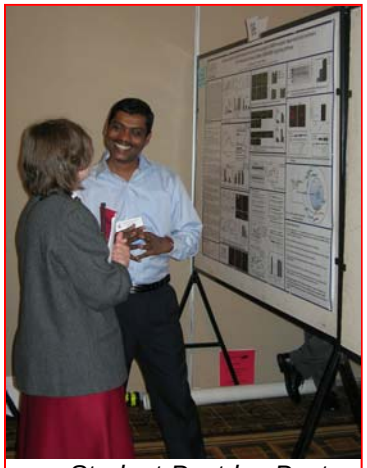
The Pharmacologist
Vol. 52
Number 2
2010
June

ASPET's Annual Meeting & Experimental Biology '10 Anaheim, CA

Award Winners

Opening Reception



Student-Postdoc Poster Competition

WIP Walk

Past Presidents Dinner

Dolores Shockley Best Abstract Award Winner

Drug Metabolism Division Mixer

The Student-Postdoc Mixer

The PHARMACOLOGIST

News

Features

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MESSAGE FROM THE PRESIDENT

Dear ASPET Members,

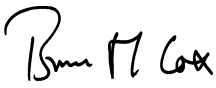
This has been a year of consolidation for ASPET. Despite a few bumps, the financial recovery has generally continued over the last year. Our finances have also benefitted from a most generous bequest to the society from the estate of a former member of the society, Dr. Vincent G. Zannoni, a Professor of Pharmacology at the University of Michigan from 1974 - 2009. The income generated by this bequest now supports our summer undergraduate fellowship (SURF) programs, allowing us to reprogram these funds for other critical activities, as well as expand the program.

One of the society's major activities this year has been the updating of the ASPET web site. This was completely redesigned last summer, and Jonathan Maybaum was appointed to the position of Web Editor. Jonathan has worked hard to re-design the ASPET Division websites and to locate them on the main ASPET server. He is now working on the development of new content for the website and will appreciate hearing your ideas. The importance of the website for the society can hardly be overestimated. Potential new members, new recruits to the profession of pharmacology, and interested members of the general public use the internet to obtain information about the society and the discipline. This is the face of the society that is visible to the world, and we need to make it as attractive and informative as possible. We also hope to make the website a valued resource for all members and encourage you to use the website to make your views known on all issues of relevance to the profession.

As my year as President comes to an end, I would like to draw attention to the outstanding work of the ASPET office staff. Rich Dodenhoff and the very skilled staff of the Publications Division continue to publish five professional journals of the highest quality. Jim Bernstein runs a very lean Public Affairs office, working very effectively with the public affairs offices of other biomedical research societies while devoting special effort to the specific interests of pharmacologists. We are progressively improving the quality of our services to members under the direction of Suzie Thompson (and we offer her our congratulations on the recent birth of her twins). This year ASPET also hired its own accountant, a task previously contracted out to FASEB. With the increasing complexity of regulations governing non-profit organizations, we need the dedicated services of our own accountant. Laine Cocca is rapidly becoming familiar with the society and its members, and we welcome her to the ASPET family. These groups have all been welded into a highly interactive team under Executive Officer Christie Carrico, who also oversees the myriad of other activities that are an integral part of the management of a professional society. It has been a pleasure to work with them.

It has been an honor and a privilege to serve as President of ASPET and I thank members for giving me this opportunity. I encourage all members of ASPET to become actively involved in the affairs of the society; the opportunities to interact with colleagues from all areas of the discipline make the commitment of time and effort seem minimal. I leave the society in the very capable hands of incoming President Jim Halpert and look forward to an exciting future for the society under his leadership.

Sincerely,



Brian M. Cox
President

EB 2010 IN REVIEW

ASPET met as part of Experimental Biology 2010 from April 24 to 28 in Anaheim, CA. With nearly 12,000 attendees, the meeting provided registrants with a mix of important science and fun networking events.

Just prior to the meeting, on Friday, April 23, attendees spent the day working at the Union Station Adult Center in Pasadena. The event was organized by ASPET's Behavioral Pharmacology Division. Volunteers prepared and served breakfast and lunch to nearly 150 people, unpacked and sorted food and other donations, and conducted routine maintenance and cleaning at the Adult Center. Started in 1973, Union Station is the largest and most comprehensive social service agency in the San Gabriel Valley. Union Station assists homeless and very low income people rebuild their lives and end homelessness.

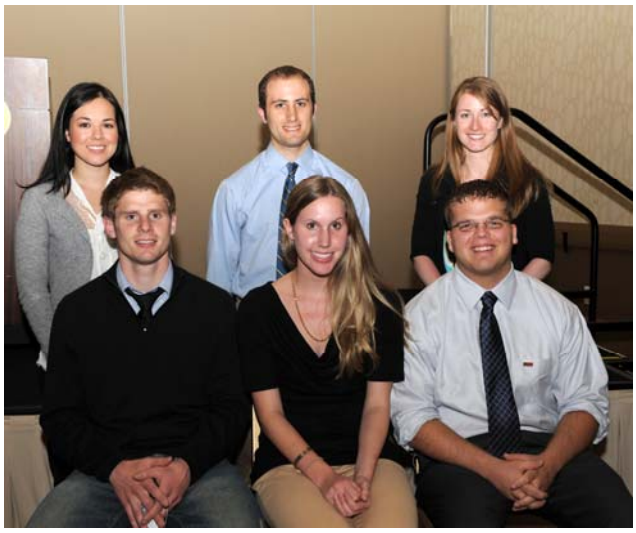
EB 2010 IN REVIEW

The ASPET Business Meeting took place on Saturday, April 24, where attendees received information about ASPET's membership, public affairs activities, finances, publications, and other business. ASPET's awards were presented at the meeting.

Recipients of ASPET's 2010 Awards

ASPET Graduate Student Travel Award Winners for 2010

ASPET Young Scientist Travel Award Winners for 2010

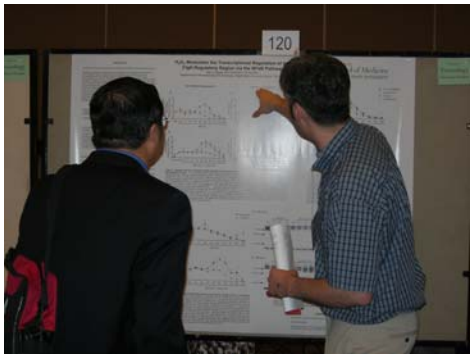


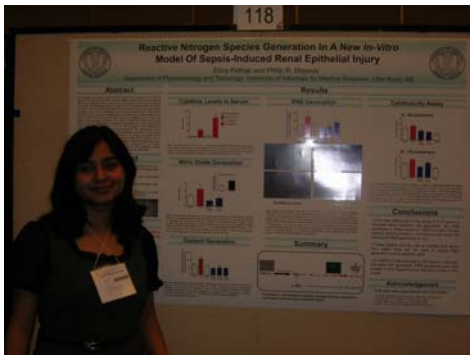
Summer Undergraduate Research Fellowship Award Winners for 2010

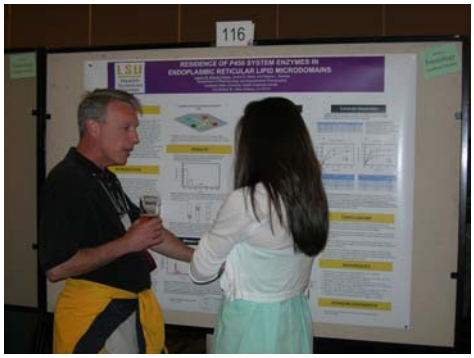
Following the Business Meeting and Awards Presentations, ASPET kicked off the 2010 Meeting with an opening reception on the roof deck of the headquarters hotel.

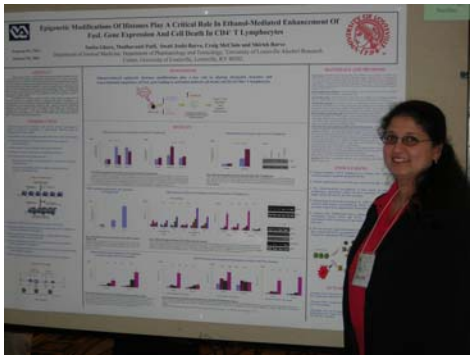
The Student/Postdoc/Mentor Mixer and Best Abstract Competition gave students and young scientists a chance to present their work and mingle with ASPET members.

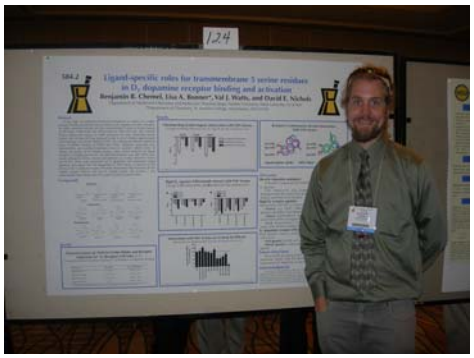






The ASPET Divisions also held mixers during the meeting. Pictures from the Division mixers and poster competitions can be found starting on page 41.

SAVE THE DATE!

2011 ASPET Annual Meeting at Experimental Biology April 9-13, Washington, DC

2011 Preliminary Symposia

- Advances in Estrogen Receptor Signaling: Potential Implications for Women's Health
- Autism & PDD: Neuropathology, Pharmacotherapies, & New Directions
- Cardiovascular KCN1 (Kv7) Potassium Channels: Physiological Regulators & Targets for Therapeutic Intervention
- Chronobiology in the Modern Curricula: Addressing Disease Linkage & Pharmacological Approaches
- Creating Effective Questions for Assessment & as Aids in Learning in Today's Pharmacology Programs
- Drug Metabolism & Action in Pathophysiological Conditions
- Gα Subtype-Selective Signaling By GPCRs as a Substrate for Functional Selectivity
- G-Protein Coupled Receptor Signaling in Stem Cell Biology
- Idiosyncratic Drug Reactions
- Julius Axelrod Symposium
- Micro-RNA Controlled Regulation of Drug Metabolism & Disposition
- New Roles for Arrestins in Signaling, Trafficking & Disease
- Novel Regulation, Physiological Roles, & Pharmacological Intervention of GPCR-Adenylyl Cyclase Signaling Systems
- Organ-Specific Toxicities Caused by Novel Metabolic Pathways
- Pharmacogenomics & Personalized Medicine
- Pharmacogenomics to Address Adverse Drug Events
- Pharmacology for Healthcare Professionals: Thirst for Knowledge
- Physiology & Pharmacology of Trace Amine Associated Receptors
- Recent Developments in the Understanding of the Biology & Physiology of the Jak Family of Tyrosine Kinases
- Regenerative Pharmacology & Translational Therapies for Repair of Nerve & Muscle Diseases/Disorders
- Role of Neuroinflammation in Psychiatric Disease
- Systems Biology of Oxidative Stress & Therapeutic Implications
- The Biological “Specifics” of the “Non-Specific” Placebo Response
- The Neurobiology of Post Traumatic Stress Disorder & Implications for Treatment
- Therapeutic Angiogenesis
- Therapeutic Peptides: Novel Approaches in Drug Development
- Therapeutic Targeting of Epoxyeicosanoids
- Too Much or Too Little: Behavioral Models & Pharmacotherapies for Eating Disorders

Division Programming

- Behavioral Pharmacology Division Symposium: Pharmacokinetic Approaches to the Treatment of Drug Abuse
- Cardiovascular Pharmacology Division Trainee Showcase & Benedict Lucchesi Distinguished Lecture
- Drug Discovery, Development & Regulatory Affairs Divison Symposium: High Impact Pharmacological Screening in Academia
- Drug Metabolism Platform Session, Gillette Award Winners & Early Career Achievement Award Lecture
- Integrative Systems, Translational & Clinical Pharmacology Division Young Scientist Awards Platform Session
- Molecular Pharmacology Division Postdoctoral Award Finalist Presentations
- Neuropharmacology Division Best Abstract Presentations
- Pharmacology Education Division Symposium: Pharmacokinetics: A Review of the Basics
- Toxicology Division Symposium: Hypoxia, Hypoxia-Inducible Factor-1α & Toxic Responses

Why Publish with ASPET?

Because ASPET gives you these advantages:

The Journal of PHARMACOLOGY and Experimental Therapeutics

MOLECULAR PHARMACOLOGY

DRUG METABOLISM AND DISPOSITION

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- **Low Color Fees** — $200/color figure for ASPET members, $400/color figure for nonmembers
- **Online Manuscript Submission** — submit your manuscript 24/7 — whenever it suits your schedule; online peer review reduces review times; track the progress of your manuscript through the review process
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Visit www.aspetjournals.org to access each ASPET journal.

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2011 Subscription Prices Set

The Board of Publications Trustees set the Society's 2011 subscription prices during a conference call on May 27. Institutional prices for *JPET* and *Molecular Pharmacology* will increase by 1%. *Pharmacological Reviews* and *Drug Metabolism and Disposition* will increase by 4%. There will be no increase for *Molecular Interventions*. The price differential between domestic and foreign institutional print-with-online subscriptions for all ASPET journals will not increase.

The member print subscription price for *DMD* will increase by 10%, raising the price from $137 to $151. Member prices for the other journals will not increase.

The minimal increases to institutional prices are possible because of cost-saving changes to our production processes made at the end of 2009 and because *JPET*, *MOL*, and *DMD* expect to publish fewer pages in 2011. *PharmRev* anticipates publishing more articles, and *DMD* continues to operate in the red; hence, their higher price increases.

The BPT decided in 2009 that member prices must cover the printing and mailing costs for print subscriptions. Thanks to the decreased costs noted above, the *JPET*, *MOL*, and *PharmRev* member subscription prices now cover those costs and did not need to increase. The 2009 *DMD* member price, however, was below actual cost and had to be raised.

The budget crises of most institutional libraries were a concern and another reason that the BPT kept subscription price increases as low as possible.

New Editorial Board Members

The following individuals joined the editorial boards of three ASPET journals following approval by the Board of Publications Trustees. The BPT is grateful for their service to their respective journals and to the Society.

Drug Metabolism and Disposition

Associate Editor:
Xinxin Ding

Journal of Pharmacology and Experimental Therapeutics

Associate Editor:
Giora Feuerstein

Editorial Advisory Board Members:

John Auchampach	Grant Drummond	Derek Hausenloy	Gerard Marek	Patrick Sexton
Bjoern Bauer	Alessandro Fatatis	Paul Heerdt	Giorgio Minotti	Ellen Walker
Ann Daly				

Molecular Interventions

Editorial Advisory Board Members:

Philip A. Cole	Jeffrey Martens	Trevor Robbins	Nancy Rusch	JoAnn Trejo

Pharmacological Reviews

Associate Editor:
Finn Olav Levy

Does your library subscribe to *Molecular Interventions*?

- Scientifically rigorous
- Cutting-edge content
- *MI* keeps you up to date in all areas related to pharmacology
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molinterv.aspetjournals.org

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Public Affairs/ Government Relations

NIH Budget Update

There has been little change for several months about future spending prospects for NIH funding. Congress is unlikely to pass a budget resolution that would set spending limits for FY 2011. There is little consensus among House Democrats on how to address deficit reduction and upcoming mid-term elections this November make any progress on a budget unlikely. Congressional appropriators will try to move forward to pass some of the 12 appropriations bills before the August recess. But with a full slate of major policy issues to address, it is virtually certain they will not be able complete action on most, not to mention all, of them. This means that a "Continuing Resolution (CR)" will most likely be needed to keep NIH and other government agencies funded through November's mid-term election. Following the elections, Congress could return for a lame-duck session and pass any unfinished appropriations bills or just wait until after January 1. The FY 2011 fiscal year begins October 1.

None of these developments are good for future NIH levels. A CR would likely maintain FY 10 spending levels. Rapidly growing pressure–political and economic—to address the federal budget deficit and national debt make the potential for any possible gains that NIH might achieve unlikely.

Both Congress and the Obama administration are responding to the difficult challenges to reduce the nation's financial deficits.

In April, the Senate Budget Committee approved a budget resolution that included a three-year freeze on all non-security discretionary spending and set a spending cap level $4 billion below the President's FY 2011 budget request. And, Labor/HHS Education Appropriations Subcommittee Chair David Obey (D-WI.) stated at a public hearing that his sub-committee, which funds the NIH, would also hold spending levels below the President's request. Obey, who is retiring from Congress, responded to an effort by advocacy groups to *increase* by $14 billion the amount for Labor/HS programs. Obey stated he may have to *cut* $3.5 billion from the president's $153 billion request for Labor/HHS appropriations.

In early June, the Office of Management and Budget (OMB) issued two memos emphasizing the need for fiscal discipline from federal agencies as they prepare their FY 12 budget proposals. One memo directs agencies to submit a budget request that is 5 percent below the discretionary total for the agency in the president's FY 11 budget. OMB does not suggest an across the board cut but instead instructs agencies to "restructure their operations strategically." The second memo directs agencies to identify "programs and subprograms that have the lowest impact on your agency's mission and constitute at least five percent of your agency's discretionary budget." The list should be included with the agency's budget submission for FY 12, "but is a separate exercise from the budget reductions necessary to meet the target for your agency's FY 2012 discretionary budget request." The White House memos can be viewed at:

http://www.whitehouse.gov/omb/assets/memoranda_2010/m10-19.pdf
http://www.whitehouse.gov/omb/assets/memoranda_2010/m10-20.pdf

New House Labor/HHS Members

The Democratic Caucus of the House Appropriations Committee made subcommittee selections for the remainder of the 111th Congress. Rep. Patrick Murphy (D-PA.) fills the seat previously held by Rep. Jack Murtha (D-PA.) who died in February. And, Rep. Jose Serrano (D-NY) joins the Labor/HHS Subcommittee replacing Rep. Jim Moran (D-VA.). A complete list of subcommittee assignments is available:

http://appropriations.house.gov/index.php?option=com_content&view=article&id=616:house-appropriations-committee-democrats-announce-subcommittee-assignments&catid=181:press-releases&Itemid=4

US Treasury Department $5 million Tax Credit Supports Research at Small Firms

The US Treasury Department issued guidelines "for applying for the new Therapeutic Discovery Project Program created by the Affordable Care Act. The program will provide tax credits and grants to small firms that show significant potential to produce new and cost-saving therapies, support good jobs and increase U.S. competitiveness." Details are at the ASPET web site at: http://www.aspet.org/PolicyUpdatesNews.aspx?id=1689

How to Measure the Impact of Federally Funded Research

OSTP, NIH, and NSF will begin a new multi-agency initiative that promises to monitor the impact of federal science investments on employment, knowledge generation, and health outcomes: "Science and Technology for America's Reinvestment: Measuring the Effect of Research on Innovation, Competitiveness, and Science" or STAR METRICS. STAR METRICS will help the federal government document the value of its investments in research and development, to a degree not previously possible. Data will come from research institutions that volunteer to participate and the federal agencies that fund them. The announcement indicates that information "will be gathered from the universities in a highly automated way, with minimal or no burden for the scientists and the university administration." The first phase of the program will calculate the employment impact of ARRA and previous federal science spending, using university administrative records. Phase two will measure the economic impact on economic growth; workforce outcomes; scientific knowledge; and social outcomes. Additional information about STAR METRICS is available at: http://nrc59.nas.edu/star_info2.cfm and http://sites.nationalacademies.org/PGA/fdp/PGA_057189. The press announcement is available at: http://www.nih.gov/news/health/jun2010/od-01.htm.

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rphipps@aspet.org

Drug Metabolism and Disposition Division News

Congratulations to the winners of the following DMD Division awards presented at ASPET's Annual Meeting during Experimental Biology 2010 in Anaheim, CA, in April.

James R. Gillette Drug Metabolism Best Papers of 2009

Metabolism Category:

NM DeVore, BD Smith, JL Wang, GH Lushington, and EE Scott (2009) Key residues controlling binding of diverse ligands to human cytochrome P450 2A enzymes², *Drug Metab Dispos* **37:**1319-1327, 2009

Pictured from left to right are Dr. Steve Leeder, Drug Metabolism Division Chair-Elect; Natasha Devore; and Dr. Jeffery Stevens, Drug Metabolism Division Chair.

Pharmacokinetics/Drug Transporters Category:

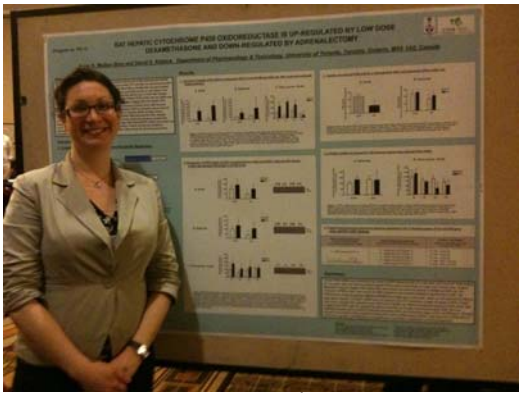
K Howe, GG Gibson, T Coleman, and N Plant (2009) In Silico and in Vitro Modeling of Hepatocyte Drug Transport Processes: Importance of ABCC2 Expression Levels in the Disposition of Carboxydichlorofluroscein, *Drug Metab Dispos* **37:**391-399

Pictured from left to right are Dr. Steve Leeder, Drug Metabolism Division Chair-Elect; Katharine Howe; and Dr. Jeffery Stevens, Drug Metabolism Division Chair.

Best Abstract Awards

Graduate Students:

First Place: Anne Mullen Gray, University of Toronto; sponsor: David Riddick. The first place prize consists of $500, meeting registration fee, $500 for travel next year, and the award winner becomes the student representative to the Drug Metabolism Executive Committee.



Second Place: Wenjun Li, University of Florida; sponsor: Margaret James. The second place prize is a $400 award.

Third Place: Kelly Clapp, University of Michigan Medical School; sponsor: Yoichi Osawa. The third place prize is a $300 award.

Honorable Mention: Ian Cook, University of Alabama-Birmingham; sponsor: Charles Falany.

Honorable Mention: Colleen Flynn, University of Kansas Medical Center; sponsor: Gregory Reed.

Postdoctoral Fellows:

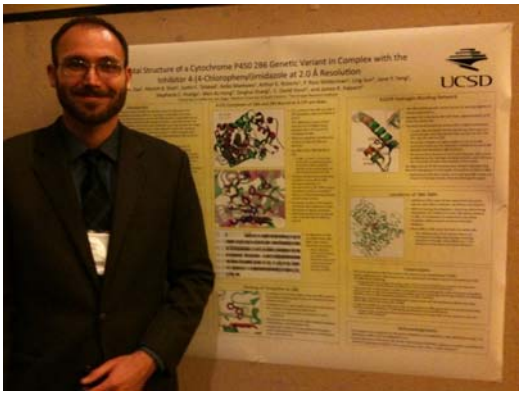
First Place: Sean Gay, University of California San Diego; sponsor: James Halpert. The first place prize consists of $500, meeting registration fee, $500 for travel next year, and the award winner becomes the postdoctoral fellow representative to Drug Metabolism Executive Committee

Second Place: Yongqiang Wang, University of California San Francisco; sponsor: Almira Correia. The second place prize is a $400 award.

Third Place: Tiangang Li, Northeastern Ohio University College of Medicine, sponsor John Chiang. The third place prize is a $300 award.

Honorable Mention: Yuzhuo Pan, State University of New York, Buffalo; sponsor: Aiming Yu.

Honorable Mention: Natasha Snider, University of Michigan Medical School; sponsor: Paul Hollenberg.



Sean Gay with his winning poster at the All Division Competition.

The Divisions' Poster Awards at the Annual Meeting

Winners of the Division for Molecular Pharmacology poster competition.

The Division of Toxicology poster competition winners and mixer.

Poster award winners from the Division of Drug Discovery, Drug Development, and Regulatory Affairs.






The Joint Mixer of the Divisions for Drug Discovery, Drug Development, and Regulatory Affairs; Integrative Systems, Translational and Clinical Pharmacology; and Pharmacology Education



The Joint Mixer for the Neuropharmacology and Behavioral Pharmacology Divisions



The Cardiovascular Division Mixer

Great Lakes Chapter
2009 Meeting Report and Abstracts

Submitted by Karie Scrogin, PhD, Past President, GLC ASPET

The 2009 meeting of the Great Lakes Chapter of ASPET was held on June 19 at the University of Chicago Gleacher Center in Chicago, IL. The central location, professional staff, beautiful facilities, excellent food, and reasonable cost made this venue highly successful. The 2010 organizing committee has already booked the same facility for next year's annual meeting.

Meeting Organization

Workshop

- "Career Opportunities in Drug Development", Marie Rock, Midwest BioResearch, LLC
- "Opportunities for BS/MS/students in Clinical Research", Alyssa O'Neill, Abbott Laboratories
- "Intellectual Property Law - At the Intersection of Science, Business, and Legal Issues", Verne A. Luckow, Neal, Gerber & Eisenberg LLP
- "Career Opportunities for Pharmacologists at Teaching-Oriented Institutions", Walter Prozialeck, Midwestern University

Afternoon Symposium – Neuroinflammation, Neurodegeneration and Drug Discovery

- "Alzheimer's Disease: Pathophysiology and New Therapeutic Approaches", Robert S. Bitner, Ph.D., Abbott Laboratories, Chicago
- "Targeting Glia Proinflammatory Cytokine Up-Regulation as a Drug Discovery Strategy for Neurodegenerative Disorders", Linda Van Eldik, Ph.D., Northwestern University, Chicago
- "Neuroinflammation-Induced Angiogenesis and Parkinson's Disease Progression", Paul M. Carvey, Ph.D., Rush University, Chicago

Keynote Address

"Reassessing Chronic Neuroinflammation in Alzheimer's Disease", Carol A. Colton, Ph.D., Duke University Medical Center, Durham, NC

Poster Session

A total of 25 posters were presented, 16 of which were submitted for the trainee poster competition: 10 for the graduate student competition and 6 for the undergraduate competition. There were no post-doctoral submissions for competition this year.

Business Meeting

Past President Bess Everett was thanked for her many years of service as she rotated off the Executive Council, and the newly elected officers were congratulated. They include Karen Snapp, President; Alejandro Mayer, Vice President; and Debra Tonetti, Councilor.

During the business meeting, awards were presented to the winners from the two categories in the poster competition. Among the pre-doctoral presenters, first place was given to Richard Gordon, Iowa State University; second place went to Juan Reyes, Northwestern University; and third place was awarded to Subhashini Srinivasan, University of Illinois at Chicago. In the undergraduate presenters category, the first place winner was Cicely Moreno of Indiana University School of Medicine-Northwest, the second place winner was Kevin Kanthasamy of Iowa State University, and the third place winner was Daryn Cass from Lake Forest College/Rosalind Franklin School of Medicine and Science.

The Great Lakes Chapter gratefully thanks the following donors: Abbot Laboratories; ASPET; Loyola University Chicago, Stritch School of Medicine; and the Rosalind Franklin University of Medicine and Science Department of Molecular and Cellular Pharmacology. The Great Lakes Chapter of ASPET also thanks the Midwestern University Chicago College of Osteopathic Medicine Department of Pharmacology and the Indiana University School of Medicine-Northwest for their in kind contributions.

Abstracts from the Great Lakes Chapter 2009 Annual Scientific Meeting

THE NOVEL CALPAIN-INHIBITOR A-705253 PREVENTS STRESS-INDUCED TAU HYPERPHOSPHORYLATION IN VITRO AND IN VIVO. R.S. Bitner[1], A.L. Nikkel[1], B. Martino[1], S. Markosyan[1], M.W. Decker[1], J.P. Sullivan[1], A. Hahn[2], H. Schoemaker[2], and A. Moeller[2]. Abbott Laboratories, [1]Neuroscience Research, Abbott Park, IL 60611; [2]Neuroscience Discovery Research, Abbott GmbH & Co. KG, D-67008 Ludwigshafen, Germany.

Calpains are a family of calcium-dependent cysteine proteases expressed by numerous cell types that participate in signal transduction, cytoskeletal architecture and apoptosis. In neuronal cells, calcium-mediated overactivation of calpain has been implicated in several neurodegenerative disorders, including Alzheimer's disease (AD). Hyperphosphorylation of the microtubule-associated protein tau and the subsequent aggregation of tau filaments resulting in the formation of neurofibrillary tangles are recognized etiological pathways in AD pathology. Cyclin-dependent kinase 5 (cdk5) is a major kinase responsible for tau hyperphosphorylation in AD that in part may involve calpain-mediated conversion of a cdk-5 regulatory activating protein (p35 to p25). In the present study, we examined the effects of calpain inhibition both in vitro and in vivo on tau phosphorylation. In hippocampal slices, lowering medium temperature to 33°C increased tau phosphorylation at the AT8 site as measured by Western blot analysis. Incubation with the novel small molecule calpain inhibitor A-705253 blocked the 33°C-induced tau phosphorylation with an IC50 of 100 nM. In vivo, pentobarbital-induced hypothermia or acute systemic LPS treatment increased tau phosphorylation in Mossy Fibers of the CA3 hippocampus of non-transgenic mice, as measured immunohistochemically at the AT8 site. A-705253 (3-10 mg/kg s.c.) pretreatment reduced the stress-induced tau hyperphosphorylation near control levels in both models. Results of these studies suggest that calpain inhibition has potential utility in reducing tau hyperphosphorylation and may represent a novel disease modifying approach in the treatment of AD.

EFFECT OF THE MARINE *VIBRIO VULNIFICUS* LIPOPOLYSACCHARIDE ON BRAIN MICROGLIA CYTOKINE AND CHEMOKINE RELEASE. Jeffrey Frenkel*[1], Monica Aldulescu[1], Mary L. Hall[1], Keith B. Glaser[1,2], Jan L. Powell[3], and Alejandro M.S. Mayer[1]. [1]Midwestern University, Downers Grove, IL 60515; [2]Abbott Laboratories, Abbott Park, IL 60064; [3]University of Maryland School of Medicine, Baltimore, MD.

Inflammation of the brain has been associated with the release of inflammatory mediators such as interleukin 1 α (IL-1α), interleukin 6 (IL-6), and macrophage inflammatory protein 2 α (MIP-2), as well as the anti-inflammatory mediator transforming growth factor β1 (TGF-β1) by microglia (BMG). Vibrio vulnificus (VvLPS) has been hypothesized to be involved in Vv (VvLPS) septicemia. We have recently reported that VvLPS stimulated BMG to release TNF-α, TXB2, O2- and MMP-9 (Mayer et al., *FASEB Journal* 21(5): A 404, 2007). The purpose of this investigation was to determine the effect of VvLPS on BMG priming and release of IL-1α, IL-6, TGF-β1, MIP-2 and lactate dehydrogenase (LDH) in vitro. BMG were isolated from neonatal rats and then treated in vitro with VvLPS or Escherichia coli (EcLPS) for 17 hours. IL-1α , IL-6, TGF-β1 and MIP-2 were determined by immunoassay, and LDH by enzyme activity. Results were the following (n=2): LDH: release observed at > 1 ng/mL VvLPS . IL-1α, IL-6 & MIP-2: concentration-dependent release detected at >1 ng/mL VvLPS ; in contrast TGF-β1 release was measured at > 1 µg/mL VvLPS . VvLPS appeared less potent than EcLPS. We conclude that VvLPS stimulated rat BMG cytokine and chemokine release in a concentration- and time-dependent manner. Further characterization of BMG response to VvLPS at both the functional and molecular level is ongoing in our laboratories. *Supported by Midwestern University and the University of Maryland.*

CORTICAL METABOLIC NEUROADAPTATION AFTER REPEATED COCAINE INJECTION AND WITHDRAWAL DEPENDS ON THE POSTNATAL AGE BY WHICH DRUG EXPOSURE ONSET IS GIVEN. D.K. Cass[1], S.R. Blume[2], K.Y. Tseng[2]. [1]Lake Forest College-Rosalind Franklin University Research Program; [2]Department of Cellular & Molecular Pharmacology, Rosalind Franklin University/The Chicago Medical School, North Chicago, IL 60064.

Exposure to a drug of abuse during adolescence significantly increases the risk for addiction when compared to the outcomes observed if drug exposure occurs during young adulthood. However, the mechanisms underlying the increased susceptibility to drug addiction during the peri-adolescent transition remains unclear. In the present study, we assessed the impact of repeated cocaine injection (5 days 15-25mg/kg/day) on cortical activity in rats, and asked whether the postnatal age at which the drug exposure onset is given plays a role in determining the neuroadaptations observed within the mesocorticolimbic system after 3 days and 3 weeks withdrawal from cocaine administration. Cortical activity was determined by assessing cytochrome oxidase (CO-I) staining as a measure of metabolic activity. In the adolescent-exposed group (PD35), we found a significant CO-I increase in the infra-prelimbic prefrontal, orbitofrontal and cingulate cortices

after 3 weeks withdrawal from cocaine injection. Such a prefrontal CO-I upregulation seems to require longer than three days to develop, as a slight increase was found after 3 days withdrawal. On the other hand, a very distinctive CO-I profile was observed when cocaine was given during adulthood (PD75). Adult exposure to repeated cocaine and withdrawal elicited an opposite pattern of CO-I activity in all frontal cortical regions, in particular at the level of the orbitofrontal cortex. The density of orbitofrontal cortex CO-I staining increases at 3 days but decreases at 3 weeks from cocaine withdrawal. Further analyses revealed that the pattern of cortical metabolic activation (adolescent-exposed) and inactivation (adult-exposed) follows a rostrocaudal pattern after 3 weeks withdrawal. Together, these results suggest that a different form of neuroadaptation may occur in the adolescent brain, in particular at the rostral portion of the frontal cortex, a cortical region that is heavily influenced by the mesocortical dopamine system. *Supported by Rosalind Franklin University and NIDA DA004093.*

THE DIABETOGENIC EFFECTS OF CADMIUM IN AN ANIMAL MODEL OF SUBCHRONIC CADMIUM EXPOSURE. J. R. Edwards, P. C. Lamar and W. C. Prozialeck. Department of Pharmacology, Midwestern University, Downers Grove, IL 60515.

It is estimated the total number of people with diabetes worldwide will rise from 171 million in 2000 to an estimated 366 million by the year 2030. The projected increase is mainly due to an epidemic of type II diabetes that has been attributed to environmental factors such as greater urbanization and industrialization. Exposure to cadmium (Cd) has been linked to diabetes in humans and in experimental animals. The mechanism(s) responsible for the Cd-induced increase in blood glucose is unclear. In this study, rats were dosed with Cd (0.6 mg/kg/day) for 12 weeks. Weekly fasting blood glucose levels were recorded, as well as serum insulin and %A1C levels at 12 weeks of treatment. Cd-treated animals showed significantly elevated blood glucose levels starting at week 8 of treatment and remained elevated until the end of the study. These changes occurred 3 weeks prior to any overt changes in renal function such as urine volume and proteinuria. After 12 weeks of treatment, fasting serum insulin and %A1C levels were significantly elevated in Cd-treated animals. This study indicates that Cd exposure contributes to the development of diabetes. Further studies need to be conducted to determine the mechanism of Cd-induced increase in blood glucose and the actions of Cd on pancreatic β-cells.

EFFECT OF *MICROCYSTIS AERUGINOSA* LIPOPOLYSACCHARIDE (LPS) ON NEONATAL RAT BRAIN MICROGLIA CYTOKINE AND CHEMOKINE RELEASE. Monica Aldulescu[1], Jeffrey Frenkel[1], Mary L. Hall[1], Keith B. Glaser[1,2], John Berry[3], and Alejandro M.S. Mayer[1]. [1]Midwestern University, Downers Grove, IL 60515; [2]Abbott Laboratories, Abbott Park, IL 60064; [3]Department of Chemistry and Biochemistry, Florida International University, Miami, FL.

Neuroinflammation has been hypothesized to involve release by brain microglia (BMG) of the inflammatory cytokines interleukin 1 α (IL-1α) and interleukin 6 (IL-6), the chemokine macrophage inflammatory protein 2 α (MIP-2), as well as the anti-inflammatory cytokine transforming growth factor β1 (TGF-β1). We have reported that M. aeruginosa LPS (MaLPS) stimulated BMG to release TNF-α, TXB2, O2- and MMP-9 (The Toxicologist 102(1):254, 2008). The purpose of this investigation was to determine the effect of MaLPS on BMG priming and release of IL-1α, IL-6, TGF-β1, MIP-2 and lactate dehydrogenase (LDH) in vitro. MaLPS (1.075 x 105 endotoxin units/mg) was isolated from M. aeruginosa strain UTCC 299 by hot phenol/water extraction. BMG isolated from neonatal rats were treated in vitro with MaLPS or Escherichia coli LPS (EcLPS) for 17 hours. IL-1α, IL-6, TGF-β1 and MIP-2 were determined by immunoassay, and LDH by enzyme activity. Results were the following (n=3-4): LDH: release was observed at > 0.1 ng/mL MaLPS; IL-6, & MIP-2: concentration-dependent release was detected at > 0.1 ng/mL MaLPS; IL-1 α: release was observed at > 1 ng/mL; and in contrast no release TGF-β1 was observed. MaLPS appeared less potent than EcLPS. We conclude that MaLPS stimulates rat BMG cytokine and chemokine release in a concentration- and time-dependent manner. Further characterization of BMG response to MaLPS at both the functional and molecular level is ongoing in our laboratories. *Supported by Midwestern University and Florida International University.*

INVESTIGATING THE CONTRIBUTION OF SERINE PHOSPHORYLATION AND ALANINE-76 TO α-SYNUCLEIN AGGREGATION, MEMBRANE ASSOCIATION, AND TOXICITY IN TWO YEAST MODELS. M. Fiske, K. Solvang, S. Valtierra, M. White, S. Herrera, M. Zorniak, A. Konnikova, and S. DebBurman. Biology Department, Lake Forest College, Lake Forest, IL 60044.

Parkinson disease (PD) is a devastating and incurable neurodegenerative disorder that afflicts over one million Americans. The universal PD pathology is the presence of aggregated α-synuclein within dying midbrain substantia nigra neurons. The role these α-synuclein aggregates play in cell death is unclear, and whether these aggregates are neuroprotec-

tive or harmful to cells is still being debated. Here, we first tested the hypothesis that serine phosphorylation promotes α-synuclein aggregation and toxicity by studying two phosphorylation-deficient (S87A and S129A) α-synuclein mutants in budding and fission yeast models. In budding yeast, we found that both S87A & S129A altered alpha-synuclein localization within cells and, surprisingly, increased cellular toxicity, but to differing extents in different strains. In fission yeast, both S87A and S129A resdistributed alpha-synuclein within the cytoplasm, and increased toxicity, but to a milder extent. Secondly, we tested the hypothesis that alanine-76 (a key residue within the middle hydrophobic domain of α-synuclein) contributes to its aggregation and plasma membrane phospholipid association by characterizing an A76E mutant in both yeasts. In support of this hypothesis, in budding yeast, significantly less A76E localized to the plasma membrane. Furthermore, in fission yeast, less A76E was aggregated in live cells. In both yeasts, more A76E was found cytoplasmically diffuse. Our data support the notion that serine-87 and serine-129 phosphorylation may make alpha-synuclein cytoprotective and alanine-76 aids in alpha-synuclein aggregation and membrane phospholipid binding. *Supported by NSF-MRI, NSF-CCLI, and NIH R15.*

FILPETTOR (FILter piPETTOR) – A SYRINGE-BASED, GENERAL SOLUTION FOR CONCENTRATION AND DESALTING OF COMPLEX BIOLOGICAL SAMPLES. Jeff Olson, Jeff Pan, Zhong Tang, and Jonathan Trumbull. Automation Engineering, Abbott Laboratories 60064; Dave Burns, William Chiou, Timothy Cloutier, Ken Comess, Scott Galasinski, Hua Tang. High throughput Screening.

The ability to separate, purify, and desalt complex biological samples is a common requirement in Pharmaceutical Research. In response, a large variety of devices have been devised and made commercially available. These include various centrifuge driven cartridges (Millipore MicroconTM, CentriconTM), pressure driven filters (Pall AcroPakTM, Millipore MillexTM), tangential filtration devices (Pall MinimateTM, UltrasetTM) and dialysis-based systems (MF-MilliporeTM). While each of these fulfills its intended purpose, a need remains for a more predictable, flexible, and fully automatable method to simultaneously process a large number of small biological samples. In response, a joint collaboration between the GPRD Automation Engineering and High Throughput Screening (HTS) departments at Abbott Park has developed a system referred to as Filpettor (FILter piPETTOR). In its current embodiment, the Filpettor is capable of fully automated filtration, concentration, and desalting of 48 parallel biological sample mixtures (30 - 300 µL volume). All fluid handling and filtration steps are conducted in custom, motor driven syringes.

The primary application thus far has been directed toward automating the Affinity Selection/Mass Spectrometry (ASMS) assay in the HTS department. Affinity Selection–Mass Spectrometry (ASMS) is one of the label-free detection technologies used in high throughput screening for identifying binders for the targets of interest. With its precise volumetric control, the Filpettor system provides 4- to 10-fold compound and protein savings as compared with traditional MicroconTM-based ASMS and could yield more accurate and reproducible results. We used two proteins (FBPase-1 and FABP-5) and five known binders for each protein to test the Filpettor-ASMS process. The results confirm that small molecule binding is protein concentration-dependent and that average KD values (N=12) are consistent with KD values generated from equilibrium dialysis.

VALIDATION OF MOUSE STEPPING TEST: FORELIMB AKINESIA INDUCED BY REPEATED MPTP INJECTION IS REVERSED BY L-DOPA TREATMENT IN A DOSE-DEPENDENT MANNER. Blume S.R, Cass D.K., Nazarian A., and Tseng K.Y. Department of Cellular and Molecular Pharmacology, Rosalind Franklin University/The Chicago Medical School, North Chicago, IL 60064.

Currently existing behavioral measures for motor impairments in rodent models with bilateral dopamine depletion have demonstrated to be difficult to assess due to the degree of task complexity. Here we adapted the stepping test, originally designed for assessing asymmetric motor deficits in rats (Olsson et al., 1995; Schallert et al., 1979), into a mouse friendly version for bilateral dopamine lesion induced by subacute MPTP injection. We found that MPTP-treated mice exhibit a significant and persistent reduction in the number of adjusting steps when compared to saline-treated animals. Typically, MPTP-induced stepping deficit becomes apparent by the fourth MPTP injection. The number of adjusting steps continues to decline throughout the injections, and by day 10 from the last MPTP injection, the stepping deficit observed is associated with ~65% TH positive cells loss in the SN. Single l-DOPA injection significantly improved the stepping performance in MPTP-treated mice as revealed by a marked increase in the number of steps. Interestingly, the duration of improved stepping is dose dependent (15, 30, 60 mg/Kg), with higher doses (60 mg/Kg) exhibiting a plateau in stepping performance lasting 6 hours post l-DOPA injection. Together, the data demonstrates that stepping test in mice is a reliable, sensitive and simple behavioral measure for assessing forelimb akinesia induced by systemic MPTP. *Supported by Rosalind Franklin University and NIDA DA004093.*

RESVERATROL PROTECTS DOPAMINERGIC NEURONS IN PARKINSON'S DISEASE MODELS BY MODULATING THE PKCD APOPTOTIC SIGNALING PATHWAY AND MICROGLIAL ACTIVATION. Kavin Kanthasamy, Richard Gordon, Arthi Kanthasamy, Vellareddy Anantharam, and Anumantha G. Kanthasamy. Parkinson's Disorder Research Laboratory, Iowa Center for Advanced Neurotoxicology, Dept. of Biomedical Sciences, Iowa State University, Ames, IA 50011-1250.

Resveratrol (3, 4', 5-trihydroxy stilbene) is a phytoalexin that has been shown to be neuroprotective in various models of neurodegeneration. Although resveratrol is generally recognized as a potent antioxidant, emerging evidence suggests this compound could exert its protective function by modulating various signaling molecules including SIRT-1 histone deacetlyase, heme oxygenase, NF-κB, COX2, and various kinases. Parkinson's disease is a multifactorial neurodegenerative disorder in which oxidative stress-induced apoptosis and neuroinflammation are recognized as important pathological mediators for initiation and progression of the disease respectively. We hypothesized that resveratrol could have a dual neuroprotective function in models of PD by protecting against dopaminergic neuron apoptosis as well as by suppressing microglia mediated neurotoxicity. Recent studies from our laboratory demonstrated that protein kinase Cd (PKCd) is an oxidative stress sensitive kinase that is proteolytically activated to induce apoptotic cell death in Parkinson's disease (PD) models. In this study, we examined whether i) resveratrol protects against dopaminergic neurodegeneration in cell culture models of PD, ii) resveratrol modulates the PKCd dependent apoptotic cascade in dopaminergic neuronal cells and iii) resveratrol attenuates microglial activation. Resveratrol treatment significantly increased cell viability in a dose-dependent manner against MPP+ induced cell death in mesencephalic dopaminergic neuronal cells (N27 cells). This protective effect was accompanied by attenuation of MPP+ induced caspase-3 enzyme activity as well as proteolytic cleavage of caspase-3. We also observed that resveratrol effectively blocked PKCd proteolytic cleavage induced during MPP+ treatment as well as the expression of the kinase in a dose dependent manner. Furthermore, we confirmed the neuroprotective effect of resveratrol in MPP+ treated mouse primary mesencephalic cultures by determining the number of TH positive neurons. Interestingly, resveratrol also attenuated LPS-induced nitric oxide production in BV-2 microglial cells in a dose-dependent manner, indicating that resveratrol can also suppress microglial activation and the subsequent neuroinflammatory response. Collectively, our results demonstrate that resveratrol protects against the degenerative process in dopaminergic neurons by modulating the PKC-delta dependent apoptotic pathway as well microglial neurotoxicity.

EVIDENCE OF A POSSIBLE PPARALPHA-INDEPENDENT VASODILATORY ACTION OF THE FIBRIC ACID ANALOGUE GEMFIBROZIL. Jacob D. Peuler and Laura E. Phelps. Pharmacology Dept., Midwestern University, Downers Grove, IL 60515.

Knocking out the gene for peroxisome proliferator-activated receptor-alpha (PPARalpha) was recently reported to abolish hypertension associated with an overactive human renin-angiotensin-aldosterone system (RAAS) transgenically expressed in mice (Hyper 50:945-51, 2007). Thus, attention is now focused on whether fibrates (PPARalpha agonists often used to lower plasma triglycerides) are capable of aggravating related forms of hypertension in humans (Hyper 50:847-50, 2007). We recently reported acute relaxant effects of high concentrations of gemfibrozil (e.g. 500 micromolar) on various smooth muscle preparations, including rat ventral tail arteries pre-contracted with either arginine-vasopressin (AVP) or serotonin (FASEB J 21:A1162, 2007). In the present work, we show that 4-hour exposure of the same arterial tissue to much lower but more therapeutically-relevant levels of gemfibrozil (e.g. 50 micromolar) also relaxes AVP- and serotonin-induced contractions. The same exposure also relaxes contractions induced by angiotensin II, norepinephrine and a high membrane-depolarizing level of extracellular potassium. Thus, we suspect that fibrates exert delayed but nonetheless direct and likely PPARalpha-independent vasodilatory activity which could offset any potential they have to exert a PPARalpha-dependent, RAAS-mediated hypertension.

ANATOMICAL STUDY OF SEROTONERGIC NUCLEI INVOLVEMENT IN CARDIOVASCULAR COMPENSATION FOLLOWING HYPOTENSIVE HEMORRHAGE. Jaimee Glasgow[1], Ling-Hsuan Kung[2], Jaime Vantrease[3], and Karie Scrogin[3]. [1]Department of Cell Biology, Neurobiology, and Anatomy; [2]Neuroscience Graduate Program; [3]Department of Pharmacology and Experimental Therapeutics, Loyola University Chicago.

Lesion of serotonin (5-HT) neurons in the hindbrain attenuates sympathetic compensation (SC) after hypotensive hemorrhage (HH) in awake rats. To determine the 5-HT nuclei involved in SC, 5-HT cells activated by HH were identified. Three days before the experiment, rats were implanted with vascular catheters under pentobarbital (65 mg/kg, i.p.) and randomized to 3 groups subjected to arterial blood withdrawal (21 ml/kg/10 min, n=7), hypotension (hydralazine, 10 mg/kg, i.v., n=8), or no treatment (n=7). After 90 min, rats were anesthetized (pentobarbital, 65 mg/kg, iv), perfused, and their brains processed for 5-HT- and c-Fos immunoreactivity. Larger numbers of double-labeled cells were observed in

the lateral extension of the B3 nucleus (P<0.05), the subependymal region lateral to B3 (P<0.05), and the raphe obscurus (P<0.01) in rats subjected to HH. In preliminary studies, adeno-associated viral-mediated knockdown of tryptophan hydroxylase 2 in the mid raphe obscurus attenuated SC (-16% vs. +72% D baseline, 15 min after HH), while knockdown in more rostral regions did not. 5-HT neurons in the brainstem, particularly in the raphe obscurus, likely contribute to SC following HH. *Supported by HL 72354*.

INDUCTION OF STRIATONIGRAL ERK1/2 PHOSPHORYLATION IN A RAT MODEL OF PARKINSON'S DISEASE. Cicely Moreno and Subbiah P. Sivam. Department of Pharmacology and Toxicology, Indiana University School of Medicine-Northwest, Gary, IN 46408.

Parkinson's disease (PD), a progressive, idiopathic disorder, typified by the symptoms of tremor, rigidity, and postural instability, results from the degeneration of nigrostriatal dopamine (DA) neurons. DA agonists, such as L-DOPA, are the main drugs for the treatment of Parkinson's disease (PD). However, their beneficial effects are invariably accompanied by serious side effects such as dyskinesias. Previous studies have suggested a role of extracellular signaling regulated kinases 1/2 (ERK1/2) with DA agonist-induced dyskinesias. ERK1/2, a MAP-kinase signaling protein, is involved in immediate-early gene induction, the regulation of gene expression, and neural adaptation. Using a hemiparkinsonism model, we examined the time-course of D1 agonist, SKF-38393-induced ERK1/2 phosphorylation in the rat striatum and substantia nigra. We unilaterally lesioned rat median forebrain bundle neurons with the neurotoxin, 6-hydroxydopamine. We screened the rats for the extent of the dopaminergic lesion by the apomorphine-induced rotation test. Fully lesioned rats were administered with the SKF-38393 and perfused at 15, 30, 60, or 120 minutes after the drug administration. The brains were removed, sectioned, and used for immunohistochemistry of tyrosine hydroxylase (TH, the rate limiting enzyme of DA synthesis), the neuropeptide substance P (SP, an indirect marker), and phospho-ERK1/2. As expected, dopaminergic lesion produced a severe decrease in TH and a modest decrease in SP levels, which did not vary with time in the striatum and substantia nigra. SKF-38393 induced a robust increase in phospho-ERK1/2 levels, which peaked at 15 min, declined at 30 min, and returned to baseline by 120 min within the lesioned striatum. We report for the first time that similar changes were observed in the substantia nigra. The time-dependent ERK1/2 activation in the striatonigral neurons may play a role in the potential mechanisms that are involved in the pathogenesis of PD and/or side effects such as dyskinesias related to the DA agonist treatment for PD.

NEURONAL LOCALIZATION OF HISTAMINE H4 RECEPTORS IN RAT AND HUMAN TISSUE. M. I. Strakhova, A. L. Nikkel, A. M. Manelli, G.C. Hsieh, T. A. Esbenshade, J. D. Brioni, and R. S. Bitner. Neuroscience Research, Global Pharmaceutical Research & Development, Abbott Laboratories, Abbott Park, IL 60064.

A series of studies were conducted in order to determine H4 receptor expression and localization in the brain, spinal cord, and dorsal root ganglia. Transcripts of the H4 receptor are present in all analyzed regions of the human CNS, including spinal cord, hippocampus, cortex, thalamus and amygdala, with the highest levels of H4 mRNA detected in the spinal cord. In rat, H4 mRNA was detected in cortex, cerebellum, brainstem, amygdala, thalamus and striatum. Very low levels of H4 mRNA were detected in hypothalamus, and no H4 signal was detected in the rat hippocampus. Low levels of H4 mRNA were detected in examined peripheral tissues including spleen and liver. Interestingly, strong expression of H4 mRNA was detected in the rat DRG and spinal cord. Immunohistochemical analysis using a selective H4 antibody revealed expression of H4 receptors on neurons in the rat lumbar DRG and in the lumbar spinal cord. These studies provide evidence of the H4 receptor presence in both human and rodent CNS and offer some insights into possible role of H4 receptors in itch and pain.

PROINFLAMMATORY CYTOKINE TNFA INDUCES APOPTOSIS IN DOPAMINERGIC NEURONAL CELLS VIA PROTEOLYTIC ACTIVATION OF PROTEIN KINASE C-DELTA (PKCD). Richard Gordon, Vellareddy Anantharam, Anumantha G. Kanthasamy, Arthi Kanthasamy, Department of Biomedical Sciences, Iowa State University, Ames, IA.

A sustained inflammatory response in CNS is a hallmark of most chronic neurodegenerative diseases. Accumulating evidence from human studies and animal and cell culture models over the last decade has identified neuroinflammation as an active pathogenic mechanism in the progression of Parkinson's disease (PD). Neuroinflammation is mainly associated with microglial activation and the production of proinflammatory factors including cytokines, chemokines, reactive oxygen species (ROS), nitric oxide (NO), and prostaglandins which contribute to the degeneration of the nigrostriatal dopaminergic neurons by augmenting oxidative stress dependent proapoptotic signaling events. In recent years, Tumor Necrosis Factor α (TNFα) has emerged as key mediator of neuroinflammation in several experimental models of PD and has also been shown to be significantly elevated in both the substantia nigra and CSF of PD cases. Notably, blocking TNFα signaling at various levels is reportedly neuroprotective in experimental models of PD. Although proapoptotic TNFα

signaling has been well studied in PD, the signal transduction events downstream of its canonical death receptor (TNFR1) in dopaminergic neurons have not been elucidated. In this study, we demonstrate that TNFα activates Protein Kinase C-delta (PKCδ), novel PKC family isoform, by caspase-8 and caspase-3 dependent proteolytic cleavage in mesencephalic dopaminergic neuronal cells (N27 cells) to induce apoptosis. Treatment of N27 cells with recombinant TNFα induced caspase-3 activation, DNA fragmentation, and time dependent proteolytic cleavage of PKCδ with a concomitant increase in its cleaved kinase activity. TNFR1 neutralizing antibody or the soluble TNF receptor Etanercept® almost completely blocked the proteolytic activation of PKCδ. Interestingly, TNFα-induced proteolytically activated PKCδ was accompanied by translocation to the nucleus. The proteolytically cleaved PKCδ is a key mediator of TNFα-induced apoptosis in these cells as a cleavage resistant mutant of PKCδ (PKCδ-CRM), or siRNA knockdown of PKCδ significantly protected against TNFα-induced caspase-3 and DNA fragmentation. Collectively, our results demonstrate that PKCδ is a key proapoptotic kinase involved in TNFα signaling through TNFR1 in dopaminergic neuronal cells. Further elucidation of PKCδ's role during neuroinflammation in the nigrostriatal dopaminergic system could provide valuable insight into development of novel therapeutic targets for PD.

KAVA-KAVA AND THE TREATMENT OF TEST-ANXIETY. Orion Biesan. The Department of Psychology and the Neuroscience Program, Baldwin-Wallace College, Berea, OH 44017.

This experiment assessed the acute effects of kava-kava on test-anxiety in an undergraduate college aged population. Test-anxiety is a psychological condition characterized by thought interference (TI) and feelings of anxiety (FI) that can have aversive latent effects on test-performance. Currently there is no strong evidence to support the use of pharmacological treatments for test-anxiety since most anxiolytic medications require daily dosages and/or induce a risk of physical dependence, memory loss, and other detrimental side-effects. Kava-kava is an herbal supplement that has been shown to have anxiolytic and nootropic effects [16]. Kava-Kava's active ingredients, kavalactones, act on several neurotransmitters including GABA, serotonin, dopamine, and norepinephrine. Thirty-seven participants were randomly assigned to one of three groups receiving a placebo, 75 mg of kavalactones or 150 mg of kavalactones. All participants completed an initial test containing the Westside Test-Anxiety Scale (WSTAS) and the Manual Test-Anxiety Profile (TAP). Students received a 10 item mathematics test and were given 20 minutes to complete the test and induce test-anxiety. After the test, participants assessed their level of TI and FI in relation to the mathematics test using the TAP. Participants in all groups showed no significant differences in WSTAS and initial TAP scores indicating a similar level of initial anxiety in each group. Participants that received 150 mg of kavalactones showed a significant reduction in FI and TI from the beginning to the end of the study and significantly less anxiety than either the placebo or 75 mg kavalactone groups when total change scores were compared. Total change scores were derived by finding the difference between post-test scores and pre-test scores in regards to FI and TI on the TAP. The placebo and 75 mg kavalactone groups showed no significant changes in TI and FI scores indicating the measure successfully induced test-anxiety. This experiment shows that an acute dose of 150 mg of kava significantly reduces test-anxiety in an undergraduate student population.

IN VIVO HISTAMINE H3 RECEPTOR OCCUPANCY STUDIES UTILIZING THE H3 RECEPTOR ANTAGONIST RADIOLIGAND [3H]A-349821. T. R. Miller, D. Strasburg, I. Milicic, J. Bauch, J. Du, S. Vaidyanathan, B. Surber, K.E. Browman, M. Cowart, J. D. Brioni, and T. A. Esbenshade. Abbott Laboratories, Global Pharmaceutical Research & Development, Abbott Park, IL 60064.

The histamine H3 receptor (H3R) antagonist radioligand [3H]A-349821 was utilized as a radiotracer for assessing in vivo receptor occupancy by unlabeled H3R antagonists. This model was established to relate H3R antagonists' receptor occupancy to blood exposure levels and efficacy in pre-clinical models. Following intravenous administration of [3H]A-349821 to adult male rats, cerebral cortical H3R occupancy by the radiotracer was measured as the ratio of its levels determined ex vivo in cortical and cerebellar tissue, the later being a brain region with very low levels of H3R. At low tracer doses and optimal exposure times, [3H]A-349821 levels were approximately 2-fold higher in cortex compared to cerebellum. With increasing [3H]A-349821 doses, cortical H3R occupancy was saturable with a binding capacity of 11.5 fmole/mg tissue, consistent with in vitro binding in rat cortex membranes. ABT-239 and other H3R antagonists, administered intraperitoneally 30 min prior to [3H]A-349821, dose-dependently inhibited H3R occupancy by the radiotracer. The ED50 for ABT-239 H3R occupancy was 0.44 mg/kg at a free blood concentration of 2.7 nM, similar to the ABT-239 equilibrium inhibition constant for the rat cortical H3R (3.3 nM) obtained from in vitro competition binding assays with [3H]-N-a-methylhistamine. Further, blood levels of ABT-239 and other H3R antagonists associated with H3R occupancy were consistent with blood levels producing efficacy in pre-clinical behavioral models of cognition. Thus, this pre-clinical model of H3R occupancy utilizing the radioligand [3H]A-349821 provides valid measures of in vivo H3R occupancy, information that may be helpful in guiding and interpreting clinical studies of advancing H3R antagonists.

A NOVEL ANTIBODY THAT TARGETS TAU TYROSINE NITRATION: IMPLICATIONS FOR ALZHEIMER'S DISEASE AND OTHER TAUOPATHIES. Juan F. Reyes[1], Yifan Fu[1], Nicholas M. Kanaan[1], Changiz Geula[2], Lester I. Binder[1,2]. [1]Department of Cell and Molecular Biology and [2]Cognitive Neurology and Alzheimer's Disease Center, Northwestern University, Chicago, IL 60611.

Alzheimer's disease (AD) is a neurological disorder typified by the formation of neurofibriallary tangles and amyloid plaques, which are composed mainly of tau and amyloid β proteins respectively. The tau protein within these structures has been identified to contain various post-translational modifications including phosphorylation, truncation and most recently, tyrosine nitration. Indeed, in a previous report, we characterized two monoclonal antibodies and identified tau that is nitrated at tyrosine 18 and tyrosine 29 in AD and other tauopathies. To determine whether tau at tyrosine 197 is nitrated in AD brains, brain homogenate samples were blotted with Tau-nY197, a new monoclonal antibody that specifically reacts with tau nitrated at tyrosine 197. Tau-nY197 identified tau proteins nitrated at tyrosine 197 in AD and control brain homogenate samples. Our immunohistochemical observations in areas of the hippocampus and frontal cortex further revealed a subset of activated, GFAP positive astrocytes containing nitrated tau at tyrosine 197. In some instances, Tau-nY197 positive astrocytes were associated with amyloid plaques. Collectively, these data suggest that tau within astrocytes is susceptible to tyrosine nitration at 197 in AD (Braak stage IV-VI) and controls (Braak stage I-III), possibly as a result of activation and /or amyloid deposition. *Supported by NIH awards AG 014449 (L.I.B.), AG 09466 (L.I.B.) and the NIH Drug Discovery and Chemical Biology (CDDCB) Training grant T-32-AG000260 awarded to J.F.R.*

EVALUATING AUTOPHAGIC REGULATION OF A-SYNUCLEIN PATHOTOXICITY PROPERTIES IN BUDDING YEAST. Ray Choi, Alina Konnikova, Daniel Sanchez, Kayla Ahlstrand, Peter Sullivan, and Shubhik DebBurman. Biology Department, Lake Forest College, Lake Forest, IL 60045.

Parkinson disease (PD) is an incurable neurodegenerative disease that results from the selective loss of dopaminergic neurons in the substantia nigra. The misfolding and aggregation of the protein a-synuclein is the likely cause of cell death in PD. A popular hypothesis is that increasing the degradation of a-synuclein may protect the cell from its toxicity and aggregation. While the lysosome is pharmacologically implicated in the degradation of a-synuclein, the genetic link between PD and autophagy is less clear. A major route for delivering proteins to the lysosome is macroautophagy (hereafter referred to as autophagy). Autophagy is a highly conserved catabolic process in eukaryotes used to recycle and/or catabolize damaged or excess protein and organelles. We hypothesized that autophagy would protect cells from a-synuclein toxicity, and tested it by genetically inhibiting the nucleation, expansion, and fusion steps of autophagy in a a-synuclein budding yeast model. We asked if a-synuclein's pathotoxicity liknked properties (accumulation, aggregation and toxicity) would be increased in yeast autophagy (Atg) gene deletion strains. Thus far, we have examined atg17D and vps15D (needed for nucleation) and atg1D, atg2D, atg8D, and atg18D (needed for expansion). Predominantly, the cellular localization of a-synuclein was subtly altered in all strains, but none of these changes induced toxicity in these autophagy yeast knockout strains. Specifically, in atg2D yeast, a-synuclein aggregated more extensively while still maintaining its plasma membrane localization. In atg17D yeast, a-synuclein became more cytoplasmically diffuse than plasma membrane localized. In these autophagy-deficient strains, a-synuclein did accumulate, but to varying and limited extents. To more completely understand autophagy-mediated alpha-synuclein degradation, we are currently analyzing knockout strains for the rest of the proteins that comprise the nucleation, expansion, and fusion steps of autophagy. *Supported by APDA, NSF-MRI, NSF-CCLI & NIH R15.*

THE ANTI-THROMBOTIC AGENT CANGRELOR (ARC69931MX), CAN INHIBIT HUMAN PLATELET ACTIVATION THROUGH A P2Y12 INDEPENDENT MECHANISM. Subhashini Srinivasan, Fozia Mir, Jin-Sheng Huang, Fadi T. Khasawneh, Stephen C.-T. Lam, and Guy C. Le Breton. Department of Pharmacology, The University of Illinois at Chicago, 835 S Wolcott Avenue, M/C 868, Chicago, IL 60612.

ADP plays an integral role in the process of hemostasis by signaling through two platelet G-protein coupled receptors, P2Y1 and P2Y12.The recent use of antagonists against these two receptors has contributed a substantial body of data characterizing the ADP signaling pathways in human platelets. Specifically, the results have indicated that while P2Y1 receptors are involved in the initiation of platelet aggregation, P2Y12 receptor activation appears to account for the bulk of the ADP-mediated effects. Based on this consideration, emphasis has been placed on the development of a new class of adenosine-based P2Y12 antagonists (separate from clopidogrel and ticlopidine) as an approach to the treatment of thromboembolic disorders. The present work examined the molecular mechanisms by which one of these antagonists, i.e., cangrelor (ARC69931MX) inhibits human platelet activation. It was found that cangrelor raises platelet cAMP to levels that substantially inhibit platelet aggregation. Furthermore, the results demonstrated that this elevation of cAMP did not require Gi signaling or functional P2Y12 receptors, but was mediated through activation of a separate G protein-coupled

pathway, presumably involving Gs. Additional experiments revealed that cangrelor did not increase cAMP through activation of A2a, IP, DP or EP2 receptors which are known to couple to Gs in platelets. Thus, these findings indicate that cangrelor interacts with an unidentified G protein-coupled receptor that stimulates cAMP-mediated inhibition of platelet function. This inhibition is in addition to that which may derive from antagonism of platelet P2Y12 receptors. *Cangrelor was kindly provided by AstraZeneca, and the authors would like to thank Lanlan Dong for her technical assistance. This work was supported in part by a grant from the National Institutes of Health HL23540-25 (to G.C.L.). Srinivasan S. is a recipient of a Predoctoral Fellowship from the American Heart Association (Grant 0815654G).*

SEROTONIN (5-HT) INNERVATION OF THE NUCLEUS TRACTUS SOLITARIUS (NTS) IS NECESSARY FOR SYMPATHETIC AND VENTILATORY RECOVERY FOLLOWING HYPOTENSIVE HEMORRHAGE. Ling-Hsuan Kung[1], Karie Scrogin[1,2]. [1]Neuroscience Institute, [2]Department of Pharmacology, Loyola University Chicago, Maywood, IL 60153.

Lesion of serotonin neurons in the caudal raphe suppresses sympathetic recovery following hypotensive hemorrhage (HH). We tested whether serotonin projections to the NTS influence sympathetic and ventilatory responses to HH, arterial baroreceptor and chemoreceptor reflexes responses. Male Sprague Dawley rats were injected with the serotonin neurotoxin 5,7-dihyroxytryptamine (N=8-12) or vehicle (N =7-9) in the medial and commissural NTS where baroreceptive and chemoreceptive sensory afferents terminate. Blood pressure (BP), heart rate (HR), renal sympathetic nerve activity (RSNA), and diaphragmatic EMG (dEMG) activity were recorded during injection of KCN (3, 10, 30, 100 µg/kg, i.v.) in conscious rats one day after instrumentation. After 24 hrs, BP, HR, RSNA and dEMG were recorded during arterial blood withdrawl (21 ml/kg/10m). Heart rate and sympathetic baroreflex gain was tested in separate, but similarly lesioned rats. Lesioned rats showed reduced pressor (9.5 ± 2.4 vs. 21.5 ± 4.9 ΔmmHg, $P<0.05$), and RSNA responses (293.0 ± 47.5 vs. 546.3 ± 65.7 Δ%baseline, $P<0.01$), as well as decreased tidal volume (324.1 ± 57.0 vs. 631.3 ± 93.2 Δ%baseline, $P<0.01$) and minute ventilation responses (234.8 ± 37.9 vs. 478.9 ± 77.7 Δ%baseline, $P<0.01$) to the highest KCN dose. Recovery of sympathetic (0.4 ± 15.4 vs. 102.2 ± 23.8 %baseline, $P<0.05$) and ventilatory responses (-30.1 ± 4.1 vs. +41.7 ± 11.5 %baseline intergrated dEMG, $P<0.01$) were reduced 30 min after termination of HH, though BP and HR responses did not differ between groups. NTS lesion increased the HR baroreflex gain at the BP set point (-4.2±0.3 vs. -3.0±0.2 ΔHR/ΔMAP, $P<0.01$) but did not affect the RSNA baroreflex. Lesion reduced the optical density of 5-HT immunoreactivity (IR) in the NTS by 93% ($P<0.01$), but did not affect tryptophan hydroxylase IR in raphe nuclei or tyrosine hydroxylase IR in the NTS. We conclude that serotonin innervation of the NTS is necessary for normal sympathetic and ventilatory recovery following HH. This effect may be related to a positive modulation of peripheral chemoreflex sensitivity by 5-HT nerve terminals in the NTS. *Supported by HLBI 72354 and AHA 0815529G.*

THE SH3A DOMAIN OF ITSN-1S REGULATES TRANSCELLULAR AND PARACELLULAR LUNG ENDOTHELIAL PERMEABILITY. *Cristina Bardita, Dan Predescu, Radu Neamu[1], and Sanda Predescu. Rush University Medical Center, Medical College, Department of Pharmacology, Vascular Biology Group; [1]Saint Raphael Hospital, New Haven, CT.*

ITSN-1 short, a multimodular protein with two NH2-terminal Eps15 homology (EH) domains coupled to five consecutive SH3 domains via a highly charged coiled–coil domain, is critically required for caveolae and clathrin-coated vesicles internalization, due to its interactions with proline-rich region of dynamin-2. The functional relevance of this interactions remains to be established. Here, using *in vitro* and *in vivo* studies complemented by biochemical and morphological analyses of endothelial cells (ECs), we investigated the role of SH3A domain of ITSN-1s in regulating endothelial permeability. Electron microscopy morphological analysis of endothelial cells expressing the SH3A domain of ITSN-1s indicated accumulation of caveolae clusters and caveolae with staining-dense rings around their narrow necks. A biotin assay for caveolae internalization, applied on SH3A-expressing ECs, showed greater than 60% inhibition of biotin uptake, by reference to controls.To asses ITSN-1s-dynamin-2 partnership *in vivo*, we expressed the SH3A domain of ITSN-1s in mouse lungs using cationic liposome delivery of a cDNA-SH3A myc-tagged sequence. Western blot analysis indicated efficient expression of the SH3A at 48 hours post-cDNA delivery. The functional effects of SH3A domain were evaluated by perfusion of 10 mg/ml dinitrophenylated albumin (DNP-BSA) through mouse lung microvasculature, followed by biochemical quantification of tracer transendothelial transport. We have found about 30% increase in the amount of BSA-DNP in all mouse lung lysates prepared from SH3A expressing mice, by reference to controls. Moreover, when 8 nm gold albumin was used as tracer, electron microscopy morphological analysis revealed open inter-endothelial junctions many of them heavily labeled throughout their length by gold-albumin complexes. Caveolae and clathrin-coated pits with very long necks, and caveolae whose narrow necks are surrounded by staining dense collar were frequently observed. Also we noticed dilated interstitial spaces, indicative of pulmonary edema. Together all these findings indicate that ITSN-1s, via its SH3A-dynamin-2 interaction, is an important regulator of endothelial permeability at both, transcellular and paracellular levels.

ION CHANNEL TOOLBOX FOR CARDIAC SAFETY EVALUATION. Ruth L. Martin, Xiaoqin Liu, James T. Limberis, Kathryn Houseman, Zhi Su, Gary A. Gintant, and Bryan F. Cox. Department of Integrative Pharmacology, Abbott, 100 Abbott Park Road, Abbott Park, IL 60064-6119.

The cardiac action potential is comprised of multiple ion channel currents acting in concert and these ion channels are important in cardiac safety liability assessment of potential drug candidates. Currently, hERG current screening is a critical part of the preclinical assessment of a drug candidate and is required before first in human (FIH) clinical studies. Through our ongoing efforts to provide efficient cardiac safety evaluation, while reducing animal usage, we have incorporated the use of several cellular ion channel whole-cell voltage clamp screens using heterologously expressed and native cardiac ion channels. With heterologously expressing cell lines, the use of planar patch technology (PatchXpress and QPatch) allows for moderate throughput by providing automated, simultaneous whole-cell voltage clamp recordings. In this study we highlight five cardiac ion channels; 3 heterologously expressed cardiac potassium channels (hERG [I_{Kr}], Kir2.1 [I_{K1}], KvLQT1/minK [I_{Ks}]) that contribute to the repolarization phase of the action potential and two additional cardiac ion channels, whose primary contribution is to the upstroke and plateau of the action potential; Nav1.5 [I_{Na}] (heterologously expressed) and the native cardiac L-type Ca channel, Cav1.2 [I_{CaL}] (cardiac myocytes). The biophysical properties of these two cardiac ion channels have been extensively characterized and each ion channel assay has been pharmacologically validated with reference compounds. In conclusion, this approach is part of our continuing effort to move to a cellular based *in vitro* safety approach to provide mechanistic SAR for solving cardiac safety issues. To date, our ion channel toolbox includes: hERG, Kir2.1, KvLQT1/minK, Nav1.5 and the native cardiac L-type Ca channel (Cav1.2); to be used on an as needed basis for cardiac safety evaluation.

DO hERG ENHANCERS AND BLOCKERS COMPETE? Xiaoqin Liu, James T. Limberis, Zhi Su, Kathryn Houseman, Gilbert Diaz, Gary A. Gintant, Bryan F. Cox, and Ruth L. Martin. Department of Integrative Pharmacology, Abbott, 100 Abbott Park Road, Abbott Park, IL 60064-6119.

HERG (human ether a go-go related gene) encodes a cardiac potassium channel that has been linked to delayed repolarization. Due to the large vestibule of the hERG channel pore, many structurally dissimilar compounds are able to block the hERG channel. This, along with specific requirement of hERG data by regulatory authorities, has added to the difficulty of drug discovery. Recently, we have discovered a series of compounds (hACTs) that do not block hERG, but actually enhance hERG current. hACT-1({4-[4-(5-trifluoromethyl-1H-pyrazol-3-yl)-phenyl]-cyclohexyl}-acetic acid) enhanced hERG current by 50 % at 60 μM. In addition, hACT-1 caused concentration-dependent shortening of the action potential duration in canine Purkinje fibers and guinea pig atrial tissue.

Preliminary studies suggest that binding of hACT-1 (60 □M) does not overlap sites of typical hERG blockers. hACT-1 did not displace radio labeled dofetilide. Also, in whole-cell voltage clamp studies, combination of hACT-1 with known hERG blockers (i.e., sotalol and terfenadine) suggest that the compounds are not competing for the same binding site. When applied simultaneously with a hERG blocker, the onset of hERG enhancement with hACT-1 occurs prior to block with either sotalol or terfenadine. Block with sotalol (150 μM) occurs at the same magnitude when used alone (42 %), or in combination with hACT-1 (44 %). Similarly, the enhancement of hERG current by hACT-1 is independent of sotalol block, just as the block of hERG current by sotalol is independent of hACT-1 current enhancement. These effects demonstrate that the binding site for enhanced hERG current is different than the binding site for block.

MEMBERS IN THE NEWS

William A. Catterall, PhD, Professor and Chair of the Department of Pharmacology at the University of Washington, has been named one of the international award winners of the Canada Gairdner Award for groundbreaking medical research behind cancer, epilepsy, and heart disease and malaria treatments. Since the inception of the award in 1959, one in four Gairdner Award winners has gone on to win the Nobel Prize. According to Dr. John Dirks, President and Scientific Director of the Gairdner Foundation, "These awards pay tribute to the passion, dedication and vision that drive these extraordinary individuals to push the boundaries of medical science."

Dr. Catterall was recognized for his discovery of the voltage-gated sodium channel and calcium channel proteins that the brain uses to receive, process, and send information. His work led to a new understanding of how these proteins work, leading to epilepsy medication for uncontrolled electrical activity. In addition, this research could lead the way to improved treatments for chronic pain and abnormal heart rhythms. He will receive his award in October. Dr. Catterall is a past editor of *Molecular Pharmacology.*

STAFF NEWS

Congratulations to new parents **Suzie Thompson**, ASPET's Director of Member Services and Marketing, and her husband, Chris, on the arrival of their twins, born June 15. Son Joshua weighed in at 4 lbs., 1 oz. and 18 inches, and daughter Taryn was 3 lbs., 12 oz. and 18 inches. Mom, dad, and the kids are doing well! Suzie will be on maternity leave until the early fall.

Congratulations are due to **Dan Collinge**, ASPET's Senior Editorial Coordinator for *Molecular Pharmacology*, who became engaged to Maura Elford in April. Dan proposed to Maura while they were in Venice, Italy. They plan to be married in August 2011.

Courtney Beardsworth joined the ASPET staff on June 7 as the editorial assistant for *JPET.* She works with Cassie Wood on the peer review process for that journal. Courtney holds a B.S. degree in English from Towson University and is working toward an M.F.A. degree in creative writing and publishing arts at the University of Baltimore. Prior to joining ASPET, she served as an editor and production manager for Cadmus Communications. Earlier positions included teaching English as a second language at the Community College of Baltimore County and at the Bead School in Prague, Czech Republic, and serving as an assistant editorial intern with Brick House Books. Courtney's writing has been published in *The Baltimore Review*, *Grub Street*, *Welter*, and *BettyConfidential.com*. In addition to welcoming her to the ASPET staff, we also congratulate Courtney on her engagement to Phil Weber on June 15.

Talia Goldman, who was the editorial assistant for *JPET*, left ASPET at the beginning of June to study massage therapy.

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Thank you for your commitment to ASPET and the discipline of pharmacology for ***50 years****!!*

ASPET Notes with Sympathy the Passing of the Following Members:

Sir James Black, MD

Jerry J. Buccafusco, PhD

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Louis R. Orkin, MD

Hans Ruelius, PhD

Jerry J. Buccafusco, PhD

Dr. Jerry Joseph Buccafusco passed away peacefully at home surrounded by his family on March 6, 2010. He was a beloved husband, father, and friend and an internationally renowned researcher at the Medical College of Georgia and the Veterans Affairs Administration Hospital. "Dr. B.," as he was known to many, was born on August 20th, 1949 in Jersey City, NJ, to Dominick and Rose Buccafusco. He was educated in Jersey City public schools, and his scientific career began with a chemistry set in his father's basement. He attended St. Peter's College, graduating with a degree in Chemistry in 1971. He then received a Master's degree from Canisius College and a Ph.D. in Pharmacology from the University of Medicine and Dentistry of New Jersey in 1978. During graduate school, he married Regina Neilan, a long-time friend, because, according to her, his car broke down and he needed transportation. In 1979, Jerry and Regina moved to Martinez, GA, when he was offered a professorship in the Pharmacology Department at MCG.

Over the next thirty years, Dr. B. was appointed director of the Neuropharmacology Laboratory at the VA, and he was the founder and director of both the Alzheimer's Research Center and the Animal Behavior Center at MCG. His research, which comprises more than 200 peer-reviewed articles in scientific journals and four edited books, has made significant contributions to a variety of fields, including hypertension, drug abuse, Gulf War Syndrome, and Alzheimer's Disease. In addition, he served as an associate editor of the *Journal of Pharmacology and Experimental Therapeutics* and various other scientific journals, as well as the chair of a National Institutes of Health study section on drug development research. For this work, Dr. B. received numerous national and international awards, most recently the Distinguished Alumnus Award from UMDNJ and the ASPET Award for Experimental Therapeutics. Last year he was appointed Regents' Professor of the University of Georgia System, and only days before his passing, he was awarded a multi-year, multi-million dollar research grant from the National Institutes of Health.

Dr. B.'s influence will live on in the work of numerous graduate students and post-doctoral fellows that he mentored who now serve in prestigious positions throughout the world. No doubt his proudest legacy, however, is his relationship with his family. Dr. B's wife, Regina, has been a member of the Columbia County Board of Education since 1998 and its Chairman since 2008. Their eldest son, Chris, was born in 1979 and is now a law professor in Chicago. Marty, born in 1982, is a filmmaker in New York City. He completed his first feature film last year. Their honorary "third son," Seth Otey, lives in Las Vegas. Dr. B. coached many of their youth sports teams, and he and Regina twice coached Marty's Odyssey of the Mind teams to the World Finals. In 1994, the Buccafuscos were named the Family of the Year by the Augusta Junior Women's Club. Jerry and Regina always maintained an open door policy for the neighborhood children, with "neighborhood" used very broadly. In addition to filling in for touch football games, Dr. B. was a trusted confidant, mentor, and inspiration to many young people. He was always happy to take time to show off his comic book collection or to introduce friends to the wonders of Dr. Who. In addition to his wife and children, Dr. B. is survived by his mother Rose Buccafusco, two brothers John & Dominick Buccafusco, a sister Roseanne Farrell, and many sister-in-laws, a brother-in-law, and nieces and nephews. He is predeceased by his father, Dominick Buccafusco.

Other information:

The research in Dr. Buccafusco's laboratory included the development of novel treatment modalities for Alzheimer's disease and related disorders. In 1988 his laboratory was the first to report the cognitive enhancing action of low doses of nicotine in non-human primates. Since that time he studied numerous novel memory-enhancing agents from several pharmacological classes in young and aged Rhesus monkeys. His most recent work was directed at the development of single molecular entities that act on multiple CNS targets to, not only enhance cognitive function, but also to provide neuroprotection, or to alter the disposition and metabolism of amyloid precursor protein.

His work in the area of drug abuse centered around the role of central cholinergic neurons in the development of physical dependence on opiates, and the expression of withdrawal symptoms and the return to drug-seeking behavior. Most recently his laboratory investigated the role of the immune system and in the production of auto-antibodies to beta-amyloid and to the receptor for advanced glycation end products (RAGE) by individuals with Alzheimer's disease.

Dr. Buccafusco's research was continually supported by federally-sponsored grants private foundations, as well as the pharmaceutical industry for more than 25 years.

Submitted by Alvin D. Terry, Jr., PhD, Medical College of Georgia, University of Georgia College of Pharmacy

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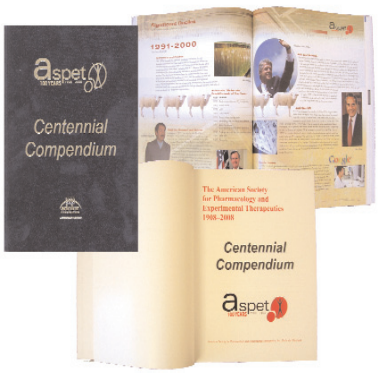

ASPET Ornament:

Holiday ornaments with the ASPET logo. Makes a great gift for colleagues! Members pay $5.00 plus shippng.

ASPET Wineglass:

A commemorative wineglass celebrating ASPET’s 100th Anniversary. Members pay $5.00 plus shipping.

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For large orders or any questions, contact Suzie Thompson, sthompson@aspet.org / 301-634-7916

ASPET-ASTELLAS AWARDS FOR TRANSLATIONAL PHARMACOLOGY

Deadline for submission of nominations is September 15, 2010

The ASPET-Astellas Awards in Translational Pharmacology are intended to extend fundamental research closer to applications directed towards improving human health. The awards will be given to 1) recognize those individuals whose research has the potential to lead to the introduction of novel pharmacologic approaches or technologies that may offer significant advances in clinical medicine in the future and 2) to facilitate that translational process. The awards are made possible by a grant to ASPET from the Astellas Foundation.

Three (3) awards of $30,000 each will be made to individuals. The money may be used for supplemental research funding, travel, training, or in any way that furthers the goals described above. In addition to the $30,000, award winners will receive travel to and lodging at the ASPET annual meeting for the winner and his/her spouse.

There are no restrictions on nominees for this award. Any ASPET member in good standing may nominate an individual for this award. Selection of the recipients will be made by a committee appointed by the President of ASPET and comprised of current and former Council members and current Division Chairs. Awards will be judged in two categories, viz. junior and senior investigators, with at least one award being given in each category. Junior investigators are defined as individuals at the assistant professor or equivalent level. In the event that there are insufficient meritorious candidates in either the junior or senior category, the committee reserves the right to make all awards in a single category. Self-nominations will not be accepted. The nominations shall be judged on submitted information and take into account:

1. The publication record of the applicant;
2. The quality and impact of the published research cited in support of the award nomination;
3. The potential of the proposed studies to advance clinical medicine through the introduction of novel pharmacological approaches to therapeutics;
4. The feasibility of the proposed research given the size of the award; and
5. The applicant's current funding to support the proposed project. For junior investigators, institutional commitment may also be a consideration.

Nominations shall be submitted **electronically** to jane.nelson@aspet.org and shall consist of:

1. A two-page summary that details the importance of the candidate's work and how it meets the criteria of the award;
2. Two additional supporting letters (need not be from ASPET members);
3. Candidate's CV and bibliography;
4. A two-page statement from the candidate of his/her plans for moving his/her research toward clinical practice and how the award money would be used to further this goal; and
5. Up to 5 articles published or submitted for publication (either PDFs or links to the articles may be submitted).

The receipt date for nominations for the ASPET-Astellas Award is September 15, 2010 for the award to be presented at Experimental Biology '11 in Washington, DC. Award winners will be notified in January 2011.

Recipients of the ASPET-Astellas Award in Translational Pharmacology

2007	2008	2009	2010
P. Jeffrey Conn	Randy D. Blakely	Piyali Dasgupta	Craig W. Lindsley
Kathryn A. Cunningham	Anthony J. Kanai	Paul A. Insel	Kenneth D. Tew
Liewei Wang	John S. Lazo	Richard R. Neubig	Gonzalo F. Torres

CALL FOR AWARD NOMINATIONS FOR 2011

JOHN J. ABEL AWARD

Deadline for submission of nominations is September 15, 2010

The John J. Abel Award in Pharmacology, named after the founder of ASPET, was established to stimulate fundamental research in pharmacology and experimental therapeutics by young investigators. The annual Award consists of $2,500, a plaque, hotel and economy airfare for the winner and spouse to the award ceremony at the annual meeting of ASPET.

Nominees for this award shall not have passed their **forty-second birthday on April 30** of the year of the Award. The candidate need not be a member of the Society; however, a nomination must be made by an ASPET member. No member may nominate more than one candidate a year, and no candidate may be nominated for more than one major ASPET award in any given year.

The Award shall be made for original, outstanding research in the field of pharmacology and/or experimental therapeutics. Independence of thought, originality of approach, clarity and excellence of data presentation are important criteria. Candidates shall not be judged in comparison with the work of more mature and experienced investigators. Quality rather than the number of contributions shall be emphasized. It shall be the responsibility of the sponsor to make clear the contribution of the candidate to any jointly authored reprints and manuscripts and the originality and independence of the candidate's research. Selection will be made by the ASPET Awards Committee, appointed by the President of ASPET.

Nominations must be submitted **electronically** to jane.nelson@aspet.org and should consist of:

1. Summary that describes the importance of the candidate's work.
2. Brief biographical sketch of the candidate.
3. Candidate's curriculum vitae and bibliography.
4. Each of six published articles or manuscripts accepted for publication that are a representation of the candidate's work (provided as PDFs or as hyperlinks to the article). **Submit each manuscript PDF as a separate attachment.**

Receipt date for nominations for the John J. Abel Award will be 5:00 pm on **September 15, 2010** for an award to be presented at Experimental Biology '11 in Washington, DC.

Recipients of the John J. Abel Award in Pharmacology

1947 George Sayers
1948 J. Garrott Allen
1949 Mark Nickerson
1950 George B. Koelle
1951 Walter F. Riker, Jr.
1952 David F. Marsh
1953 Herbert L. Borison
1954 Eva K. Killam
1955 Theodore M. Brody
1956 Fred W. Schueler
1957 Dixon M. Woodbury
1958 H. George Mandel
1959 Parkhurst A. Shore
1960 Jack L. Strominger
1961 Don W. Esplin
1962 John P. Long
1963 Steven E. Mayer
1964 James R. Fouts
1965 Eugene Braunwald
1966 Lewis S. Schanker
1967 Frank S. LaBella
1968 Richard J. Wurtman
1969 Ronald Kuntzman
1970 Solomon H. Snyder
1971 Thomas R. Tephly
1972 Pedro Cuatrecasas
1973 Colin F. Chignell
1974 Philip Needleman
1975 Alfred G. Gilman
1976 Alan P. Poland
1977 Jerry R. Mitchell
1978 Robert J. Lefkowitz
1979 Joseph T. Coyle
1980 Salvatore J. Enna
1981 Sydney D. Nelson
1982 Theodore A. Slotkin
1983 Richard J. Miller
1984 F. Peter Guengerich
1985 P. Michael Conn
1986 Gordon M. Ringold
1987 Lee E. Limbird
1988 Robert R. Ruffolo, Jr.
1989 Kenneth P. Minneman
1990 Alan R. Saltiel
1991 Terry D. Reisine
1992 Frank J. Gonzalez
1993 Susan G. Amara
1994 Brian Kobilka
1995 Thomas M. Michel
1996 John D. Scott
1997 David J. Mangelsdorf
1998 Masashi Yanigasawa
1999 Donald P. McDonnell
2000 William C. Sessa
2002 Steven A. Kliewer
2003 David S. Bredt
2004 David P. Siderovski
2005 Randy Hall
2006 Christopher Counter
2007 Michael D. Ehlers
2008 Katarina Akassoglou
2009 John J. Tesmer
2010 Russell A. DeBose-Boyd

CALL FOR AWARD NOMINATIONS FOR 2011

JULIUS AXELROD AWARD IN PHARMACOLOGY

Deadline for submission of nominations is September 15, 2010

The Julius Axelrod Award in Pharmacology was established to honor the memory of the eminent American pharmacologist who shaped the fields of neuroscience, drug metabolism and biochemistry and who served as a mentor for numerous eminent pharmacologists around the world. The Julius Axelrod Award is presented annually for significant contributions to understanding the biochemical mechanisms underlying the pharmacological actions of drugs and for contributions to mentoring other pharmacologists.

The award consists of an honorarium of $5,000, a medal, hotel and economy airfare for the winner and spouse to the annual meeting. The formal presentation of this award and medal will be made at the annual meeting of ASPET. The recipient will be invited by the President of the Society to deliver the Julius Axelrod Lecture and organize the Julius Axelrod Symposium at the annual meeting a year hence. The recipient will also be invited by the Catecholamine Club to give a less formal presentation at its annual dinner meeting that year of the award.

There are no restrictions on nominees for this award. However, a nomination must be made by a member of the American Society for Pharmacology and Experimental Therapeutics (ASPET) or the Catecholamine Club. No member may nominate more than one candidate in a year, and no candidate may be nominated for more than one major ASPET award in any given year. The award shall be made on the basis of originality and uniqueness of accomplishments throughout a long career distinguished by sustained, significant contributions to research and mentoring in pharmacology. Selection of the recipient will be made by the Axelrod Award Committee, appointed by the President of ASPET and comprised of members of ASPET and the Catecholamine Club.

Nominations shall be submitted **electronically** to jane.nelson@aspet.org and shall consist of:

1. Letter of nomination describing the research and mentoring contributions to pharmacology of the candidate that make him/her eligible for this Award, listing major contributions. Up to two additional letters of support would be welcome (need not be from ASPET members).
2. Brief biographical sketch of the candidate.
3. List of individuals mentored by the individual. Up to two letters from former trainees describing the quality of their training with the nominee and its impact on their careers would be welcome (need not be from ASPET members).
4. Candidate's curriculum vitae and bibliography.

Receipt date for nominations for the Julius Axelrod Award will be 5:00 pm on **September 15, 2010** for an award to be presented at Experimental Biology '11 in Washington, DC.

Recipients of the Julius Axelrod Award

The Julius Axelrod Award was initiated in 1991 by the Catecholamine Club. ASPET assumed the award in 2007.

1991 Ullrich Trendelenberg
1992 Arvid Carlsson
1993 Norman Weiner
1994 Robert Furchgott
1995 Irvin Kopin
1998 Sidney Spector
1999 Solomon Snyder
2000 Erminio Costa
2001 Toshi Nagatsu
2002 Salomon Langer
2003 Richard Weinshilboum
2004 Richard Palmiter
2005 Marc Caron
2006 Susan Amara
2007 Tong H. Joh
2008 Randy D. Blakely
2009 Palmer W. Taylor
2010 Brian Kobilka

CALL FOR AWARD NOMINATIONS FOR 2011

PHARMACIA-ASPET AWARD FOR EXPERIMENTAL THERAPEUTICS

Deadline for submission of nominations is September 15, 2010

The Pharmacia-ASPET Award in Experimental Therapeutics is given annually to recognize and stimulate outstanding research in pharmacology and experimental therapeutics—basic laboratory or clinical research that has had, or potentially will have, a major impact on the pharmacological treatment of disease. The award is supported in perpetuity by a gift from Pharmacia (now Pfizer). The winner will receive a $2,500 honorarium, a plaque, hotel and economy airfare for the winner and spouse to the award ceremony at the ASPET annual meeting.

There are no restrictions on nominees for this award. No one may nominate more than one candidate a year, and no candidate may be nominated for more than one major ASPET award in any given year. The Award shall be made on the basis of published reprints, manuscripts ready for publication, and a two-page summary. Selection will be made by the ASPET Awards Committee, appointed by the President of ASPET.

Nominations shall be submitted **electronically** to jane.nelson@aspet.org and shall consist of:

1. Two-page summary that details the importance of the candidate's work.
2. Six articles published or ready for publication by the candidate that have direct bearing on the Award (provided as PDFs or as hyperlinks to the article). Submit each manuscript PDF as a separate attachment.
3. Brief biographical sketch of the candidate.
4. Candidate's curriculum vitae and bibliography.

Receipt date for nominations for the Pharmacia ASPET Award for Experimental Therapeutics will be 5:00 pm on **September 15, 2010** for an award to be presented at Experimental Biology '11 in Washington, DC.

Recipients of the ASPET Award for Experimental Therapeutics

1969 John A. Oates
1970 Joseph R. Bertino
1971 Elliot S. Vesell
1972 Francois M. Abboud
1973 Dean T. Mason
1974 Leon I. Goldberg
1975 Mackenzie Walser
1976 Louis Lasagna
1977 Allan H. Conney
1978 Attallah Kappas
1979 Sydney Spector
1980 Sanford M. Rosenthal
1981 David G. Shand
1982 William H. Prusoff
1983 Marcus M. Reidenberg
1984 Sir James Black
1985 Louis Lemberger
1986 Alan C. Sartorelli
1987 Albrecht Fleckenstein
1988 Jean-Francois Borel
1989 Benedict R. Lucchesi
1990 Albert Sjoerdsma
1991 Theophile Godfraind
1992 James W. Fisher
1993 V. Craig Jordan
1994 Susan Band Horwitz
1995 Henry I. Yamamura
1996 Robert F. Furchgott
1997 Michael M. Gottesman
1998 Phil Skolnick
1999 Yung-Chi Chen
2000 Salomon Z. Langer
2001 George Breese
2002 Darryle D. Schoepp
2003 William C. De Groat
2004 Philip Needleman
2005 Donald P. McDonnell
2006 John C. Lee
2007 P. Jeffrey Conn
2008 Jerry J. Buccafusco
2009 Kenneth A. Jacobson
2010 Garret A. Fitzgerald

ASPET DIVISION FOR DRUG METABOLISM EARLY CAREER ACHIEVEMENT AWARD

Deadline for submission of nominations is September 15, 2010

The ASPET Division for Drug Metabolism Early Career Achievement Award has been established to recognize excellent original research by early career investigators in the area of drug metabolism and disposition.

The award is presented biennially in odd-numbered years. The award consists of $1,000, a plaque, and complimentary registration plus travel expenses (to a maximum of $1,000) for the winner to attend the award ceremony at the annual meeting. The awardee will deliver a lecture at the annual meeting describing their relevant research accomplishments. The awardee will be invited to publish a review article on the subject matter of the award lecture in *Drug Metabolism and Disposition*.

Nominees for this award must have a doctoral degree (e.g., Ph.D., M.D., Pharm. D., D.V.M.) and must be within 15 years of having received their final degree, as of December 31 of the year of the award. There are no restrictions on institutional affiliation and a candidate need not be a member of ASPET. There is a requirement for two nominators, although more are acceptable. Nominators must be members of ASPET. An individual cannot nominate more than one candidate per award cycle.

Candidates who have made their research contributions in any sector (e.g., academia, industry, government) of the drug metabolism community may be nominated for the award. The primary criterion for the award is the level of excellence and originality of the research conducted by the candidate in the field of drug metabolism and disposition. Independence of thought, originality of approach, clarity of communication, and the impact of the work on the drug metabolism field are important considerations. Candidates shall not be judged in comparison with the work of more experienced investigators. Selection will be made by the Executive Committee of the Division for Drug Metabolism.

Nominations shall consist of the following components:

1. Two or more letters of nomination and support.
2. The five most significant published papers authored by the candidate. A detailed examination of these publications will form a primary basis for evaluation.
3. The candidate's curriculum vitae and publication list.

All materials must be submitted as e**-mail attachments in PDF format**. Submit e-mail attachments to jane.nelson@aspet.org. Nominations for this award must be received no later than **September 15, 2010.**

Recipients of the ASPET Division for Drug Metabolism
Early Career Achievement Award

2007 Qiang Ma
2009 Wen Xie

ASPET DIVISION OF PHARMACOLOGYY EDUCATION TRAVEL AWARD FOR PHARMACOLOGY EDUCATORS

Deadline for receipt of applications is January 3, 2011

The ASPET Division of Pharmacology Education is pleased to announce the opening of applications for the 2011 Travel Award for Pharmacology Educators. The primary goal of this travel award is to promote participation in a FASEB-ASPET Meeting by pharmacology educators and to foster career development in pharmacology education.

Although there are no restrictions on faculty rank, the eligibility criteria are that the applicant (1) has significant teaching responsibilities in the area of pharmacology; and (2) is a member of ASPET (primary or secondary membership in the Division of Pharmacology Education is encouraged). The areas of teaching responsibilities in pharmacology can include instruction in graduate and undergraduate college classes as well as professional schools. In addition to curriculum delivery, preference will be given to the applicant demonstrating efforts in creative aspects of pharmacology education, e.g., curricula design assessment and faculty development.

The award (not to exceed $1,000) can be used to defray any of the following as needed: ASPET dues, travel expenses, registration, hotel accommodations, and cost of meals. All reimbursement expenses must be consistent with the guidelines of ASPET. The applicant will receive a plaque in recognition of his/her receipt of the award.

Application, updates and submission information may be found at: http://divisions.aspet.org/education/.

CALL FOR AWARD NOMINATIONS FOR 2011

EPILEPSY RESEARCH AWARD FOR OUTSTANDING CONTRIBUTIONS TO THE PHARMACOLOGY OF ANTIEPILEPTIC DRUGS

Deadline for submission of nominations is September 15, 2010

The International League Against Epilepsy (ILAE) has sponsored an award of $5,000 and a Certification of Citation to be awarded by the American Society for Pharmacology and Experimental Therapeutics for the purpose of recognizing and stimulating outstanding research leading to better clinical control of epileptic seizures. This research may include the basic screening and testing of new therapeutic agents, studies on mechanisms of action, metabolic disposition, pharmacokinetics, and clinical pharmacology studies.

The recipient will be selected by the Epilepsy Award Committee appointed by the President of ASPET, with representation of ILAE. Judgment will be based upon the significance of the candidate's contribution to the treatment of epileptic seizures in human subjects. Selection will be free of restrictions on age, sex, nationality, institutional affiliation, or membership in scientific societies. The nominee must be actively engaged in research for which the Award is made, and primary emphasis will be placed upon work accomplished in the five-year period prior to each Award.

Nominations for the Award may be submitted by members of any recognized scientific association, domestic or foreign. Nominations shall be submitted **electronically** to jane.nelson@aspet.org and shall consist of:

1. A summary describing the nominee's major achievements.
2. Candidate's curriculum vitae and bibliography.
3. Six of the nominee's most significant papers, published or accepted for publication (provided as PDFs or as hyperlinks to the article). Submit each manuscript PDF as a separate attachment.

The biennial award shall be presented to the recipient at the annual meeting of ASPET. Hotel and economy airfare to the annual meeting for the recipient and his/her spouse will be provided.

Receipt date for nominations for the Epilepsy Research Award will be **September 15, 2010** for an award to be presented at Experimental Biology '11 in Washington, DC.

Recipients of the Epilepsy Research Award

1978 Alan Richens
1979 Paolo L. Morselli
1980 Dixon M. Woodbury
1981 James A. Ferrendelli
1982 Ewart A. Swinyard
1983 Arthur Camerman
1984 Phillip C. Jobe
1985 Robert J. DeLorenzo
1986 Robert L. Macdonald
1987 James O. McNamara
1988 Harvey J. Kupferberg
1989 Frank C. Tortella
1990 Robert Naquet
1991 Raymond Dingledine
1992 O. Carter Snead III
1994 Michael A. Rogawski
1995 Karen N. Gale
1997 Marc A. Dichter
1999 Robert Sloviter
2001 Wolfgang Löscher
2003 Brian S. Meldrum
2005 J. Victor Nadler
2007 Robert Schwarcz
2009 Tallie Z. Baram

BENEDICT R. LUCCHESI DISTINGUISHED LECTURESHIP IN CARDIAC PHARMACOLOGY

Deadline for submission of nominations is September 15, 2010

The Benedict R. Lucchesi Award in Cardiac Pharmacology was established to honor Dr. Lucchesi's lifelong scientific contributions to our better understanding and appreciation of pharmacological treatment and prevention of cardiovascular disease and for his mentoring of countless prominent functional (in vivo) cardiovascular pharmacologists.

The Benedict R. Lucchesi Award is a biennial award consisting of an honorarium of $1,000, a custom-designed crystal bowl depicting the named Lectureship, and up to $2,000 travel expenses including registration to the annual spring ASPET meeting. A recipient will be selected and invited to deliver a state-of-the-art lecture on recent advances in the field of cardiac and electropharmacology at the spring ASPET meeting (Division's programming session). The presentation of his/her research should be of broad interest and contribute to the growth of the Cardiovascular Pharmacology Division.

CALL FOR AWARD NOMINATIONS FOR 2011

There are no restrictions on institutional affiliation, nationality, or age of the candidate, but the recipient must be a member of the ASPET. Nominations must be made by a member of the ASPET, and no member may nominate more than one candidate per year. Final selection of the recipient will be made by the Award Committee of the Division for Cardiovascular Pharmacology.

Nominations should consist of not more than five letters from nominators describing the contributions to cardiac and electropharmacology of the candidate that make him/her eligible for this Award and listing of his/her major contributions, together with a complete curriculum vitae. To ensure consideration, all information must be submitted **electronically** to Jane Nelson (jane.nelson@aspet.org) no later than **September 15, 2010**.

Previous Recipients of the Benedict R. Lucchesi Award

2007 Garrett J. Gross
2009 Joan Heller Brown

TORALD SOLLMANN AWARD IN PHARMACOLOGY

Deadline for submission is September 15, 2010

The Torald Sollmann Award in Pharmacology was established to commemorate the pioneer work of Dr. Torald Sollmann in the fields of pharmacological investigation and education. The Torald Sollmann Award is presented biennially in odd years for significant contributions over many years to the advancement and extension of knowledge in the field of pharmacology.

The award consists of an honorarium of $3,500, a medal, hotel and economy airfare for the winner and spouse to the annual meeting. The formal presentation of this biennial award and medal will be made at the annual meeting of ASPET. The recipient will be invited by the President of the Society to deliver a lecture to the membership that may be published in an appropriate ASPET journal.

There are no restrictions on nominees for this award; however, a nomination must be made by a member of the American Society for Pharmacology and Experimental Therapeutics (ASPET). No member may nominate more than one candidate in a year, and no candidate may be nominated for more than one major ASPET award in any given year. The award shall be made on the basis of originality and uniqueness of accomplishments throughout a long career distinguished by sustained, significant contributions to education, research, and service in pharmacology. Selection of the recipient will be made by the ASPET Awards Committee, appointed by the President.

Nominations shall be submitted **electronically** to jane.nelson@aspet.org and shall consist of:

1. No more than five letters from nominators describing the contributions to pharmacology of the candidate that make him/her eligible for this Award, listing major contributions.
2. Brief biographical sketch of the candidate.
3. Candidate's curriculum vitae and bibliography.

Receipt date for nominations for the Torald Sollmann Award will be 5:00 pm on **September 15, 2010** for an award to be presented at Experimental Biology '11 in Washington, DC.

Recipients of the Torald Sollmann Award in Pharmacology

1961 Otto Krayer
1963 Bernard B. Brodie
1966 Arnold D. Welch
1969 Earl W. Sutherland, Jr.
1973 Julius Axelrod
1975 Sidney Udenfriend
1978 Karl H. Beyer, Jr.
1981 Avram Goldstein
1984 K. K. Chen
1986 Walter F. Riker
1988 James A. Bain
1990 George B. Koelle
1992 E. Leong Way
1995 Theodore M. Brody
1997 William W. Fleming
2001 Benedict Lucchesi
2003 Palmer W. Taylor
2005 Kenneth E. Moore
2007 Sue P. Duckles
2009 S. J. Enna

Definitions of Categories of ASPET Membership

Regular Members: Any doctoral level investigator who has conducted and is the primary author on at least one publication of an original study in the area of pharmacology published in a peer-reviewed journal is eligible for membership in ASPET. Exceptions may be made for someone who does not meet the degree requirement but who has made major research contributions to pharmacology. Dues for regular members are $140/year. Regular members must be nominated by one (1) Regular or Retired ASPET member.

Affiliate Members: An investigator who does not meet the requirements for Regular membership because of the lack of a degree or lack of publication is eligible to apply for Affiliate membership. Affiliate members receive all the same member benefits as Regular members except that they may not vote in ASPET elections. Dues for Affiliate members are $105/year. Affiliate members must be nominated by one (1) Regular or Retired ASPET member.

Student Members: Individuals who are enrolled in undergraduate, graduate, or professional degree programs are eligible for Student membership in ASPET. Student members receive all the same benefits as Regular Members except that they may not vote in ASPET elections. Individuals may remain in the Student Member category for up to two (2) years following completion of their research doctoral degree. Undergraduate students pay no dues. Dues for second year and above Student members are $30. Student members must be nominated by one (1) Regular or Affiliate ASPET member.

Sponsors should send an email or letter addressing the applicant's qualifications for ASPET membership directly to the ASPET office (rphipps@aspet.org).

Regular Member Benefits (Dues $140):

- Reduced page charges for corresponding authors to publish in ASPET journals – pay $40/page instead of $80/page and save enough with one four-page article to pay your annual ASPET dues!
- Half-price color fees to publish color figures in ASPET journals.
- Free full-text access to all five online ASPET journals, including all back issues.
- Free subscription to *Molecular Interventions* (print) and *The Pharmacologist (*online).
- Reduced subscription rates for ASPET print journals.
- Reduced registration fees for ASPET meetings.
- Sponsorship of papers at the ASPET meeting.
- Best abstract awards for young scientists at the ASPET meeting.
- Free listing in the FASEB Directory.
- Membership in multiple ASPET Divisions for no additional dues.

Affiliate Members (Dues $105) have all the benefits of Regular Members except they may:

- Sponsor candidates for Student membership only.
- Not sponsor a paper for a non-member at a Society meeting.
- Not vote in Society elections.
- Not hold an elected office in the Society.

Student Members (Dues $30) have all the benefits of Regular Members except they:

- Pay no dues their first year.
- Pay only $30 annual dues thereafter. Undergraduate student members pay no dues and get their first graduate year free.
- Must have their papers at Society meetings sponsored by a member.
- May not vote in Society elections nor hold an elected office in the Society.

2010 Publication Subscription Rates for Members

All Society Members qualify for the following reduced print publication subscription rates:

- *Journal of Pharmacology and Experimental Therapeutics* (Monthly) - $220/year
- *Pharmacological Reviews* (Quarterly) - $89/year
- *Drug Metabolism and Disposition* (Monthly) - $137/year
- *Molecular Pharmacology* (Monthly) - $180/year
- *Molecular Interventions* (Bimonthly) – included with dues

APPLICATION INSTRUCTIONS

Submit the completed Application for Membership form or use the online application form on the ASPET web site at **http://www.aspet.org/membership/apply/.** Submit a current *curriculum vitae* including bibliography for Regular and Affiliate Membership. You may e-mail the CV to the ASPET Membership Coordinator, Robert Phipps, **rphipps@aspet.org**.

Sponsor Statements: Submit a statement of qualifications of the applicant from one Regular/Retired Member of ASPET for Regular Membership, Affiliate Membership and Student Membership (Affiliate Members may also sponsor student applicants). In addition to the statement certifying that the applicant is qualified for ASPET membership, sponsors should provide their own current address, phone, fax, and email. It is the responsibility of the applicant to insure that these documents are submitted to the ASPET office.

American Society for Pharmacology and Experimental Therapeutics
9650 Rockville Pike, Bethesda, MD 20814-3995 USA
Phone: 301-634-7060 ♦ Fax: 301-634-7061 ♦ www.aspet.org

Membership Application – TP0610

Please Complete All Sections:

Section 1: Application Details

Application for:

❑ **Regular Membership**

❑ **Affiliate Membership**

❑ **Graduate Student – Expected Date of Graduation:** _____

❑ **Undergraduate Student - Year:** ❑ **Fr** ❑**Soph** ❑**Jr** ❑**Sr**

Section 2: Source

How did you hear about ASPET:

❑ **Meeting** _____

❑ **ASPET Journal** _____

❑ **Mentor** _____

❑ **Other** _____

Section 3: Personal Information

Name:

Institution:

Address:

Telephone:

Fax:

Email:

Section 4: Optional Demographics (Not Required)

Date of Birth: _____

Sex: ❑ Female ❑ Male

Ethnicity:

❑ Asian

❑ Black or African American

❑ American Indian or Alaskan Native

❑ Hispanic or Latino

❑ Native Hawaiian or Pacific Islander

❑ White

❑ Other: _____

The information in this section will be used by ASPET to collate statistics and will be kept private. Completion of this section is voluntary.

Section 5: Sponsor (Must be an ASPET Member)

Name and email of your sponsor:

Please have your sponsor send us a brief letter or e-mail outlining your qualifications for membership in ASPET to the Membership Coordinator , Robert Phipps, (rphipps@aspet.org).

Section 6: Division Selection

Divisions: *Division membership is a benefit of ASPET membership and there is no additional charge to belong to a division. It is highly recommended that you join a division so that you may take full advantage of Society participation. Joining a division allows you to participate in creating the scientific program for the annual meeting, network with people in your field at mixers and divisional programs, and receive special notices and newsletters about items and activities of interest in your field. Be sure to pick a division!*

Indicate primary (1) and as many secondary (X) divisions to which you wish to belong:

___Division for Behavioral Pharmacology
___Division for Cardiovascular Pharmacology
___Division for Drug Discovery, Development & Regulatory Affairs
___Division for Drug Metabolism
___Division for Integrative Systems, Translational, & Clinical Pharmacology
___Division for Molecular Pharmacology
___ Division for Neuropharmacology
___ Division for Pharmacology Education
___Division for Toxicology

Section 7: Curriculum Vitae

Regular, Affiliate, and Graduate Student applicants: Please send your *Curriculum Vitae* (including bibliography) by email to the Membership Coordinator, Robert Phipps (rphipps@aspet.org).

Undergraduate Student Applicants Only:

Current Education :

Expected Degree & Date: School: City/State/Country: Major Field:

Applications are reviewed on a rolling basis. Please DO NOT send payment with your application.
Upon membership approval, you will be sent a dues statement and welcome package.
Student Membership is FREE for the first year, Regular members pay $140, Affiliate Members pay $105.

Call or e-mail the ASPET Membership Department for additional information: 301-634-7135 / rphipps@aspet.org.

Future Meetings

Mid-Atlantic Pharmacology Society
Annual Meeting
December 10, 2010
Fox Chase Cancer Center
(Philadelphia)

Experimental Biology '11
Washington, DC
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