Hogan Elected President for 2009
Goodson, Green, Sternberg, and Waterman-Storer to Serve on Council

Brigid Hogan of Duke University Medical Center was elected by the ASCB membership to serve as Society President in 2009. Hogan will serve on the Executive Committee as President-Elect in 2008, and will succeed Robert Goldman as President.

Elected from among eight candidates for Council are Holly V. Goodson of the University of Notre Dame, Kathleen J. Green of Northwestern University Feinberg School of Medicine, Paul W. Sternberg of the California Institute of Technology/HHMI, and Clare Waterman of the NIH/National Heart, Lung, and Blood Institute. Each member of Council will serve a three-year term beginning January 1, 2008.

The ASCB Bylaws revisions were approved by the membership. Thirty-nine percent of eligible members voted in this year’s election.

Celldance 2007

The ASCB Public Information Committee will hold its third annual Celldance competition at the ASCB Annual Meeting on Tuesday, December 4. See page 6 for more details.

Did You Know …?

- July 26 is the Regular Abstract Submission deadline for the ASCB Annual Meeting, December 1–5, in Washington, DC.
  - Abstract sponsorship is required.
  - Current members and member-applicants may sponsor their own abstract.
  - All regular, postdoctoral, and emeritus members may sponsor another’s abstract if not submitting an abstract themselves.
- Now is the time to encourage others to join ASCB.
  - Member-applicants can sponsor their own abstract and be eligible for the discounted member-only Annual Meeting registration rate by submitting applications at www.ascb.org.
  - New benefits are now available at www.ascb.org: Click on “Membership” and look for “Membership Advantages.”
- July 26 is also the deadline for Annual Meeting Travel Award Applications.
  - Awards are available for undergraduates, predoctoral students, and postdoctoral fellows.
  - Minorities Travel Awards are also available. To learn more, visit www.ascb.org/meetings/ and click on “Travel Awards.”
Science in a World at War

Introducing a new ASCB Newsletter column that will highlight cell biology around the world as well as training and collaboration opportunities

For me, one of the greatest things about the life of a scientist is the window it opened up to the world. I have had a chance to meet colleagues from many nations, and to travel to places I only dreamed about as a boy growing up in Minnesota. (I didn’t leave my home state until high school and never flew on a plane until college.) In my mind, however, this opportunity comes with a corresponding need to take seriously our responsibilities as world citizens in an increasingly globalized yet divided world. Meeting colleagues or reading papers from distant nations puts human faces on international headlines—for me this has become increasingly true for news from the Middle East.

A Traveling Education

Like many in the world, I am saddened by the escalating violence throughout the Middle East in the last decade, and frustrated by the failure of the U.S. government to take action to help bring a just peace to the region. In 1978, while a junior in college, I spent a term living in the Old City of Jerusalem, traveling through the countryside of Palestine and Israel, and learning history from ancient times through yesterday. I met and became friends with many Palestinians. Since then, peace sometimes seemed close and, at other times, impossible. When I left the region in 1979, I vowed not to return until a just peace was in sight. However, as my career progressed, I got to know many Israeli scientists.

Thus in 2000, I visited the Weizmann Institute (www.weizmann.ac.il) and attended a remarkable scientific meeting on the Dead Sea; that was a relatively hopeful time. For those of you who have never had a chance to visit Israel, the Weizmann is a remarkable, world-class research institute, rivaling the best in the U.S. Visiting there was a great scientific experience for someone working, as I do, at the boundary between cell and developmental biology.

Finding Similarities

In 2002 an invitation to the Weizmann brought me back to the region, and provided an opportunity to visit friends in Palestine who work at a Catholic primary/secondary school. While there, I visited the neighboring Arab American University of Jenin (www.aauj.edu), another remarkable place. It was established by an entrepreneurial, expatriate Palestinian in 1995 as the first private university there, with 5,000 students. I met the Chair of the Biology and Biotechnology Department and many faculty. I was impressed to find a vibrant program, with equipment better than that at the top-notch liberal arts college I attended. The faculty was better trained, and the curriculum more rigorous and, incidentally, entirely in English in the sciences. They use the same textbooks we use in our classes at the University of North Carolina.

However, what was even more striking were the similarities between the two visits. In both Israel and Palestine I found a group of superb scientists, well trained in labs entirely in English in the sciences. They use the same textbooks we use in our classes at the University of North Carolina.

Scientists need to use their privileged positions and international connections to act as world citizens.

[Scientists need to use their privileged positions and international connections to act as world citizens.]

maintained in Europe were remarkable given the circumstances. Palestinian scientists also face tremendous challenges in seeking outlets for their students. Though as well trained as the students coming out of our own liberal arts colleges, Palestinian scientists and students find it almost impossible to obtain a visa to visit the U.S. Most important, the political and economic hardships of the Occupation affect everyone in the West Bank, including the scientific community.
The situation for the Israeli scientists was much less grim, but even for them, the ongoing conflict has an impact. My friends at the Weizmann told of their difficulty in getting colleagues from the U.S. or Europe to present seminars, in large part because of fears about the likelihood of terrorism. In addition, hostility among some Europeans against the policies of the Israeli government has led to some calls for boycotts of Israeli scientists. Sadly, the Israeli and Palestinian scientific communities are composed of those whose wish for peace was the greatest.

A Deteriorating Situation
Since my 2002 visit, the situation has deteriorated. In Palestine, travel restrictions and slow but steady violence are wearing down people. Many of the educated are fleeing to the West. After the Palestinian elections, when the candidates the U.S. government supported were not elected, the U.S. led international efforts to cut off money for the new Palestinian government. The Israeli government stopped distributing tax revenues collected on the West Bank. This led to the suspension of paychecks for government employees in March 2006, putting further stress on an economy near collapse. In early 2006 the Israelis imposed new restrictions on travel by those with foreign passports to the West Bank. This meant that American and Palestinian faculty with foreign passports have had difficulty returning to work and have been threatened with deportation.

Meanwhile, the Israelis found themselves fighting a war on two fronts, when the conflict in Gaza escalated and border skirmishes in Lebanon led to full-scale war. The Technion in Haifa was one university within range of the Hezbollah missiles. Concern about Iranian actions and its nuclear weapons program adds to the anxiety. In addition, many Israeli colleagues have children in the military, or are doing reserve service themselves, in a nation where military service is mandatory for most.

Of course, the Palestinian and Israeli scientific communities represent only a microcosm of the impact the violence in the Middle East has had. Imagine being a scientist from Iran, trying to maintain contacts with the West while one’s government and that of the U.S. seem to be dedicated to inflaming tensions between our nations. What of scientists in Lebanon? Their nation was finally emerging from a decades-long civil war when suddenly the bombs fell once more, and the airport, roads, and infrastructure were targeted. Competing factions, many of them supported by outside powers, threaten to tear the nation apart. Or perhaps, worst of all, imagine being a scientist in Iraq, where a mass kidnapping of scientists at a research institute became just another depressing headline.

Becoming Informed, Using One’s Privilege
It’s not a pretty picture. We may differ in our views of who is “at fault” and how we should move forward, but I think all of us can agree that innocent civilians on all sides, including many of our scientific colleagues, are often those suffering the most. The magnitude of the conflict can leave one feeling powerless. Yet, as I said at the beginning, scientists need to use their privileged positions and international connections to act as world citizens.

What can each of us do? First, become informed. Learn about the world situation, and don’t turn away from it. Second, reach out to colleagues from these war-torn regions, both expatriates living in your communities and those you meet at scientific meetings or other venues. Finally, consider a visit to Israel and Palestine, if invited, or offer to present a lecture there. Rehovot, home of the Weizmann, is a safer place to visit than most big-city medical schools in the U.S., including my own alma mater. During my visit to the West Bank, the only time I felt endangered was by the driving style of the Jordanian priest who ferried me there from Jerusalem; he offered a great impression of a Formula 1 race-car driver.

We also need to consider what we can do as an international community of cell biologists—the ASCB—in these and other areas of international need and conflict. As a member of the International Affairs Committee, I would be happy to hear from you with suggestions for how we can help combat the isolation of our colleagues from Israel and its Arab or Muslim neighbors. Many scientists from these nations now live and work in the U.S., and provide a tremendous resource. We are proud to belong to the same international cell biology society, the ASCB. Can we arrange international scientific exchanges, raise funds to bring students to our meetings, and even remind our governments about the plight of our colleagues in other nations? If we don’t act, who will?

—Mark Peifer
International Affairs Committee

International Authors Wanted
The ASCB International Affairs Committee (IAC) is seeking to serve ASCB international members better and inform all members about the opportunities and challenges in cell biology around the world. The new ASCB Newsletter IAC column will focus on international issues.

As one part of this effort, the IAC seeks members’ submissions about the state of cell biology research in their countries/regions. Articles of approximately 600–650 words should address:

1. The approximate size, infrastructure, resources, strengths, concerns, and future hopes of the cell biology community there
2. Opportunities for collaboration, noting approximate number of students, size of labs, and types of collaboration possible (e.g., postgraduate fellowships, residencies for senior scientists)

Columns should not focus on an individual’s own research, but might note “hot areas” of study and provide resources for more information.

If interested, please write or submit to iac@ascb.org.
The ASCB 47th Annual Meeting
December 1–5, 2007
Washington Convention Center, Washington, DC
Bruce M. Alberts, President ■ R. Dyche Mullins, Program Chair ■ John Hammer, Local Arrangements Chair

Keynote Symposium
Saturday, December 1
New Biologists for the New Biology—6:00 pm
William Bialek, Princeton University
Shirley Ann Jackson, Rensselaer Polytechnic Institute

Symposia
Sunday, December 2
Membrane Dynamics—8:00 am
Pietro De Camilli, Yale University School of Medicine/HHMI
Kirit Pouglaio, University of California, San Diego
Kai Simons, Max Planck Institute, Dresden

Architecture of Signaling Systems—10:30 am
Richard M. Loeic, Harvard University
Tobias Meyer, Stanford University School of Medicine
Pamela A. Silver, Harvard Medical School

Unconventional Organelles—10:30 am
Martina Brueckner, University of Illinois
Richard Harland, University of California, Berkeley
Deborah Hogan, Stanford University

Tuesday, December 4
Geography of Signaling—8:00 am
Howard Chang, Stanford University
Deborah Hogan, Dartmouth Medical School
Elly Tanaka, Max Planck Institute, Dresden

Force and Form in Cell Biology—10:30 am
Dennis Discher, University of Pennsylvania
Michael P. Sheetz, Columbia University
Valerie M. Weaver, University of California, San Francisco

Wednesday, December 5
Single Molecule Studies—8:00 am
Steve Kowalczykowski, University of California, Davis
Paul Selvin, University of Illinois
Michelle Wong, Cornell University

Cell Biology in Ten Years—10:30 am
Benjamin F. Cravatt, III, The Scripps Research Institute
David Haussler, University of California, Santa Cruz
Stanislas Leibler, Rockefeller University

Minisymposia

Apoptosis and Organelles
Seamus J. Martin, Trinity College Dublin, Ireland
Donald Neumaier, La Jolla Institute for Allergy and Immunology

Assembling Complex Cytoskeletal Structures
Jack Gaertig, University of Georgia
Dave Kavet, The University of Chicago

Biological Oscillators
Jay C. Dunlap, Dartmouth Medical School
Hideo Iwashita, Waseda University

Cell Biology and Disease
Lucy A. Godley, The University of Chicago
Timothy J. Mitchison, Harvard Medical School

Cell Biology of the Synapse
Eugene R. Chapman, University of Wisconsin–Madison
Gezame W. Davis, University of California, San Francisco

Cell Cycle
Michael Glotzer, The University of Chicago
Sue L. Jasper, Stowers Institute for Medical Research

Cell Migration/Motility
Jeff Hardin, University of Wisconsin–Madison
Irina Kaverina, Vanderbilt University Medical Center

Chromatin Architecture and Remodeling
Laura Reisch, Duke University Medical Center
Jerry Workman, Stowers Institute for Medical Research

Cytoskeletal Dynamics and Polarity
Ed Muolo, Center for Cell Dynamics, University of Washington
William Saxton, University of California, Santa Cruz

Epithelial Morphogenesis
M. Thomas LeCuit, Developmental Biology Institute of Marseilles-Luminy
Jennifer Zallen, Memorial Sloan-Kettering Cancer Center

Evolution of Eukaryotic Endomembrane Systems
John A. Fuerst, University of Queensland
Trevor Lithgow, University of Melbourne

Extracellular Matrix as a Memory Storage Device
Linda Gay Griffith, Massachusetts Institute of Technology
Patricia Keely, University of Wisconsin–Madison

High-Tech Cell Biology
Grant Jensen, California Institute of Technology
Kendall Knight, University of Massachusetts Medical School

Host-Pathogens Interactions and Innate Immunity
Joanne Engel, University of California, San Francisco
Jean Greenberg, The University of Chicago

Intermediate Filaments and Nuclear Lamins
Pamela K. Geyer, University of Iowa
Birgit Lame, IMB Singapore and University of Dundee

Making ‘omics Useful to Cell Biologists
John D. Atkinson, Institute for Systems Biology
Nevan J. Krogan, University of California, San Francisco

Mechanisms of Cytoskeletal Systems
Margaret L. Garidel, The University of Chicago
Wolfgang Loerter, University of Maryland, College Park

Mechanisms of Epigenetic Regulation
Gary Felzenfeld, National Institute of Diabetes & Digestive & Kidney Diseases/NIH
Cynthia Wolberger, Johns Hopkins School of Medicine/HHMI

Mechanisms of Membrane Trafficking
Juan Benitez, National Institute of Child Health & Human Development/NIH
Elizabet Conibear, University of British Columbia

Mitosis and Meiosis
Sue Biggins, Fred Hutchinson Cancer Research Center
Dean Davison, Oklahoma Medical Research Foundation

Molecular Motors: Alone and in Groups
Gisèle Koenen, Institute for Atomic and Molecular Physics
Daniela Nicastro, Brandeis University

Neuronal Cell Biology
Michael D. Ehlers, Duke University Medical Center/HHMI
Franc Pellegrini, University of North Carolina at Chapel Hill

Nuclear Import and Export
Charles N. Cole, Dartmouth Medical School
Richard W. Wisniewski, University of Alberta

Nuclear Organization and Dynamics
Sui Huang, Northwestern University Feinberg School of Medicine
Susan R. Wente, Vanderbilt University Medical Center

Prokaryotic Cell Biology
Zemer Gitai, Princeton University
David Z. Rudner, Harvard Medical School

Protein Folding
Elizabeth Craig, University of Wisconsin–Madison
Suzannah L. Rutherford, Dartmouth Medical School

RNA Silencing Mechanisms
Natasha J. Caplen, University of California, San Francisco

Results of Working Group Discussion
R. Dyche Mullins, University of California, San Francisco, Moderator

Regulatory Roles of Lipid Microdomains
Barbara A. Baird, Cornell University
Michael Edidin, Johns Hopkins School of Medicine

Signaling through Cell Adhesion Proteins
David A. Calderwood, Duke University Medical Center/HHMI

Stem Cell Niches
Leanne Jones, Salk Institute for Biological Studies
Hasfan Lin, Yale University

X-rayl and Cell Signaling
Holly A. Ingraham, University of California, San Francisco
Kim Orb, University of Texas Southwestern Medical Center

For more information, contact the ASCB:
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www.ascb.org/meetings

NEWSLETTER JULY 2007
More MetaMorph

The metamorphosis continues! Introducing MetaMorph® 7 imaging software from Molecular Devices. Combining the most flexible and powerful tools for image acquisition, processing, and analysis, MetaMorph 7 offers a complete solution for even the most demanding live-cell imaging needs. New in MetaMorph 7:

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Expect more. We'll do our very best to exceed your expectations.
PIC Volunteers, Meet the Press (Book)!

The Society’s Public Information Committee (PIC) is looking for PIC Associates to “peer screen” Annual Meeting abstracts and spot this year’s breaking science news for ASCB’s year-ly press guide. The top science stories will be featured in Cell Biology 2007, the press book that goes to science journalists who cover the ASCB meeting.

PIC Associates will be briefed for the two screening rounds, which run from August 2–20. All work will be accomplished by phone or by email. PIC Associates are also needed for “CellSlam II, the DC Throwdown,” the sequel to last year’s blockbuster science slam inaugural. CellSlam II will be held on Tuesday, December 4, at the ASCB Annual Meeting. PIC Associates are needed at the meeting and to work on postmeeting outreach, encouraging local ASCB members to mount their own CellSlams back home, as well.

To volunteer, contact PIC Chair Rex Chisholm (r-chisholm@northwestern.edu). Questions? Contact PIC Science Writer John Fleischman (jfleischman@ascb.org).
What Scientists and Engineers Can Do to Change the Course of Science Education

Systemic Improvement
- Lead education reform through school district, state, or national organizations.
- Advocate for strategic, research-based education redesign and improvement within school districts.
- Attend a Strategic Planning Institute

Policy
- Be involved in the discussion about appropriate standards.
- Demand that all curriculum materials have a research base.
- Help establish a vision for local and national science education goals.

Management
- Join the school board.
- Advocate for strategic, research-based education redesign and improvement within your organization and local school.
- Teach schools how to use current standards to enable learning.

Professional Development
- Train teachers through professional development.
- Be a content resource for local school districts.
- Communicate your passion for science to teachers and to the public.

Classroom Enrichment
- Receive training in research-based teaching methods in Professional Development Institutes.
- Volunteer regularly in classrooms (retirees).
- Apply educational research in your own classroom.
- Be a content resource for individual teachers.
- Model the inquiry process to students and teachers in a classroom visit.

Special Services
- Advocate for strategic, research-based education redesign and improvement to your peers.
- Write letters to the editor.
- Become informed about the research behind science education.
- Lead family science activities.

Adapted from a diagram from the National Alliance of Business’s The Fourth R

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**Differential Association of Phosphatidylinositol 3-kinase, SHIP-1, and PTEN with Forming Phagosomes**

Lynn A. Kamen, Jonathan Levinsohn, and Joel A. Swanson

Fcγ receptor–mediated phagocytosis is regulated by enzymes that synthesize or hydrolyze phosphatidylinositol phospholipids (PIPs). These include the type I PI3-kinase (PI3K), which phosphorylates \( \text{PI}(4,5)P_2 \) to generate \( \text{PI}(3,4,5)P_3 \), and the lipid phosphatases PTEN and SHIP-1, which remove the 3’ and 5’ phosphates from \( \text{PI}(3,4,5)P_3 \) to generate \( \text{PI}(4,5)P_2 \) and \( \text{PI}(3,4)P_2 \), respectively. Overexpression of either phosphatase inhibits phagocytosis, whereas their inhibition or depletion accelerates phagocytosis. Here the authors have examined the dynamic localization of these enzymes and their products on the forming phagosome by means of cyan fluorescent protein (CFP) and citrine (YFP) fusions of the enzymes or of PH domains that specifically recognize distinct PIP species. They find that PI3K and SHIP-1 are recruited early in phagocytosis during initial cup formation, but that SHIP-1 is subsequently concentrated at the leading edge of the phagocytic cup. Interestingly, PTEN is excluded from the phagosome. An initial shallow gradient of \( \text{PI}(3,4,5)P_3 \) is lost upon SHIP-1 redistribution. Thus, SHIP-1 appears to regulate and coordinate \( \text{PI}(3,4,5)P_3 \)-dependent progression through distinct stages of phagocytosis, while PTEN functions as a global suppressor of \( \text{PI}(3,4,5)P_3 \) signaling.

**The Transmembrane Domain of Acid Trehalase Mediates Ubiquitin-independent Multivesicular Body Pathway Sorting**

Ju Huang, Fulvio Reggiori, and Daniel J. Klionsky

In the yeast *Saccharomyces cerevisiae*, the disaccharide trehalose, which provides membrane protection under stress conditions, accumulates during stationary phase but is rapidly mobilized when nutrients are resupplied. Trehalose hydrolysis is catalyzed by two enzymes: a cytosolic neutral trehalase (Nth1) and an acid trehalase (Ath1) believed to be localized in the vacuole. Here the authors confirm that Ath1 resides in the vacuole and show that its localization is independent of endocytosis yet requires the sequential activities of three ESCRT (endosomal sorting complex required for transport) complexes, the so called multivesicular body (MVB) machinery. ESCRT complexes are known to recognize and package ubiquitin-conjugated cargo molecules into intralumenal vesicles of MVBs. Although Ath1 has ubiquitinated lysines in its cytoplasmic domain, these are not required for its vacuolar localization; instead, the transmembrane domain of Ath1 is both necessary and sufficient. These data suggest that alternate, ubiquitin-independent mechanisms exist to trigger MVB formation, a finding consistent with previous observations that MVB internal vesicles are generated even in the absence of ubiquitinated cargo.

**The SRP RNA Links Conformational Changes in the SRP to Protein Targeting**

Niels Bradshaw and Peter Walter

Efficient Interaction between Two GTPases Allows the Chloroplast SRP Pathway to Bypass the Requirement for an SRP RNA

Peera Jaru-Ampornpan, Sowmya Chandrasekar, and Shu-ou Shan

The cotranslational targeting of newly synthesized secretory or transmembrane proteins across biological membranes is mediated by the interactions of two related GTPases, SRP54 (Ffh in *Escherichia coli*) and the SRP receptor (SR; FtsY in *E. coli*) that are highly conserved across evolution. Ffh and FtsY bind through their GTPase domains and reciprocally activate each other’s GTPase activity, which drives their dissociation following transfer of the nascent protein–ribosome complex to the translocon. Ffh consists of two domains joined by a flexible linker: the M domain, which recognizes signal sequences, and the NG domain, which includes the GTPase module. A universally conserved SRP RNA is bound to the M domain. The SRP RNA catalyzes the interaction between Ffh and FtsY by 400-fold and enhances the GTPase activity of the Ffh•FtsY complex. Through mutagenesis of Ffh, Bradshaw and Walter suggest an interesting role for the SRP RNA as a kinetic regulator of protein targeting. Mutations in conformationally dynamic regions of the M domain and the linker of Ffh abolish or diminish the catalytic effects of SRP RNA. These data suggest that the SRP RNA senses conformational changes in Ffh due to signal sequence binding and coordinates the interaction of the two GTPases with cargo loading and unloading events during protein targeting.

Jaru-Ampornpan *et al.* find support for this model in their study of chloroplast SRP (cpSRP), which as an interesting exception lacks an SRP RNA. The authors show that cpSRP54 and cpFtsY, like other SRP54/ SR family members, interact and reciprocally activate each other’s GTPase reaction. However, despite the absence of an SRP RNA, these interactions occur at rates equivalent to the SRP RNA–catalyzed rate of Ffh– FtsY interaction. Formation of the Ffh•FtsY complex involves an unfavorable rearrangement of both GTPases from an “open” conformation to a “closed” conformation in which the GTPases exhibit higher nucleotide binding specificity. Analysis of the nucleotide binding specificity of cpFtsY suggests that this protein is pre-organized in the closed conformation, bypassing the rate-limiting conformational changes otherwise catalyzed by SRP RNA and accounting, in part, for the more rapid association of cpSRP54 and cpFtsY. The authors suggest that the SRP RNA may provide a “checkpoint” to enhance specificity and fidelity of protein targeting, which is less essential for translocation in the chloroplast given the restricted number of substrates destined for the thylakoid membrane.
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Cross-Contamination of Cell Cultures: A Call for Vigilance and Authentication

We are all familiar with the pressures of life and what can happen as we rush what are considered to be routine tasks in the lab. Mistakes become more frequent under these conditions, sometimes leading to the misidentification and/or cross-contamination of cell lines—a serious problem that plagues contemporary biomedical research. The problem is magnified further by poor technique having its origin in inadequate training and a lack of vigilance in the preparation and maintenance of cell cultures.

Enduring Crisis, Costly Problem
The crisis of misidentification and cross-contamination of cell lines is not new nor is it diminishing. The problem was first identified and widely publicized by Gartler (1967) and Nelson-Rees throughout the 1970s and 1980s. For example, Nelson-Rees reported having received—at a cell culture repository he was maintaining for the National Cancer Institute (NCI)—279 cross-contaminated cultures from 45 different laboratories (Nelson-Rees et al., 1981). Other major repositories had similar experiences. For example, the German DSMZ Cell Bank reported that 14% of human hematopoietic cell lines submitted were cross-contaminated (Drexler et al., 1999). In another survey conducted by the DSMZ repository, 45 of 326 submissions (17.9%) were cross-contaminated. Of that group 42 were intraspecific contaminants (Drexler et al., 2003). More recently, Liscovitch and Ravid (2006) reported that one of the 60 NCI cell lines distributed for the evaluation of drugs and other cancer-related studies was not a drug-resistant breast cancer cell line (as listed) but an ovarian cancer cell line. The authors estimate that about 300 research papers have been published with the erroneous cell line designation.

The consequences of widespread misidentification and cross-contamination of cell lines are immeasurable. In addition to the waste of money, time, and intellectual resources, there is the loss of confidence in published work, and the integrity of science suffers.

Embracing Commitment to Change
Improved laboratory practices are needed to help stem the tide. Professional societies and research institutions, through education initiatives, can play an important role. Research supervisors should ensure that their staff adhere to the following guidelines for good cell culture work and the avoidance of cross-contamination and misidentification.

1. If possible, do cell culture work at a time of day when distractions and fatigue will be minimal.
2. Before and after working with cultures, thoroughly wipe the hood with a disinfectant.
3. Work with only one cell line or cell lineage in the hood at one time.
4. Disinfect the hood prior to and after each cell line is fed, subcultured, or handled for other purposes.
5. Each cell line or lineage must be fed, rinsed, or trypsinized, etc., with reagents specifically dedicated for that line. In other words, never share reagents among different cell lines, even if the cells are maintained in the same medium formulation.

Each cell line must have a dedicated set of reagents and supplies.
6. Plan ahead to ensure that you have an adequate supply of reagents and other materials. If you find yourself short, you may be tempted to break the cardinal rule: Never Share Reagents.
7. A pipette should be used only once, regardless of the number of flasks to be fed. Never go back into a bottle of reagent with a used pipette.
8. Examine and evaluate the cultures before they are brought to the hood. Check the cultures for growth and morphological characteristics.
9. Whenever possible, use authenticated cell lines obtained from a repository with high standards for certification and quality control.

- Establish and adhere to a logical schedule for re-evaluation of authenticity.
- Do not distribute cell lines if those cell lines can be procured from a repository.
- Do not distribute cell lines unless they have been authenticated.

More important than education initiatives in correcting the current deplorable situation is cell line authentication. Robust, relatively inexpensive methods now exist to authenticate cell lines, that is, to verify that a cell line actually is what it is purported to be (Nardone, 2007). Yet investigators, for the most part, ignore this important quality control measure (Chatterjee, 2007). Some are unaware of cross-contamination problems, others are in denial (it cannot happen in my lab), and others morph into ostriches and pretend that a problem does not exist. Against this backdrop, profession-wide compliance will never be achieved. It is for this reason that a bold proposal, namely that authentication be a condition for the receipt of research grants and for publication in our best journals, is under consideration by an independent expert panel (Nardone, 2007). We should not tolerate any longer published cell culture-based research, 15–19% of which is tainted by misidentification and cross-contamination.

References
Bush Vetos Stem Cell Bill—Again

Since coming to office in January 2001 President Bush has signed over 1,400 bills into law and only vetoed three. Two of those vetoes were vetoes of bills to expand the Bush stem cell policy. H.R.810, the Stem Cell Research Enhancement Act of 2005, was the first bill the President vetoed, in July 2006. S.5, the Stem Cell Research Enhancement Act of 2007—his third veto—was vetoed last month. Both H.R.810 and S.5 would have expanded the Bush policy to allow federally funded researchers to use human embryonic stem cells derived from excess IVF embryos that would otherwise be destroyed.

In his veto message to the Senate, the President took credit for being the first U.S. President to provide federal funds for embryonic stem cell research. “My policy did this in ways that would not encourage the destruction of embryos,” he added. Curiously, President Bush went on to boast that his policy has provided $130 million for embryonic stem cell research, and over $3 billion for research on other forms of stem cells (from adult and other non-embryonic sources).

The President also echoed a claim made by congressional opponents of embryonic stem cell research that nonembryonic stem cells are already producing results. In his remarks during the White House veto ceremony, Bush said, “Destroying human life in the hopes of saving human life is not ethical—and it is not the only option before us. We’re already seeing remarkable advances in the science and therapeutic uses of stem cells drawn from adults and children, and the blood from umbilical cords—with no harm to the donor.”

After vetoing S.5, Bush issued an Executive Order that would direct the National Institutes of Health (NIH) to support research into the development of pluripotent stem cells “without creating a human embryo for research purposes or destroying, discarding, or subjecting to harm a human embryo or fetus.” Unfortunately, the Executive Order does not provide any funds for research. Such research is already supported by the NIH. The order is viewed by many as a political device to make it appear that the President supports research.

To implement S.5, Congress would need to override the President’s veto. While there are not enough votes to override the veto in the House of Representatives, it is very possible that the Senate could override it. Strong support for embryonic stem cell research, and shrinking support for the President, could provide the 67 votes necessary to override his veto. In an unusual move, conservative Republican Sen. Judd Gregg (R-NH) has already issued a press release saying that he will vote to override. Sen. Gregg said, “We’ve heard from a number of people, including the head of the National Institutes of Health, that further research from stem cells is needed to successfully pursue cures for a number of diseases. In order to accomplish that, additional stem cell lines are needed. If we proceed in a very ethical and moral way, stem cell research can be done appropriately. Under strict guidelines, we should continue research in the stem cell area, and I will not support the President’s position on this issue.”

—Kevin M. Wilson

To read the President’s Veto Statement and Executive Order, go to http://www.whitehouse.gov/news/releases/2007/06/20070620-8.html.

Committees Craft NIH Funding Bills

Offering the National Institutes of Health (NIH) different funding levels and open access language, recently completed House and Senate bills pave the way for future negotiation. Neither, however, offers NIH advocates reason for celebration.

In the House bill, the overall budget for the NIH is $29.65 billion, $750 million or 2.6% more than appropriated for FY07 and $1.029 billion—or 3.6%—more than what President Bush requested for the NIH. The actual NIH budget would drop, however, after NIH’s increased portion of the U.S. contribution to the Global HIV/AIDS Fund is expended. In the House FY08 budget, the NIH contribution to the Fund is increased from $99 billion in FY07 to $300 billion this year. After deducting that allocation, the House NIH budget drops to $29.449 billion, which is $549 million or only 1.9% more than in FY07.

The Senate figure for the NIH budget is $29.9 billion, which is $1 billion or 3.5% more than appropriated for the current year. The NIH budget,
after the $300 billion contribution to the Global HIV/AIDS Fund is deducted, declines to $29.699 billion. This is $799 million or 2.8% more than in FY07, still below the most recent Biomedical Research and Development Price Index (inflation). Once the bills are passed by the House and Senate, House and Senate Conferees will work out a compromise bill to be presented to the President.

Open Access, Stem Cells Addressed
Both the House and Senate bills also include a requirement that any scientific manuscript based on NIH-funded research be released to the National Library of Medicine’s PubMed Central immediately upon journal acceptance for posting 12 months after publication. The language in the House and Senate bills differs only slightly. The Senate bill requires that the policy follow copyright law but the House bill makes no mention of these protections.

After President Bush vetoed S.5, a bill to expand the Bush policy regarding federally funded embryonic stem cell research, Sen. Tom Harkin (D-IA) responded by adding a provision to the Senate LHHS appropriations bill. The provision would allow federally funded researchers to use human embryonic stem cells derived before June 15, 2007. The deadline in effect in the current policy is August 9, 2001. The provision requires that the cell lines be derived from human embryos donated from IVF clinics, are in excess of clinical need, would never be implanted, and would otherwise be destroyed. This provision would expire at the end of FY08. New language would have to be added to future appropriations bills. The President is more likely to veto a bill with this provision attached to it.

—Kevin M. Wilson
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Consult an ancient cell biology textbook—ancient being anything more than 10 years old—and there it is in black and white: Prokaryotes do not have a cytoskeleton. The dynamic cellular infrastructure of tubulin, actin, and intermediate filaments (IF) belongs solely to eukaryotes, or so say the old texts. Today the prokaryotes are striking back.

The rewriting began early in the decade when researchers working with bacteria identified prokaryotic homologs for both eukaryotic tubulin and actin. In 2003, the Yale laboratory of Christine Jacobs-Wagner filled out the cast of principal actors in the cytoskeleton with the discovery in the bacterium *Caulobacter crescentus* of a prokaryotic IF-like protein. Jacobs-Wagner and her postdoc, Nora Ausmees, called the protein crescentin.

It was a startling find, according to William Margolin of the University of Texas Health Science Center at Houston. “Her discovery that *Caulobacter* contains an intermediate filament-like protein [crescentin] required for its curved shape was stunning, showing the world that bacteria now have homologs in all three of the major cytoskeletal proteins of eukaryotes.” Moreover, Jacobs-Wagner’s discovery of crescentin is only one of a string of major papers from her lab, which has been at Yale only since 2001. In Margolin’s opinion, her scientific record and her early career stage made Jacobs-Wagner a prime candidate for the ASCB’s Women in Cell Biology (WICB) Career Recognition Junior Award.

Bags of Enzymes No More

It was an opinion—and a nomination—seconded by Lucy Shapiro of Stanford University. The winner of the 2007 WICB Junior Award, Jacobs-Wagner, notes that she had the good fortune to arrive in the brave new world of prokaryotic cell biology at the right time. New technologies and new curiosity were in play. Shapiro described her former postdoc as “an absolutely first-rate scientist” and listed a string of “significant discoveries” in Jacobs-Wagner’s short career. These discoveries address how bacteria signal internally, move proteins, set landmarks, manage asymmetry, and control cell division. Jacobs-Wagner’s productivity also shows how radically prokaryotic biology has changed, according to Shapiro. No longer were bacteria considered little more than “bags of enzymes with DNA and ‘schmutz’ [debris] swimming around inside.”

Shapiro believes that Jacobs-Wagner’s discovery of the IF-like protein crescentin is yet another chapter in the increasingly complex story of bacterial cell organization. “The major point is not that these [IF] molecules have been found in bacteria but that the [prokaryotic] cell is highly organized,” Shapiro declares. “It really does exist as a complex, three-dimensional organization, and it behooves us to understand how it is regulated and what its functions are.”

Jacobs-Wagner’s discovery in *Caulobacter* of the IF-like protein crescentin is yet another chapter in the increasingly complex story of bacterial cell organization.

Yet in this strange prokaryote’s life cycle are all the big, fundamental issues of cell biology—polarization, localization, differentiation—but in one well-defined, if small, model organism, says Jacobs-Wagner.

Not the Smart One

Jacobs-Wagner was born in Liège, Belgium, in 1968, the middle child between an older sister and younger brother. “My mother was extremely supportive of education in general, but after that, it was up to us,” Jacobs-Wagner recalls. “For me and my sister, it was science. For my brother, it was art. You know how every family has a label for each child? Well, my sister was the smart one. I was the athlete. And my brother—I guess he was the troublemaker.”

The family athlete, however, was good enough in science to follow her sister into biology at the University of Liège but branched off into biochemistry, whereas her “smart” sister took the other curriculum track into immunology. In 1990, she went to fulfill her research requirement with a subsidized internship in the lab of microbiologist Staffan...
Normark at Washington University in St. Louis. There she discovered her passion for bacteria and the problem of beta-lactamase induction.

Under attack by common antibiotics such as penicillin or cephalosporin, resistant bacteria secrete the enzyme beta-lactamase to cleave a four-atom beta-lactam ring common to these drugs. Jacobs-Wagner returned to Belgium to enroll in a doctoral program in protein chemistry. However, she quietly determined to follow the problem wherever it led. Over the next five years, her quest led her to labs in Liège, St. Louis, Paris, Boston, and finally Stockholm.

Jacobs-Wagner discovered that beta-lactamase induction was part of a pathway regulating the bacterial cell's recycling of breakdown products shed internally by the cell wall. By monitoring the ratio of breakdown products to cell wall precursor, the beta-lactamase pathway could “see” an antibiotic attack as degradation products increased inside the cell. Jacobs-Wagner's discovery of this “unexpected mechanism has opened the door to new antibiotic targets that bypass some routes of drug resistance,” according to Shapiro. It also earned Jacobs-Wagner's thesis the Grand Prize at the 1997 Pharmacia/Science Magazine Young Scientist Award.

**Asymmetrical Swarmers**

For her postdoc, Jacobs-Wagner went to one of the powerhouse of the new prokaryotic cell biology, the Shapiro lab at Stanford. There careful genetics and new imaging technologies had made *Caulobacter* the hot new model organism. Yet in this strange prokaryote's life cycle are all the big, fundamental issues of cell biology—polarization, localization, differentiation—but in one well-defined, if small, model organism, says Jacobs-Wagner.

It was in *Caulobacter* that Jacobs-Wagner and Ausmees discovered crescentin, the IF-like protein that so startled the eukaryotic community. Jacobs-Wagner says that they stumbled upon it. She gives full credit to Ausmees for realizing that a peculiar “straight” *C. crescentus* mutant in a mass visual screen might be worth closer study. In turn, Ausmees credits Jacobs-Wagner with fostering a lab atmosphere where an unexpected phenotype was not brushed aside as a distraction. “You know, later on when we talked with other *Caulobacter* people about this,” Ausmees remembers, “many of them said, ‘Oh yeah, we've seen straight *Caulobacter* before.’ But nobody thought it was anything worth pursuing. They didn't want to get sidetracked.” Jacobs-Wagner was not afraid of chasing a weird phenotype, says Ausmees. Ausmees is now on the faculty at Uppsala University in her native Sweden.

“Christine is terrific to work with because she's so enthusiastic and so open,” Ausmees continues. “She knows how to inspire people, to give credit, and to encourage you. There are places where everyone is secretive, but Christine wants everything to be very democratic and out in the open. She's a wonderful scientific leader.”

**Bio Hazards Take Cup**

Jacobs-Wagner admits that, outside the lab, her old family identity as the athlete lives on, at least on the soccer fields of Yale's intramural league. This year, Jacobs-Wagner played for the Bio Hazards, the biology graduate coed soccer team that defeated both the medical and law school teams for the championship. The team includes “graduate students plus a few old people like me,” she explains. “We're not very good but we have a lot of fun. And I'm very proud that we're the champions.” Playing soccer was also how she met her husband, Matt Wagner, a web developer. They married in 2000, and they later joined last names and both became Jacobs-Wagner. Today they live just outside New Haven in Hamden.

Jacobs-Wagner loves her job nearly as much as she loves bacteria but admits that her original decision to pursue a career in the United States was a “no-brainer.” “I miss Belgium in many ways,” Jacobs-Wagner allows, “but what I really like here is that even though starting a lab can be brutal, at least you get a nice jump start. Here's your set-up money. You're on your own. If you crash, you crash, but it's your own thing.”

If scientists crash and burn, so do model organisms. “People used to study bacteria,” she points out. “Before that it was viruses. Then they moved on to study ‘higher organisms’—eukaryotes. But they left all this fascinating biology behind.” Now some of the most exciting cell biology today is being done on lower organisms that a decade ago were barely considered organized. “I think there is a boom but that we're just at the beginning.” She likens the buzz surrounding the prokaryotic cytoskeleton to the excitement 40 years ago following the breakthrough discoveries in eukaryotes of tubulin and actin. “And you know where that led. It was huge. Two-thirds of the ASCB meeting is still about the eukaryotic cytoskeleton,” she laughs. “But even that's changing now.”

—John Fleischman
Dear Labby,

I am a tenured Associate Professor in a Department of Cell Biology. At this point in my career I consider myself reasonably experienced, but something has happened for which I was not prepared. Two months ago I reviewed a paper very close to my own work (but not directly overlapping or in head-to-head competition). I rated the paper quite highly, as did the other two reviewers. (This journal shares all the comments with the reviewers.) The paper was promptly revised and accepted, and is now in press.

A week ago in my lab, we made a totally unanticipated discovery that is not only very interesting, but reveals that the entire interpretation made by the authors of the manuscript I reviewed is incorrect, as is their bottom line conclusion. I am not sure what to do. If I reveal this to the editor and she communicates this matter to the authors, they will eventually learn my identity as a reviewer when we publish our findings. On the other hand, if I keep quiet, these authors will end up unknowingly publishing a paper that is wrong. In due course, they will have to publish a Correction. The point they innocently missed in their work was nothing they could have anticipated, so they will not be thought less of when this all comes out. I would love to hear Labby’s advice.

—Puzzled

Dear Puzzled,

This happens occasionally, although your case is particularly dramatic in that the new finding so decisively erases the authors’ interpretation and conclusion. The simple scenario of letting events proceed without communication sounds relatively benign; for, as you say, they will suffer little embarrassment given how unanticipated your finding was. But Labby believes there is a much better solution.

You should communicate with the editor and authorize her to reveal your identity, for you have almost nothing to gain by remaining anonymous. You filed what sounds like a very fair-minded and supportive review. Any residual reason you might hold for wishing to remain anonymous is more than offset by the collegial service you would provide these authors by sharing your lab’s finding.

A logistic issue is whether, once your new finding is disclosed, the authors would be able to replicate it quickly and publish before your paper’s publication. That should not happen, of course. The editor might be willing to act as a negotiator on this point. At a minimum, if you proceed as Labby is suggesting, the editor and/or the authors will have to suspend publication; this will allow you to write up your lab’s finding and, perhaps, offer the paper’s authors the opportunity to modify their paper as well.

Finally, whether the authors publish their findings or not, if there is an occasion for you to report your new finding at a meeting in the next couple of months, you should take it. This would also alert any others in the field who might be led astray prior to your publication, if the other work is published as is.

—Labby

Direct your questions to labby@ascb.org. Authors of questions chosen for publication may indicate whether or not they wish to be identified. Submissions may be edited for space and style.

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**ASCB Annual Meeting Deadlines**

Reminder: many deadlines for the 47th ASCB Annual Meeting are **Thursday, July 26**. These include:

- Regular Abstract Submission
- Special Interest Subgroup Applications
- First-Time Membership Applications (to qualify for self-sponsorship of a regular abstract)
- Minorities Affairs Committee Travel Award Applications
- Undergraduate/Predoctoral Student/Postdoctoral Fellow Travel Award Applications

Also, remember to reserve early for the best hotel/room selection. Rooms begin at $118 per night.

To assist our members, the preliminary program, registration, abstract submission, and housing sites are available at www.ascb.org/meetings.

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An Elite University in Populist Times

The following commencement speech was presented in May 1999 at Washington University in St. Louis by Ursula W. Goodenough.

We are here to celebrate the scholarly achievements of the outstanding men and women who are graduating with honors from Washington University. We hereby send you forth to join the intellectual elite of our society.

Whoa! What did she say? Intellectual elite?

Most of you are likely starting to feel pretty uncomfortable. Intellectual and elite are tricky words. But why? I’d like to explore what these words mean, why they are tricky, and how we might get past feeling uncomfortable about them, and hence become more comfortable with who we are.

First, the words. Both intellectual and elite, I have discovered, come from the same Latin root, legere, which means to pick out, choose, select. Elite refers to those who stand out overall; intellectual refers to those who stand out in wisdom and understanding.

In the history of human culture, the intellectual elite were celebrated unabashedly. Whether in Africa, or Oceania, or Asia, or Western Europe, or in Native American traditions, enormous status and reverence have always been accorded to the wise ones, the healers, the poets, the interpreters of the histories and the religions and the natural world. True, the warriors and the politicians usually wound up usurping the economic resources. But in the end, their worth was tightly coupled to their association with the intellectual members of their societies, and many of the great leaders in human history have been both shrewd manipulators and deep thinkers.

Given that you have been singled out for superior performance at an elite university, does this mean that you should consider yourselves intellectuals? To be sure, people often distance themselves from the term, preferring instead something like intelligent. But if we get specific about what we mean by intellectual, I think that most of you would find the concept acceptable.

An intellectual can be characterized by four traits. First, intellectuals are curious. They are eager to figure out the answers to the questions they are asking, questions that they find extremely interesting. Second, intellectuals are flexible. They are comfortable about changing their minds when they are presented with a better idea than the one they have been holding. Third, intellectuals are interested in abstraction, in generalizing about things, rather than just categorizing specific experiences. And fourth, intellectuals have a well-developed sense of irony. They deeply appreciate both the pathos and the humor of human frailty, including their own.

I rather suspect that most of you, and indeed most graduates of Washington University, could self-identify with these four traits. So let us proceed with the assumption that your inherent disposition, and your training, allow you to feel comfortable with the idea that you are an intellectual, at least in the privacy of your own mind.

Then why is it that we feel vaguely embarrassed, and cast down our eyes, and perhaps even try to offer a refutation when a commencement speaker reminds us that we belong to an intellectual elite?

An obvious answer is that the intellectual elite is under siege in this country. In fact, the intellectual elite has never fared too well in America. The siege has been pretty continuous.

But if we focus on our current detractors, whom we can call populists, they include two groups. The first are the flat-out anti-intellectuals, including, I’m afraid, a number of our elected political representatives, who openly snigger at the liberals sitting around in their faculty lounges getting a free ride. The second perspective is more subtly anti-intellectual: It holds that the intellectual approach is somehow effete, irrelevant, and in fact responsible for our global dilemma, and claims that we must instead embrace something that is far more intuitive, right-brained, and informed by our emotions.

All of this strikes me as most unsettling. Why is it that curiosity, flexibility, abstraction, and irony are so threatening that large sectors of our society seek to deconstruct our perspective?
Irony are so threatening that large sectors of our society seek to deconstruct our perspective?

I can suggest some answers.

There are those who are afraid of the questions we are asking, and the answers we are offering, since they all too often challenge convention and hence predictability.

There are many who have learned to think in concrete slogans, indeed, who need to think in concrete slogans, and they are uncomfortable with flexibility and abstraction.

And finally, there are those who unfortunately interpret what we call irony as a manifestation of amorality. In fact, of course, our insistence that individuals can and should develop their own system of values doesn’t mean that we don’t have deeply held values of our own.

Given this distrust of the intellectual agenda, the Great American Compromise has been to put us intellectuals in a separate class, give us our universities and books and professions, and let us talk to one another while the “real folks” get on with “real things” like politics and power. In exchange, we are expected to feel vaguely apologetic about our privileged, if marginalized, position, and not make waves. Indeed, if we presume to suggest too strongly that our perspective might be of value, the word elite is transmuted to elitist and we are told that we are self-serving, out of touch, arrogant. A dear friend of mine, a distinguished professor at Columbia University, testified eloquently before a House subcommittee several weeks ago on the importance of government support for our universities. When he finished, he anticipated specific questions on his remarks. Instead, a long silence was finally broken by a single query: “Tell me, professor, what’s your current salary?”

Given the gist of my remarks, my bottom line will not be surprising. I think we are acting like wimps, which is, in fact, the way we are commonly perceived. We intellectuals somehow have to develop the courage, and the conviction, to reassert our rightful place as deeply valued members of our culture.

Importantly, we need to do something about our fear of leadership, both our fear of exerting leadership ourselves and our fear of responding positively to the leadership of those who show courage, conviction, and wisdom.

Intellectuals do not live exclusively in universities. They do just about everything, and crop up in the most unexpected locations and vocations. No matter what you proceed to do, let the intellectual flourish, and things will never get dull.

—Ursula Goodenough
for the Women in Cell Biology Committee

Let the intellectual flourish, and things will never get dull.
IN Memoriam

Charles Leblond

Charles Leblond, a pioneer in the fields of stem cell research and molecular and reproductive cell biology, passed away on April 10 at the age of 97. Throughout his career, largely spent at McGill University, Leblond was a pioneer. He was responsible for such accomplishments as showing how cells continuously renew themselves, regardless of age, and developing autoradiography. Leblond was an ASCB Councilor from 1969–1971 and longtime Society member. In 1982, he received the ASCB’s E.B. Wilson Medal, the Society’s highest honor for science.

Leblond was born in 1910 in Lille, France. He received his doctorate from the University of Paris, the Sorbonne. After graduating, Leblond came to Yale University to complete a Rockefeller Fellowship, and then returned to Paris to join the lab of Antoine Lacassagne at the Institut du Radium.

In 1941, Leblond moved to McGill University as a lecturer in histology, and quickly rose to assistant (1943), associate (1946), and then full professor of anatomy (1948). He served as the chair of the Department of Anatomy from 1957–1974.

Leblond’s early career at McGill was interrupted by WWII, during which he served in the Free French Forces. He was dispatched first to Rio de Janeiro, then to London, where he conducted medical exams of would-be soldiers.

After returning to McGill, in collaboration with Leonard Bélanger, he developed the technique of autoradiography, which permits the exact localization of radioactive molecules in tissues and cells. This procedure continues to be used today by molecular biologists to detect RNA molecules in situ, and to study the localization of genes and DNA sequences.

Leblond used autoradiography to introduce radioactive precursors of DNA, and then examine the renewal and fate of cells of several basic tissue types. He demonstrated for the first time that most cells and tissues in the adult body undergo continued renewal. Using mathematical models and modern methods of quantitation, Leblond and his colleagues estimated with remarkable accuracy the turnover and mitotic rates of numerous cell types. He and his colleagues made fascinating discoveries that resulted in the introduction of “time dimension” to cells and tissues, opening the doors to the understanding of the cell cycle and to the identification of stem cells.

During his career, Leblond published 430 scientific papers and was named a Fellow of the Royal Society of London, the Royal Society of Canada, and the American Academy of Arts and Sciences. He received honorary doctorates from Acadia University (1972), McGill University (1982), l’Université de Montreal (1985), York University (1986), and l’Université de Sherbrooke (1988), and was inducted into the Canadian Medical Hall of Fame. In 1992, he won Quebec’s Prix Marie-Victorin for his contributions to science. In 2000, he was named a Companion of the Order of Canada; the following year, he was elevated to the rank of Grand Officer of the Ordre National du Québec.

To mark his 65th birthday in 1975, Leblond was honored at an international symposium on the existence of stem cells in adult tissues; the resulting book, Stem Cells of Renewing Cell Populations, was the first formal, comprehensive account on the subject.

Instead of retiring, Leblond continued his research with an NIH Fogarty Scholarship at the National Institute of Dental Research, where he learned about immunohistochemistry. Leblond then began what became a 20-year molecular exploration culminating in his conceptualization of the basement membrane as an integrated polymer. Initially, the membrane was theorized by others to comprise layers of separated macromolecules. In late September 2006, he published his final article—about detecting the MMP9 cysteine activation switch for the first time in remodeling cartilage.

Leblond was preceded in death by his wife of 64 years, Gertrude Sternschuss, who died in 2000. After Gertrude died, Leblond married a childhood friend, Odette Lengrand, in 2001; they were both 91. Odette died in 2004. He is survived by his sons Philippe, Paul, Pierre, and his daughter Marie-Pascale—all from Montreal—and many grandchildren, other relatives, and friends.

—John Saville
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The Council of Life Sciences, which is coordinating curriculum reform for the Departments of Botany, Microbiology & Immunology, and Zoology at the University of British Columbia, invites applications for postdoctoral researchers for the Life Sciences Carl Wieman Science Education Initiative (LS-CWSEI), a five-year program for study of and innovation in science education (http://www.vpacademic.ubc.ca/CarlWieman/).

We seek individuals to work with faculty teams from the three departments and CWSEI staff to develop and coordinate biology courses and curriculum: Teams will develop course and program level learning objectives, assessments, and pedagogy; administer and evaluate assessments of student learning and of student attitudes toward science; supervise and guide development and testing of teaching and learning materials including web-based learning resources; and teaching assistant training programs. Faculty teams are focusing on areas such as first and second year biology courses, and upper-level courses such as those in genetics, ecology and evolution, cell biology, immunology, or physiology.

Candidates should have: completed their Ph.D. within the last three years in a biology discipline; excellent organizational, interpersonal, and communication skills; and a strong personal commitment to science education. English fluency is also required. Experience in educational materials or curriculum development, online teaching, project management, and familiarity with current pedagogical research at the post-secondary level will be considered assets. Candidates with a Ph.D. in Education and an M.Sc. in biology will also be considered.

The appointments will be for one year initially, and may be renewable for up to three years at the postdoctoral level. The LS-CWSEI expects to hire multiple researchers who will work with each other and with researchers in other disciplines participating in the CWSEI across the Faculty of Science.

Applicants should submit a resume, statement of teaching interests, and the names and complete contact information (including phone, fax and email) of three references to: Dr. George B. Spiegelman, Department of Microbiology & Immunology, 2506 Life Science Centre, 2350 Health Sciences Mall, University of British Columbia, Vancouver B.C. V6T 1Z3 (spie@interchange.ubc.ca). The initial deadline for applications (May 15), has been extended and we will continue to review applications until the positions are filled.

UBC hires on the basis of merit and is committed to employment equity. We encourage all qualified persons to apply; however, Canadians and Permanent Residents of Canada will be given priority. The positions are subject to final budgetary approval. Salary and title will be commensurate with qualifications and experience.
**GRANTS & OPPORTUNITIES**

**AAA Award Nomination.** The American Association of Anatomists is accepting nominations for its R.R. Bensley Award in Cell Biology. Nominees need not be members of AAA. Nomination deadline is August 1; nomination materials are due September 15. [www.anatomy.org/forms/award_forms/Bensley_Award_Nomination.asp](http://www.anatomy.org/forms/award_forms/Bensley_Award_Nomination.asp).

**National Centers for Systems Biology.** The NIH/NIGMS invites applications for Systems Biology, which promotes new conceptual and technical approaches used to discover the underlying principles of biological systems (i.e., computational analysis of data from genomics, proteomics, and other high-throughput technologies). Letters of Intent deadline is September 21; application deadline is October 22. [http://grants.nih.gov/grants/guide-files/RFA-GM-08-004.html](http://grants.nih.gov/grants/guide-files/RFA-GM-08-004.html).

**Stem Cell Research Grants.** NIH/NIGMS is accepting applications for its Human Embryonic Stem Cell (hESC) research program, which is intended to stimulate research that will lead to a better understanding of the unique properties of HESC and its potential use in regenerative medicine and clinical applications. Letters of Intent deadline is September 23; application deadline is October 23. [http://grants.nih.gov/grants/guide-files/RFA-GM-08-004.html](http://grants.nih.gov/grants/guide-files/RFA-GM-08-004.html).


**NIH Biodefense Fellowships.** Applications are being solicited from biodefense training and development researchers of prevention, detection, diagnosis, and treatment of diseases caused by potential bioterrorism agents. Grants, fellowships, and career development awards. Multiple deadlines. [www.niaid.nih.gov/biodefense/research/funding.htm](http://www.niaid.nih.gov/biodefense/research/funding.htm).

**NIGMS Grants.** The NIH/NIGMS is accepting applications for funding research in which several interdependent projects offer significant advantages over support of these same projects as individual research. Standard NIH application dates apply. [http://grants.nih.gov/grants/guide/par-files/PAS-07-381.html](http://grants.nih.gov/grants/guide/par-files/PAS-07-381.html).

**NIH Director’s Bridge Awards.** New program to provide certain investigators with continued, but limited, funding to allow additional time to strengthen their revised R01 competing renewal applications. NIH components will nominate investigators to receive this support. More information is available at [http://grants.nih.gov/grants/guide/notice-files/NOT-OD-07-056.html](http://grants.nih.gov/grants/guide/notice-files/NOT-OD-07-056.html).


**SCORE Awards.** The NIH/NIGMS is accepting applications for its Support of Competitive Research (SCORE) developmental awards designed to increase faculty research competitiveness at minority-serving institutions. The program announcement, as well as three other program announcements (PAR-06-491, PAR-06-492, PAR-06-493), can be found at [http://grants1.nih.gov/grants/guide/par-files/PA-06-490.html#PartI](http://grants1.nih.gov/grants/guide/par-files/PA-06-490.html#PartI).

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**The ASCB 2007 Call for Award Nominations**

**Norton B. Gilula Memorial Award**

Who is Eligible: An outstanding graduate or undergraduate student who has excelled in research.

How to Apply: The student or advisor should submit a one-page research statement, a list of publications, if any, the abstract submitted to the current year’s Annual Meeting, and the advisor’s letter of recommendation. Duplicate applications from graduate students may be submitted for the Gilula and Bernfield Memorial Awards.

Awards: The winner is presented a plaque. Expenses to attend the Annual Meeting are paid.

Deadline: August 1

**Merton Bernfield Memorial Award**

Who is Eligible: An outstanding graduate student or postdoctoral fellow who has excelled in research.

How to Apply: The student or postdoc or his or her advisor should submit a one-page research statement, a list of publications, a copy of the abstract submitted to the current year’s Annual Meeting, and the advisor’s letter of recommendation. Duplicate applications from graduate students may be submitted for the Gilula and Bernfield Memorial Awards.

Awards: The winner is presented a plaque and will speak in a Minisymposium at the Annual Meeting and receives financial support to attend the Annual Meeting.

Deadline: August 1

All applications and nominations should be submitted to:

**The American Society for Cell Biology**

8120 Woodmont Avenue, Suite 750

Bethesda, MD 20814-2762

ascbinfo@ascb.org

For names of prior awardees or more information, visit [www.ascb.org](http://www.ascb.org), or contact the ASCB at (301) 347-9300, or ascbinfo@ascb.org.
MEETINGS Calendar

ASCB Annual Meetings

2007
Washington, DC
December 1–5

2008
San Francisco
December 13–17

2009
San Diego
December 5–9

August 5–8. Boston, MA
Engineering Cell Biology II. www.engconfintl.org/7ak.html.

August 23–26. Vienna, Austria
EMBO Workshop, “Molecular Medicine, Drug Action and Chemical Biology in the Post-genomic Era.”
http://cwp.embo.org/w07-27/.

September 1–4. Dresden, Germany
European Life Scientist Organization Annual Meeting.
www.elsol.org.

September 17–20. Chicago, IL

September 20. Paris, France

October 3–6. Aspen, CO
The Aspen Health Forum.
www.aspeninstitute.org/healthforum.

October 8–9. Irvine, CA
National Academy of Sciences Sackler Colloquium,
“Therapeutic Cloning: Where Do We Go From Here?”

January 5–9, 2008. San Diego, CA
Genetics Society of America meeting, “Analysis: Model Organisms to Human Biology.” Abstract deadline:

February 2–6, 2008. Miami, FL


June 7–11, 2009. Zürich, Switzerland
VIII European Symposium of The Protein Society

August 22–27, 2010. Kobe, Japan
14th International Congress of Immunology.
www.ici2010.org/.

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