



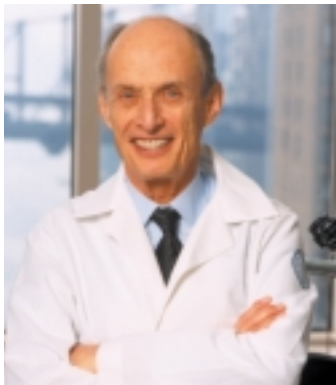
news & notes

FRIDAY, MARCH 15, 2002

THE NEWSLETTER FROM THE ROCKEFELLER UNIVERSITY'S OFFICE OF COMMUNICATIONS AND PUBLIC AFFAIRS

TODAY'S EVENTS

Paul Greengard on
Signal Transduction



Professor Paul Greengard, head of the Laboratory of Molecular and Cellular Neuroscience, will discuss "Signal Transduction in the Brain" at the Friday lecture today (March 15) at 3:45 p.m. in Caspary Auditorium.

Greengard, Vincent Astor Professor and director of the Fisher Center for Alzheimer Research at Rockefeller, received the 2000 Nobel Prize in Physiology or Medicine for his discovery of how dopamine and several other transmitters in the brain exert their action in the nervous system.

Greengard's discoveries have provided a conceptual framework for understanding how the nervous system functions at the molecular level. He also has demonstrated that many effects — both therapeutic and toxic — of several classes of common antipsychotic, hallucinogenic and antidepressant drugs can be explained in terms of distinct neurochemical actions that affect the transmission of nerve signals in the brain, a process called signal transduction.

Over the last 30 years, Greengard and his colleagues have developed a general model that provides a rational explanation, at the molecular and cellular levels, of the mechanism by which stimuli — both electrical and chemical — produce physiological responses in individual nerve cells.

Recent research of Greengard and his colleagues helps explain the molecular mechanism behind Prozac's effect on depression and why estrogen is beneficial in preventing the buildup of senile plaques in the brain in Alzheimer's disease (see related story, page 2).

Graduate Student Open House Poster Session

1:30 to 3 p.m.

17th Floor,
Weiss Research Building

Mouse Studies Shed Light on How Prozac Works

Nobel laureate Paul Greengard and other Rockefeller University scientists have illuminated, in laboratory mice, new details of the complex chemical interaction in the brain that is generated by Prozac, the widely prescribed drug for depression.

Their findings are reported in a pair of papers in the March 5 issue of *Proceedings of the National Academy of Sciences (PNAS)*.

For many years, scientists have known that Prozac and similar "selective serotonin re-uptake inhibitor" drugs boost the levels of the chemical messenger serotonin in the brain by blocking its reabsorption. But, serotonin levels alone could not be the only explanation for Prozac's effectiveness in relieving symptoms of anxiety and improving mood in people suffering from depression.

Now, for the first time, Greengard and his Rockefeller colleagues have demonstrated that serotonin increases the effectiveness of glutamate, another key chemical messenger that acts as an on-off switch for nerve impulses.

"We now can explain that Prozac works on depression in part by increasing signaling through the glutamate pathway, but we don't know why an increase in glutamate signaling alleviates depression in people," says Greengard, Vincent Astor Professor and head

of the Laboratory of Molecular and Cellular Neuroscience. "We know enormously more about how Prozac works than before we did this study, but we don't have all the answers.

"The brain is such an incredible instrument. To say that you know how it works would be nothing short of arrogance."

The research reported in *PNAS* helps opens the door to a wider understanding of brain chemistry and its effects on depression and related health disorders.

An imbalance in the brain's serotonin system plays a role in causing depression. Normally, a "sending" brain cell releases serotonin into what is called the synapse. This is the space between the axons of the "sending" nerve cell and dendrites of the "receiving" nerve cell. When serotonin reaches the surface of the dendrite, it stimulates or activates receptors that can interact only with this chemical messenger. Stimulation of these receptors generates an electrical impulse in the "receiving" cell that allows brain cells to communicate with each other. The serotonin is removed and recycled through a molecule called a serotonin re-uptake pump on the "sending" cell.

By blocking the action of the serotonin re-uptake pump, Prozac increases the amount of active

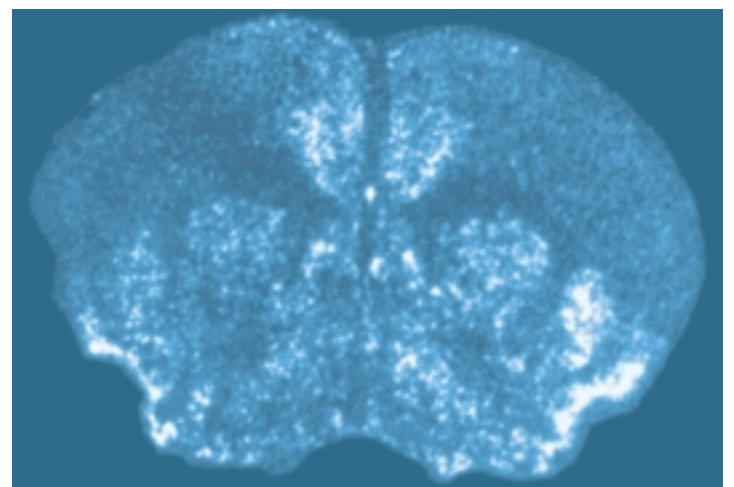
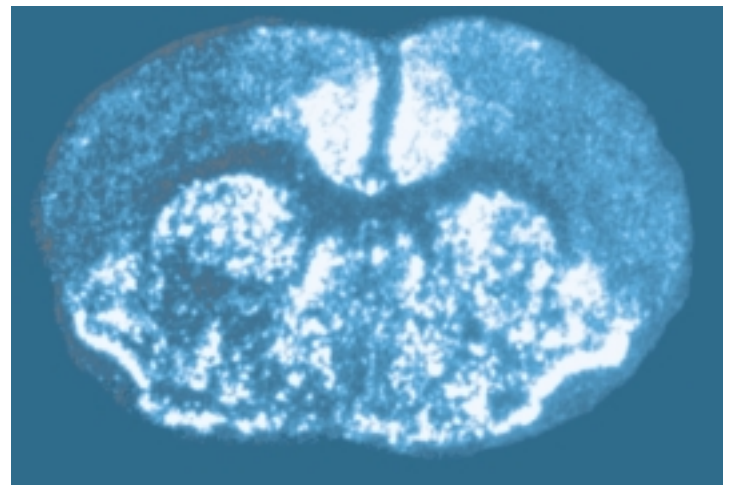


Photo at top shows a section of a normal mouse brain after serotonin was administered to it; photo below shows a section of a DARPP-32 knockout mouse after serotonin administration. Bright spots in the normal mouse indicate activation of a molecule called c-fos (which is used as a marker for serotonin activity). This activation is reduced in the DARPP-32 mouse.

serotonin that can be delivered to the dendrite. Scientists theorize that increased serotonin in the brain somehow helps restore nor-

mal message transmission among brain cells, thereby relieving symptoms of depression.

continued on page 3

"Superbug" update:

Few Strains Cause Most Drug-resistant Disease

Targeting What Makes These Pervasive Bugs Unique May Generate New Therapies

The culprits behind antibiotic-resistant diseases now plaguing hospitals worldwide have been harboring a secret — one that Rockefeller scientists have recently exposed. It seems these infectious microbes, termed *Staphylococcus aureus* or *S. aureus*, are not independent criminals working alone. Rather, they are members of only a few massive "superbug" families, which have spread out across the globe.

The findings, reported in the March 1 issue of *Lancet Infectious Diseases*, suggest that a close examination of what makes these particular pathogens so powerful may allow scientists to locate and target their weaknesses, and subsequently develop novel disease-fighting drugs.

"The secrets of the spectacular success of these *S. aureus* lineages may be hidden in their unique genetic background and may ultimately lead to new strategies to help fight these dangerous microbes," says Alexander Tomasz, head of the Laboratory of Microbiology at The Rockefeller University and second author of the paper.

Scientists in the Laboratory of Microbiology at The Rockefeller University and the Laboratory of Molecular Genetics at the Instituto de Tecnologia Quimica e Biologica, Universidade Nova de Lisboa, Portugal, conducted a major study of methicillin-resistant *S. aureus* (MRSA) — the primary antibiotic-resistant bacterium or "superbug" dominating



The map illustrates the extent to which only a few families of the drug-resistant superbug *Staphylococcus aureus* have spread across the globe. The red squares represent the Iberian strain; the red dots signify the Brazilian/Hungarian strain; the yellow dots indicate the Pediatric strain; and the blue dots are the NY/Japan strain.

hospitals worldwide — beginning six to seven years ago.

The study involved collecting a large number of MRSA strains, or clones, from diseased patients

in 160 hospitals in southern and eastern Europe, five Latin American countries, the United States and Japan. Hospitals cho-

continued on page 3

How Prozac Works *continued*

Serotonin is one of many neurotransmitters. Another chemical messenger, dopamine, affects brain processes that control movement, emotional response and ability to experience pleasure and pain. Abnormalities in dopamine signaling are associated with Parkinson's disease, schizophrenia, attention deficit hyperactivity disorder and substance abuse. Because dopamine plays a role in these and other neurological and psychiatric disorders, Greengard and his research colleagues at The Rockefeller University reasoned that this neurotransmitter may interact in some way with serotonin and thus regulate emotional behavior.

To probe this possible connection, the Rockefeller scientists studied the effects of Prozac in mice genetically altered to lack a protein called DARPP-32 (the mouse is called a "DARPP-32 knockout mouse"). Previous research by Greengard and his colleagues has shown that DARPP-32 is a major player in the mechanisms by which the neurotransmitter dopamine produces its effects in the brain.

The scientists found that the behavioral antidepressive effect and several other biochemical effects of Prozac were abolished when Prozac was administered to the DARPP-32 knockout mice.

"These results show for the first time an important role for DARPP-32 in mediating the actions of serotonin in mice," says first author Per Svenningsson,

research associate in the Greengard lab. "Moreover, the data provide the outline of a molecular mechanism for the behavioral actions of psychostimulants and antidepressant agents that achieve their effects through perturbation of transmission of serotonin."

In the second *PNAS* paper, Svenningsson, Greengard and colleagues mimicked the effects of Prozac observed in the DARPP-32 knockout mice in a test tube by applying serotonin to brain slices.

The scientists discovered that Prozac and serotonin regulate protein phosphorylation at three distinct sites on DARPP-32. Proteins are activated through phosphorylation, the process by which a phosphate molecule is attached to a target protein. A phosphorylated protein, through one or more biochemical steps, produces the physiological response characteristic of neurotransmitters.

Interestingly, serotonin increases DARPP-32 phosphorylation at two sites, while decreasing phosphorylation at a third site. The researchers identified three serotonin receptors responsible for the changes in phosphorylation of DARPP-32. The three receptors work synergistically through DARPP-32 to block an enzyme called protein phosphatase-1 (PP-1). PP-1 regulates many crucial biological processes by dephosphorylating, or removing phosphate molecules from phosphorylated proteins, thereby inactivating them.

"Inhibition of PP-1 means a lot of proteins won't be dephosphorylated and thus that their physiological properties will be altered," says Greengard. "Our next step is to identify these proteins and determine how PP-1 inhibition might be related to the action of antidepressant agents."

The researchers focused on a target of PP-1 called AMPA, which is a receptor for the amino acid glutamate, the primary excitatory neurotransmitter in the mammalian central nervous system. Studies by other researchers have indicated that classes of drugs effective in animal models of depression also regulate the AMPA receptor, and other antidepressant drugs are known to work directly on glutamate to increase glutamate signaling. They found that increased glutamate activity resulted from a complex signaling cascade initiated by serotonin: serotonin activates DARPP-32, which inhibits PP-1, increasing AMPA receptor phosphorylation, which in turn increases the effectiveness of glutamate.

Svenningsson's and Greengard's co-authors are Eleni T. Tzavara, Jeffrey M. Witkin, Allen A. Fienberg, George G. Nomikos and Feng Liu.

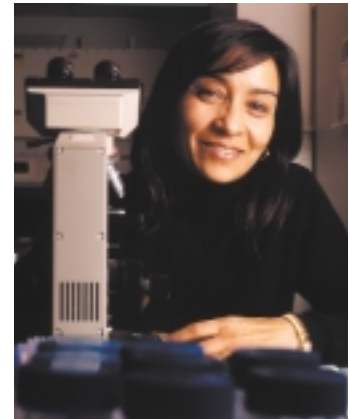
This research was supported in part by grants from the U.S. Public Health Service.

— Joseph Bonner

NEWS BRIEFS

Bhardwaj Receives Duke Foundation Clinical Research Award

Nina Bhardwaj, associate professor of clinical investigation, received the Doris Duke Charitable Foundation's 2001 Distinguished Clinical Scientist Award (DCSA) for Excellence in "Bench to Bedside" Research. The DCSA Program recognizes outstanding physician-scientists who apply the latest basic science advances to the prevention, diagnosis, treatment and cure of disease.



Bhardwaj, one of seven award recipients in the U.S., receives \$1.5 million over five years to support her project titled "Enhancement of Anti-HIV Immunity." Research in

Bhardwaj's lab includes clinical work at The Rockefeller University Hospital, and these studies may provide important insights for developing a vaccine to combat HIV.

M.D.-Ph.D. Student Wins Weintraub Award



Agata Smogorzewska, a biomedical fellow in the Tri-Institutional M.D.-Ph.D. Program, has received the 2002 Harold M. Weintraub Graduate Student Award, sponsored by the Basic Sciences Division of the Fred

Hutchinson Cancer Research Center in Seattle.

Smogorzewska studies TRF2, a telomeric protein essential for the protection of mammalian chromosome ends, in the laboratory of Professor Titia de Lange.

Smogorzewska will participate with the 16 other award winners in North America and Europe in a scientific symposium May 3 and 4 at the Hutchinson Center.

Award recipients receive a certificate, travel expenses and an honorarium from the Weintraub and Groudine Fund, established to foster intellectual exchange through the promotion of programs for graduate students, fellows and visiting scholars.

"Superbug update" *continued*

sen for the study were known to contain a high percentage of drug-resistant MRSA cases. Using DNA fingerprinting techniques similar to those for forensic medicine and criminal investigations, the researchers probed the genetic backgrounds of the bacterial culprits responsible for hospital-borne MRSA infections.

The findings were astonishing: in more than two thirds of the 3,067 MRSA samples analyzed, the researchers identified the "fingerprints" of as few as two drug-resistant clones of MRSA. "We kept seeing the same two bugs again and again," says Duarte Oliveira, a visiting scientist in the Tomasz lab who is first author of the study.

"It was like finding the same two fingerprints in crimes committed on four continents," says Herminia de Lencastre, senior research associate at Rockefeller and head of the Laboratory of

Molecular Genetics at the Instituto de Tecnologia Quimica e Biologica, Universidade Nova de Lisboa, Portugal, and senior author of the *Lancet Infectious Diseases* report.

One of these clones already had a long "criminal record" in hospitals: it was a direct descendent of the very first MRSA strain detected in the United Kingdom in 1961. Members of this family of bacteria have since then spread in truly pandemic fashion to cause disease in such faraway places as Argentina, Brazil, the Czech Republic, Hungary, Portugal and Spain.

The second clone was identified as the cause of MRSA disease in 60 percent of the 258 isolates collected from 29 health care facilities in Connecticut, New Jersey and Pennsylvania in 1998. The same clone was found in abundance in a hospital in Tokyo, and represents almost half of the MRSA recovered

from 12 New York City hospital patients infected with the bacteria in 1996.

S. aureus is the most versatile of human pathogens, readily surviving and proliferating in today's hostile, antibiotic-laden environment. It causes a wide range of potentially life-threatening afflictions, from skin infections to infections of the central nervous system, and is the number-one cause of the estimated two million hospital-borne infections in the United States each year.

Today, at least half of *S. aureus* infections in U.S. hospitals are caused by strains resistant to several antibiotics, including penicillin, tetracycline, erythromycin and methicillin. Although the antibiotic vancomycin can be used as a therapy of last resort against these dangerous microbes, history indicates that altogether novel methods to fight them are imperative. Now



Herminia de Lencastre (left), Alexander Tomasz and Duarte Oliveira detected the genetic signatures or "fingerprints" of two massive families of drug-resistant *Staphylococcus aureus* in hospitals around the world.

that de Lencastre and colleagues have uncovered the extent of these insidious superbug dynasties, scientists might be able to home in on an unforeseen Achilles' heel.

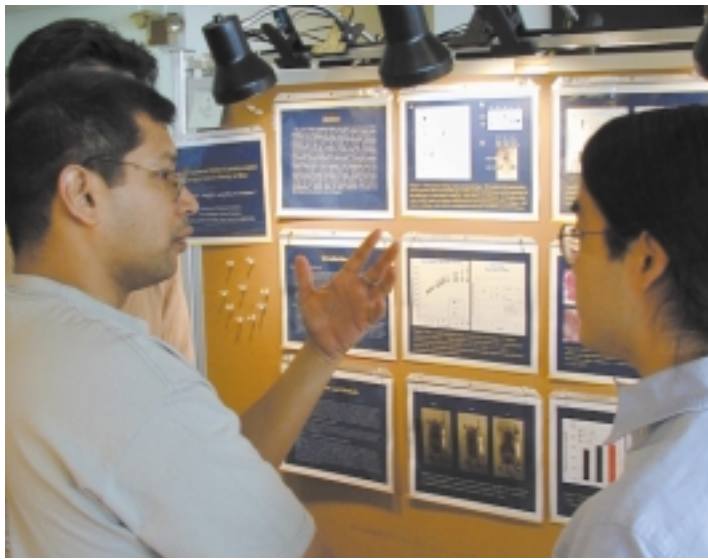
This work was supported by grants from the National

Institutes of Health, Fundação para a Ciencia e Tecnologia (Ministry of Science and Technology) and Fundação Calouste Gulbenkian, Portugal.

— Whitney Clavin

Poster Session Showcases Grad and Postdoc Science

Prospective graduate students — as well as current students, postdocs and faculty — had the chance to learn about the various research projects taking place at Rockefeller at the Graduate Program Open House Poster Session last Friday on the 17th floor of Weiss. More than 30 laboratories were represented. A second poster session takes place from 1 to 3:30 p.m. today (March 15).



Andre Ragnauth (left), a postdoctoral associate in the Pfaff lab, inquires about the research of Makoto Ishii, a biomedical fellow in the Friedman lab.



Joshua Rappaport (left), a postdoctoral associate in the Simon lab, explains his work to Saul Kivimäe, a graduate fellow in the Young lab. The computers in the foreground display the research of Professor Sanford Simon.

Latest on Fruit fly DNA Sequence



Gerald Rubin, director of the *Drosophila* Genome Center at the University of California, Berkeley, gave an overview of computational and experimental approaches for annotating the *Drosophila* genome sequence at the Centennial symposium titled "From Gene to Organism" last Thursday. The final Centennial symposium, "Genes and Disease: Opportunities for Human Genetics in Modern Medicine," will occur from 1 to 5 p.m., Wednesday, April 3, in Caspary Auditorium.

Researchers Pinpoint How Estrogen May Prevent Alzheimer's "Plaques"

Speed of Pathway Changes Metabolic Fate of Protein

Estrogen prevents the buildup of Alzheimer's disease's "senile plaques" in the brain by scooting key proteins through their normal pathways before they can form the debilitating plaques.

This finding, by scientists in the laboratory of The Rockefeller University's Paul Greengard, along with colleagues at Weill Medical College of Cornell University (WMCCU), provides evidence that the speed of a pathway can change the metabolic fate of a protein.

The results, derived from mouse cell lines and primary cultures of rat and human neurons, may have clinical applications for Alzheimer's prevention in women.

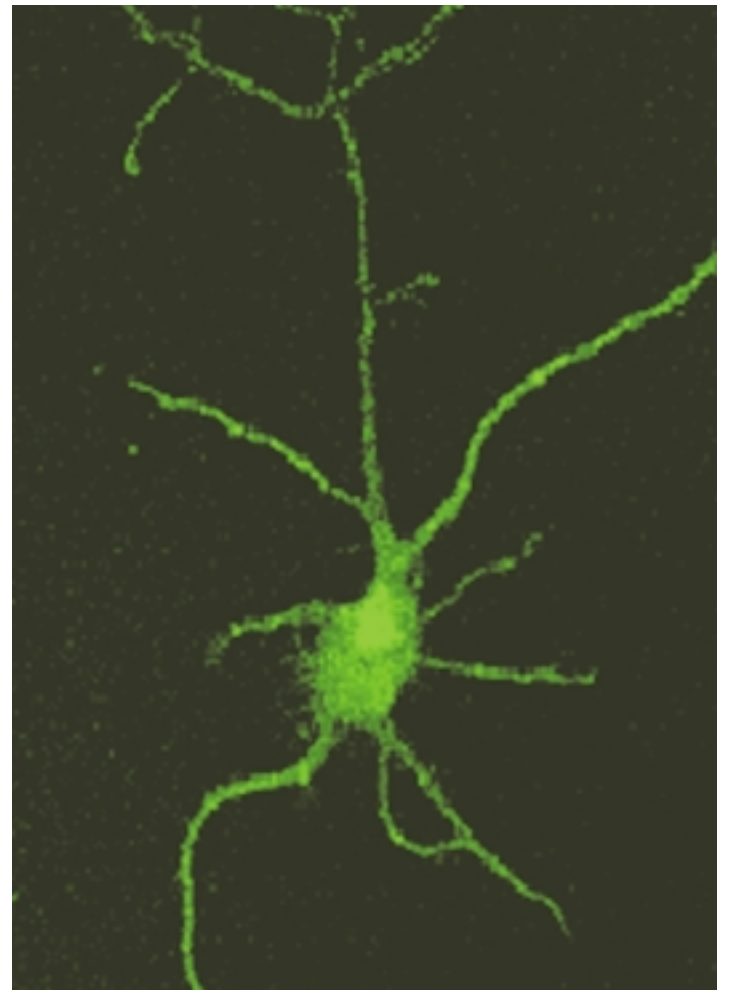
"These results suggest a new approach in the development of therapeutic agents for the treatment of Alzheimer's disease," says Greengard, Vincent Astor Professor, head of the Laboratory of Cellular and Molecular Neuroscience and director of Rockefeller's Fisher Center for Alzheimer Research.

Alzheimer's — whose devastating effects include memory loss, dementia and ultimately death — is characterized by these so-called senile plaques that build up in the brain. Most researchers believe these plaques are the cause of the disease. In the late 1990s, Greengard's laboratory, using mouse, rat and human neurons, was the first to demonstrate that estrogen reduces the production of the protein which accumulates to form these plaques. Several researchers, including Huaxi Xu in Greengard's lab, then showed that estrogen administration resulted in similar reductions in this protein in animal models of Alzheimer's disease.

The new findings, reported in the *Journal of Biological Chemistry*, provide the first step-by-step explanation of how estrogen prevents senile plaque buildup.

Senile plaques in the brain consist of clumps of protein fragments known as beta-amyloid peptides. These are formed when certain proteins (called amyloid precursor proteins, or APP) travel through a part of the cell known as the trans-Golgi network (TGN) on their way to the cell surface. Most scientists believe that the longer the proteins stay in the TGN, the more likely they are to develop into beta amyloid.

The Rockefeller team used an advanced "cell-free" laboratory



Researchers in Paul Greengard's laboratory have shown that the hormone estrogen stimulates the passage of amyloid precursor proteins (APP) — which contribute to the formation of senile plaques found in Alzheimer's disease — through a part of the cell known as the trans-Golgi network, thus reducing beta-amyloid formation in that compartment. Above, a neuron from the cortical region of a rat brain shows APP predominantly localized in the Golgi compartment (bright area).

technique that allowed them to look at individual components of cells (rather than whole cells) to analyze discrete steps in the APP pathway. They found that estrogen ushers the precursor proteins through the TGN before they can form beta amyloid. Once the proteins reach the cell surface, they are cut in half by enzymes and can no longer form senile plaques.

"Merely by speeding up APP's pathway, estrogen can reduce the amount of protein secreted as beta amyloid," says author Xu, an assistant professor in Greengard's laboratory and the Fisher Center for Alzheimer Research. "Over a lifetime, even a small percentage decrease could have significant health consequences."

"Alzheimer's is a disease of old age because it usually takes decades for these beta-amyloid plaques to build up," adds first author Jeffrey P. Greenfield, a former graduate student of Greengard's who is now an M.D. candidate at WMCCU. "That's also why it's a disease of the 20th century — in the past, few people lived long enough to get Alzheimer's."

The new findings may explain the lack of clinical effectiveness

of estrogen in women who already have Alzheimer's. Explains Greenfield: "Estrogen can help keep the senile plaques from forming, but does not, so far as anyone knows, act to reverse the buildup. Like heart disease or osteoporosis, Alzheimer's may be easier to prevent than to treat."

The researchers caution, however, that estrogen supplementation in women has potential health complications. "Most currently prescribed hormonal supplements are a combination of estrogen and progesterone, which is considered safer than estrogen alone," says Greenfield. "We don't yet know how the combination of these two hormones might affect the development of the Alzheimer's plaques, and we don't know how doses of pure estrogen might affect human patients."

Other co-authors are Dongming Cai, Krista Kaasik and Rachel S. Gross at Rockefeller, and Lawrence W. Leung and Enrique Rodriguez-Boulan at WMCCU.

Funding for this research was provided by the National Institutes of Health, Alzheimer's Association, Ellison Medical Foundation and American Health Assistance Foundation.

— Lisa Stillman



calendar

MARCH FIFTEENTH THROUGH APRIL TWELFTH

WWW.ROCKEFELLER.EDU/RUCAL

Friday Lectures and Thesis Presentations

These events are held in Caspary Auditorium at 3:45 p.m. and preceded by tea at 3:15 p.m. in Abby Aldrich Rockefeller Lounge. All are welcome.

FRIDAY, MARCH 15

Signal Transduction in the Brain.

Paul Greengard, Vincent Astor Professor and head of laboratory, RU.

MONDAY, MARCH 25

Thesis Presentation. Stress and Hippocampal Plasticity.

Kara Pham, graduate fellow, McEwen laboratory, RU.

WEDNESDAY, MARCH 28

Thesis Presentation. Lysogenic Bacteriophage: An Effector of Bacterial Evolution in the Human Throat.

Thomas Broudy, graduate fellow, Gotschlich and Fischetti laboratories, RU.

FRIDAY, MARCH 29

RNA Catalysis: With and without Proteins.

The Richard M. Furlaud Memorial Lecture. Thomas Cech, Distinguished Professor, University of Colorado, Boulder, and president, Howard Hughes Medical Institute.

FRIDAY, APRIL 5

Reciprocal Coupling of Metabolism and Circadian Rhythm.

Stephen L. McKnight, professor and chairman, Department of Biochemistry, University of Texas Southwestern Medical Center at Dallas.

FRIDAY, APRIL 12

Dynamics and Mechanics of Meiotic Chromosomes.

Nancy Kleckner, Herchel Smith Professor of Molecular Biology, Harvard University.

Scientific Events

FRIDAY, MARCH 15

12 p.m. Shr3p Ensures the Packaging of Amino Acid Permeases and the Extracellular Nutrient Sensor Ssy1p into ER-derived COPII Vesicles.

Per Ljungdahl, Ludwig Institute for Cancer Research, Stockholm, Sweden. Cellular Biochemistry and Biophysics Seminar. 116 Rockefeller Research Laboratories, MSKCC, 430 East 67th St.

1-3:30 p.m. Graduate Program Open House Poster Session.

Weiss 17th Floor (Entire). Open to RU community and guests.

2 p.m. Standardization and Validation of Microarray Experiments.

David Chung, Stratagene. Microarray Technology Seminar. 301 Weiss. Contact Greg Khitrov, 327-7064. Open to RU/WMCCU/NYPH/MSKCC community and guests.

MONDAY, MARCH 18

1:30 p.m. Regulatory Mechanisms That Control the Immune Response to Normal Self Tissues and Tumors.

Richard Flavell, Yale University School of Medicine and Howard Hughes Medical Institute. Immunology Seminar. 116 Rockefeller Research Laboratories, MSKCC, 430 East 67th St.

TUESDAY, MARCH 19

4 p.m. Why Express Three NO Synthases? Understanding the Basis and Potential Advantage of Their Distinct Behaviors.

Dennis J. Stueh, Lerner Research Institute, Cleveland Clinic. WMCCU Winter/Spring 2002 Research Seminar Series. Weill Auditorium, WMCCU, 1300 York Ave. Coffee and Cookies at 3:45. Contact Lisett Checo, 746-6250.

4 p.m. HCV: What Determines Success and Failure of Immune Responses?

Paul Klenerman, Nuffield Department of Medicine, John Radcliffe Hospital. Center for the study of Hepatitis C Seminar. 301 Weiss. Open to RU/WMCCU/NYPH/MSKCC community and guests.

WEDNESDAY, MARCH 20

11 a.m. Molecular Machines for Protein Degradation.

Robert Huber, Department of Structural Research, Max Planck Institute for Biochemistry. Student- and Postdoc-sponsored Seminar Series. 301 Weiss. Pizza Luncheon at 12 p.m. on the 17th Floor of the Weiss Research Building. Open to RU/WMCCU/NYPH/MSKCC community and guests.

12 p.m. Does Stress Damage the Brain?

Bruce S. McEwen, professor and head of laboratory, RU. Seminar in Clinical Research. 110B Nurses Residence. Contact Dale Miller, 327-8411.

12 p.m. Combining Bioinformatics and Genomics to Gain Insight into the Etiology of Cancer.

Kenneth H. Buetow, director, Center for Bioinformatics, National Cancer Institute. Auditorium, Rockefeller Research Laboratories, MSKCC, 430 East 67th St.

4:30 p.m. Hepatitis C: Toward New Therapies and Vaccines.

Charles Rice, professor and head of lab, and scientific and executive director, Center for Study of Hepatitis C, RU. MSKCC President's Research Seminar. Auditorium, Rockefeller Research Laboratories, MSKCC, 430 East 67th St. Tea at 4 p.m.

THURSDAY, MARCH 21

8 p.m. P53, Mdm2 and Cancer: The Good, the Bad and the Dead.

Moshe Oren, Weizmann Institute of Science, Rehovot, Israel. Harvey Society Lecture. Caspary Auditorium.

MONDAY, MARCH 25

4 p.m. Membrane-Cytoskeleton Adhesion: Role of PIP2.

Mike Sheetz, Columbia University. PBMM Research Seminar. Weill Auditorium, WMCCU, 1300 York Ave. Tea at 3:45 p.m.

4:30 p.m. The Origins and Programming of Blood and Vasculature in *Xenopus* and Zebrafish.

Roger Patient, Queens Medical Center Nottingham, University of Nottingham, UK. Cell Biology and Genetics Seminar. Papanicolaou Library, A-106, WMCCU. Open to RU/WMCCU/NYPH/MSKCC community and guests.

TUESDAY, MARCH 26

11 a.m. Genetic and Environmental Models of Anxiety and Depression.

Renee Hen, Columbia University. Neuroscience Development Seminar. 305 Weiss.

11 a.m. Information Flow in ERK Signaling Modules.

Melanie Cobb, University of Texas Southwestern Medical Center at Dallas. Pels Family Center for Biochemistry and Structural Biology Seminar. 301 Weiss. Refreshments at 10:45 a.m. Contact Sarah Cullins, 327-7221.

4 p.m. ITP and Hepatitis C: A Discussion.

James Bussel, Cornell University. Center for the study of Hepatitis C seminar series. 301 Weiss. Open to RU/WMCCU/NYPH/MSKCC community and guests.

4 p.m. Structural DNA Nanotechnology.

Ned Seeman, New York University. Center for Studies in Physics and Biology Seminar. B Level Conference Room, Smith Hall Annex. Coffee at 3:30 p.m.

WEDNESDAY, MARCH 27

12 p.m. Angiogenesis and Lymphangiogenesis in Skin Inflammation and Tumor Progression.

Michael Detmar, Harvard Medical School. Seminar in Clinical Research. 110B Nurses Residence. Contact Dale Miller, 327-8411.

THURSDAY, MARCH 28

12 p.m. RNA-binding Proteins in Spermatogenesis.

Mike Kiledjian, Rutgers University. Endocrinology and Reproductive Biology Seminar. 301 Weiss.

THURSDAY, MARCH 28

12 p.m. RNA-binding Proteins in Spermatogenesis.

Mike Kiledjian, Rutgers University. Endocrinology and Reproductive Biology Seminar. 301 Weiss.

FRIDAY, MARCH 29

10 a.m. Toward a Biological Dissection of NK Cell Differentiation.

James P. Di Santo, Institut Pasteur. Immunology Seminar. 101 Rockefeller Research Laboratories, MSKCC, 430 E. 67th St.

TUESDAY, APRIL 2

11 a.m. Late Differentiation in *Drosophila* Photoreceptor Cells.

Bertrand Mollereau, assistant professor, Steller lab, RU. Neuroscience Development Seminar. 305 Weiss.

4 p.m. Inhibition of NK cells by HV-E2.

Nick Valiante, Chiron Corporation. Center for the study of Hepatitis C Seminar. 301 Weiss. Open to RU/WMCCU/NYPH/MSKCC community and guests.

WEDNESDAY, APRIL 3

1-5 p.m. Genes and Disease: Opportunities for Human Genetics in Modern Medicine.

Centennial Symposium. Caspary Auditorium. Admission is free and no registration is required. A complete symposium schedule is available at www.rockefeller.edu/lectures.html. For more information contact Bobbie Larraga, 327-7240.

11 a.m. Amplification and Attenuation of Dendritic Cell Responses Via Cognate Interactions with Autologous NK cells.

Nick Valiante, Chiron Corporation. Center for the Study of Hepatitis C Special Seminar. 301 Weiss. Contact Patricia Holst, 327-7047. Open to RU/WMCCU/NYPH/MSKCC community and guests.

6 p.m. P53 Action During Tumor Development and Therapy.

Scott Lowe, Cold Spring Harbor Laboratory. **Retinoids in Redox Regulation of Signaling: Implication in Apoptosis.** Irina Koritcheva, MSKCC. Cell Death Society Meeting. 116 Rockefeller Research Laboratories, MSKCC, 430 East 67th St. Refreshments at 6 p.m. Contact Adriana Haimovitz-Friedman, 639-5109. Open to RU/WMCCU/NYPH/MSKCC community and guests.

The Arts and Other Events

FRIDAY, MARCH 15

12 p.m. Tri-Institutional Noon Recitals.

Auryn Quartet. Performing Haydn: *Last Seven Words of Christ on the Cross*. Open to RU/WMCCU/NYPH/MSKCC community and guests.

MONDAY, MARCH 18

5:30 p.m. Narrative and Science.

Oliver Sacks, Albert Einstein School of Medicine. The Lewis Thomas Prize: Honoring the Scientist as Poet. Caspary Auditorium. Contact Gloria Phipps, 327-8967.

8 p.m. Rockefeller University Film Series.

Shoot the Piano Player (directed by François Truffaut, 1960). Caspary Auditorium. Open to RU/WMCCU/NYPH/MSKCC community and guests.

WEDNESDAY, MARCH 20

8 p.m. Peggy Rockefeller Concerts Series.

Anthony Dean Griffey, tenor. Caspary Auditorium. Contact Meridith Egyes, 327-8437.

THURSDAY, MARCH 21

4 p.m. Presentation of Postdoctoral Association Representative Candidates.

305 Weiss. Contact Daniel Besser, 327-8793. Open to RU community and guests.



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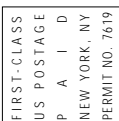
Writers: Whitney Clavin, Lynn Love, Holly Teichholtz, Lisa Stillman

Editors: Joseph Bonner, John Haubrich, Anna Sobkowski, Cathy Yarbrough

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