One-on-One with Professor Bengt Samuelsson: Keynote Speaker, 36th Annual Meeting

Professor Bengt Samuelsson was born in Halmstad, Sweden in 1934. He studied medicine at the University of Lund and then entered the Karolinska Institutet in Stockholm to do his graduate studies in biochemistry and medical studies. He completed his graduate studies in 1960 and then his medical degree in 1961.

Professor Samuelsson spent a year at Harvard University before returning to Stockholm, first to serve as professor of medical chemistry at the Royal Veterinary College and then to become professor and chairman of the department of physiological chemistry at Karolinska Institutet. Dr. Samuelsson is also a member of the Royal Swedish Academy of Sciences and is currently Chairman of the Nobel Foundation.

In 1982, Professor Samuelsson shared the distinguished Nobel Prize in Medicine with Sune Bergstrom and Sir John Vane for their groundbreaking research on prostaglandins and related biologically active substances. The Society for Leukocyte Biology is honored to have Professor Samuelsson open the 36th annual meeting in Philadelphia as the keynote speaker. Professor Samuelsson will deliver his lecture on Oct. 2, 2003 at 2:00 PM.

Can you speak about specific influences that motivated you early in your scientific career?
I started to do research very early. And, when I started to study medicine, I think biochemistry was the subject that I really got interested in. The Department of Biochemistry was very active, very good. And, I was offered the possibility to do part-time research while I continued to do medical studies.

How did you first get interested in arachidonic acid and prostaglandin biosynthesis? Before your research efforts, most investigators in the biomedical sciences at the time thought that lipids played structural roles in membranes and were solely the storage form of energy. The concept that arachidonic acid is a precursor leading to the formation of many bioactive lipids broke the dogma of the time and developed new concepts around the understanding of chemical mediators in autacoids. How did you arrive at the hypothesis that arachidonic acid was the precursor to the prostaglandins?
I've always thought that structure and function are key elements in biomedical research. The prostaglandin activity was known from the 1930s from the work of Dr. Ulf von Euler and the British pharmacologist Goldblat. But, nobody had really done any structural work. After my Ph.D., I worked with Dr. Sune Bergstrom on the elucidation of the prostaglandin structures. And so, we determined the structures of PGE1 and PGE2 and corresponding prostaglandin F-compounds.

And, then I finished my M.D., and went to the U.S. to spend the year in the chemistry department at Harvard University with E.J. Corey. There, I learned a lot of synthetic and structural chemistry and learned new techniques like NMR for small molecules, this technique was used a lot by the organic chemists at that time.

When I returned to the Karolinska Institutet, I was able to buy an NMR instrument through the department. With this equipment and also other means, I could determine the structure of another prostaglandin called PGE3. And, this was essentially done by NMR and some degradation work. This prostaglandin has three double bonds: two in the same place as PGE2; and one additional double bond in the omega-3 position. And, when that structure came out, it struck us that the double bond positions in prostaglandin E3 corresponded to those in eicosapentaenoic acid and in PGE2 to arachidonic acid; and in PGE1 to bis-homo-gama-linolenic acid. So, suddenly, with the PGE structure, we could see a pattern, which was similar for the polyunsaturated fatty acids and the prostaglandins. We then — continued on page 5
President's Podium

In January, I began my term as President of the Society, after serving as a member of SLB Council for five years. As a council member, I was able to see first hand how the society runs, and where the society was heading. Unfortunately, I also came to appreciate just how little a president can do in a single year to influence these directions. For this reason, one of my first orders of business at the recent council meeting was to recommend that the tenure of the president be extended from one to two years, after my term concludes. This motion received unanimous approval of council and will be brought up at the business meeting at this year's annual meeting. Speaking of which, the annual meeting this October will be in Philadelphia, a town where I spent my formative scientific years. I have a great fondness for this city, and I'm very excited about the meeting that Charles Serhan and Linda McPhail have organized. The program looks super, including two Nobel Prize winners. Well done, Charlie and Linda! I am looking forward to a great turnout for this excellent meeting. As president, one of my primary goals is to perpetuate high scientific standards, to assure that the SLB name is always associated with the best science in leukocyte biology. The annual meeting (for which I take no credit) is certainly a step in the right direction. I have proposed that we continue to reach out to other societies to broaden awareness of leukocyte biology. Along these lines, the SLB held a satellite symposium at the AAI Meeting this past May in which myself, Alan Ezekowitz, Doug Golenbock, and Michele Swanson presented; the session was so successful that it was “standing room only”. We look forward to organizing more of these meetings and to getting more of our members involved in them. My major goal for the year pertains to the membership. I hope to increase society membership, and to get a broader section of the members actively involved in the society. I have extensively reviewed our current membership list, and I have contacted several of you, inviting you to serve on committees or asking you to consider being on Council. If any of you out there is interested in becoming more involved with the Society please contact us. The more involvement we have, the stronger the Society will be. I would also like to make a direct plea to the current members to help me increase our membership. I’m sure that all of you know at least one person who would be an excellent fit in the Society. Invite that person to join! Did you know that our student membership is only $10? For this small price, students become eligible for more than $10,000 in student travel awards. If this sounds like a deal that it too good to pass up, it is. My goal for the coming year is to increase the depth and breadth of our membership, to participate in more meetings and to let more of our members spread the word about leukocyte biology.

— David Mosser

Unraveling Inflammation

36th Annual Meeting of the Society for Leukocyte Biology
October 2-5, 2003
Philadelphia, PA

Scientific Program Chair:
Dr. Charles N. Serhan,
Harvard University
Meeting Co-Chair:
Dr. Linda C. McPhail,
Wake Forest University

For updates, information about registration, program, and additional details: www.leukocytebiology.org
e-mail: slb@faseb.org
2003 Marie T. Bonazinga Award

The Awards Committee of the Society for Leukocyte Biology is pleased to announce the selection of Dr. Siamon Gordon of the Sir William Dunn School of Pathology, University of Oxford as the 2003 Marie T. Bonazinga Award winner. This annual award has been sponsored by Accurate Chemical and Scientific since 1980; the award recognizes a member of the Society who has shown consistent excellence in research. Dr. Gordon exemplifies such an individual, demonstrating commitments both to research in macrophage biology and to the Society for Leukocyte Biology.

A native of a small village near Cape Town, South Africa, Dr. Gordon earned his M.B., Ch.B. from the University of Cape Town in 1961. He continued his training at The Rockefeller University in New York with Zanvil Cohn, a previous Bonazinga Award winner, and received his Ph.D. in 1971. Dr. Gordon also holds an honorary D.Sc. from University of Cape Town, South Africa and was honoured as a Fellow of the Academy of Medical Sciences in 2003.

Dr. Siamon Gordon

Since 1976, Dr. Gordon has been at the Sir William Dunn School of Pathology, University of Oxford. He has held his current position, as Professor of Cellular Pathology, since 1991. His primary research interests focus on the functions of professional phagocytes. His research has been instrumental in understanding monocyte/macrophage surface receptor expression and elucidating the roles that these receptors play in innate immunity. The recent work from Dr. Gordon’s laboratory has described the function of Dectin-1 as a major b-glucan receptor on macrophages. The laboratory identified that Dectin-1 is a pleiotropic receptor, with one binding site for endogenous ligand on T cells and another for exogenous carbohydrates. These studies have helped researchers in the field of macrophage immunobiology to begin understanding how Dectin-1 mediates the biological effects of b-glucans, interactions that are essential in the inflammatory response to fungal pathogens.

Dr. Gordon has authored more than 300 articles in journals and books chapters.

Dr. Gordon’s other distinctions include his role as the Chairman, Scientific Advisory Committee, Institute for Infection, Immunity & Molecular Medicine, University of Cape Town and as a co-organizer of the M.Sc. course, Global Medicine, at the University of Oxford. He has also been involved in HIV/AIDS education: in 1999 and 2001, Dr. Gordon organized workshops in Cape Town for sub-Saharan medical scientists. In a collaboration with Fran Balkwill, Mic Rolph, and John Inglis, Siamon Gordon organized the publication of a cartoon booklet, ‘Staying Alive: Fighting HIV/AIDS’, for adolescents in South Africa. At the end of 2002, Dr. Gordon completed his tenure as a counselor for the Society for Leukocyte Biology and he is a Co-chair with Alan Ezekowitz for the Society’s 2005 Annual meeting.

Dr. Gordon is married to Lyndall and they have two children, Anna, a teacher; and Olivia, a budding writer. He enjoys reading biography, history and literature, and listening to classical music.

Dr. Gordon will present the Bonazinga Award lecture on Friday, Oct. 3, 2003 at the 36th annual meeting of SLB in Philadelphia, PA.

2003 Dolph Adams Awards


2004 SLB Elections

Candidates:

President-elect (one to be elected):
Matthew Fenton, Univ. of Maryland School of Medicine

Philip Murphy, NIH

Secretary (one to be elected):
Lesley Smythies, Univ. of Alabama Dept. of Medicine

Martine Torres, Children's Hospital, Los Angeles

Councilors (two to be elected):
Robert Clark, Univ. of Texas Health Science Center

Mark Quinn, Montana State Univ.

Steven Holland, NIH

Elizabeth Kovacs, Loyola Univ. Medical Center

Marcus Thelen, Inst. for Research in Biomedicine, Switzerland

Please look for your ballot in the mail and return to the SLB office by Sept. 15, 2003
Charles A. Janeway, Jr. 1943-2003

We lost an outstanding member of the Society for Leukocyte Biology and good friend with the passing of Dr. Charles A. Janeway Jr. on April 12, 2003. Many of us saw Charlie in action as the co-organizer and leading spirit at the SLB meeting in Boston on October 5-8, 2000. He took the opportunity to teach us about the evolution of the innate immune system; a concept he fathered over the past 14 years. Although already suffering from lymphoma for over five years and dependent on a cane, he exhibited his enormous intellectual vigor and commitment to scientific ideas at the meeting and enriched the proceedings with his wise and witty comments. Charlie Janeway effectively enlarged the scope of the field of immunology. He was the first to propose the concept that pattern recognition receptors defend us against pathogens. Together with Dr. Ruslan Medzhitov, he made the pivotal observation that toll receptors are crucial in the host defense of mammals as well as insects. The activation of toll receptors induces costimulatory molecules resulting in immunoadjuvanticity. (1) Although the host defense, unlike the adaptive immunity of T and B lymphocytes, is non-clonal and fixed in the genome, he chose to call it “innate” immunity. Several years ago, I asked him why he chose such a non-sequitur. He told me that it was intended to broaden the perspective and interests of “classical immunologists”. In this he succeeded and as a result broadened the studies of immunology to the study of host defense. This coincidentally increased the acceptance by immunologists of other mediators of “innate” immunity, including our favorite monocytes, neutrophils and cytokines; all thanks to Charlie Janeway and Ruslan Medzhitov.

Of course Dr. Janeway made many more notable research contributions to immunology. He was a coauthor of the most readable and successful immunology text (2). As a professor at Yale, he taught many students and mentored many young scientists.

Ironically, as indicated in his presidential address to the American Association of Immunologists (3), Charles Janeway’s scientific bent was probably innate as well as adaptive. His great grandfather was a professor of medicine and pathology at Bellevue Hospital. His grandfather was a professor of medicine at Columbia College of Physicians and Surgeons and Johns Hopkins. His father, Charles Janeway was a professor of Pediatrics and chairman of the department at Children’s Hospital Medical Center in Boston who discovered that treatment with pooled serum gamma globulins protected immunodeficient children from infections. This exposure led to Charlie’s interest in immunology. He studied with some of the most eminent immunologists, namely Hugh McDevitt, John Humphrey, Robin Coombs, Bill Paul and Hans Wigzell, before becoming a professor at Yale. To learn more about this outstanding scientist I highly recommend the eminently readable articles that elegantly trace the development of Charlie Janeway’s concepts and adventures in science and in life. (1,3,4)


— Joost J. Oppenheim

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SLB welcomes our newest members (from January 1-June 15, 2003)

Futwan Al-Mohanna
Kathy Amir
Joshua M. Astern
Seth L. Blumerman
Eric D. Boehmer
Katherine C.
Brittingham
Jared M. Brown
Pere Joan Cardona
Britsi E. Chatterjee
Keylon L. Cheseman
Li C. Chen
In-Hong Choi
Yatein Chung
Robert L. Coffman
Sean M. Conrad
Pamela Correll
Andrew W. Craig
Penelope C. Davey
Valeria Do Mello-Coelho
Mauricio Di Fulvio
Luisa A. DiPietro
Vishwa D. Dixit
Adokutuho Omolola
Eniola
Jon Fleming
Ann E. Field
Kate A. Fitzgerald
Josephine H. Fox
Seiji Fokuda
Ada Funaro
Katherine A. Gauss
Gohava Geller
Douglas T. Golenbock
Maureen M. Goodenow
Susan A. Gregory
Chang-Jiang Guo
Shervanthi Homer-
Vanniaskam
Jessica S. Hook
Chuanqang Jiang
Stephen B. Jones
Young Ho Kim
Connie W. Lam
Andrew C. Larner
Krzyzstof Ludanski
Donald W. Lawrence
Carlos G. Leon
Francisco J. Leyva
Yuan Li
Nicholas Lukacs
Michael A. Lutcz
Liza Makowska
Chris T. Migliaccio
Suzanne A. Miles
Maria Chiara G. Monaco
Margaret A. Morris
Lata Mukundan
Takaya Murakami
Sang-Yun Nam
Masakatsu Nanamori
Sean A. Parsons
Eric Pearlman
William F. Pendergraft
Lanya L. Perez
David G. Perregraux
Mark J. Peters
VanessaPinho
Thorkil Reimer
Maria Cecilia Rodriguez-Galan
Helene Rosenberg
Thomas J. Sayers
Mariasofa Segura
Valeria Sorba
Alan Sher
Aristobulo M. Silva
Li Song
Xiao-Yu R. Song
Matthew A. Stark
Timothy H. Sabin
Kenneth Swanston
Jaya Tareja
Seth L. M. Tommas
Benjamin J. Thompson
Illy Tietzel
Jill C. Toth
James E. Tomlinson
Florin Tuluc
Yukiko Ueda
Sonia Varadarajan
Michelle L. Varney
John P. Vasilakos
Ji Ming Wang
Tiehui Wang
Xu Wang
Anthony V. Washington
Jami D. Willette-Brown
Kristi L. Williams
Jeannette M. Willman
Celine A. Wishcamper
Ning Zhang
Ting Zhang
Tienming Zhao
Joseph S. Zhou
Ye Zhou
Margot Zoeller

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used labeled arachidonic acid, in about one day we could demonstrate that arachidonic acid with an enzyme preparation gave about 80% yield of prostaglandin E\(_2\). That was a great moment. There were people who had worked on this in the lab for about two years with a number of hypothetical precursors and never got more than one tenth of a percent conversion. And, here, suddenly, it fell into place.

The discovery of the role of arachidonic acid in prostaglandin biosynthesis connected two fields, both discovered in the 1930s — the prostaglandins and the essential fatty acids. And, the old dogma that lipids only have a structural role had to be abandoned. This was a very exciting time. The polyunsaturated fatty acids suddenly got a metabolic functional role besides that old structural role.

And, at this time, my mentor, Dr. Bergstrom became Dean of the medical faculty and subsequently president of the Karolinska Institutet. And, so he essentially closed his laboratory, and instead pursued work on the clinical use of prostaglandins in obstetrics and fertility control and especially related to the programs of WHO. At that time, I decided to set up my own research group and work on the mechanisms of prostaglandin biosynthesis and also on prostaglandin metabolism.

The structure of thromboxane is a very unusual structure in biological systems and has proven to be one of the most important of the eicosanoids in coagulation and cardiovascular disease. Can you recall what drew your attention to carrying out studies to elucidate thromboxane?

The discovery of thromboxane was a result of our work on the mechanism of prostaglandin biosynthesis. A key finding was that two oxygen atoms of the five-membered ring in the prostaglandins were derived from the same molecule of oxygen. And, this led me to introduce the identified concept of an endoperoxide structure as intermediate in the formation of prostaglandins. And, eventually, the two endoperoxides, prostaglandin PG\(_{2}\) and H\(_2\), were identified and isolated. They were very unstable structures and difficult to handle. But, with the structures in hand, we could define the chemical reactions involved in the transformation of arachidonic acid to prostaglandins.

And, the first reaction involved was oxygenation and cyclization. So, we coined the term cyclooxygenase in 1974. And, of course, the abbreviation is COX. This has become a household word and the enzymes are now referred to as COX-1 and COX-2. And, that was the basis for the isolation and cloning of cyclooxygenase, which was done in Bill Smith’s laboratory. Later, COX-2 was discovered. It is published in a paper from 1974 in PNAS that we introduced the term cyclooxygenase.

So to come to thromboxane. At the time we had the endoperoxides in pure form and were interested in the biological activity. And, to our great surprise, the endoperoxides had biological activities that were different from the prostaglandins known at that time, PGE\(_2\) and PGF\(_2\). But, endoperoxides caused aggregation of platelets. Our conclusion was that there must be other derivatives of the endoperoxides that are responsible for these new biological activities.

With my collaborator, Mats Hamberg, we started to look at products formed from arachidonic acid in human platelets. And, this led us to the identification of thromboxane B\(_2\). However, thromboxane B\(_2\) turned out to be biologically inactive. In a very complex series of trapping experiments, we were able to demonstrate the existence of a very unstable derivative, which was responsible for the biological inactivity. And, this we named thromboxane A\(_2\). Then, the structure we proposed for thromboxane A\(_2\) was not really confirmed until more than a decade later when Still Clark, an organic chemist from Columbia University, synthesized thromboxane A\(_2\). In the meantime, many chemists, very talented chemists, tried to synthesize the compound and, when failing, they always questioned the structure of thromboxane A\(_2\), which is very unusual.

There is actually an interesting story related to that, which led to prostacyclin. When John Vane heard of our work, he was then working for the drug company Wellcome, he became interested in developing inhibitors of the enzyme that caused formation of thromboxane A\(_2\) from PGH\(_2\). So we sent him some PGE-H\(_2\), the endoperoxide. When he incubated it with a preparation of arteries, where he thought thromboxane A\(_2\) was, he found to his surprise, a vasodilator and an anti-aggregatory derivative. Then, with chemists at the Upjohn company, they worked out the structure of prostacyclin, or PG\(_I_2\).

Do you recall what you were doing at the time and what you might have been thinking when you and your colleagues first recognized that eicosanoids were rapidly inactivated by cells and in tissues? We were interested in the slow-reacting substance of anaphylaxis (SRS-A), which is formed in lung tissue. So, with Erik Anggard, who was my first graduate student, we decided to look at the transformation of arachidonic acid and prostaglandins in the lung. And, we did not find the structure of SRS-A, which we did 15 years later. But, we discovered the 15-hydroxy-dehydrogenase and the rapid inactivation of prostaglandins in lung tissue and many other tissues. Of course, the dehydrogenase, which is responsible for the inactivation, is present in many tissues. That was a sort of serendipitous finding while we were looking for something else.

It has been stated by some that the eicosanoid area is forever “green”. Do you think that this is the case? Do you foresee major therapeutic breakthroughs in the near future, and do you anticipate additional breakthroughs in the area of lipid mediators? I will restrict myself to the arachidonic cascade. I think that there will be major therapeutic breakthroughs in several areas. One area that has not really been explored very much is prostaglandin receptors. A lot of knowledge has accumulated regarding prostaglandin receptors, and I’m sure that antagonist and agonists will be developed. In the leukotriene receptor area there are LT1 receptor antagonists, but the LT2...
receptor is beginning to look very interesting, especially from a cardiovascular point of view. The other parts of the leukotriene pathway, like leukotriene B4 are very interesting. And, there are the lipoxins and the possibility of developing anti-inflammatory drugs based on that. The most recent, exciting finding, which I actually will talk quite a lot about at the meeting in Philadelphia, is on the membrane prostaglandin E-synthase. This is induced in a similar way to COX-2. And, there are now knockout studies that indicate that they have a very essential role in inflammation. I think that this enzyme will be the next target for development of anti-inflammatory drugs.

In the United States graduate students and post-docs are strongly encouraged to depart from the area of research of their mentors. I noticed that you shared the Nobel Prize with you mentor, Dr. Sune Bergstrom. Would you encourage mentorship of post-docs and students to maintain research efforts in the same area for longer periods of time? I think training and mentorship should be for relatively short periods of time — three or four years, or so. That way, you get exposed to many new ideas, new problems, new techniques, new milieus, which I think play a very important role in the development of a scientist. In my own case, I did work on my thesis with Dr. Bergstrom for three/four years. And, then I collaborated with him on the prostaglandin structures for about two years. But, then I went abroad for a post-doc at Harvard. And, that was a very, very important year for me because I suddenly got exposed to completely new things, organic chemistry and mechanistic organic chemistry. I think that played a very important role for me when I returned and started my own laboratory on the mechanism of prostaglandin biosynthesis. The experience at Harvard was crucial in approaching it in the way I did; it played a very important role in shaping the rest of my career and the approach of the problems. I think you should not stay too long in any one place, you need to move around and see other things.

In today’s research, many investigators/researchers no longer have to take trips to their university library to view the literature, but instead do swift literature search via their desktop computers and retrieve almost all of the published literature by command of keystroke. This is a very convenient development in our communication between biomedical scientists. Would you still advocate paper libraries and sending graduate students, as well as senior researchers to the library on a regular basis with a chance of unexpected findings of relevant literature? There is no doubt that a literature search via computer is very productive and everybody is using that a lot. But, I still go to the library, or I have my own subscriptions to some journals, which I sort of like to look through. Of course, you can look through the table of contents of everything. But, journals like Science, Nature, PNAS, New England Journal of Medicine, or so — I always have paper copies of and look at them. You always find unexpected things.

Do you have any advice for graduate students, fellows, and junior faculty? Try to get involved in the most exciting research you can think of. Go for something very exciting. And also, usually, in biomedical research, go for structure and function. And try to apply the best tools. And don’t try to solve problems that are not at the time approachable. Put realistic goals. I think that’s very important. Of course, you can work on something that is very esoteric, but you’ll never get there. Then you can work on very trivial things. You’ll always get the results, but they may not be very exciting. So, try to find an exciting problem that is researchable and usually try to look for structure and function. And, work long-term.

In this time of incredible technological advances, what do you see as some of the greatest advances? I still like the concept of structure and function. And, now, of course, you can use genomics and proteomics to help you in studying questions of structure and function. Let us take COX-2 as an example. Most drugs have previously been discovered by finding a compound, elucidating a pathway, isolating enzymes and receptors; and then developing antagonists or enzyme inhibitors. For COX-2 inhibitors, a gene was identified that coded for a protein that had a function that was different from COX-1. And suddenly, by approaching the problem from the opposite side, there was a target for developing a new drug. And, it’s the same thing with membrane prostaglandin E-synthase. The gene was found, and the protein was expressed. And, we found to our surprise and delight that the protein catalyzed the transformation of endoperoxide into prostaglandin E-2. This is now a potential target for the development of novel drugs.

What are some of your outside interests/hobbies? I’m very interested in art — I spend a lot of time looking at art and reading about art. And, I’m interested in music, opera and literature. And, dining and wine is also something I treasure. And, I love to play golf.

With best wishes from
Berlex Laboratories, Inc.

Berlex Biosciences
2600 Hilltop Drive
Richmond, CA 94806
Workshop on Innate Immunity and Aging
Satellite Symposium to be held at the 36th Annual SLB Meeting

Sponsored by the National Institute on Aging (NIA)
Thursday, October 2, 2003, 8:30 am - 1:00 pm
The Doubletree Hotel on Broad Street at Locust, Philadelphia, PA
Co-chairs: Rebecca Fuldner, Ph.D. (fuldnerr@nia.nih.gov) and
Elizabeth J. Kovacs, Ph.D. (ekovacs@lumc.edu)

Invited Speakers
• Kohtaro Fujihashi DDS, Ph.D., Univ. of Alabama, Birmingham, Impairment of Mucosal Immunity with Aging
• Elizabeth J. Kovacs, Ph.D., Loyola University Chicago, Aging, Macrophage Mediators, and Injury
• Simin N. Meydani, DVM, Ph.D., Tufts University Boston, Anti-oxidants, Infection and Aging
• Robert D. Stout, Ph.D., University of Louisville, Macrophage Dysfunction in Immunosenescence
• Suryaprakash Sambhara, DVM, Ph.D., CDC, Atlanta, Impaired Toll-Like Receptor Function in Aging
• Rebecca Fuldner, Ph.D., National Institute on Aging, Funding Opportunities

Pre-Registration for Workshop
Please register in advance to attend the Satellite Workshop on Innate Immunity and Aging by contacting Rebecca Fuldner at fuldnerr@nia.nih.gov by September 15, 2003. There is no additional charge for this workshop.

Pre-View

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So, whether you are a reader or an author, go to the Journal of Leukocyte Biology website to use this feature at www.jleukbio.org.

A publication of the Society for Leukocyte Biology
Calendar of Upcoming Events and Deadlines

July 2003
Elections for 2004 Officers and new Council Members. Look for your ballots in the mail.

August 2-6, 2003
Vancouver Convention & Exhibition Center
www.inflammation2003.com

August 28-30, 2003
17th European Macrophage and Dendritic Cell Society Meeting University of Leicester Oadby Campus, U.K. Organising Committee: emds@le.ac.uk

September 2, 2003
Deadline for late-breaking abstracts. Please contact SLB office for details: slb@faseb.org or 301.634.7810.

October 1, 2003
Satellite on “Aging and Immunity” preceding 36th Annual Meeting at the Doubletree Hotel, Philadelphia, PA

October 2-5, 2003
36th Annual SLB Meeting “Unraveling Inflammation” Philadelphia, PA

May 8-11, 2004
Toll 2004
Taormina, Sicily, Italy
register at www.umassmed.edu/Toll2004

October 3-7, 2004
12th Biennial International Inflammation Research Association Conference, Sagamore Hotel and Conference Center, New York

October 21-24, 2004
37th Annual SLB Meeting “Host Response to Pathogens” Toronto, Ontario CANADA

www.leukocytebiology.org
for more information on these SLB announcements and membership application

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