

*Theme:*

***Environmental Etiologies of Neurological Disorders:  
Scientific, Translational and Policy Implications***

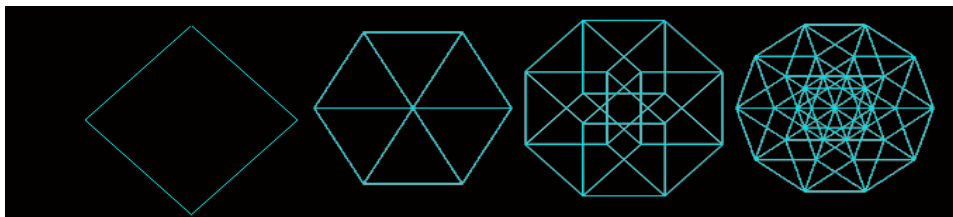
*October 12-16, 2008 in Rochester, New York*

**CONFERENCE PLAN**

**Conference Title, Theme, Dates, Location, and Facilities:** This proposal requests your sponsorship of the *Twenty-Fifth International Neurotoxicology Conference* (NEUROTOX 25) addressing the theme of “Environmental Etiologies of Neurological Disorders: Scientific, Translational and Policy Implications”. NEUROTOX 25 will be held October 12-16, 2008 in Rochester, New York. The meeting locations are the Hyatt Regency Conference Center and the University of Rochester Medical Center (1 day) located nearby. We have negotiated ideal arrangements and greatly reduced room rates for NEUROTOX 25 participants, including additionally reduced rates for students.

**Background on the Conference Series.** The *International Neurotoxicology Conference* is an annual event that focuses on a timely theme while providing an opportunity for presenting new data related to the general interdisciplinary field of neurotoxicology. We place special emphasis on promoting, nurturing and mentoring students/young investigators in the field and employ novel mechanisms to provide networking with experts. The Conference is a free-standing academically-related entity of the Department of Pediatrics in the UAMS College of Medicine founded, organized and convened annually by Professor Joan Cranmer, a UAMS faculty member for 32 years. Most of the time the Conference has convened in either Little Rock or Hot Springs, Arkansas. However it also has convened in Chicago (x2), Colorado Springs (x2), Honolulu, Research Triangle Park, and San Antonio. Each year the conference focuses on a timely theme that is identified on an annual basis by the Editor, Associate Editors and Editorial Board of the international specialty journal *NeuroToxicology* and other members of the Society of Toxicology’s Neurotoxicology Specialty Section.

**Significance, Justification and Rationale for the Theme:** The International Neurotoxicology Conference Series will reach its silver anniversary in 2008, with its 25<sup>th</sup> meeting. The history of this series reflects the expansion of neurotoxicology into domains that had hardly been conceived of in 1982, the date of the first conference. There is no better way to depict its evolution than to show it geometrically.



**The Geometry of Neurotoxicology:** The series began with essentially one topic, as in the first square, which depicts a single dimension. This first conference featured metals—manganese, tin, and lead—still prominent in later meetings, and the question of chlordecone. The next expanded the conference scope by examining a critical endpoint, axonopathies that could be the outcome of many different kinds of exposures, followed by one that embraced a variety of pesticides; the second figure represents the scope of these two meetings. The fourth conference, *Neurotoxicology in the Fetus and Child* (1985), for the first time addressed a topic with multiple dimensions, and might be depicted by the third figure. The scope of each successive conference until the 24<sup>th</sup>, held in 2007, varied from single topics to multiple perspectives on an issue such as children’s development. The 24<sup>th</sup> conference (NEUROTOX 24) represented a large evolutionary step. It encompassed not just the science of neurotoxicology, but its societal implications, ranging from how it affected (and reflected) social order, how it might move health and environmental policy, and how it might influence clinical practice. The fourth figure depicts its dimensional expansion. NEUROTOX 25 continues this evolution. It will take up issues that, in earlier conferences, were treated, if not in isolation, at least as principals in their own domains. NEUROTOX 25 will be looking, not just at the multiple surfaces in the fourth figure, but at their intersections, which are multiples of surfaces. Science, policy, and translation can no longer be treated as independent entities. NEUROTOX 25 is poised to view them as they truly are: a problem in multidimensional geometry.

**Overall Objective.** The overall objective of this conference is to bring together experts from academia, governmental organizations, non-governmental organizations, and industry, from around the world, to discuss the state-of-the-science of what is known, identify information gaps, and help define future directions for research to advance our fundamental knowledge of the causes, mechanisms, diagnosis, related new technologies, treatment

and prevention of environmentally-induced or exacerbated diseases and disorders of the nervous system in children and adults. This long-standing conference series has a strong history of convening internationally-recognized experts, neuroscientists, research clinicians, basic scientists, epidemiologists, regulatory scientists and other informed and interested individuals to provide data and information to draw upon and apply to important scientific, environmental, clinical, treatment, methodological, epidemiological, policy, public health, regulatory, risk assessment and other contemporary issues related to the theme. Our overall goal is to accomplish this in 2008 and disseminate the results to the scientific community and health related professions.

**Specific Aims.** The purpose of NEUROTOX 25 is to:

**Aim 1.** Convene the identified international experts and other established and new researchers anywhere in the world to present, discuss and evaluate the most recent research data and information related to causes, mechanisms, diagnosis, treatment, and prevention of environmentally-linked nervous system diseases and disorders.

**Aim 2.** Encourage and enhance communication, understanding and collaboration between practicing clinicians, basic scientists, clinical researchers, epidemiologists, special interest groups, risk assessors and others working to further our understanding of all aspects of neurological diseases/disorders.

**Aim 3.** Organize the conference participants, tutorials, invited and free presentations, discussions, debates, demonstrations and interactions to document major new research advances, facilitate identification of specific research gaps, formulate hypotheses and potential experimental designs to answer questions.

**Aim 4.** Facilitate interdisciplinary collaboration of practitioners and scientist/investigators to help foster or initiate new multidisciplinary research approaches necessary to advance the science related to the theme.

**Aim 5.** Include representation from academia, government, industry, foundations and private organizations on the program as organizers, session chairs, invited speakers, poster presenters, discussion facilitators or participants.

**Aim 6.** Rapidly peer-review and disseminate findings to the scientific community. All presenters are invited to submit original research papers of new data presented as well as Reviews or Mini-Reviews of recent findings related to the specific topics. Guest Editors will be selected from the Session Chairs and Invited Experts participating in the conference. Papers will be peer-reviewed and accepted papers will be published in less than one year in *NeuroToxicology*, an international scientific journal that is widely disseminated, indexed, abstracted and available online. This Specific Aim has been accomplished for each of the previous 24 conferences in this series (see Appendix 5 "Report of Publications").

**Conference Methods, Format to Achieve Aims:** The Specific Aims identified above will be accomplished by organizing and conducting a 4 ½ day conference according to a varied format similar to previous conferences in this series which has proven to be highly successful. Each conference has its own "special touch" and consists of Tutorials, Symposia, Workshops, Panel Discussions, Roundtables, Debates, Poster Session, Pre- and Postdoctoral Student Competition and Awards and social events as well as novel approaches to facilitate networking and mentoring. NEUROTOX 25 will not be simply an occasion to present data. It will also coalesce human, experimental animal, methodological, and epidemiological research, as well as risk assessment, social and policy implications, by offering a well-recognized venue where previously unconnected investigators can come together and where commonalties can emerge. To promote camaraderie there will be a Welcoming Reception and Buffet Sunday evening, finger foods, dessert bar, refreshments and cash bar during the evening Poster Session, and a Conference Dinner/Awards Banquet and *Festschrift* for Bernard Weiss.

**Mentoring of Students/Traditional Sessions Not Related to the Theme.** In addition to the Sessions devoted to the theme there will be a General Poster Session, platform presentations to be selected from submitted abstracts (by a sub-group of the Program Committee) as well as Social events for networking and camaraderie. As always there will be a pre- and postdoctoral Student Award Competition. Traditionally the Neurotoxicology Conference places special emphasis on nurturing, recognizing and rewarding pre- and post-doctoral students and young investigators. Extra effort is extended by the Organizers and all participants to mentor students and help them network with the leaders in the field. As always, there will be cash, plaque and certificate awards for 1<sup>st</sup>, 2<sup>nd</sup>, and 3<sup>rd</sup> place competition in both the pre- and postdoctoral student award competition. (See Appendix 4 "Report of Student Awardees").

**NEUROTOX 25 will consist of 12 sessions as shown on the Overview (Appendix 2) and the Detailed Draft Program that follows:**

**NEUROTOX 25**

# *Environmental Etiologies of Neurological Disorders: Scientific, Translational and Policy Implications*

October 12 - 16, 2008

Hyatt Regency and University of Rochester

Rochester, New York

Sunday Afternoon 12 October 2008 1:30 PM – 3:00 PM

Grand Ballroom, Hyatt Regency Rochester

Opening

## **Session I-A: OPENING, WELCOME AND OVERVIEW OF CONFERENCE**

Conference Chair: Joan Cranmer, PhD

Conference Co-Chair: Deborah Cory-Slechta, PhD

- Opening
- Introductions and Acknowledgements
- Welcome ~ Dean, University of Rochester Medical Center
- A Word From Our Sponsors (Conference Expectations of Major Sponsors)

Plenary Session

## **Session I-B: HISTORICAL HIGHLIGHTS: "LIGHT" LESSONS FROM LUMINARIES**

A First Person account by luminaries in the field of their historical contributions to the interdisciplinary science of neurotoxicology. Luminaries will be introduced by their students – who are also luminaries in the field – just younger!

- Thomas Clarkson – *Introduced by Michael Aschner*
- Toshio Narahashi – *Introduced by William Atchison*
- Herb Needleman – *Introduced by David Bellinger*
- Bernard Weiss – *Introduced by Deborah Rice*

Sunday Afternoon 12 October 2008 3:15 PM – 5:30 PM

Opening Plenary Session

## **Session II: CRITICAL ISSUES IN NEUROTOXICITY RESEARCH.**

### **HOT NEW TOPICS**

Sunday Evening 12 October 2008 5:30 PM – 9:30 PM

*Hosted Welcoming Reception, Buffet and 25<sup>th</sup> Anniversary Party*

Monday Morning 13 October 2008 8:30 AM – 12:00 PM

Symposium

## **Session III: ACCELERATED AGING ASSOCIATED WITH EARLY ENVIRONMENTAL EXPOSURES**

Session Chair: M. Christopher Newland, Auburn University

Co-Chair: TBA

**Theme and Rationale:** The idea that the consequences of neurotoxicant exposure not appear until much later in life has been with neurotoxicology since its inception. When the theory of silent damage was first articulated by Bernard Weiss and William Simon in the first text on Behavioral Toxicology, it was only a proposal. Empirical support was provided by the accidental discovery that a meperidine contaminant, MPTP, causes specific lesions to nigral dopamine neurons and hastens the onset of Parkinson's Disease. In recent years we have come to recognize that neurotoxicant exposure during early adulthood or even during early development increases the vulnerability of the nervous system to aging. This symposium is

designed to bring together investigators whose research is aimed at linking the diseases of aging to environmental exposures that ends well before the first signs appear. The symposium is envisioned to draw from human studies and animal models. Proposed topics and speakers will be selected from the following:

### **Introduction and Session Overview**

Chris Newland, *Auburn University*

### **Early Lead Exposure and Risk of Dementia**

Brian Schwartz, *Johns Hopkins University (invited)*

He's identified relationships between markers of dementia in older people and markers of early lead exposure.

### **Methylmercury and Aging in Animal Models**

Chris Newland, *Auburn University*

He has published work on gestational exposure to methylmercury and aging in a rodent model.

### **Parkinson's Disease and Occupational Exposure to PCBs.**

Rich Seegal, *Wadsworth Center, N.Y. Dept. of Health*

He's been tracking PCB workers coming down with Parkinson's Disease.

### **Economics of Aging and Environmental Exposures.**

Jessica Reyes, *Amherst College*

An economist who has tracked the removal of lead and crime. She is developing an interest in aging.

### **Methylmercury and Aging in the Minamata Cohort.**

Mineshi Sakamoto, *Minamata Institute*

He has been involved in aging and MeHg exposure. Sakamoto has been tracking the Minamata cohort.

### **Ca++ mediated Aging and Potential Relationships to Aging. Late breaking research related to ALS.**

William Atchison, *Michigan State University*

### **Late breaking research (to be chosen from poster presentations)**

### **Panel Discussion**

**Monday Afternoon 13 October 2008 1:00 PM – 3:15 PM**

*Platform Session*

### **Session IV: THEME TBA AFTER ABSTRACTS RECEIVED.**

### **Papers Selected from Abstracts**

**Monday Afternoon 13 October 2008 3:15 PM – 5:00 PM**

*Roundtable/Debate (Tentative. Being planned)*

### **Session V: PARAQUAT AS A CONTROVERSIAL ANIMAL MODEL FOR PARKINSON'S DISEASE: A ROUNDTABLE DEBATE (THIS SESSION IN PLANNING STAGES) (PROPOSED SPEAKERS)**

*Session Co-Chairs:* **Richard Seegal and Pierluigi Nicotera**

**Theme and Rationale:** The use of experimental animal models of human diseases often raises important issues that can lead to spirited debate. How closely should a "good" animal model mimic features of a disease? How reasonable is it to extrapolate from animal models to humans? How should we interpret data (often conflicting) from different animal models of the same disease? A recent example of a controversial animal model is the administration of paraquat (alone or in combination with other pesticides) to rodents as a tool for reproducing features of Parkinson's disease (PD). Paraquat kills nigrostriatal dopaminergic neurons (the same neuronal

population highly vulnerable to the neurodegenerative process of PD), but its mechanism of action appears to differ from that of other PD-relevant toxicants. Exposure of experimental animals to paraquat reproduces some (e.g. aggregation of the protein alpha-synuclein) but not other (e.g. severe dopamine depletion in the striatum) features typical of PD. It is likely that paraquat neurotoxicity involves oxidative damage (oxidative injury has also been suggested to play a role in PD pathogenesis); however, other critical events leading to paraquat-induced neurodegeneration remain relatively undefined (e.g. how does paraquat enter neuronal cells?). How should these uncertainties be interpreted? Do they actually detract from the model or do they warrant a more concerted research effort to elucidate the toxic properties of paraquat?

The debate about the paraquat PD model is even more relevant considering its potential public health implications. If paraquat kills dopaminergic neurons in *in vivo* animal models, is this a “red flag” for human exposure, given that paraquat is still extensively used as an herbicide? How relevant is the neurotoxicity observed following administration of paraquat by an acute, high-dose regimen to human exposure and to the development of a late-life, chronic disease such as PD? How important are data from human population studies (i.e. epidemiological investigations) to corroborate or confute the view of paraquat as a PD risk factor? This roundtable, involving basic researchers, epidemiologists, industry representatives and policy makers, will attempt to address these questions, which are not unique to the paraquat model but have widespread relevance to experimental research on human diseases.

#### **Proposed Participants:**

Deborah Cory-Slechta, Donato DiMonte, Abby Li, Ted Locke, Richard LoPachin, Gary Miller, Richard Seegal, Perluigi Nicotera

#### **5:00 – 7:00 PM Break for dinner**

Monday Evening 13 October 2008 7:00 PM – 9:30 PM

*Heavy Hors d'œuvres & Dessert Bar, No-Host Bar*

### **Session VI: POSTER SESSION**

*Session Chairs: TBA*

The Poster Session is a highlight of this conference series. It has proven to be an effective venue for informal, in-depth discussion and collaboration building -- as well as an important networking opportunity for all participants. Papers on any aspect of neuroscience, toxicology, children's environmental health, public health & policy are welcome! Posters may be put up as early as 9:00 AM on Sunday and should remain up for the conference duration for maximum exposure.

### **STUDENT AWARD COMPETITION**

Chairs and Committee: **TBA**

The Student Award Competition is divided into two groups: one for post-doctoral and one for pre-doctoral competition. Awards will consist of a cash prize, plaque or certificate, plus a one year subscription to the international journal *NeuroToxicology*. A winner(s) will be chosen from each group. Competing students are asked to give an overview of their work in 2-3 minutes to the judges followed by a brief set of questions and answers. Originality, significance, hypothesis, presentation material and style, as well as knowledge of the subject, will be considered in selecting the winners. All papers in competition for the Student Awards must be presented from poster. Judging will be done during this time.

Tuesday Morning 14 October 2008 8:30 AM – 12:00 Noon

*Concurrent Session*

### **Session VII-A: MODELS OF NEUROPROTECTANT SCREENING FOR CHEMICALLY-INDUCED NEURODEGENERATION**

Session Chair: **David Jett, NINDS**

*This session is in the planning stages. Speakers to be confirmed.*

### **Introduction and overview of Session**

David Jett, NINDS

### **Age and Sex Effects on Nerve Agent Damage to the Brain and Antidotal Therapies**

Edson X. Albuquerque, *University of Maryland School of Medicine*

### **Efficacy of GluR5 Antagonists Against Soman-Induced Seizures and Neuropathology**

Maria F.M. Braga, *Uniformed Services University of the Health Sciences*

### **Neuroprotective Roles for Neuregulins in Neurotoxin-Mediated Neuronal Injury**

Byron D. Ford, *Morehouse School of Medicine*

### **Neuroprotective Screens for Nerve Agents Neurodegeneration**

John McDonough, *US Army Institute of Chemical Defense*

### **An *in vitro* Slice Model for Screening Neuroprotectants**

Kathy Wilcox, *University of Utah*

### **Panel Discussion**

**Tuesday Morning 14 October 2008 8:30 AM – 12:00 Noon**

*Concurrent Sessions - Platform Session*

**Session VII-B: THEME TBA AFTER ABSTRACTS RECEIVED.**

**Papers Selected from Abstracts**

**Tuesday Afternoon 14 October 2008 1:00 PM – 5:00 PM**

*Concurrent Sessions – Platform Session*

**Session VIII-A: THEME TBA AFTER ABSTRACTS RECEIVED.**

**Papers Selected from Abstracts**

**Tuesday Afternoon 14 October 2008 1:00 PM – 5:00 PM**

*Concurrent Sessions - Symposium*

**Session VIII-B: GENETIC BASIS OF BEHAVIORAL NEUROTOXICOLOGY**

Session Co-Chairs: **Annette Kirshner, NIEHS**  
**Edward Levin, Duke University**

**Theme and Rationale:** Considerable progress has been made over the past couple of decades concerning the molecular bases of neurobehavioral function and dysfunction. The field of neurobehavioral genetics is becoming mature. Genetic factors contributing to neurologic diseases such as Alzheimer's have been found and evidence for genetic factors contributing to other diseases such as schizophrenia and autism are likely. This genetic approach can also benefit the field of neurobehavioral toxicology. It is clear that there is substantial heterogeneity of response to the behavioral impairments of neurotoxicants. Many factors contribute to differential sensitivity but it is likely that genetic variability plays a prominent role. Important discoveries concerning genetics and behavioral neurotoxicity are being made on a broad front from work with invertebrate mutant models to classic mouse knockout models and human epidemiologic studies of polymorphisms. Discovering genetic factors of

susceptibility to neurobehavioral toxicity not only helps identify those at special risk, it also advances our understanding of the mechanisms by which toxicants impair neurobehavioral function in the larger population. This symposium brings together researchers from invertebrate, rodent and human studies to determine the role of genetic factors in susceptibility to behavioral impairment from neurotoxic exposure.

## Introduction and Overview

Annette G. Kirshner

## C. Elegans Models

Michael Aschner, *Vanderbilt University*

## Drosophila Models

Ulrike Heberlein, *University of California San Francisco*

## Mouse and Zebrafish Models

Edward D. Levin, *Duke University*

## Humans

Kathleen A. Welsh-Bohmer, *Duke University*

## Panel Discussion

**Tuesday Evening 14 October 2008 7:00 PM – 9:00 PM**

*Free Evening*

**Wednesday Morning 15 October 2008 8:00 AM – 11:45 AM**

8:00 AM Buses to URMC

8:30 AM – 12:00 Noon

*The Rochester Conference ~ Festschrift for Dr. Bernard Weiss*

## **Session IX: CRITICAL ISSUES IN NEUROTOXICITY RESEARCH**

Recognizing the Critical Contributions of URMC to Neurotoxicology: Festschrift for Dr. Bernard Weiss

*Organizers:*           **Deborah Cory-Slechta**, Chair  
                              **Philip W. Davidson**, Co-Chair  
                              **Lisa Opanashuk**  
                              **Kim Tieu**

**Theme and Rationale:** The Program on Wednesday is sponsored by the University of Rochester Medical School. It is ideally integrated into the overall conference program.

On October 15<sup>th</sup>, 2008, the University Of Rochester Department Of Environmental Medicine will sponsor and host the 21<sup>st</sup> Rochester Conference on Environmental Toxicity in conjunction with NEUROTOX 25. The 2008 Rochester Conference will focus on highlighting the *State of the Science* in the field of neurotoxic exposures and diseases and disorders of the nervous system. Many of the critical issues facing the field today had their origins in research conducted in Rochester. The meeting will showcase the many roles played by Rochester colleagues whose work has had a major impact on the field of neurotoxicology (See working draft program attached). The *Rochester Conference* series started in 1968. Each symposium involves 15 to 20 distinguished scientists in the field of environmental toxicology and typically results in a major publication which this year will be combined with NEUROTOX 25. Historically, publication of these proceedings has had a substantial impact on ongoing research, research funding, and policy making. All papers are peer-reviewed.

This *Rochester Conference* will be dedicated as a *Festschrift* for Dr. Bernard Weiss, who, for the past half century, has been a seminal force within the behavioral and developmental neurotoxicology field. Much of his career has been spent at the University of Rochester. The *Festschrift* will take place in conjunction with the 25<sup>th</sup> Annual International Neurotoxicology Meeting, which is being held in Rochester on October 12<sup>th</sup> through 16<sup>th</sup>. About 200 attendees are expected. The *Festschrift* is still being planned. It will focus on areas of research stimulated by Bernie Weiss. The Department plans to invite about 20 of Bernie's present and former students, fellows, and colleagues as presenters and discussants. In addition to the scientific

meeting, the *Festschrift* will culminate in a reception and dinner honoring Bernie on his home ground. Funding for the *Festschrift* will be borne by the University of Rochester.

8:30 - 9:00 AM

**The Neurotoxicology Research Contributions from URM**

Joan Cranmer, *University of Arkansas for Medical Sciences*

Themes for the Day and Proposed Speakers:

**Session IX-A: DEVELOPMENT AND METALS/LEAD EXPOSURE**

*Session Chair:* Deborah Cory-Slechta, *University of Rochester Medical Center*

9:00 - 9:25 AM

**Retrospective on Lead: Then and Now**

Herbert Needleman, *University of Pittsburgh*

9:25 - 9:50 AM

**Synergistic Consequences of Combined Lead and Maternal Stress in Experimental Models**

Deborah Cory-Slechta, *University of Rochester Medical Center*

9:50 - 10:15 AM

**Combined Effects of Lead and Stress in Children: Translational Studies**

Brooks Gump, *SUNY Oswego*

10:15 - 10:30 AM

**Discussion of Papers**

10:30 -10:45 Coffee Break

**Session IX-B: NEURODEVELOPMENT AND NEURODEGENERATIVE DISEASES AND AIR POLLUTION**

*Session Chair:* Deborah Cory-Slechta, *University of Rochester Medical Center*

Colleagues from Pulmonary/Cardiovascular Core, *University of Rochester Medical Center*

10:45 - 11:10 AM

**Translocation of Ultrafine Manganese to Brain**

Allison Elder, *University of Rochester Medical Center*

11:10 - 11:35 AM

**Pediatric Effects of Chronic Air Pollution**

Lilian Calderon-Garciduerias, *Instituto Nacional de Pediatría, Mexico*

11:35 - 11:45 AM

**Discussion of Papers**

**11:45 AM – 1:15 PM** Hosted Lunch at University of Rochester Medical Center

**Introduction to Bernie Weiss's Contributions to Neurotoxicology**

Thomas Clarkson, *University of Rochester Medical Center*

**Wednesday Afternoon 15 October 2008 1:15 PM – 4:00 PM**

*The Rochester Conference ~ Continued*

**Session X-A: MERCURY**

Session Chair: **Philip W. Davidson**, *University of Rochester Medical Center*

1:15 - 1:40 PM

**Session 1 Potential Benefits of Maternal Fish Consumption to Fetal and Child Development**

Sean Strain, *University of Ulster, Northern Ireland*

1:40 - 2:05 PM

**Session 2 Distribution of Hg in Rat Brain Following Prenatal Exposure to Two Species of Hg**

Hiomi Ishitobi, *University of Rochester Medical Center*

2:05 - 2:30 PM

**Session 3 New Data on Combined Prenatal Exposures to MeHg and Hg vapor from Dental Amalgams in the Seychelles Child Development Study**

Gene Watson, *University of Rochester Medical Center*

2:30 - 2:45 PM

**Discussion of Papers**

2:45 - 3:00 PM Coffee Break

**Session X-B: ENVIRONMENTAL CHEMICALS AND THE PARKINSON'S DISEASE PHENOTYPE**

Session Chair: **Lisa Opanashuk**, *University of Rochester Medical Center*

Co-Chair: **Kim Tieu**, *University of Rochester Medical Center*

3:00 - 3:25 PM

**Astrocyte-Neuron Interactions in a Parkinson's Disease Model**

Kim Tieu, *University of Rochester Medical Center*

3:25 - 3:50 PM

**Developmental Pesticide Parkinson's Disease Model**

Mona Thiruchelvam, *EOSHI & Robert Wood Johnson Medical School, UMDNJ*

3:50 - 4:15 PM

**PCBs and Dopaminergic System Dysfunction**

Lisa Opanashuk, *University of Rochester Medical Center*

4:15-4:30 PM

**Discussion of Papers**

4:30 - 5:15 PM

**The First 80 Years and the Next 80 Years**

Wrap Up Talk by Person of Honor Bernard Weiss

5:30 PM Buses from URMC to Hyatt Hotel

6:00 - 7:00 PM Free Time

**Wednesday Evening 15 October 2008 7:00 PM - 10:00 PM**

**Hyatt Regency Ballroom**

**Festschrift Dinner honoring Bernard Weiss**

**Student Awards Presentations.**

**Testimonials of colleagues at various points through the dinner/evening**

Symposium

**Session XI: ANIMAL MODELS OF AUTISM SPECTRUM DISORDERS: INFORMATION FOR NEUROTOXICOLOGISTS**

Session Chair: **Isaac Pessah**, *University of California, Davis*

Co-Chair: **Pamela Lein**, *Oregon Health & Science University*

**Theme and Rationale:** Recent findings derived from large scale datasets and biobanks link multiple genes to autism spectrum disorders (ASD). Consequently, novel rodent mutants with deletions, truncations and in some cases, overexpression of these candidate ASD genes have been developed and studied both behaviorally and biologically. The aim of this symposium is to present up to date information regarding the genetics of autism and provide a background on mutant mice being tested as animal models. In addition, research involving other approaches (*in vitro*, *Drosophila*, *C. elegans*) will be presented to identify potential opportunities beyond vertebrate genetic mutant models. Finally, a discussion following the symposium will explore the opportunities and challenges of using these transgenic mouse models and other model systems to explore gene x environment interactions. propose a daylong symposium with 3-4 presentations of approximately 45 minutes each with a 30 minute break in the morning to introduce the general topic; 3 presentation of approximately 30 minutes each in the afternoon with a 30 minute break concluding with an hour long panel discussion with subsequent audience participation to address directed questions regarding the development of animal models for studying gene x environment interactions in ASD.

8:30 - 8:35 AM

**Session Overview**

Isaac Pessah, *University of California, Davis*

8:35 - 9:05 AM

**The Clinical Heterogeneity of Autism Spectrum Disorders**

Susan Hyman, *University of Rochester*

9:05 - 9:35 AM

**Overview and Description of Relevant Transgenic Mouse Models**

Isaac Pessah, *University of California, Davis*

9:35 - 10:05 AM

**The Relevance of Studying Social Behaviors in a Mouse to ASD**

Valerie Bolivar, *Wadsworth Center*

10:05 - 10:35 AM

Break

10:35 - 11:05 AM

**Strategies and Projects for Creating Mutant Mouse Models**

David Threadgill, *University of North Carolina*

11:05 - 11:20 AM

**Late breaking research (to be chosen from posters)**

11:20 AM - 12:00 pm

**Discussion**

**Lunch Break**

1:30 - 1:35 PM

**Session overview**

Pam Lein, *Oregon Health & Science University*

1:35 - 2:05 pm

**Primate Models of Environmental Exposures and How They Relate to Autism**

David Amaral, *University of California, Davis*

2:05 - 2:35 pm

**Using *In Vitro* Models to Study Gene X Environment Interactions in Autism**

Pam Lein, *Oregon Health & Science University*

2:35 - 3:00 pm

Break

3:00 - 3:30 pm

**Interactions Between Reelin Haploinsufficiency, Hormonal Environment, and Exposure to Pesticides in the Heterozygous Reeler Mouse**

Flavio Keller, *Università Campus Bio-Medico di Roma, Italy*

3:30 - 4:00 pm

***Drosophila Melanogaster* as a Neurogenetic Model System for Drug Screening**

Linda Restifo, *University of Arizona*

4:00 - 4:15 pm

**Late breaking research (to be chosen from poster presentations)**

4:15 - 4:45pm

**Panel Discussion**

Michael Aschner, *Vanderbilt University*, Discussion Leader

Panel members will include all Session presenters

*Panel Discussion and Objectives*

1. What is the “state of the science” with regards to animal/in vitro models of neurodevelopmental disorders?
2. What are the challenges to identifying the appropriate behavioral and neurobiological and neuropathological outcomes?
3. How can alternative models (in vitro, drosophila) be utilized for better screening approaches
4. What are the most promising models to be used, and what is the best strategy for utilizing these models to study gene x environment interactions?
5. How can modifiers of environmental toxicity be added to the equation, and is the field ready for an additional layer of complexity?

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***Closing of the Conference***

**Friday:**

**Post Conference Wine Tour of Finger Lakes Region**

***To Be Scheduled:***

**CNS Effects of Arsenic**

Veronica Rodriguez, *Queretaro, Mexico*

**Manganese Transport and Neurotoxicity**

Michael Aschner, *Vanderbilt University Medical Center*