed. Bill can be contacted at 617-301-7225, or by email at wselig@combinatorx.com.

Loran Killar (former IRA Board member & Officer) reports that she is enjoying her retirement and is actively involved in community service activities including working as part of the Organizing Committee and as a captain of a team for the American Cancer Society Relay For Life®. Support Loran and the ongoing fight against cancer by donating to the Relay for Life. Stay in touch with Loran using lorankillar@msn.com.

Doug Miller joined Wyeth Research in Collegeville as Director, Discovery Translational Medicine in April 2006. Doug was previously with Merck in Rahway where he was a Senior Investigator in the Department of Cardiovascular Diseases. Doug's new contact information is by email at millerd5@wyeth.com, and by telephone using 484-865-9687.

Mike Parnham has been appointed Director of Preclinical Discovery at the Centre of Excellence in Macrolide Drug Discovery (CEMDD) within the GSK Research Centre Zagreb, Croatia, following the acquisition of PLIVA Research Institute by GlaxoSmithKline in 2006. Also announced as of January 2007 were the appointments of Mladen Mercep as Director of Strategy, and Vesna Erakovic Haber as Director of Biology. Email Mike using michael.y.parnham@gsk.com.

Jilly Evans joined Amira Pharmaceuticals in June 2005 as Vice President, Biology. Prior to joining Amira, Jilly was Director of Cardiovascular Diseases at Merck. Jilly can be contacted at 858-228-4667, or by email at Jilly.Evans@amirapharm.com.

Acknowledgements

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