

ISNV

International Society for NeuroVirology

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Associate Editor: Michael Nonnemacher, Ph.D.

Assistant Editor: Leslie Marshall, Ph.D.

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11th International Symposium on NeuroVirology

David Volsky

On behalf of the International Society for NeuroVirology and our local Meeting Organizing Committee, it is my pleasure to invite you to the combined 11th International Symposium on NeuroVirology and the 2012 Conference on HIV in the Nervous System which will be held from May 29th through June 2nd at the newly renovated NY Grand Hyatt, at 42nd Street and Grand Central Station, New York, NY, USA.

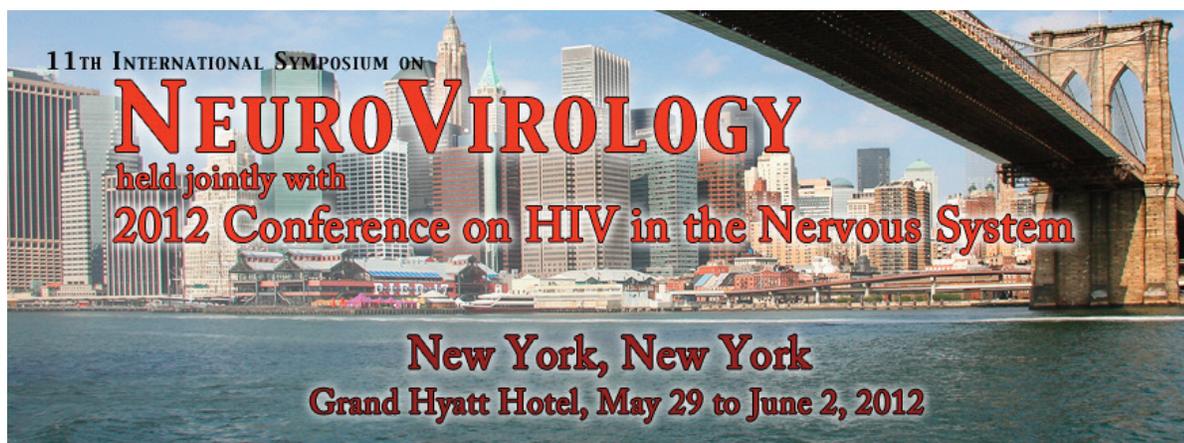
We anticipate participation of more than 350 basic scientists, clinicians and junior investigators working in areas of neurology, neuropathology, neuropathogenesis and molecular virology. In addition, NIMH will hold a Global NeuroAIDS Round Table on Tuesday, May, 29th, from 1 – 5 pm.

As is our tradition, the Pioneer Award in NeuroVirology will be presented during the Gala Dinner, to be held in the hotel on Friday evening.

We have been fortunate to attract leading experts from around the world to present their cutting-edge research and provide overall perspectives on the molecular mechanisms of viral diseases and advancements in treatment and prevention.

While visiting the big apple, I invite you to experience everything that the “city that never sleeps” has to offer. Take a stroll in Central Park, go shopping on Fifth Avenue, take in a Broadway show, visit one of the many museums and experience some of the best dining the world has to offer!

I look forward to seeing you soon in New York City!



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Science News

Feathers, Ferrets & Flu

Dianne Langford



A virologist has made it onto TIME magazine's list for the "100 Most Influential People in the World" (TIME, April 30, 2012, page 77). In September 2011, Ron Fouchier, Ph.D. from Erasmus Medical Center, Netherlands presented data at the 4th European Scientific Working group on Influenza (ESWI) conference describing experiments in his laboratory that had successfully engineered an H5N1 variant capable of infecting and being transmitted in mammals. Studies from another laboratory led by Yoshihiro Kawaoka, Ph.D., D.V.M., from the University of Wisconsin and the University of Tokyo also identified mutations that would allow H5N1 to spread readily among mammals. Both studies were scheduled to be published in December, Fouchier's in *Science* and Kawaoka's in *Nature*. However, neither the details of experimental design nor the findings from the studies have been made public. On the other hand, the global debate surrounding whether or not publication of these findings could provide would-be terrorists with information needed to recreate an H5N1 capable of spreading among mammals has resulted in dozens of commentaries and editorials.

In December 2011, the U.S. government's National Science Advisory Board for Biosecurity (NSABB) recommended delaying dissemination of details describing the mutations that had rendered H5N1 transmissible by mammals. In February 2012, a special panel consisting mainly of influenza experts met with Fouchier and Kawaoka at the World Health Organization (WHO) in Geneva and they concluded that it was neither practical nor scientifically

reasonable to withhold findings.

In an interview with *ScienceInsider* in November 2011, Fouchier described the lab-derived virus as "probably one of the most dangerous viruses you can make". *Science* published a March 9th update in the News and Analysis section where Fouchier cited new data showing that the virus wasn't as "transmissible" as he once believed (Cohen, J., *Science*, 335: 1155-1156, 2012). He described experiments using a tried-and-true method for determining transmissible infection whereby the noses of four ferrets were swabbed with the mutant virus. Ferrets in adjacent cages became ill. The virus was re-isolated from the ferrets that "caught" the flu from their neighbors. This virus was then used to infect other ferrets via nasal swabbing. Their neighbors also came down with the flu. Importantly, both Fouchier and Kawaoka point out that the ferrets didn't die. They just got sick. According to WHO, since its emergence in 1997 as a human infectious pathogen, the bird flu (H5N1) has infected 577 people in 15 countries and 340 of those people died (Kuehn, B., *JAMA*, 307: 1009, 2012 and Pavai, A., *Ann. Intern. Med.*, 156: 463-465, 2012).

Fouchier addressed issues surrounding the controversy in *Journal of Infectious Diseases* in March (*JID* March 27, 2012, PMID: 22454474) where he describes the launching of a worldwide debate surrounding the possibility of release of the mutant virus, whether accidental or intentional. Questions at the core of the debate address issues carrying important implications for the scientific community: Should scientists be allowed to conduct this type of research? And if so, are we taking appropriate precautions against accidental release? Moreover, should we worry about providing bioterrorists with the recipe for the mutated virus?

Among the key issues cited by the NSABB leading to the exhaustive six month debate were 1) misunderstandings due to the shortened format of papers published in *Nature* and *Science* that may have sensationalized the findings and precluded detailed descriptions of biosafety regulations, 2) failure to emphasize the use of sequences for epidemiological surveillance, 3) unrealistic views regarding sharing the information with the entire scientific community, and 4) insufficient regulations for the oversight of such research. At the end of the day, the NSABB decided that the research should be published in its entirety. This decision, although slow to be reached, will certainly serve as a precedent on which many other cases of research with security aspects will be argued for years to come.

ISNV congratulates Georgette Kanmogne

Dianne Langford



Please join the ISNV in congratulating Dr. Georgette Kanmogne on receiving the University of Nebraska Medical Center (UNMC) 2011 Distinguished Scientist Award. Georgette's research focuses on HIV-induced vascular dysfunction, neuroAIDS and viral genetic diversity (with a focus on sub-Saharan Africa). Dr. Kanmogne, Associate Professor and Vice Chair for

Faculty Development and Resources Allocation in the Department of Pharmacology and Experimental Neuroscience, was recognized on May 22, 2012 in a ceremony honoring 23 scientists for outstanding research achievements and sustained contributions to UNMC's research mission.

A Note from NIDA

Diane Lawrence

The National Institute on Drug Abuse (NIDA) is pleased to participate in this year's ISNV conference. It is estimated that 3 million injection drug users (IDU) worldwide are living with HIV infection—nearly 10% of the total estimated number of people living with HIV. In addition, HIV prevalence among non-injecting drug users has grown to similar levels as IDU, possibly due to high risk sexual behavior associated with use of methamphetamine, amyl nitrates, and cocaine, as well as sexual transmission from IDU to non-IDU drug users. Multiple studies have shown that the prevalence of cigarette smoking among persons living with HIV infection is 2- to 3-fold higher than that of the general population, and a recent study of HIV+ adolescents reported that a majority of study participants used alcohol, marijuana, and tobacco/cigarettes. The National HIV Behavioral Surveillance (NHBS) reports 38% prevalence of marijuana use among men who have sex with men, currently one of the highest HIV risk groups in the U.S. and many other countries. Polydrug use is also common in HIV+ populations. Therefore, it is crucial to understand any potential impact of substance use on HIV prevention, treatment, and pathogenesis, including HIV-associated neurological and neurocognitive complications (HAND).

Given the lack of clinical success thus far in developing treatments to combat HAND, there is a clear need to understand the neurobiological basis between HIV infection of the brain and neurocognitive impairment, as well as the basis for resistance to impairment. Given that a major site of action of substance abuse is the brain and specific neurotransmitter systems, it is crucial that substance use be considered in the understanding of HIV neuropathogenesis. Chronic substance use is associated with a variety of neurocognitive impairments, and

basic research using both in vitro and in vivo approaches, as well as neuroimaging studies and clinical case reports, have begun to define ways in which substance use may exacerbate or even protect from neuronal damage induced by HIV. The effects of substance use on immune function, chronic immune activation, response to antiretroviral treatment, and HIV latency are also of interest with respect to both immunopathogenesis and neurological complications. As individuals are living longer with HIV while taking antiretroviral medications, it is important to study how a history of drugs of abuse affects the molecular and cellular mechanisms with low level, chronic viral expression in the brain along with the effects of ART on neural cells to further our understanding of HIV-associated neurodegeneration. In addition, further clarification of the neurobiological link between these molecular, cellular, and inflammatory effects to specific behavioral and cognitive impairments, both in humans and in model systems, would be an important next step in this research area.

NIDA has a number of potential funding opportunities now and in the near future that can support research on substance use and HIV. Our priorities are broad, but recent initiatives on systems biology, viral and host genetics, epigenetics, and proteomics are indicative of some of our highest priorities in basic research. NIDA also can provide guidance on incorporating substance use assessment into human subjects research. ISNV members are encouraged to talk with NIDA program staff regarding possible research ideas, as well as potential collaborators in the substance use field. Watch for new programs and opportunities on our website, www.drugabuse.gov/AIDS.

An update from the Women in Neuroscience (WIN) Committee

Lynn Pulliam

The Women in Neuroscience Committee will sponsor a special Networking cocktail reception at the upcoming 11th International Symposium on the evening of Thursday May 30, 2012. This year's event will include a special panel discussion featuring panelists from different career paths in NeuroVirology. The panelists will have representation from NIH, industry, and

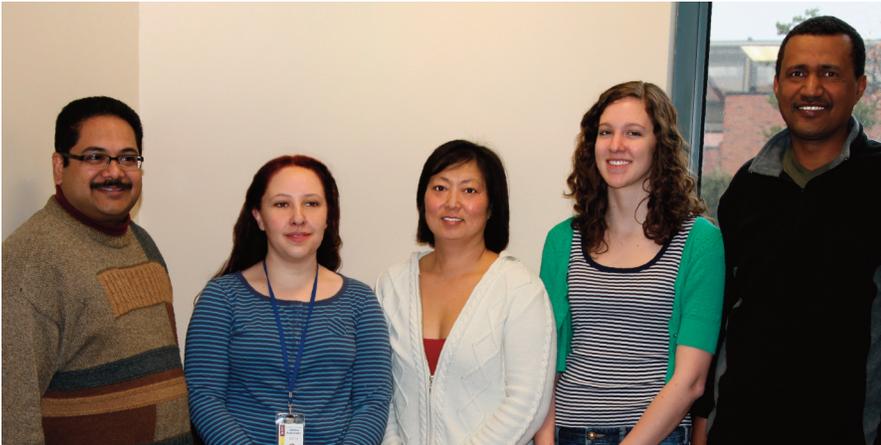
scientists and clinicians from academia. Dr. Joan Berman, our WIN Lectureship recipient this year, will moderate the discussion. We anticipate a very informative and enjoyable evening that will provide an excellent opportunity for junior and senior investigators to network.

Women in Neuroscience (WIN) Committee

Joan Berman (Chair)
Shilpa Buch
Jennifer Gordon
Ruth Brack-Werner
Lynn Pulliam

ISNV Highlights – Maxim Cheeran, D.V.M., Ph.D.

Leslie Marshall



From left to right: Maxim Cheeran, Jessica Rotschafer, K. Yeon Choi, Dianna Cheney-Peters, and Gamal Elmubarak

Dr. Maxim Cheeran's laboratory utilizes a murine model of herpes simplex encephalitis (HSE) to study the interactions between immune responses to viral infection and neural stem cell (NSC) response to injury. Viral encephalitis often causes significant long-term brain damage that may result in life-long neurological disabilities. It is widely believed that brain damage is in part, a direct consequence of exacerbated immune responses to infection. Over the last decade, it has become clear that adult mammalian brains have some potential to regenerate following injury. Adult neurogenesis is reminiscent of embryonic development and is engendered by a small population of neural stem cells (NSCs) in specialized brain niches that support their function. NSCs within these niches respond to both normal physiological and injury-induced cues, many of which originate from the immune system. However, little is known about immune mechanisms that influence neurogenesis during viral infections and their roles in modulating the reparative potential of NSCs.

Studies from Dr. Cheeran's group showed that animals surviving an acute infection exhibit a spectrum of chronic cortical and hippocampal lesions, which are associated with severe spatial memory deficits (Armien, *et. al.*, *Brain Pathol.*, 20: 738-750, 2010). Both viral infection and inflammatory responses followed spatially and temporally distinct courses within the brain but did not directly infringe into NSC niches. Interestingly, the chronic neuro-inflammatory response was dominated by persistent CD8-T lymphocytes that are retained during recovery from acute infection, in addition to the increased microglia activation (Marques, *et. al.*, *J. Immunol.*, 181: 6417-6426, 2008).

More recent unpublished studies in Dr. Cheeran's laboratory show that the initial response to HSV-1 brain infection is an increase in NSC proliferation. Surprisingly, this increase in proliferation is significantly suppressed during the recovery phase. Concomitant

with modulation of NSC proliferation is a rise and subsequent fall in the expression of neurotrophic factors in infected brains compared to uninfected animals. This is particularly prominent with fibroblast growth factor expression, which plays a critical role in maintaining the subventricular zone NSC niche. These findings indicate that changes in the tissue microenvironment during the recovery phase may not be conducive for neurogenesis. This work supports the hypothesis that the change in neurogenic programming is mediated by persistent CD8 T-cells retained in the brain subsequent to viral infection. In vitro, activated CD8 T-cells suppress NSC proliferation through the production of interferon- γ and in the process induce an altered NSC differentiation state. Current work in the Cheeran laboratory addresses the role of specific T-cell populations in

modulating NSC responses during HSE, in an effort to identify points of intervention that could be translated into new therapeutic approaches for the debilitating neurological deficits seen in the wake of viral encephalitis.

Dr. Cheeran's laboratory has recently embarked on an investigation into the role of cell-mediated immune responses in the emergence of swine influenza virus. While it is perceived that host responses, in particular humoral (antibody) responses, impose selection pressure on emerging influenza viruses, the mechanisms that drive selection of viral mutations in the pig are at best speculative. This gap in knowledge is particularly prominent in the lack of understanding of T-cell immunity in pigs. Maxim's lab is currently developing methods to begin bridging this knowledge gap by identifying swine influenza virus epitopes recognized by porcine T lymphocytes, which is an important component of protective immunity against heterologous viral strains. Given that pigs are considered a source for emerging influenza viruses, understanding mechanisms by which immune pressure directs antigenic variation in swine influenza virus may be critical for predicting and protecting against the next influenza pandemic.

Dr. Maxim Cheeran received a Ph.D. in Veterinary Medicine in 2000 from the University of Minnesota, an M.S. in Veterinary Biotechnology in 1995 from the Indian Veterinary Research Institute, and a D.V.M. in 1992 from Kerala Agriculture University in India. He currently serves as an Assistant Professor in the Department of Veterinary Population Medicine at the College of Veterinary Medicine in St. Paul, Minnesota. Dr. Cheeran is an active member of the neurovirology community and serves as an ad-hoc reviewer for many prominent journals in the field. His laboratory is funded by NINDS to study the fate of neural stem cells during viral encephalitis. In addition, Maxim mentors numerous undergraduate, graduate, and medical students.



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ISNV Highlights - Glenn Rall, Ph.D.

Dianne Langford

Dr. Glenn Rall's laboratory studies basic aspects of neuroimmunology and neurovirology, primarily using measles virus (MV) infection of a receptor-expressing transgenic mouse model. This model, NSE-CD46, expresses a human vaccine strain receptor transcriptionally restricted to CNS neurons, allowing both an *in vivo* pathogenesis model of neuronal infection, as well as the ability to isolate and culture MV-permissive neurons. The Rall lab has two primary objectives. The first explores how viruses, made in the neuronal cytoplasm, are transported to the synapse, where egress occurs, and whether traditional receptor-ligand interactions are needed for trans-synaptic spread. The second addresses how the host immune response recognizes and resolves neuronal infections, and how the neuronal response to potent antiviral cytokines, such as interferon- γ (IFN- γ), may differ from the classical pathway initially defined in fibroblasts. For example, recent studies from Glenn's laboratory showed that despite a requirement for IFN- γ in protection from MV-triggered neuropathology, the canonical STAT-1-mediated pathway in neurons is not required (O'Connell *et al.*, *J. Immunol.*, 188: 1915-1923, 2012). Rather, alternative signaling pathways are operative, inducing genes that allow neurons to survive the anti-viral host response.

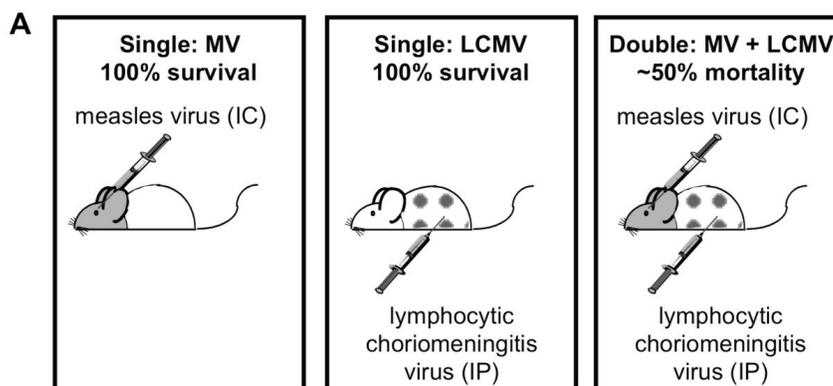
A departure from these primary themes was recently reported in *PLoS Pathogens*, and reflects a third new interest in the laboratory (Matullo *et al.*, *PLoS Pathogens*, 7: e10002462, 2011). Using the same transgenic model, the Rall laboratory explored lymphocyte trafficking under conditions of poly-viral challenge. Immunocompetent adult mice survive either a neuronal MV challenge or a peripheral lymphocytic choriomeningitis virus (LCMV) challenge. However, when mice were challenged with both viruses, 100% of the doubly infected mice exhibited signs of CNS disease, with ~50% dying. A "mis-recruitment" of anti-LCMV CD8⁺ T cells to the CNS was responsible for this pathology, but how T cells cause disease is unclear, especially since no LCMV antigens were found in the brain following peripheral challenge. The key



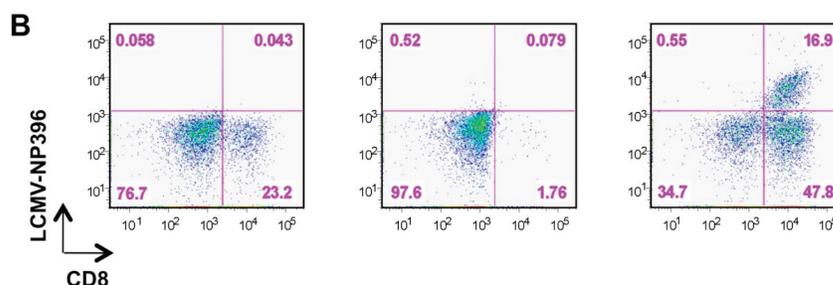
From left to right: Chris Matullo, Jazz Skipworth, Glenn Rall, Alicia Holmgren, Sarah Cavanaugh, Kristen Henkins

message from these studies is the proof-of-principle observation that CNS diseases may result from immune challenges (such as viral infections) that are not within the brain. This may be relevant to human CNS diseases of unknown etiology, but that possess pathological hallmarks of neuroinflammation.

Glenn received a Ph.D. from Vanderbilt in 1990 and completed four years of post-doctoral training at the Scripps Research Institute under the direction of Dr. Michael Oldstone. He joined the Fox Chase Cancer in 1995 where he is an Associate Professor. Currently, the Rall laboratory consists of 4 postdoctoral fellows (Christine Matullo, Kevin O'Regan, Kristen Henkins and Karen Lancaster), three graduate students (Sarah Cavanaugh, Alicia Holmgren and Andreas Solomos) and one absolutely amazing scientific technician, Jazz Skipworth. In addition, the laboratory routinely hosts summer college students, high school volunteers, and various pets.



As described in a recent paper (Matullo *et al.*, *PLoS Pathogens*, 7: e10002462, 2011) Dr. Rall's laboratory has developed a mouse model of viral coinfection, using measles virus as a CNS activator and immune cell recruiter and lymphocytic choriomeningitis virus as a peripheral inducer of the immune response (A). This coinfection results in significant morbidity and mortality which is coincident with recruitment of LCMV-specific CD8⁺ T cells into the brain (B).



ISNV Highlights - Ravi Mahalingam, Ph.D.

Dianne Langford

Dr. Ravi Mahalingam's laboratory at the University of Colorado School of Medicine conducts cutting edge research on varicella zoster virus (VZV) that has contributed significantly to our current understanding of the pathogenesis of virus latency and reactivation in humans. Having studied VZV for 25 years, Dr. Mahalingam is a leader in the field and boasts more than 100 publications addressing several important aspects of VZV infection. After completing graduate studies at Southern Illinois University, Ravi joined the University of Colorado in 1983 as a post-doctoral fellow. In 1986, he began working with Dr. Don Gildea as a Research Associate and is currently a Professor in the Department of Neurology.

VZV belongs to the alpha herpesvirus family and causes chickenpox (varicella) in children, establishes latency in ganglia and may reactivate decades later in the elderly to cause shingles (zoster). Neurological complications associated with zoster include post-herpetic neuralgia, paralysis (VZV myelitis), stroke (VZV vasculopathy) and blindness (VZV retinitis). Ravi was the first to use PCR technology to demonstrate that VZV DNA is present in multiple ganglia in humans along the entire neuraxis. Before the advent of real-time PCR, in 1993, Ravi used competitive PCR to show a low abundance (as few as 36 copies in 105 cells) of latent VZV DNA in human ganglia. Over the next few years, he reported for the first time, the expression of a VZV ORF63 protein in the cytoplasm of human ganglia that were latently infected with VZV. He also demonstrated persistence of VZV DNA in blood mononuclear cells from elderly patients with post-herpetic neuralgia.

Later, he established an animal model of VZV latency in non-human primates infected with simian varicella virus (SVV). By generating SVV expressing green fluorescent protein (GFP), the identification of cells infected with SVV both in vitro and in vivo was made possible, and were used to study of varicella pathogenesis and latency in experimentally infected animals. Using this model, Ravi demonstrated that varicella enters ganglia before the appearance of rash. Ravi and his colleagues sequenced the complete 125,000 base pair SVV genome that paved the way for molecular analysis of latency and reactivation. In 2007, they reported the reactivation of SVV in immunosuppressed Cynomolgus monkeys, the pathogenic features of which are shared with human VZV patients. In a recent publication in *Journal of NeuroVirology*, Ravi showed that SVV ORF63 is essential for viral replication in culture.

Currently, Ravi's laboratory is involved in the identification of cell types that transport varicella to the skin and ganglia during primary infection (chickenpox). Other studies in his laboratory are aimed at characterizing the phenotype of the T-cells that respond to varicella reactivation and also to identify signaling molecules that recruit T cells in ganglia. Ravi's laboratory is utilizing BAC

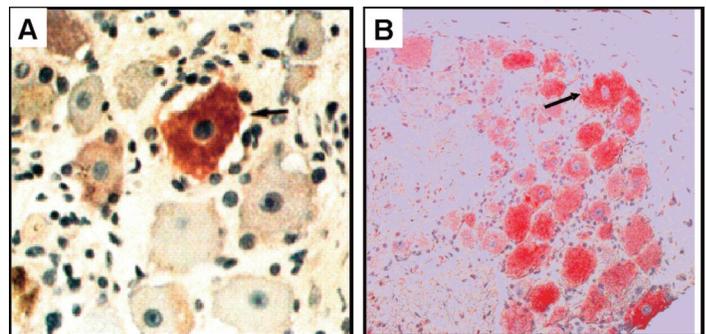


From left to right: Mary Wellish (research assistant), Dr. Stephanie James (postdoctoral fellow), Thomas Ogas (student), Martin Lapel (student) and Jennifer Manfredo (student)

technology to generate a conditional SVV ORF63 mutant virus to characterize the role of ORF63 in virus infection.

VZV and SVV are highly species-specific. Ravi's laboratory is also involved in the preparation of a VZV-SVV chimeric virus which will enable direct studies of VZV infection in primates. Additionally, the chimeric virus will help identify specific regions of VZV and SVV genome that are important in their species specificity.

Finally, Ravi has mentored and trained numerous students at all levels of study. He serves on the Editorial Board for the *Journal of NeuroVirology* and *BioMed Central infectious Diseases* and reviews for many other peer-reviewed scientific journals.



Detection of VZV and SVV ORF63 proteins (arrows) in the cytoplasm of neurons in ganglia of (A) a human latently infected with VZV and (B) a rhesus macaque latently infected with SVV.

JOURNAL OF NEUROVIROLOGY



The Neurological Infections Lectureship features prominent investigators who study viral and non-viral pathogens that infect and damage the human peripheral and central nervous systems. Established at the 7th International Symposium on NeuroVirology Held in Philadelphia, PA, USA this will be the fifth Neurological Infections Lectureship.

For information on submitting abstracts, subscribing to newsletters, and general information about the Journal of NeuroVirology, please visit www.jneurovirol.com

2012 Upcoming Conferences

April

American Academy of Neurology: [April 21-28, New Orleans, Louisiana](#)
 Society on NeuroImmune Pharmacology (SNIP), 18th Scientific Conference:
[April 25-28, Honolulu, Hawaii](#)

Gordon Research Conferences (GRC)

CNS Myelination and Remyelination: [April 28-29, Lucca, Italy](#)
 Biology and Pathobiology: [April 29-May 4, Lucca, Italy](#)

May

International Symposium on HIV and Emerging Infectious Diseases (ISHEID):
[May 23-25, Marseille, France](#)

11th International Symposium on NeuroVirology held jointly with the 2012 Conference on
 HIV in the Nervous System: [May 29-June 2, New York, New York](#)

June

American Society for Microbiology: [June 16-19, San Francisco, California](#)

Gordon Research Conferences (GRC)

Barriers of the CNS: [June 16-17, New London, New Hampshire](#)
 Cell Biology of the Neuron: [June 24-29, Waterville Valley, New Hampshire](#)

American Society for Microbiology: [June 16-19, San Francisco, California](#)

July

Gordon Research Conferences (GRC)

Synaptic Transmission: [July 29-August 3, Waterville Valley, New Hampshire](#)

American Society for Virology: [July 21-25, Madison, Wisconsin](#)

August

Gordon Research Conferences (GRC)

Neurobiology of Brain Disorders: [August 5-10, Easton, Massachusetts](#)

September

Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC):
[September 9-12, San Francisco, California](#)

October

American Neurological Association: [October 7-10, Boston, Massachusetts](#)

Society for Neuroscience: [October 13-17, New Orleans, Louisiana](#)

ID Week: joint meeting of IDSA, SHEA, HIVMA, PIDS:
[October 17-21, San Diego, California](#)

Classifieds

Postdoctoral fellow position

A postdoctoral position is available in the Department of Pharmacology and Experimental Neuroscience, University of Nebraska Medical Center, to investigate the signaling and molecular mechanisms of HIV infection, viral-bacterial interactions and disease progression. The ideal candidate will be creative, highly motivated, have a doctoral degree with a strong background in microbiology / infectious diseases, molecular and cellular biology, and signal transduction. Prior experience working with laboratory animals preferred. The candidate should be able to design and perform experiments with minimal supervision, critically analyze the results, and help write manuscripts for publications. Interested candidates should send their curriculum vitae and names of three references (including telephone numbers and e-mail addresses) to:

Georgette D. Kanmogne, Ph.D., MPH.
Associate Professor,
Vice-chair for Faculty Development
& Research Development,
Department of Pharmacology &
Experimental Neurosciences,
University of Nebraska Medical Center
Tel: 402-559-4084
Fax: 402-559-8922
email: gkanmogne@unmc.edu

Postdoctoral fellow position

A postdoctoral position is available in the Department of Pharmacology and Experimental Neuroscience, University of Nebraska Medical Center, to investigate HIV genetics and disease neuropathogenesis, using applied genomics and system biology approaches. The ideal candidate will be highly motivated, have a doctoral degree, with background in genetics, bioinformatics/functional genomics, strong computational and analytical skills. The candidate should be able to design and perform experiments with minimal supervision and help write manuscripts for publications. Interested candidates should send their curriculum vitae and names of three references (including telephone numbers and e-mail addresses) to:

Georgette D. Kanmogne, Ph.D., MPH.
Associate Professor,
Vice-chair for Faculty Development
& Research Development,
Department of Pharmacology &
Experimental Neurosciences,
University of Nebraska Medical Center
Tel: 402-559-4084,
Fax: 402-559-8922,
email: gkanmogne@unmc.edu

Postdoctoral fellow position

The Pharmacology Neuroimmunology (PNI) program at the University of Minnesota is now accepting applications from post-doctoral fellows seeking laboratory-based research training in neuroimmune interactions or neurobehavior in addition in an NIH/NIDA funded training program. The program is specifically looking for recent doctoral degree recipients (within the past 4 years) interested in areas that intersect four disciplines: Pharmacology, Psychology/Psychiatry, Neuroscience, and Microbiology/Immunology. Since this is a federally funded program, only U.S. citizens and those foreign nationals who possess a permanent residency visa (green card) may be supported. Individuals from underrepresented groups are encouraged to apply. Salary will be based on NIH NRSA stipend rates for postdoctoral trainees. To request an application form or to ask further questions please contact:

Yorie Smart
smart006@umn.edu,
Veterinary Population Medicine
University of Minnesota

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11TH INTERNATIONAL SYMPOSIUM ON

NEUROVIROLOGY

held jointly with

2012 Conference on HIV in the Nervous System



New York, New York

Grand Hyatt Hotel, May 29th to June 2nd, 2012

Contact the ISNV Administrative office at:

Tel: (215) 707-9788

Fax: (215) 707-9838

Email: mail@isnv.org

www.isnv.org



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Drexel University College of Medicine

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