**DIRECTORY ATTRACTS CRITICAL MASS TO MAKE A DAY OF IT**

The Hispanic Scientists Directory currently contains the names and biographical data of about 70 scientists at NIH and FDA—surely not the actual number of people of Hispanic origin that populate NIH and FDA labs, but a sufficient and growing number of self-subscribed individuals to build Hispanic Scientists Day at NIH.

Whether this day will occur every year—and what forms recognition of the research of Hispanic scientists may take—is not certain, said Nancy Vázquez-Maldonado, a prime mover of both the directory and the day, but the professional and personal connections established through the directory are here to stay.

The directory gives each person’s phone, fax, and e-mail; institute and lab; job title, degree, and degree-granting institution; dates of arrival and expected departure from NIH or FDA; research areas of interest; and country of origin. Most scientists now listed come from Puerto Rico and the countries of South America, with the remainder representing Mexico, Spain, Central America, Cuba, and the United States. The address is

<ftp://helix.nih.gov/felcom/latino2.html>

Most scientists who have listed themselves in the directory, Vázquez-Maldonado said, credit it for having enabled them to connect with other Hispanic scientists—to collaborate or just to be able to speak Spanish.

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**CON MUCHO GUSTO: HISPANIC SCIENTISTS AT NIH SHOWCASE THEIR RESEARCH**

by Cynthia Delgado

In what its wearied but exhilarated organizers hope will be a continuing event at NIH, the first Hispanic Scientists Day (June 5) captured the diversity of research pursuits among NIH Hispanic scientists who grew up in countries or homes where Spanish was the first or second language.

A brief nine months after the creation of an NIH Hispanic Scientists Directory, the research of 30 self-identified Hispanic scientists and their multinational colleagues from 15 different institutes across campus joined in a poster display following a keynote lecture by Juan Rivera, senior investigator and head of the Signal Transduction Group (see The NIH Catalyst, January–February 2000, page 13). NIAMS.

Rivera’s NIH career started when he was a first-year college student and included working as a lab tech here while he was getting advanced degrees in immunology. His talk on Fc receptor signaling was further elaborated in one of the posters of the day, presented by Jesus Buonomo, a postbac student in his lab (see page 5).

Following is a small sample of the research presented that day.

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Alonzo García, postdoc, Laboratory of Viral Diseases, NIAID: Identification and characterization of bacterial-type DNA Holliday junction resolvase in poxviruses

In 1964, Holliday proposed a model of homologous DNA recombination to explain the sequence of molecular events that occur between two chromosomes involved in genetic exchange. A structural intermediate of the model, and a catalytic “hot spot,” was dubbed a Holliday junction (HJ). Since that time, the key molecular components of homologous recombination have been dissected in *Escherichia coli*. The dimeric endonuclease RuvC is one such component that resolves the HJ into duplex products by symmetrically nicking two of the four DNA strands. García and his colleagues at NIAID and the NLM’s National Center for Biotechnology Information are the first to identify a bacteria-like HJ resolvase in viruses that infect eukaryotic cells. He notes that “until now it had only been found in bacteriophages, bacteria, and yeast.” His team is now exploring what role the enzyme plays in virus infection.

The team reported that within the aid of “protein alignment and threading programs, they identified critical structural features within the enzyme that are conserved across a wide range of organisms.” Furthermore, the team proposed that this enzyme could be a potential target for the development of new antiviral drugs.

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**MISSION ACCOMPLISHED**

(left to right): Nancy Vázquez-Maldonado, FDA, Carmen Collazo-Cristado, NIAID, Carolina Nadel, NCBI, and OIR Assistant Director Arlyn García-Pérez, NHLIB, await the start of the day they worked months to put together.

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**JUAN RIVERA (left), NIAMS, DELIVERED THE KEYNOTE LECTURE AT THE NIH HISPANIC SCIENTISTS DAY. RAY MEJIA, PRESIDENT OF THE NIH HISPANIC EMPLOYEE ORGANIZATION, ANNOUNCED TWO SYMPOSIA ON HEALTH DISPARITIES IN MINORITY POPULATIONS SEPTEMBER 13 AND OCTOBER 4.**

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**ALONZO GARCÍA, POSTDOC, LABORATORY OF VIRAL DISEASES, NIAID:**

Identification and characterization of bacterial-type DNA Holliday junction resolvase in poxviruses

In 1964, Holliday proposed a model of homologous DNA recombination to explain the sequence of molecular events that occur between two chromosomes involved in genetic exchange. A structural intermediate of the model, and a catalytic “hot spot,” was dubbed a Holliday junction (HJ). Since that time, the key molecular components of homologous recombination have been dissected in *Escherichia coli*. The dimeric endonuclease RuvC is one such component that resolves the HJ into duplex products by symmetrically nicking two of the four DNA strands. García and his colleagues at NIAID and the NLM's National Center for Biotechnology Information are the first to identify a bacteria-like HJ resolvase in viruses that infect eukaryotic cells. He notes that “until now it had only been found in bacteriophages, bacteria, and yeast.” His team is now exploring what role the enzyme plays in virus infection.

The team reported that within the aid of “protein alignment and threading programs, they identified critical structural features within the enzyme that are conserved across a wide range of organisms.” Furthermore, the team proposed that this enzyme could be a potential target for the development of new antiviral drugs.
Becoming "Title 42" is becoming something of a household word around NIH—and many of our recent senior recruits have arrived under the Title 42 umbrella—it seems fitting to elaborate on some of its features and new uses.

Generally, Title 42 (the shorthand we use to cite a personnel authority under the Public Health Service Act, which appears in Title 42 of the U.S. Code) is a measure that has enhanced NIH's ability to bring scientists into the intramural research program (IRP) and to advance those already here. It now gives NIH greater flexibility to appoint and pay senior scientists and has simplified the personnel process. The authority was first put into law more than 30 years ago and may also be applied to visiting intramural scientists and some extramural staff, but those uses will not be addressed in this column. A recent interpretation by the HHS General Counsel's Office now lets us use this legal authority to appoint and pay scientists at all levels, including our senior ranks.

What does Title 42 authority mean for the IRP and why is it beneficial?

NIH can now use the authority for scientists appointed as staff scientists and clinicians, for tenure-track investigators, and, when it is the best option, for tenured senior investigators. Once the usual competitive search has been completed, we can appoint the scientist without further administrative red tape. Thus, Title 42 saves time and allows the hiring lab to snap up the very best researchers and make a formal offer much more quickly—a process that fits the scientific environment better.

Another advantage of Title 42 is that its pay structure is much more flexible than other systems. Unlike the “lock-step” system of the General Schedule (GS), Title 42 allows us to set pay at the appropriate level, based on the individual's scientific expertise and competitive market rates.

What are the relationships between Title 42, permanence, and tenure?

Intramural tenure at NIH denotes independent scientific resources and salary that are continued throughout a scientist's productive career. The decision to grant tenure at NIH is independent of hiring authority. Title 42 appointments must be renewed every 1 to 5 years, whereas most GS appointments do not require renewal. Title 42 positions may seem somewhat tenuous, but this is largely a matter of appearance. Many of the scientists appointed under Title 42—such as scientists in the Senior Biomedical Research Service—are leading lights in the intramural program and we expect they will spend their entire career at NIH.

How are Title 42 salaries set? What criteria are used to set pay?

First and foremost, the individual's scientific credentials must be peer-reviewed to ensure that she or he meets the requirements for the position. This is done in one of several ways. For outside recruits, the search committee reviews the accomplishments of all applicants and recommends a group of finalists to the selecting official. A number of institutes and centers (ICs) ask their promotion review boards to provide input on these recommendations. At higher salary levels, if Title 42 is applied to a current NIH scientist, the deputy director for intramural research (DDIR) asks at least two senior scientists with knowledge of the area to review the nominee. Their recommendations will be weighed along with a recent Board of Scientific Counselors review.

Once the credentials review is completed, the next step is to set salary. The scientific director and the institute director make salary recommendations for scientists in their ICs. The ICs may approve pay up to Executive Level 1—the salary of the HHS secretary and the NIH director—currently $157,000.

When the proposed salary is higher than Executive Level 1, or where a proposed increase is greater than $30,000, a board chaired by the DDIR and the deputy director for management also looks at pay. For clinical scientists, this board includes the director of the Clinical Center, several clinical directors, and the director of human resources.

Thanks to the pay flexibility of Title 42, salary decisions may take into account pay levels for comparable senior positions in universities and other private research environments. Considerations may also include the current salary of individuals recruited from outside and competing offers.

The board also considers pay equity within NIH—what scientists with similar experience, accomplishments, and duties are making. After these deliberations, most cases proceed to the NIH director for final approval; in rare instances, we must obtain approval from the office of the HHS secretary.

What is likely in the future with Title 42?

I foresee several things. First, there is growing interest in more guidelines for setting competitive salaries. What shape such guidelines would take is not clear yet, but a set of overlapping salary bands for different professional designations may evolve.

Pay equity among scientists is another very important issue—the more flexible the system, the greater the chance of imbalances. We will have to remain vigilant to ensure that salaries across institutes and centers are based on scientific accomplishments, responsibilities, and experience.

Finally, we need to ensure that our Title 42 authority is not compromised by inappropriate uses. We want to be sure that our pay structure can pass muster internally and during any outside independent review. To safeguard this mechanism that has helped us attract, recruit, and retain many outstanding scientists, we will continue careful oversight of Title 42 at NIH.

—Stephen Benowitz
NIH Director of Human Resources
The FAES (Foundation for Advanced Education in Science) Graduate School at NIH launches its fall semester, the week of September 18. The deadline for mail-in registration is August 31. On-site registration will be held from September 6 through 12. (The fall catalogue is online at the FAES web site: <http://www.faes.org>.)

Each year, about 3,000 individuals from NIH and the surrounding community participate in the school’s nearly 200 continuing education courses. The faculty is made up primarily of NIH permanent scientific staff, but FAES has a new dean who would like to expand the presence of NIH fellows at the school—not only as students but, more significantly, as teachers.

Constance Tom Noguchi, chief of the Molecular Cell Biology Section in the Laboratory of Chemical Biology, NIDDK, stepped in as FAES dean at the end of the Fall 1999 semester. Her predecessor, Paul Torrence, had worked with the NIH Fellows Committee (FELCOM; see page 10) to create an online database, accessible via the FAES web site, of postdoctoral fellows qualified to teach specific topics in biomedical research, a resource that FAES faculty and others could plumb for guest lecturers.

The next step, Noguchi says, is for more fellows to organize their own courses—an activity she helped foment by issuing a general call to NIH Interest Groups (see Directory, starting on page 6) for new courses. Among those taking up the challenge is Carol Torgan, a senior staff fellow in NHLBI, who, together with Christine Winters of NINDS, is offering a new course (Biology 431) that explores recent advances and controversies in skeletal and cardiac muscle.

Subject areas missing from the FAES curriculum or expandable, in Noguchi’s view, include such timely topics as genomics and bioinformatics, as well as more classical subjects such as developmental biology. At the moment, there are only two course offerings in bioinformatics, which—based on the overflow that greeted the new Introduction to Bioinformatics course offered last spring—may not be nearly enough.

A perusal of the new 2000-2001 catalog shows 17 new science and medicine courses, including topics in neurophysiology and development, stem cells, clinical carcinogenesis, women’s health, biomedical ethics, impacts of biotechnology, and clinical preventive medicine. And as testimony to the eclectic nature of the FAES learning environment, there are also new courses in such subjects as French and darkroom techniques.

For more information about the FAES graduate school and the fall session, call Audrey Lyons at 301-496-7976; e-mail: <lyonsa@mail.nih.gov>.

Women’s Health Research Comes of FAES Age

One of the FAES newcomers is called “Women’s Health: A Clinical Research Update”—a course, say its coordinators, whose time has come as surely as the rush to close the chasms in women’s health research that surfaced a decade ago.

Emerging on the 10th anniversary this fall of the NIH Office of Research on Women’s Health (to be celebrated September 10 and 11), the course will cover such areas as immunity and autoimmune diseases, endocrinology, cardiovascular health, bone and musculoskeletal disorders, cancer, digestive disorders, infectious diseases, behavioral and social sciences, neuroscience, and reproductive health. These issues will be addressed in the context of developmental stages—from infancy and childhood through adolescence, reproductive and middle years, and old age. Due consideration will be paid to the effects of racial, ethnic, and cultural diversity.

Meant to foster communication among biomedical and behavioral scientists in fields related to women’s health, the course is coordinated by Carmen Pastor, adjunct scientist, and Lawrence Nelson, senior clinical investigator, Gynecologic Endocrinology Unit, Section on Women’s Health Research, NICHD, with input from a planning committee headed by Vivian Pinn, NIH associate director for research on women’s health.

“Women’s Health: A Clinical Research Update,” will be held Thursday evenings, starting September 21, from 5:30 to 7:30 p.m. and will extend over the fall and spring semesters, for two credits each. It will be offered in alternate years. Prerequisites are graduate level training or experience in the biomedical or health sciences or consent of the coordinators. For more information, contact Carmen Pastor or Larry Nelson: <pastorca@mail.nih.gov> or <nelson@cc1.nichd.nih.gov>.

Ye Olde Bookshoppe

For about 25 years of the more than 40 years FAES has existed, the FAES bookstore has been the place on campus to buy the books required for the courses. But the bookstore goes beyond the required FAES texts, offering a comprehensive array of biomedical and other scientific books, including books related to the teleconferencing classes sponsored by the Office of Education and off-campus universities. Some materials are available in CD form. And some titles have little to do with scientific courses—like travel guides, cookbooks, some best sellers, and children’s books. Browsing, says store manager Jerryann Gilbert, is welcomed. The bookstore special orders titles, reserves books, and handles shipments within the United States. Government credit cards and institute purchase orders can be used, and FAES members get a 5 percent discount on all purchases.

The bookstore is located on the B1 level of building 10, room L101, opposite the cafeteria and bank and adjacent to and a bit behind the gift shop. It’s open Monday through Friday, 8:30 am to 4:00 pm. Its web address is <www.faes.org/bookstor.htm>.

Research Festival Poster Deadline

The NIH Research Festival—the annual showcase of the Intramural Research Program—is scheduled for October 10 through 13. The deadline for online poster registration is 5:00 p.m., Monday, August 7. Applicants will be notified of acceptance by August 21. The festival organizing committee, co-chaired this year by NHLBI scientific directors Robert Balaban and Elizabeth Nabel, requests a limit of one poster submission per first author. Plenary sessions feature nitric oxide, angiogenesis, and genome analysis. A schedule of events and online poster registration can be found at <http://festival2000.nih.gov>.
elements of RuvC . . . in uncharacterized open reading frames from all poxviruses and an iridovirus." The group noted that the gene product of the Vaccinia virus gene A22R had sequence and structural similarities to the RuvC bacterial resolvase.

Further, they found that the recombinant protein rA22 specifically bound to and cleaved a synthetic HJ, yielding duplex molecules. This catalytic activity was not present against single- or double-stranded DNA, Y-junctions, or duplex-branched molecules. Although mutations in conserved amino acids in rA22 required for endonuclease activity abrogated catalytic activity, specific binding to the HJ was not inhibited. They postulated that the HJ resolvase plays a role during Vaccinia virus infection through its effects on viral DNA processing and resolution of recombinant DNA intermediates. This theory was supported by the absence of resolution when they co-transfected a mutated A22R and a cruciform-structure-containing plasmid (which has a "Holliday-like" junction) in cells infected with wild-type virus.

Carmen M. Collazo-Custodio, postdoc, Laboratory of Parasitic Diseases, NIAID:

The IFN-γ-inducible GTPase (IGTP) is required for in vivo control of Toxoplasma gondii infection in both hematopoietic and nonhematopoietic host cells.

Toxoplasma gondii is an intracellular protozoan parasite and the etiologic agent responsible for toxoplasmosis. The early acute phase of infection is characterized by rapid replication in a variety of nucleated host cells. A potent cell-mediated immune response by the host is thought to result in a shift to the chronic phase in which slow-growing forms called bradyzoites are contained within tissue cysts. These cysts convey protective immunity but can also be the source of reactivated infections. Although the precise mechanisms are not fully understood, numerous studies have shown the importance of interferon-γ (IFN-γ) in both acute and chronic phases of T. gondii infection.

Collazo-Custodio and her NIAID colleagues, in collaboration with Gregory Taylor, a former NCI postdoc now at Duke University Medical Center in Durham, N.C., studied mice that lacked the IFN-γ-inducible GTPase (IGTP) to determine the nature of its role in the intracellular control of T. gondii. They found that IGTP-deficient mice exhibited a complete loss of host resistance to infection, as well as the inability to clear the parasites from host tissues. These animals did, however, retain normal IL-12, IFN-γ, and nitric oxide levels associated with essential mechanisms of the immune response.

Reciprocal bone marrow chimeras between knockout and wild-type mice revealed the tissue compartments in which IGTP functions in host resistance. In the control group, wild-type mice reconstituted with wild-type bone marrow were resistant to T. gondii infection and displayed long-term survival. But wild-type mice reconstituted with knockout bone marrow and knockout mice reconstituted with either wild-type or knockout bone marrow exhibited similar profiles when infected, dying within 9 to 11 days of exposure. These observations, the team reported, indicate that "IGTP is required for control of T. gondii infection by both hematopoietic and nonhematopoietic cells and . . . is likely to be involved in the common intracellular pathway used by these two lineages to limit tachyzoite growth."

The group has now shifted its focus to the chronic phase of infection and to localization studies of IGTP in normal vs. infected cells.

Maria Ruiz-Hidalgo, Fogarty fellow, Laboratory of Immunobiology, CBER, FDA:

Notch-1 is required for adipogenesis of 3T3-L1 cells and affects the way these cells interpret the Ras signal.

The Notch family of genes encodes transmembrane receptors, which are responsible for intracellular signals that affect cell fate during differentiation. Numerous reports have demonstrated that in some systems Notch expression is a characteristic of the undifferentiated state of many cell types during development. Ruiz-Hidalgo and her colleagues have found the contrary in their system.

Examining adipogenesis of 3T3-L1 cells, a well-characterized system of differentiation, the team showed that Notch-1 acts as an accessory to differentiate the cells. When Notch-1 signaling was blocked by ligand interference or transfection with constructs encompassing Notch-1 cDNA in antisense orientation, the cells remained in an undifferentiated state despite hormone induction. To explain this phenomenon, the team explored downstream events in the differentiation pathway, specifically, the expression of transcription factors of the helix-loop-helix family, such as PPARα and PPARγ. They found that when Notch-1 expression was decreased, so was the expression of these transcription factors. "PPARγ is especially important," says Ruiz-Hidalgo, "and PPARγ expression is decreased when Notch-1 is not in the cells."

Their recent investigations suggest that the levels of expression of Notch-1 are important for the interpretation of extracellular signals. In opposition to previous reports indicating that constitutively active Ras induces adipogenesis of 3T3-L1 in the absence of inducers, they observed that activated Ras expression induced transdifferentiation and no differentiation in control 3T3-L1 cells and in cells with low Notch-1 expression. Interestingly, properties associated with oncogenic transformation were inversely proportional to the level of Notch-1 expression in these cells. This effect appeared to be mediated, at least in part, through a different pattern of activation of MAP kinase.

"With much more work," Ruiz-Hidalgo says, light could possibly be shed on new strategies to block malignant transformation or control the differentiation of cells grown for transplantation and cell therapy.

Carolina Nadel, postdoc, NCBI, NLM: Genes and disease—a web site of short descriptions of inherited disorders

Nadel is a member of the team that provides ongoing update of the NCBI "Genes and Disease" web site, <http://www.ncbi.nlm.nih.gov/disease/>, a collection of short reports on human genetic diseases. Although the site was established to disseminate information to the general public, it’s useful for physicians as well, she says. Each disease page includes a clinical synopsis, followed by the condition’s genetic underpinnings and, when applicable, information about the involved proteins. The last paragraph, Nadel says, "usually looks to the future" and may describe mouse models, diagnostic factors, and treatment options.

Among the links that connect users to pertinent databases are OMIM,
Yesenia Rivera, pre-IRTA cancer research trainee, Tumor Cell Biology Section, Medicine Branch, NCI.
Geldanamycin, an Hsp90 antagonist, blocks both NFkB and AKT-dependent survival pathways in tumor cells

Rivera cites the wording in her group's abstract to explain the rationale of their research: “Several anticancer drugs, including Adriamycin, have been shown to induce survival pathways in cancer cells, thus counteracting their own cytotoxicity. The transcription factor NFkB and kinase AKT mediate two of the survival pathways.” She and her teammates are examining ways to block these pathways.

The group's latest data demonstrate that geldanamycin (GA) can augment the cytotoxic effects of Adriamycin. GA is an antagonist to the chaperone protein Hsp90, which impairs stability and function to kinases involved in both pathways. GA exerts its effects by removing apoptotic blocks induced by Adriamycin along both pathways. For example, GA inhibited Adriamycin-induced NFkB activation, possibly by destabilizing IKK kinases (IKKα and IKKβ), thereby blocking NFkB accumulation in the nucleus and promoting induction of apoptosis. Similarly, they found that GA destabilizes AKT, thus preventing the phosphorylation of an apoptotic transcription factor, FKH1, resulting in its nuclear accumulation and the triggering of apoptosis.

The combination of Adriamycin and GA scored higher than either alone in assays for toxicity and apoptotic activity. A Phase I clinical trial of a GA derivative in the treatment of solid tumors is underway, and a Phase II trial of a combination GA-Adriamycin arm is being considered.

Nancy Vázquez-Maldonado, postdoc, Division of Therapeutic Proteins, CBER, FDA:
IL-4 suppresses IFN-γ-induced FcγRI (CD64) gene expression in human monocytes by inhibiting STAT1 activation.

The IFN-γ signal transduction pathway is initiated by a specific ligand-receptor interaction that triggers a series of phosphorylating and signaling events that culminate in transcription stimulation of genes such as, that influence the final phenotype of activated macrophages, such as FcγRI, ICAM-1, and B7.

IL-4 is a cytokine known to inhibit several IFN-γ-inducible genes in monocytes. Vázquez-Maldonado and FDA colleagues, with collaborators at Virginia Commonwealth University in Richmond, believe they have deciphered one mechanism by which IL-4 mediates this inhibition.

Pretreatment of monocytes with IL-4 significantly reduced both the mRNA levels of FcγRI and the expression of its protein, CD64. These findings correlated with reduced tyrosine phosphorylation and nuclear translocation of STAT1 (signal transducers and activators of transcription), signal transduction events associated with IFN-γ-inducible gene expression.

Several lines of evidence suggested that de novo protein synthesis was required for the inhibitory effects of IL-4.

First, inhibition of STAT1 activity required pretreatment of cells with IL-4 an hour before stimulation with IFN-γ. Second, IL-4 inhibition was abrogated by the addition of actinomycin D, a potent transcription inhibitor, and correlated with increased expression of the SOCS-1 (suppressors of cytokine signaling) gene.

Further studies in a murine system indicated that IL-4 inhibition was dependent upon SOCS-1 protein and STAT6 as well. SOCS-1 is known to prevent transcription initiation by blocking STAT1 activation in the JAK (Janus kinase)/STAT signaling pathway. The team found that "IL-4 induced the expression of SOCS-1 and inhibited activation of STAT1 in macrophages from wild-type but not STAT6 knockout mice."

These studies, Vázquez-Maldonado says, should further elucidate the counter-regulatory pathways of cytokines in human monocytes and may have implications in the management of chronic inflammatory diseases.

Jesus Buonomo, postbac, Arthritis and Rheumatism Branch, NIAMS:
Studies on Vav localization and function: Vav is a proto-oncogene that functions as a guanine nucleotide exchange factor for the Rho family of proteins and is believed to play an essential role in signal transduction of hematopoietic cells. The protein product of the Vav1 gene exhibits several structural motifs, including a conserved Dbl homology domain and an SH2 domain that interact with kinases, adaptor molecules, and negative regulators of signal transduction. Vav1 becomes associated with the plasma membrane of activated mast cells, in cholesterol-rich lipid rafts upon antigen cross-linking of the high-affinity receptor for Ige (FcεRI).

Buonomo and his colleagues began their investigation by asking the question: "Is Vav1 localization to the plasma membrane and/or lipid rafts sufficient to activate function?" They looked for answers using colocalization studies with fluorescent or epitope-tagged constructs of Vav1. These constructs included sequences targeting Vav1 either to the plasma membrane (PM) or the lipid raft, as well as mutations in functional domains.

Preliminary results indicated that Vav1 localized to the PM as a micro-aggregate upon FcεRI stimulation, but was not PM-associated in unstimulated cells. Further, they observed that FcεRI and Vav1 co-localization was dependent on the SH2 domain of Vav1.

They also discovered that Vav1 co-localized with LAT (linker of the activation of T cells), a protein found in lipid rafts. The group now plans to study the effect of Vav1 localization and function on signaling via the small GTPase Rac1 and the c-Jun NH2-terminal kinase (JNK).

The research team, including Nancy Vázquez-Maldonado, Cynthia Delgado, and Yesenia Rivera, is pushing the boundaries of understanding the intricate pathways that govern inflammation and disease. Their work promises to provide new insights into the mechanisms that underlie these conditions and potentially point the way to more effective therapeutic strategies.

[Image of Nancy Vázquez-Maldonado, postdoc, Division of Therapeutic Proteins, CBER, FDA]

Nancy Vázquez-Maldonado

[Image of Jesus Buonomo, postbac, Arthritis and Rheumatism Branch, NIAMS]

Jesus Buonomo

[Image of Yesenia Rivera, pre-IRTA cancer research trainee, Tumor Cell Biology Section, Medicine Branch, NCI]

Yesenia Rivera

[Image of Cynthia Delgado]

Cynthia Delgado
INTERINSTITUTE INTEREST GROUP DIRECTORY

Web Access
Note: Although not all the sites are up to date, nearly all the Interest Groups have web sites that can be accessed through the NIH Home Page (http://www.nih.gov/) by clicking on "Scientific Resources," then "Special Interest Groups," and then the targeted group(s).

MAJOR INTEREST GROUPS

Cell Biology Interest Group
Meeting time: Once every four months
Meeting place: Building 32, Library
Contact: Jennifer Lippincott-Schwartz
Phone: 402-1010, 402-1009
E-mail: <jlippin@helix.nih.gov>
Listserv: subscribe to CELBIO-L

Clinical Research Interest Group
Meeting time and place: sponsors Clinical Center Grand Rounds once every other month
Contact: Cliff Lane
Phone: 496-7196
E-mail: <clane@nih.gov>

Genetics Interest Group
Meeting time: Usually 2nd Tuesday, 4:00 pm
Meeting place: Building 49, Conference Room A and B
Contact 1: Heinz Arnheiter
Phone: 496-1645
E-mail: <ha3p@nih.gov>
Contact 2: Beverly Mock
Phone: 496-2360
E-mail: <bev@helix.nih.gov>
Listserv: subscribe to GIG-L@list.nih.gov

Immunology Interest Group
Meeting time: Each Wednesday (except summer), 4:15 pm
Meeting place: Building 10, Lipsett Auditorium
Contact: William Paul
Phone: 496-5046
E-mail: <wpaul@niaid.nih.gov>
ListServ: subscribe to IMMUNI-L at ListServ@LIST.NIH.GOV

Molecular Biology/Biochemistry Interest Group
Meeting time: Yearly to consider speakers
Meeting place: Building 8, Room 122
Contact: Reed Wickner
Phone: 496-3542
E-mail: <wickner@helix.nih.gov>

Neuroscience Interest Group
Meeting time: alternate Fridays, 4:30 pm
Meeting place: Cloisters, Rathskeiler
Contact 1: Chip Gerfen
Phone: 496-1341
E-mail: <gerfen@helix.nih.gov>
Contact 2: Betsy Murray
Phone: 496-5625, X-227
E-mail: <eam@ln.nimh.nih.gov>

Structural Biology Interest Group
Meeting time and place: Announced to members by e-mail and regular mail
Contact 1: Adrian Parsegian
Phone: 496-6561
E-mail: <parsegi@helix.nih.gov>
Contact 2: Marius Clore
Phone: 496-0782
To register for e-mail announcements:
E-mail <cch@discus.niams.nih.gov>

OTHER INTEREST GROUPS

AIDS Interest Group
Meeting time and place: Varies
Contact: Fulvia Veronese
Phone: 496-3677
E-mail: <veronese@od.nih.gov>
ListServ: subscribe to AIDSINTG-L

Apoptosis Interest Group
Meeting time: 1st Monday, 4:00 pm
Meeting place: Building 30, Conference Room 117
Contact 1: Colin Duckett
Phone: 594-1127
E-mail: <duckett@helix.nih.gov>
Contact 2: Yves Pommier
Phone: 496-7944
E-mail: <ypom@nih.gov>

Behavioral and Social Sciences Interest Group
Meeting time: Varies, in the fall and spring
Meeting place: See NIH Calendar of Events
Contact 1: Jaylan Turkkan
Phone: 443-1263
E-mail: <jaylan@nih.gov>
Contact 2: Ronald Abeles
Phone: 496-7859

BSSR Methodology and Measurement Interest Group
Meeting time: First or second Tuesday, 8:30 am
Meeting place: Building 45, Room 3AS10
Contact: Jared Jobe
Phone: 496-3137
E-mail: <jared_jobe@nih.gov>

Bioethics Interest Group
Meeting time: 1st Monday (except 2nd Monday, September; usually does not meet during summer), 3:00 pm
Meeting place: Natcher, Room D, or Building 31, conference room 7, check yellow sheet or web site
Contact: Miranda Kelty
Phone: 496-9222
E-mail: <mk46u@nih.gov>

Biophysics Interest Group
Meeting time and place: Varies (often Building 10, Bunim Room
Contact: Peter Bassler
Phone: 435-1949
E-mail: <pbassler@helix.nih.gov>

Birth Defects and Teratology Interest Group
Meeting time: Quarterly seminars
Meeting place: Videoconference between Bethesda and Research Triangle Park, N.C.
Contact 1: Megan Adamson
Phone: 443-1354
E-mail: <madamson@willco.niaaa.nih.gov>

Breast Cancer Think Tank
Meeting time and place: Varies
Contact 1: Joanna Zukewski
Phone: 402-0985
E-mail: <zukewski@nih.gov>
Contact 2: Patricia Steeg
Phone: 496-9753

Calcium Interest Group
Meeting time: Usually Tuesday, 3:00 pm
Meeting place: Building 49, Room 1A50
Contact 1: Arthur Sherman
Phone: 496-4325
E-mail: <asherman@nih.gov>

To subscribe to the listservs for most of these groups, e-mail listserv@listserv.nimh.nih.gov with subscribe listserv eqlistname as the body of the message.
Cornea Interest Group
Meeting time: 1st Monday, 8:30 am
Meeting place: Building 6, Room 409
Contact 1: Joram Piigtorsky
Phone: 496-9467
E-mail: <joramp@intra.nei.nih.gov>
Contact 2: Janine Davis
E-mail: <davisj@intra.nei.nih.gov>

Cultural and Qualitative Research Interest Group
Meeting time: 1st Wednesday, 9:00 am
Meeting place: Neuroscience Center, B1/B2
Contact 1: Suzanne Heurtin-Roberts
Phone: 443-0369
E-mail: <sheurtin@willco.niaa.nih.gov>
Contact 2: Wendy Smith
Phone: 443-8771

Cytokine Interest Group
Meeting time: Quarterly symposia
Meeting place: Varies
Contact 1: Rachel Caspi
Phone: 435-5455
E-mail: <rcaspi@helix.nih.gov>
Contact 2: Howard Young
Phone: 1-301-846-5700
E-mail: <youngh@mail.ncifcrf.gov>

Developmental Biology Interest Group
Meeting time and place: Varies
Contact 1: Tom Sargent
Phone: 496-0369
E-mail: <tsargent@nih.gov>
Contact 2: Peggy Zelenka
Phone: 496-7490
E-mail: <zelenkap@intra.nei.nih.gov>

DNA Repair Interest Group
Meeting time: 3rd Tuesday, 12:30 pm
Meeting/Videoconference: Natcher, Room H; GRC (Baltimore), Room 1E03; FCRDC, Building 549, Conf. Rm. A; NIEHS (Research Triangle Park, NC) Building 101, Room B20; SUNY, Stony Brook; University of Texas, MD Anderson Cancer Center, Smithville, TX; Lawrence Livermore (CA) National Laboratory; University of Michigan, Ann Arbor; University of Kentucky, Lexington
Contact 1: Kenneth Kraemer
Phone: 496-9033
E-mail: <kraemerk@nih.gov>
Contact 2: Wilhelm Bohr
E-mail: <vbohr@nih.gov>

Domestic Violence Research Interest Group
Meeting time and place: To be announced
Contact: John Unhau
Phone: 496-7515
E-mail: <unhau@nih.gov>

Drosophila Interest Group
Meeting time: 3rd Tuesday, 1:15 pm
Meeting place: Building 6B, Room 4B129
Contact 1: Sue Haynes
Phone: 295-9791
E-mail: <shaynes@usuds.mil>
Contact 2: Jim Kennison
E-mail: <kennisonj@exchange.nih.gov>

Drug Discovery Interest Group
Meeting time: Usually one Thursday a month, 3:00 pm
Meeting place: Building 37, Room 5A21
Contact: John Weinstein
Phone: 496-9571
E-mail: <weinstein@dtpax2.ncifcrf.gov>

Economics Interest Group
Meeting time and place: Varies
Contact 1: James A. Schuttinga
Phone: 496-2229
E-mail: <js12@nih.gov>
Contact 2: Agnes Rupp
E-mail: <ar2f@nih.gov>

Endocrinology Interest Group
Meeting time and place: Varies
Contact 1: George Chrousos
Phone: 496-5800
E-mail: <George_Chrousos@nih.gov>
Contact 2: Phil Gold
Phone: 496-1945

Epidemiology and Clinical Trials Interest Group
Meeting time and place: Varies (subscribe to ListServ for notices)
Contact 1: Martina Vogel-Taylor
Phone: 496-6614
E-mail: <martinav@nih.gov>
Contact 2: Bill Harlan
Phone: 496-1508
ListServ: subscribe to Epidem-L at listserv@listserv.nih.gov

Epilepsy Interest Group
Meeting time: Wednesdays, 3:00 pm
Meeting place: Building 10, Room 5S235
Contact: Timothy Dunn
Phone: 402-2978
E-mail: <dunn@ninds.nih.gov>

Fluorescence Interest Group
Meeting time: 2nd and 4th Friday, 4:00 pm
Meeting place: Building 10, usually Room 5N261
Contact: Jay Knutson
Phone: 496-2557
E-mail: <jaysan@helix.nih.gov>
Contact 2: Dan Sackett
Phone: 594-0358
E-mail: <sackettdi@mail.nih.gov>
### Interinstitute Interest Group Directory

#### Gene Therapy Interest Group
- **Meeting time:** 2nd and 4th Thursday, 2:00 pm
- **Meeting place:** Building 10, Lipsett Auditorium
- **Contact:** Richard Morgan
  - Phone: 402-1833
  - E-mail: <rmorgan@nhgri.nih.gov>
- **Contact:** Fabio Candotti
  - Phone: 402-1833
- **E-mail:** <weinstein@harden.od3.ncifcrf.gov>

#### Genomics and Bioinformatics Interest Group
- **Meeting time:** Usually one Thursday a month, 3:00 pm
- **Meeting place:** Building 37, Room 5A21
- **Contact:** John Weinstein
  - Phone: 496-9571
  - E-mail: <weinstein@dtpax2.ncifcrf.gov>
- **E-mail:** <steinbac@helix.nih.gov>

#### Glycobiology Interest Group
- **Meeting time and place:** Varies
- **Contact:** Diana Blithe
  - Phone: 435-6990
  - E-mail: <blithed@nih.gov>
- **ListServ:** Subscribe to GLYCO-L@LIST.NIH.GOV

#### GTP Binding Proteins Interest Group
- **Meeting time:** Irregular
- **Meeting place:** FAES Social & Academic Center
- **Contact:** R. Victor Rebois
  - Phone: 496-2007
  - E-mail: <rebois@box-r.nih.gov>

#### Hard Tissue Disorders Interest Group
- **Meeting time:** Day varies; 9:30 am
- **Meeting place:** Building 30, Room 117
- **Contact:** Pamela Robey
  - Phone: 496-4563
  - E-mail: <probe@yoda.nidr.nih.gov>
- **Contact:** Michael Collins
  - Phone: 496-4913

#### Head and Neck Cancer Interest Group
- **Meeting time:** To be announced
- **Meeting place:** Building 30, Room 117
- **Contact:** Adrian Senderowicz
  - Phone: 594-5270
  - E-mail: <adrian.senderowicz@nih.gov>
- **Contact:** Wendy Weinberg
  - Phone: 301-827-0709
  - E-mail: <weinberg@ebcr.fda.gov>

#### History of Biomedical Research Interest Group
- **Meeting time:** Second Tuesday, 3:30 pm
- **Meeting place:** Varies; check web site
- **Contact:** Richard Morgan
  - Phone: 496-0610
  - Contact: Victoria Harden
  - E-mail: <hardenv@od3.ncifcrf.gov>

#### Human Development Across the Lifespan Interest Group
- **Meeting time and place:** By e-mail alert
- **Contact:** Kim Roberts
  - Phone: 496-0420
  - E-mail: <kim_roberts@nih.gov>

#### Image Processing Interest Group
- **Meeting time:** 3rd Thursday, 11:00 am
- **Meeting place:** Building 10, Room B1N256
- **Contact:** Benes Trus
  - Phone: 496-2250
  - E-mail: <trus@helix.nih.gov>
- **Contact:** Matt McAuliffe
  - Phone: 594-2432

#### Integrative Neuroscience Interest Group
- **Meeting time:** Alternate Thursdays, 4:00 pm
- **Meeting Place:** Building 49, Room 1A51
- **Contact:** Betsy Murray
  - Phone: 496-5625, X-227
  - E-mail: <betsy@ln.nimh.nih.gov>

#### In Vivo NMR Interest Group
- **Meeting time:** Varies
- **Meeting place:** Building 10, Room B1N256
- **Contact:** Jeff Duyn
  - Phone: 594-7305
  - E-mail: <jhd@helix.nih.gov>

#### Java Interest Group
- **Meeting time:** 2nd Thursday, 4:00 pm
- **Meeting place:** Building 12B, second floor Conference Room
- **Contact:** Jai Evans
  - Phone: 594-2900
  - E-mail: <evansj@helix.nih.gov>

#### Knowledge Management Interest Group
- **Meeting time and place:** To be determined
- **Contact:** Geoffrey Marsh
  - Phone: 594-2423, ext. 213
  - E-mail: <geoff@mail.nih.gov>

#### Lambda Lunch (Bacterial and Phage Genetics)
- **Meeting time:** Each Thursday, 11:00 am
- **Meeting place:** Building 30, Room 1B13
- **Contact:** Susan Gottesman
  - Phone: 496-3524

#### Light Microscopy Interest Group
- **Meeting time:** Monthly, Tuesday, noon
- **Meeting place:** Building 10, Room 4B51
- **Contact:** James McNally
  - Phone: 435-6402
  - E-mail: <geoff@mail.nih.gov>

#### Lymphoma and Leukemia Interest Group
- **Meeting time:** 1st and 3rd Thursday, 11:00 am
- **Meeting place:** Building 10, Room 7C101
- **Contact:** Lewis Pannell
  - Phone: 435-6402
  - E-mail: <L_Pannell@nih.gov>

#### Mass Spectrometry Interest Group
- **Meeting time:** Varies
- **Meeting place:** Building 10, Room 9S235 (Bunim Room)
- **Contact:** Dan Sackett
  - Phone: 594-0358
  - E-mail: sackett@mail.nih.gov

#### Microarray Users Group
- **Meeting time and place:** Varies
- **Contact:** Katherine Peterson
  - Phone: 496-3059
  - E-mail: katherine.peterson@nih.gov

#### Microtubule Interest Group
- **Meeting time:** Varies
- **Meeting place:** Building 10, Room 9S235 (Bunim Room)
- **Contact:** Dan Sackett
  - Phone: 594-0358
  - E-mail: sackett@mail.nih.gov

#### Mitochondria Interest Group
- **Meeting time:** 1st Monday, 3:00 pm
- **Meeting/Videoconference:** Natcher, Room H; NIEHS, Research Triangle Park, NC; GRC, Baltimore; University of California at Davis, University of Maryland, Baltimore
- **Contact:** Steve Zullo
  - Phone: 435-3576
  - E-mail: <zullo@helix.nih.gov>

#### Molecular Modeling Interest Group
- **Meeting time:** See <http://mignet.nih.gov/MMIG>
- **Meeting place:** Building 12A, conf. rooms
- **Contact:** Peter Steinbach
  - Phone: 496-1100
  - E-mail: <steinbach@helix.nih.gov>
Motility Interest Group
Meeting time: 1st Monday, 4:00 p.m.
Meeting place: Building 10, Banim Room 98235
Contact: Jim Sellers
Phone: 496-6887

Mouse Club
Meeting time: 1st Tuesday, 4:00 pm
Meeting place: Building 31, Room 2A52, or Building 6A, Room 405
Contact: Heiner Westphal
Phone: 402-0545
E-mail: hw@helix.nih.gov

Mycobacterial Interest Group
Meeting time: Alternate Mondays, 10:30 am
Meeting place: Building 29, Room 121, or Twinbrook II, 2nd-floor conference room
Contact 1: Clifton Barry
Phone: 435-7509
E-mail: clifton_barry@nih.gov
Contact 2: Mike Brennan
Phone: 496-9559

Nerve-Muscle Interest Group
Meeting time: Alternate Wednesdays, 9:00 am
Meeting place: Building 36, Room 1B07
Contact 1: Matt Daniels
Phone: 496-2898
E-mail: mdaniels@codon.nih.gov
Contact 2: Zuhang Sheng
Phone: 435-1496

Neural-Immune Interactions Interest Group
Meeting time: One Tuesday a month (except July and August), 4:00 pm
Meeting place: Building 10, Room 11S235
Contact 1: Craig C. Smith
Phone: 496-4561
E-mail: ccs@codon.nih.gov

Neurobiology Interest Group
Meeting time: alternate Fridays, 4:30 pm
Meeting place: Cloisters, Rathskeller
Contact 1: Chip Gerfen
Phone: 496-1341
E-mail: cgerfen@helix.nih.gov
ListServ: http://intra.nih.gov/nig/

Neuroinformatics Interest Group
Meeting time: 2nd Tuesday, 12:00 noon
Meeting place: Building 49, Conference Room 1A/B
Contact 1: Rochelle Small
Phone: 402-3464
E-mail: rochelle_small@nih.gov
Contact 2: Yuan Liu
Phone: 496-3108

PET Interest Group
Meeting time: Each Friday, 2:00 pm
Meeting place: Building 10, Room 1C520
Contact: Peter Herscovitch
Phone: 402-4297
E-mail: herscovitch@nih.gov

Phage-Tech Interest Group
Meeting time and place: Varies
Contact 1: Steve Zuollo
Phone: 435-3576
E-mail: zuollo@helix.nih.gov
Contact 2: Carl Merril
Phone: 435-3583

Pigment Cell Research Interest Group
Meeting time: 3rd Monday, 3:00 pm
Meeting place: Building 49, first-floor Conf. Room
Contact 1: Bill Pavan
Phone: 496-7584
E-mail: bpavan@nih.gov
Contact 2: Vincent Hearing
Phone: 496-1564

Protein Trafficking Interest Group
Meeting time: 2nd Tuesday, 3:30 pm
Meeting place: Building 10, Room 9S235
Contact 1: Harris Bernstein
Phone: 402-4770
E-mail: harris_bernstein@nih.gov
Contact 2: Peng Loh
Phone: 496-3239

RNA Club
Meeting time: 1st Tuesday (except August), 4:00 pm
Meeting place: Building 41, Room C509
Contact 1: Carl Baker
Phone: 496-2078
E-mail: ccb@nih.gov
Contact 2: Susan Haynes
Phone: 301-295-9791
E-mail: shaynes@usuhls.mil

Science Writing Interest Group
Meeting time and place: To be announced
Contact 1: Edward McSweeney
Phone: 402-8370
E-mail: emcsweeney@niaid.nih.gov
Contact 2: Alisa Machalek
Phone: 496-7301

Signal Transduction Interest Group
Meeting time: Alternate Fridays, 4:30 pm
Meeting place: 5 Research Court, Room 2A08
Contact 1: John Northup
Phone: 496-9167
E-mail: jnorthup@codon.nih.gov
Contact 2: James Battey
Phone: 402-0900

Synaptic and Developmental Plasticity Interest Group
Meeting time: Wednesday, 12:00 noon
Meeting place: Building 49, Room 1A50
Contact 1: Serena Dudek
Phone: 402-7195
E-mail: sdudek@helix.nih.gov
Contact 2: Bai Lu
Phone: 435-2970

Therapeutic Oligonucleotides Interest Group
Meeting time: Last Thursday, 4:00 pm
Meeting place: Building 10, Room 2C16
Contact: Yoon Cho-Chung
Phone: 496-4020
E-mail: chochung@helix.nih.gov

Transcription Factors Interest Group
Meeting time: 1st Thursday (except July-Sept.), 1:30 pm
Meeting place: Building 49, Conf. Rm. B
Contact 1: Soney Simons
Phone: 496-6796
E-mail: steroids@helix.nih.gov
Contact 2: Uli Siebenlist
Phone: 496-7662
ListServ: subscribe to TFACTORS

Tumor Angiogenesis & Invasion Working Group
Meeting time and place: Posted at website
Contact 1: William Figg
Phone: 402-3622
E-mail: wdfigg@helix.nih.gov
Contact 2: Steven Libutti
Phone: 496-5049

Viral Hepatitis Interest Group
Meeting time: One Monday a month, 3:30 pm
Meeting place: Building 10, 9S235 (Bunin)
Contact: T. Jake Liang
Phone: 496-1721
E-mail: tliang@nih.gov

Virology Interest Group
Meeting time: Mini-symposia 1-2 times/year
Meeting place: to be announced
Contact 1: Claus Strebel
Phone: 496-3132
E-mail: kstrebel@nih.gov
Contact 2: John Patton
E-mail: jpatton@niaid.nih.gov
ListServ: Contact <Buckler@nih.gov>
**INTERINSTITUTE INTEREST GROUP DIRECTORY**

**Washington Area Yeast Club**  
Meeting time: 2nd Wednesday, 5:15 pm  
Meeting place: Building 6B, Room 4A05  
Contact: Reed Wickner  
Phone: 496-3452  
Email: <wickner@helix.nih.gov>  
Contact: Alan Hinnebusch  
Phone: 496-4480  
Email: <ahinnebusch@nih.gov>

**Women’s Reproductive Health Interest Group**  
Meeting time and place: Every four months at times decided by the group.  
Contact: Phyllis Leppert  
Phone: 496-6515  
Email: <leppertp@nih.gov>

**WorldWideWeb Interest Group**  
Meeting time: 2nd Tuesday, 2:30 pm  
Meeting place: Building 10, Lipsett Auditorium  
Contact 1: Sandy Desautels  
Phone: 402-6553  
Email: <sandy_desautels@nih.gov>  
Contact 2: Dale Graham  
Phone: 402-1905  
Email: <degraham@helix.nih.gov>

**Xenopus/Zebrafish Interest Group**  
Meeting time: Last Monday (except summer), 3:30 pm  
Meeting place: Building 6B, Room 429  
Contact 1: Brant Weinstein  
Phone: 435-5760  
Email: <bw96w@nih.gov>  
Contact 2: Ajay Chitnis  
Email: <ajay.chitnis@mail.nih.gov>

**X-ray Crystallography Interest Group**  
Meeting time: Quarterly, announced by e-mail, 2:00 pm  
Meeting place: Building 5, Room 127  
Contact: Xinhua Ji  
Phone: (301) 846-5035  
Email: <jix@ncifcrf.gov>

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**FELCOM: An Intro**

As its name implies, the NIH Fellows committee (FELCOM) was established to serve the NIH fellows community. It therefore draws its members from each NIH Institute and from both basic and clinical science. FELCOM representatives are elected for a one-year term, act as liaisons between postdoctoral fellows and NIH administrative bodies, and serve as ad hoc members of several NIH-wide committees, including the CME committee and the Committee on Scientific Conduct and Ethics.

A major FELCOM program is the Fellows Award for Research Excellence (FARE). Held annually in May, this competition is based on abstracts submitted for blind peer review by study sections composed of two postdoctoral fellows and one senior scientist. The winners receive a $1,000 travel award to attend and present their work at a scientific meeting, and they also present a poster to the NIH community in conjunction with a Wednesday Afternoon Lecture Series (WALS) event. FELCOM also sponsors up to three WALS lectures each year.

Ongoing events include the Survival Skills workshops, aimed at teaching fellows how to become independent investigators, and a series on Scientific Careers for the Millennium. FELCOM also tackles such issues as visas, and it promotes the following teaching activities:

- **Adventure in Science** shows children ages 8–11 the excitement of scientific discovery. Meets Saturdays October through March. Volunteers select their own material. Contact Edward Max (<max@eber.fda.gov>).

- **NIH Resident Teacher Program** enlists fellows who want to teach science in Montgomery County public schools at the middle and high school levels. Offers a chance to earn state teacher certification. Contact Sandra Shmookler (phone: 301-279-3432; fax: 279-3428; or e-mail: <shmookler@fc.mcps.k12.md.us>).

- **FAES** seeks teachers for its college-level evening courses—from guest lecturers to course developers; monetary compensation is possible. Contact Constance Noguchi at 301-496-1163 or see website: <http://www.faes.org>.

Current FELCOM chairs are Patricia Van Bergen and James Gulley; meetings (first Thursday each month at 4, Building 1) are open. For more info, see: <ftp://helix.nih.gov/FELCOM/index.html>.

—Elizabeth McNeil, Ph.D., NCI  
Suzanne Dashiell, NINDS

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**Addenda**

**Considering starting a new Interest Group?** Contact Celia Hooper (fax: 301-402-4303; e-mail: <booperc@nih.gov>).  

**Need to correct your group’s listing?** Contact CIT’s web publishing group: <publish@cit.nih.gov>.

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**An Apple For Excellence**

The NIH Fellows Committee is requesting nominations for the 2000 Distinguished Clinical Teacher Award. Each year an NIH senior clinical investigator who has demonstrated excellence in clinical training involving direct patient care is publicly honored. A brief supporting statement should accompany nominations. Please fax or e-mail your nomination letter to:

- Distinguished Clinical Teacher Award  
  Attention: Deborah Cohen, NIH Office of Education  
  Fax: 301-402-8975; e-mail: <fellows@box.f.nih.gov>.  

The **deadline** for receipt of nomination letters is **August 30, 2000**.
programs that we are establishing toward these goals include a microarray facility at the NCI's Division of Clinical Sciences Advanced Technology Center in Rockville, Md., and an in vitro and in vivo facility at FCRC to study and assess radiation-drug interactions.

My personal primary research interest is in developing new therapeutics based on molecular targeting. Although radiation is typically described as an average dose to a tissue, at the end of its path in tissue, the energy is delivered in dense packets. Furthermore, radiation can be focused to tissue via external beam, implanted sources, and systemically administered isotopes. Thus, our conceptual model is that radiation-induced agents that can perturb a wide range of cellular targets. This perturbation can be exploited with molecular therapeutics. Moreover, because many of the newly developed cancer treatments—such as antiangiogenesis agents or radiation-targeting agents—may be cytostatic and not cytotoxic, radiation may be essential to make the agents effective in killing tumor cells. My research interest over the past decade has been targeting the tumor microenvironment, including tumor hypoxia, which is present in about half the tumors studied. This not only leads to radiation resistance, but the hypoxic stress and hypoxia itself can alter cellular phenotype, for example, the induction of HIF-1-responsive genes. Hypoxic perturbation within tumors, such as an abnormal redox state, may alter protein structure and function.

James Mitchell's group in the Radiation Biology Branch is advancing this work by developing techniques to image hypoxia in real-time that would help guide therapeutics. My own laboratory interest has turned to an understanding of the cyclooxygenase inhibitors as enhancers of tumor cell killing. We have demonstrated that the nonspecific nonsteroidal anti-inflammatory drug ibuprofen is an effective radiation sensitizer in vitro and in vivo. We are investigating possible mechanism(s) for this effect, including perturbation of arachidonic acid metabolism, inhibition of NFkB, which is constitutively active in a number of cancers, and possibly through the action of PPAR (peroxisome proliferator-activated receptors) family.

ROSP is now recruiting basic molecular biology researchers, physicists, and engineers for the molecular sensor program and clinicians interested in translational research. We welcome inquiries and look forward to collaborations with colleagues at NIH.

Barney Graham received his M.D. from the University of Kansas, Kansas City, in 1979 and completed clinical training and a fellowship in infectious diseases at Vanderbilt University in Nashville, Tenn., in 1986; in 1991, he completed his Ph.D. in microbiology and immunology at Vanderbilt, where he is currently a professor of medicine in the Division of Infectious Diseases and an associate professor of microbiology and immunology. He will join the NIH Vaccine Research Center as the director of clinical trials, commencing on a regular basis as soon as the building is ready and setting in next spring.* My primary research interests are viral pathogenesis and vaccine development. I have focused on two major areas of work: AIDS vaccine evaluation in clinical trials and pathogenesis of respiratory syncytial virus (RSV) in a murine model. My specific recent interest in this area is in the role of RhoA, a small GTPase, in RV entry, morphogenesis, and pathogenesis. I am also interested in science education and expanding research opportunities for African-Americans and other underrepresented minorities. In this regard, I am a member of the Development Committee for National Medical Fellowships, Inc., and a mentor in the Fellowship Program in Academic Medicine for Minority Students sponsored by Bristol-Myers Squibb.

Since 1987 I have been involved in the development of the AIDS Vaccine Evaluation Group, recently renamed the HIV Vaccine Clinical Trials Network (HVTN). This group has collectively conducted more than 40 Phase I and II clinical trials of candidate AIDS vaccines. I chaired the initial human studies evaluating recombinant Vaccinia virus vectors, including studies combining a live recombinant

* The September-October issue of the NIH Catalyst will include a story on new VRC recruits and programs. A ceremony to mark the official opening of the VRC is planned for October.
**RECENTLY TENURED**

...virus with a subsequent subunit protein booster.

In addition, I chaired studies evaluating novel adjuvants for subunit envelope antigens, and multivalent peptide vaccines combining both B- and T-cell epitopes. I also chaired the studies evaluating subjects who developed breakthrough HIV-1 infection despite prior vaccination, and I served in leadership positions for all the major subcommittees in the HIVTN. I am particularly interested in the use of cytokines, chemokines, and other natural immuno-modulators as vaccine adjuvants, and in the evaluation of vaccine approaches that combine immunogens, delivery vehicles, and routes of administration.

My laboratory focus is to define basic mechanisms of RSV pathogenesis and to apply this knowledge to vaccine development. RSV is an important cause of respiratory disease that has received high priority for vaccine development. Previous vaccine candidates have failed in clinical trials, and a formalin-inactivated whole virus (FI-RSV) preparation was associated with vaccine-enhanced illness.

After early studies characterizing a murine model of RSV infection and defining the role of T cells and antibody in primary RSV infection, we addressed the pathogenesis of the FI-RSV vaccine-enhanced disease. My laboratory demonstrated that priming with inactivated RSV induces a type 2 T-helper lymphocyte (Th2) response (dominant IL-4 expression), whereas priming with live RSV induces a Th1-like response (dominant IFN-γ expression) in mice following RSV challenge.

We have subsequently used the murine RSV model to evaluate new vaccine approaches and to investigate how immunization determines the composition of immune responses. Specific projects include defining the mechanisms by which: 1) the RSV G glycoprotein induces IL-5 and eosinophilia, 2) IL-4 modulates the cytokine mechanism of CD8+ cytokine T cells, 3) RSV-induced immune responses interact with allergic inflammation to cause airway hyperresponsiveness, 4) co-administered antigens influence RSV-induced immune responses and lung pathology, and 5) integrins mediate lymphocyte trafficking and activation during RSV infection. The studies specifically aim to answer questions about in vivo phenomena, taking advantage of a variety of knockout and transgenic mouse strains and assays designed to avoid in vitro artifacts.

A recent discovery in my laboratory of a cellular ligand for the RSV F glycoprotein has led to new projects in the areas of RSV-induced membrane fusion and viral entry and the role of intracellular signaling pathways triggered by the RSV F interaction in virus morphogenesis, immunity, and disease.

Our belief is that understanding basic mechanisms of antiviral immunity and viral pathogenesis will lead to novel and more effective approaches to vaccine development. Both laboratory and clinical studies yield essential information that must be integrated to inform the process of vaccine development.

Michael Krause received his Ph.D. from the University of Colorado in 1986 and did postdoctoral work with the late Harold Weintraub at the Fred Hutchinson Cancer Research Center in Seattle. He joined the NIDDK Laboratory of Molecular Biology in 1993 and is now a senior investigator heading the Section on Developmental Biology.

I am interested in transcriptional regulation of cell fate during development. I use the nematode Caenorhabditis elegans as an experimental system. C. elegans adults have only 959 somatic cells and develop from an essentially invariant cell lineage over the course of three days. The nearly complete genome sequence of C. elegans became available last year, revealing about 19,000 genes. Recent techniques, using traditional mutagens and/or molecular approaches, make it possible to quickly create loss-of-function phenotypes for most genes. The combination of relatively simple anatomy, defined cell lineage, complete genomic information, and powerful forward and reverse genetics makes this organism an ideal system in which to study the transcriptional regulation of cell fate.

My lab focuses primarily on the regulation of muscle cell fates in C. elegans, including the early events of mesodermal patterning that underlie the formation of the correct type, number, and position of muscle cells. Some of the muscle types in C. elegans can be correlated with muscle types found in mammals and many of the transcription factors involved in mammalian myogenesis have homologs in C. elegans. The simple anatomy of C. elegans allows us to study the function of these transcription factors in a single cell, sometimes revealing functions obscured in more complex animal systems.

A landmark discovery in the field of myogenic cell fate was the identification of the protein MyoD. MyoD is a basic helix-loop-helix (bHLH)-type transcription factor restricted primarily to skeletal muscle cell precursors; MyoD is not present in cardiac or smooth muscle. MyoD activates transcription of multiple downstream genes to cause proliferating cells to exit from the cell cycle and initiate the skeletal muscle program of differentiation. MyoD is one of four highly related proteins that together make up a family of myogenic factors in mammals. The identification of MyoD validated the notion of "master" regulatory genes capable of initiating whole programs of differentiation and marked the beginning of an intense research effort on skeletal muscle differentiation.

I arrived at the NIH shortly after identifying the only C. elegans homolog of MyoD (CeMyoD) and having shown that the expression profile of CeMyoD was consistent with its playing a key role in myogenesis. In collaboration with Andy Fire's group at the Carnegie Institution of Washington in Baltimore, we used CeMyoD knockouts to show that skeletal muscle-like cells in the nematode (body wall muscle) can be formed in the absence of CeMyoD activity. This observation is surprising, because mammalian tissue culture studies and mouse knockout experiments demonstrate that some members of the mammalian MyoD family are essential for skeletal myogenesis. Although CeMyoD is not essential for body wall muscle formation in C. elegans, it is required for these cells to function; homozygous CeMyoD-null animals cannot move normally and die shortly after the embryo hatches.

Through work in my lab at NIH, we have now shown that the CeMyoD finding is just one in a series of observations revealing both conservation and divergence in the function of several highly conserved, muscle-associated transcription factors in C. elegans. Through further dissection of the functions of these factors we have found that embryonic and postembryonic periods of myo-
genes in *C. elegans* use different regulatory pathways. These results have implications beyond the nematode and help to explain some of the dramatic differences between *C. elegans* and other organisms in the function of these evolutionarily conserved factors.

More recently we have turned our attention to another bHLH transcription factor, Twist, which acts early in the myogenic pathway. We have identified mutants in Twist that reveal that it is required for patterning a subset of the muscles in *C. elegans*. Twist also functions in a subset of mesodermal tissues during mammalian development, and Twist mutations in humans result in Saethre-Chotzen syndrome, which affects craniofacial development. We are interested in studying CeTwist in more detail to identify interacting factors and downstream target genes.

Together with other labs, we have begun to identify the transcriptional hierarchy regulating postembryonic muscle cell fates in *C. elegans*. Future work is aimed at adding additional detail to the regulatory network of genes and defining pathways functioning in other muscle cell types. It should be possible to describe in complete molecular detail how each muscle cell of *C. elegans* arises, beginning with fertilization of the egg. By defining developmental paradigms, this will be an important milestone, not only for myogenesis but for developmental biology in general.

Chris McBain received his Ph.D. from the University of Cambridge, England, in 1988 and did postdoctoral work at the University of North Carolina and Duke University, both at Chapel Hill, before joining the Laboratory of Cellular and Molecular Neurophysiology of NICHD in 1993. He is now a senior investigator heading the Section on Cellular and Synaptic Physiology.

It is becoming clear that to begin to understand the coordinated activity of large ensembles of neurons, we must first understand the nature of transmission between individual pre- and postsynaptic elements within a circuit and each and every neural element involved. My interests are in elucidating the precise nature of excitatory and inhibitory synaptic transmission between specific identified neural populations within the hippocampal and cortical formations.

For the hippocampal neuronal network, the net flow of information is strongly modulated by the action of the local-circuit GABAergic inhibitory interneurons, whose cell bodies are distributed throughout all layers of the hippocampus and comprise about 10 to 15 percent of the total neuronal population. The importance of these inhibitory neurons is underscored by the number of animal models of epilepsy that result from manipulation or loss of these inhibitory pathways.

My research has found that, rather than act as GABAergic counterparts of glutamatergic excitatory cells, interneurons possess both intrinsic and extrinsic properties that set them apart from principal cells of the cortical formation. This divergent cell population can be distinguished by the molecular identity of receptors and channels they express, by their mechanisms of short- and long-term synaptic plasticity, and by signal transduction mechanisms associated with both ligand- and voltage-gated receptors.

Work in my lab has focused primarily on three areas of research. The first is the nature of synaptic plasticity regulating hippocampal inhibitory interneuron excitability. We found that, in contrast to principal cells, interneurons do not possess conventional forms of N-methyl-d-aspartate (NMDA)–receptor postsynaptic long-term plasticity, but are influenced indirectly by a "passively propagated" form of plasticity occurring within a polysynaptic pathway.

In addition, we demonstrated cell-specific expression of presynaptic forms of plasticity at synapses formed by the axons of dentate gyrus granule cells, the so-called mossy fibers. Target specificity of presynaptic plasticity results from the segregated expression of AMPA-dependent signaling cascades. This signaling pathway is targeted to presynaptic axons associated with principal cells but is absent at the same synapses made onto inhibitory interneurons. Expression of glutamate receptor proteins is similarly segregated on the postsynaptic side of interneuron synapses. Within a single cell the α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA)–preferring class of glutamate receptors, which permit the entry of calcium ions, are expressed exclusively at mossy fiber synapses, while Ca-impermeable AMPA receptors are associated with inputs arising from the CA3 pyramidal neurons.
ing a deletion of the Lis1 gene. Mechanisms of synaptic transmission were severely disrupted, revealing hyperexcitability of the hippocampus and a predisposition for electrographic seizure activity. These data are the first to pinpoint the neuronal basis of seizures associated with lissencephaly within the hippocampus. We will continue to use clinically relevant animal models to investigate the intrinsic properties, synaptic transmission, and receptor targeting in defined neuronal networks.

**Toru Miki** received his Ph.D. from Kyoto University, Japan, in 1979. He taught at Yamaguchi University School of Medicine, Ube, before joining the Laboratory of Cellular and Molecular Biology, NIC, in 1985. He is now chief of the Molecular Tumor Biology Section in the Basic Research Laboratory, NIC.

My research interests are in molecular mechanisms that convert normal cells to cancerous cells. To explore this field, I have developed an efficient expression cloning system and used it to isolate cDNAs that can convert normal fibroblasts into morphologically transformed cells. In an earlier study, we introduced an epithelial cell expression library into NIH3T3 cells and identified three morphologically distinct foci of transformed cells, from which we rescued three transforming cDNA clones designated ECT (epithelial cell transforming genes). We soon identified ECT1 as a receptor of keratinocyte growth factor (KGF). KGF is related to fibroblast growth factors (FGFs), but specifically targets epithelial cells. Several research groups were in the process of isolating FGF receptors at that time, and structural comparison of KGF receptor and FGF receptor-2 (FGFR2) revealed a difference only in a small region of the ligand-binding domain, suggesting that these receptors are encoded by alternative transcripts of a single gene. This finding established that ligand binding specificity can be determined by alternative splicing.

We also isolated a constitutively activated FGFR2 from rat osteosarcoma cells. Acquisition of a new sequence, designated FRAG1, at the COOH-terminus of FGFR2 played a major role in the activation by overriding the negative regulatory effect of the normal ligand-binding domain. Finally, we found that FGFR2 isoforms with an acidic amino acid stretch were heavily modified with glycosaminoglycans. The modified receptor exhibits sustained activation of MAP kinase and stimulation of DNA synthesis. This modification event is also regulated by alternative splicing, because the modification site is encoded by an alternative exon.

In contrast to ECT1, which exhibits structural homology to several protein kinases, ECT2 initially lacked structural information. However, our continued attempts to isolate novel transforming genes from different cell types, as well as accumulation of new protein sequences in databases, led us to new members of this group of oncogenes, which share structural motifs of the guanine nucleotide exchange factors for the Rho family GTPases. While these oncogenes, ECT2, OST, T1M, NET1, and NY5, share common motifs, they also display distinct features. OST comprises multiple isoforms, catalyzes guanine nucleotide exchange on RhoA and Cdc42, and associates with Rac1 in its GTP-bound form. OST also regulates the JNK/SAPK MAP kinase pathway and induces actin reorganization in fibroblasts. NET1 is one of the genes induced with oxidative stress. NY5, an exchange factor for Rac1, is a major nuclear antigen expressed in proliferating, but not resting, cells.

Most of my recent interests are in the biological functions of ECT2. Besides the Rho exchange factor homology domain, ECT2 also contains domains related to several molecules involved in cell-cycle checkpoint control and repair. We have recently found that ECT2 is a critical molecule regulating cell division. ECT2 functions as a guanine nucleotide exchange factor for Rho GTPases and is phosphorylated specifically in G2 and M phases. Interestingly, this phosphorylation is required for the exchange activity of ECT2. Unlike other exchange factors, ECT2 localizes in the nucleus in interphase, but spreads to the entire cell after nuclear membrane breakdown. During cell division, ECT2 localizes at the cleavage furrow and then the midbody. Inhibition of ECT2 activity in cultured cells either by microinjection of anti-ECT2 antibody or expression of dominant-negative isoforms strongly inhibits cytokinesis. ECT2 also localizes in spindle microtubules during mitosis.

We have recently cloned Xenopus ECT2 and are studying its function in mitosis using a Xenopus in vitro cell-cycle control system. We are also increasing our attempts to clarify the role of Rho exchange factors in cell-cycle control.

**Bob Seder** received his M.D. from Tufts University in Boston in 1986 and completed a residency in internal medicine at Cornell University-New York Hospital. He came to NIH in 1989 as a postdoctoral fellow in the Laboratory of Immunology, NIAID, and is currently chief of the Clinical Immunology Section in the Laboratory of Clinical Investigation, NIAID.

Cellular immunity plays an important role in protecting hosts from a variety of infectious pathogens. These include fungal (such as Histoplasma capsulatum), parasitic (Leishmania major, Toxoplasma gondii), mycobacterial (Mycobacterium tuberculosis, M. avium complex), and bacterial (Listeria monocytogenes) pathogens. Over the past 15 years, the increase in opportunistic infections in individuals infected with HIV has underscored the importance of the cellular immune response in mediating protection against some of these pathogens. Despite a greater understanding of the factors regulating immune responses against these pathogens, patients often died from these infections due to an inadequate immune response. The worldwide pandemic of M. tuberculosis infection further attests to the need for understanding protective primary and memory cellular immune responses, which could lead to effective vaccines.

Protective immunity to many intracellular pathogens requires a complex sequence of events involving cells of the innate and acquired immune response. While innate immune response does provide some protection, acquired cellular immune response appears essential for sterilizing immunity and maintaining effective memory responses. Cytokines, such as IL-12, operate as a critical link between innate and acquired immunity. Their effect on the immune response overall and specifically on CD4+ T-cell differentiation has been the major focus of my career and current laboratory.

The importance of CD4+ T cells in regulating immune responses was highlighted by the seminal observation that
CD4+ T-cell clones could be segregated into specific subsets—Th1 and Th2—based on their pattern of cytokine production. This distinction and its importance in vivo provided a framework to examine the factors involved in the generation of these subsets. In my postdoctoral lab, we showed that cytokines such as IL-12 and IL-4 were potent regulators of Th1 and Th2 differentiation, respectively. Based on these observations, my current laboratory is using a variety of experimental infectious disease models to further understand the role of cytokines in immune regulation in vivo. To this end, we have studied three human pathogens (H. capsulatum, M. tuberculosis, L. major) in vivo. We also use these models to study cytokines as the basis for new therapies and vaccines.

Our recent work applies our understanding of how cytokines alter specific immune responses to a daunting challenge—the development of vaccines against intracellular infection. The great success of all existing vaccines depends on long-lived humoral immune response via antibodies. These responses to bacteria and viruses are readily achieved by many different vaccine formulations. By contrast, there are no uniformly effective vaccines for infections such as tuberculosis, malaria, and HIV, which now account for a majority of the deaths worldwide from infection. For all of these infections, a cellular immune response mediated by CD4+ and/or CD8+ T cells is required for protective immunity.

We have used the mouse model of Leishmania major infection, which requires Th1 responses, to study how long-term cellular immunity can be induced in vivo. Our results so far have shown that plasmid DNA vaccination encoding a specific leishmanial antigen induces long-lived protective responses in mice. This work has led us to explore whether this approach can work in nonhuman primates. If successful, we will follow up these studies in humans to determine safety and efficacy. We hope that this model will also help guide a rational approach for vaccination against M. tuberculosis, which requires a similar type of immune response.

Wei Yang received her Ph.D. from Columbia University in New York in 1991 and did postdoctoral work at Yale University in New Haven, Conn. In 1995, she joined the Laboratory of Molecular Biology of NIDDK, where she is now a senior investigator.

I have been interested in protein-DNA interactions for quite some time. Trained to be a biochemist and X-ray crystallographer in graduate school, I became fascinated by DNA recombination during my postdoc period at Yale University. At that time, I determined the crystal structure of a site-specific recombinase, gd-resolve, complexed with a 34-bp substrate DNA. Since joining NIH, my group has widened the research target from DNA recombination to mismatch repair and has taken a combinatorial approach, including X-ray crystallography and biochemistry, and mutagenesis, to study mechanisms of both processes.

DNA repair is an essential biological process in all living organisms. Mistakes in base pairing occur during DNA replication and are routinely repaired. MutS, MutL, and MutH are necessary and sufficient to initiate methyl-dependent mismatch repair in Escherichia coli. MutS possesses an ATPase activity and initiates repair by recognizing DNA containing mispaired or unpaired nucleotides. After binding to a mismatch, MutS recruits MutL to mediate the activation of MutH endonuclease, which cleaves the newly synthesized strand 5' to a transiently unmodified d(GATC) sequence. Both MutS and MutL also play essential roles in the subsequent removal and resynthesis of the daughter strand. The mismatch repair proteins MutS and MutL are conserved from prokaryotes to humans, and defective human homologues, for example, MSH, PMS, and MLH1 proteins, have been directly implicated in hereditary nonpolyposis colon cancers (HNPCC) and other familial and sporadic cancers.

During the past four and a half years, we have determined the crystal structures of MutH, a 40-kD fragment of MutL (LN40), and LN40+ in complex with an ATP analog and with the product ADP. Most recently, in collaboration with Peggy Hsieh's group (GBB, NIDDK), we have determined crystal structures of Thermus aquaticus MutS alone, its complex with DNA, and a ternary complex of MutS, DNA, and ADP-Mg++. Based on these structures, we identified the active site residues of MutH and the MutS ATPase, and we confirmed the functional importance of these residues by mutagenesis. The crystal structures of MutS and its complex with DNA revealed the mode of mismatch recognition, which is based on the instability of heteroduplex DNA at mispaired or unpaired bases. We also uncovered an ATPase activity intrinsic to MutL that previous investigators had concluded did not exist.

Using hydrodynamic methods and crystallography, we have shown that binding of ATP triggers a structural transformation of MutL. Finally, we have developed an in vitro assay for initiation of mismatch repair. Combining our structural, mutagenesis, and biochemical results, we have proposed a proofreading mechanism of mismatch recognition by MutS—similar to that found in protein synthesis and DNA replication, but the first example identified in DNA repair processes. Our studies of mismatch repair proteins also have shed light on defects of mutations in HNPCC kindreds.

The other project my research group has been focused on is V(D)J DNA recombination, which is a site-specific event essential for the assembly of the immunoglobulin (Ig) and T-cell receptor (TCR) genes in vertebrate immune system. The Ig and TCR genes are encoded in separate V, D, and J segments. Each segment has multiple copies with slight variations in sequence. By combinatorial means of joining V, D, and J segments into Ig and TCR genes and modifying the junctions between segments in somatic cells, immune systems are able to make millions of different Ig and TCRs from a limited set of coding sequences.

The V(D)J recombination is initiated by the combined action of two lymphoid-specific proteins, RAG1 and RAG2, which introduce double-strand breaks at the borders of the V, D, and J segments. The second stage of V(D)J recombination is to process and rejoin the cleaved coding ends of V, D, and J segments to form the mature Ig and TCR genes. Several proteins involved in the general repair of DNA double-strand breaks, including the catalytic subunit of DNA-dependent protein kinase, Ku protein, XRCC4 and ligase IV, are required for the second stage of V(D)J recombination. Mistakes at any stage of the V(D)J can cause cancers, growth and developmental defects, and premature death in humans. We are studying almost all of the involved proteins and in collaboration with Martin Gellert's group (LMB, NIDDK) have succeeded in determining the crystal structure of XRCC4. We are continuing to unravel the structure and function of macromolecular complexes involved in DNA repair and recombination.
**Call for Catalytic Reactions**

In this issue, we are asking for your reactions in four areas: retirement, NIH programs for graduate students, Interest Group participation, and Hot Methods.

Send your responses on these topics or your comments on other intramural research concerns to us via e-mail: <catalyst@nih.gov>; fax: 402-4303; or mail: Building 2, Room 2W23.

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1) This is a repeat question: We are planning an issue that concentrates on retirement and how retired scientists conduct their lives after they leave the NIH campus, and we still want to hear your anecdotes about other scientists, plans of your own, retirement philosophy—really almost anything on this potentially thorny transition.

Send your responses on these topics or your comments on other intramural research concerns to us via e-mail: <catalyst@nih.gov>; fax: 402-4303; or mail: Building 2, Room 2W23.

2) With a new director of graduate program partnerships, Mary DeLong, NIH will be looking for ways to improve and expand programs for graduate students. What would you recommend?

3) What role does your participation in other groups have in your life as an NIH scientist?

4) Are there any “Hot Methods” out there?