Fall 2011
Vol. 11 No. 3

ISNV

International Society for NeuroVirology

Editor: Dianne Langford, Ph.D. Associate Editor: Michael Nonnemacher, Ph.D. Assistant Editor: Leslie Marshall, Ph.D.

11th International Symposium on NeuroVirology

held jointly with

2012 Conference on HIV in the Nervous System

Greetings everyone! It is with much enthusiasm that I announce the 11th International Symposium on NeuroVirology to be held jointly with the 2012 Conference on HIV in the Nervous System, May 29 through June 2, 2012, at the newly renovated Grand Hyatt Hotel at Grand Central Station, New York, NY, USA. More than 350 basic scientists, clinicians and trainees working in the areas of neurology, neuropathology, neuropathogenesis, neuroimmunology and molecular virology will attend these meetings. In addition to leading experts from around the globe presenting their cutting edge research during our main sessions and workshops, we are excited to announce that there will be a Global NeuroAIDS Round Table Satellite Symposia sponsored by NIMH that will occur during the afternoon of Tuesday May 29th. **Abstract submission is open with abstracts due March 1, 2012**. For updates and details on the agenda, registration information and venue, please visit the ISNV web site, www.isnv.org. Our first gathering in New York City will surely provide a backdrop for an exciting and memorable event and I look forward to seeing everyone in Spring 2012!

Lynn Pulliam
President of ISNV

Science News

HIV Vaccine Update

Jay Rappaport
Professor and Vice Chair in the Department of Neuroscience at Temple University School of Medicine and the Director of Graduate Programs for the TUSM Department of Neuroscience

It has been more than 27 years since the April 23rd, 1984 announcement from Margaret Heckler (Health and Human Services Secretary at the time) that based on the work of Dr. Robert Gallo and his team, the human lentivirus, now known as HIV, was the cause of AIDS. The fact that an effective vaccine has not yet been developed is a major scientific disappointment to say the least, and underscores the resilient persistence of this virus in the infected host.

A major obstacle appears to be the inability to induce broadly reactive neutralizing antibody responses capable of durable protection against a highly variable envelope protein. Furthermore, multiple lines of evidence emphasize the role of virus-specific cytotoxic T-cell responses in the effective control of HIV infection. Based on these considerations, it was a further disappointment when the international vaccine trial "STEP", utilizing an adenovirus based vector to induce virus specific cytotoxic T-cells, was halted in 2007 based on interim results analyzed by the data safety monitoring board. The failure of the vaccine trial may have been due to characteristics of the adenovirus vector such as its inability to replicate, or something more fundamental regarding the overall approach.

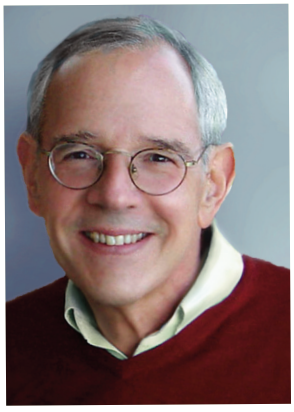
Immunologic control of HIV presents somewhat of a dilemma in that the virus infects helper T-cells, which are essential for both effective antibody production and cytotoxic T-cell responses. Moreover, since activated memory helper T-cells are readily infected by HIV, the virus is likely capable of overwhelming pre-existing responses, depending on the magnitude of the virus-specific immune cell reservoir versus the level of virus an individual is exposed to. With this in mind, it is of interest that the recent analysis of the RV144 vaccine trial in Thailand suggests that some level of protection is provided, as discussed in an article by Jill Kresge in the September 2011 International AIDS Vaccine Initiative report. The positive correlation of anti-Env IgG with protection and the negative correlation of anti-Env specific IgA appear to suggest a mechanism involving antibody dependent cytotoxicity (ADCC), where IgA antibodies may be interfering with IgG mediated ADCC. However, other interpretations are possible and further studies are needed to determine if these biological correlates are mechanistically meaningful. Parallel non-human studies by Genoveffa Franchini indeed showed the protective effect of RV144 could be over-ridden by an increase in the magnitude of the viral challenge.

Approaches using live vectors capable of inducing and maintaining high levels of memory and/or effector cells may be required to overcome repeated exposures to HIV over-time, and in individuals at high risk for infection. Recent studies reported in the May 26th issue of *Nature* by Louis Picker's laboratory demonstrate that SIV replication in macaques can be effectively controlled using a persistent virus, cytomegalovirus, as a vaccine vector, by generating robust effector/memory responses.

It will be interesting to determine if there is a threshold level required for effective anti-HIV immunity and if live/persistent vectors are necessary to achieve such responses. Conceptual aspects such as these are important to address so that issues regarding broad-based and cross-clade immune responses can be investigated in feasible and informative experimental systems.

ISNV Highlights - Michael Chopp, Ph.D.

Leslie Marshall



Dr. Michael Chopp is a distinguished scientist that has pioneered the role of cell-based therapies in the treatment of stroke and neural injury and has recently demonstrated that tissue plasminogen activator (tPA) is upregulated by cell therapy and promotes neurite outgrowth and functional recovery. He serves as the Zolton J. Kovacs Chair in Neuroscience at the Henry Ford Health System in Detroit, Michigan where he is also the Scientific Director of the Neuroscience Institute and Vice Chairman of the Department of Neurology. In addition, he is a distinguished Professor in the Department of Physics at Oakland University in Rochester, Michigan. In the thirty years since completing his doctoral work in physics at New York University, Dr. Chopp has authored 500 scientific publications, has presented his work in over 339 scientific meetings, has mentored over 30 Ph.D. candidates and 11 post doctoral fellows, and has served as a reviewer for countless study sections and scientific journals. He has received many awards for both research and mentoring, and in 2001 he was recognized by the American Heart Association for one of the Top 10 advances in Medical Science.

Dr. Chopp's laboratory primarily studies ways to enhance neurological recovery after neural injury, stroke, and neurodegenerative diseases, such as multiple sclerosis. Studies range from basic molecular and cell biology to preclinical development of neurorestorative agents. In addition, his laboratory studies the interaction of neurogenesis, angiogenesis, oligodendrogenesis, and neurite outgrowth to understand the molecular pathways involved in these restorative processes. Recent studies demonstrate that after a brain injury or stroke, substantial plasticity and neurite outgrowth occurs in the brain-contralateral to the injured tissue and spinal cord, and neurorestorative agents greatly amplify this brain/spinal cord ***Continued on page 3***

ISNV Highlights - Michael Chopp, Ph.D. (continued)

(Continued from page 2) plasticity that correlates to functional benefit. Some of the pharmacological agents tested for the treatment of stroke and traumatic brain injury include agents that increase HDL (Niaspan, carbamylated erythropoietin and Thymosin beta 4 (TB4)- an actin binding protein). The laboratory has shown that these drugs are highly restorative and can be translated from preclinical animal models to the patient. Particularly for TB4 and HDL, oligodendrogenesis is highly upregulated and promotes recovery of function after stroke, traumatic brain injury and experimental autoimmune encephalomyelitis. Dr. Chopp's laboratory also focuses on the diabetic brain, which is an understudied area with great clinical importance. His laboratory is investigating the response of the diabetic brain to stroke and restorative therapies including both cell and pharmacological restorative therapies. Along with these studies, is an active MRI program that seeks to develop non-invasive means to monitor structural vascular and axon/white matter changes as an index and biomarker of functional recovery. Finally, the laboratory has also been investigating the roles of miRNA in stroke, neural damage, and neurorestoration. In addition to studies on neural injury, stroke and MS, his laboratory studies glioma, and has recently shown that miRNA can be readily transferred via gap-junctions between glioma cells.

2012 Upcoming Conferences

March

Conference on Retroviruses and Opportunistic Infections: March 5-8, Seattle, Washington

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

July

Gordon Research Conferences (GRC)
Synaptic Transmission: July 29-August 3, Waterville Valley, New Hampshire

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

ISNV Highlights – David Volsky, Ph.D.

Mary Jane Potash

Dr. Potash is an Associate Professor of Pathology in the Molecular Virology Division, St. Luke's-Roosevelt Hospital Center and Columbia University

David J. Volsky received a B.Sc. in Biology from Ben-Gurion University, Israel, and M.Sc. and Ph.D. in Biochemistry in 1979 from Hebrew University in Jerusalem. Following postdoctoral research on Epstein-Barr virus (EBV) receptors and cell transformation at the Karolinska Institute in Stockholm in 1981, Dr. Volsky moved to the University of Nebraska Medical Center and continued research in EBV and viral carcinogenesis. With the advent of the AIDS epidemic, Dr. Volsky changed his research interests to retrovirology and AIDS and by 1985 dedicated his laboratory exclusively to HIV research. In 1987, he moved to St. Luke's-Roosevelt Hospital Center and Columbia University in New York and became Director of the Molecular Virology Division and Professor of Pathology and Cell Biology at Columbia University.

Since his move to New York, Dr. Volsky's HIV/AIDS research has expanded to include various aspects of HIV biology and viral pathogenesis with a particular interest in HIV interactions with brain-derived cells and molecular mechanisms of viral neuropathogenesis. These studies focus on HIV infection and cellular signaling in human fetal astrocytes, gene expression profiling of infected astrocytes and autopsy brain tissues from patients with dementia, and other aspects of HIV function in the CNS that may contribute to brain disease. Although antiretroviral therapy (ART) has proved to preserve immune function in HIV-infected persons, neurocognitive disorders persist, raising questions about the virally-induced neuropathogenic events during HIV infection. With Dr. Alejandra Borjabad from the Molecular Virology Division and Dr. Susan Morgello, Director of the Manhattan HIV Brain Bank, and others, Dr. Volsky recently investigated the influence of ART on changes in cellular gene expression in brains from HIV infected persons with HIV associated neurocognitive disorders (HAND) using large scale microarray analysis among other bioinformatics approaches. Compared to brains from uninfected persons, brains from untreated HAND patients show highly significant dysregulation of expression of 1470 genes most prominently in antiviral and immune responses and suppression of synaptic transmission and neurogenesis. In distinct contrast, depending on treatment adherence, brains from ART-treated patients showed dysregulation in 83-93% fewer genes and significantly lower dysregulation of biological pathways compared to untreated patients, with particular improvement indicated for nervous system functions. These findings indicate that similar to its action in the periphery, ART can significantly alleviate aberrant gene expression in brain tissues of patients with HAND and by extension improve brain functions (Fig.1 and 2). It is noteworthy that a group of about 100 genes were similarly affected in brains of HAND patients, independent of treatment, compared to brains of uninfected persons. These core changes may underlie the neurocognitive defects defying ART. To facilitate his neuroAIDS research, Dr. Volsky and collaborators recently developed a murine model of infection with chimeric HIV and applied it to the study of experimental HIV neuropathogenesis including induction of neurocognitive deficits in mice. Most current efforts focus on identifying parallels between gene dysregulation in the brains of patients with HAND and gene expression changes in HIV infected mice.

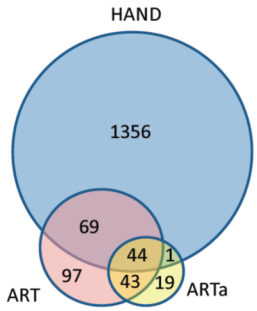


Figure 1. The pattern of changes in brain cell gene expression relative to uninfected subjects differs significantly between untreated HAND patients and HAND patients treated with ART. The Venn diagram displays the number and overlap of transcripts differentially expressed relative to uninfected subjects from HAND, ART, and HAND patients excluding low ART adherence patients (ARTa). (Reprinted from Borjabad *et al.*, *PLoS Pathog.* 2011 September; 7(9): e1002213).

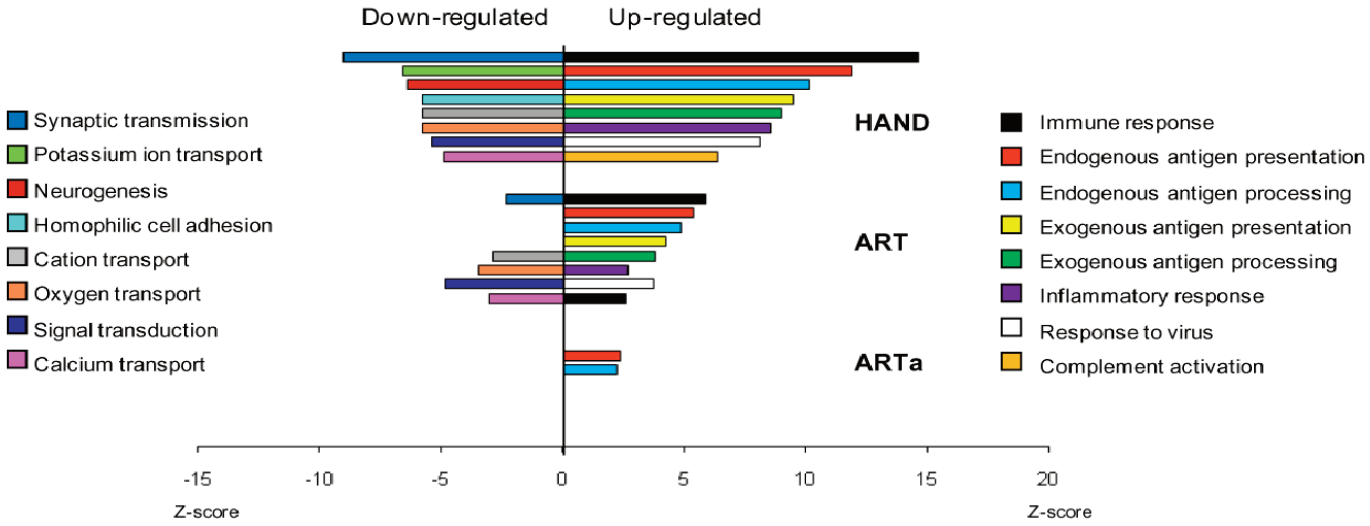


Figure 2. GAzer was employed to identify the eight most dysregulated biological pathways relative to uninfected subjects in the HAND dataset and the extent of change in the same pathways in ART and ARTa datasets are shown for comparison with Z-scores plotted for the significantly up-regulated (right panel) and down-regulated (left panel) pathways. (Reprinted from Borjabad *et al.*, *PLoS Pathog.* 2011 September; 7(9): e1002213).

ISNV Highlights – Dana Gabuzda, M.D.

Dianne Langford

Front row (left to right): Anupa Kamat, Postdoctoral Fellow; Edana Cassol, Postdoctoral Fellow; Joya Mukerji, graduate student; Dana Gabuzda, MD, Professor; Megan Mefford, graduate student; Will Yen, graduate student. Back row (left to right): Alex Holman, PhD, Bioinformatics Engineer; Vikas Misra, MS, Lab Manager; Erez Pery, Postdoctoral Fellow.

Dr. Dana Gabuzda received her M.D. from Harvard Medical School, and completed clinical training in neurology at Massachusetts General Hospital. She then completed four years of postdoctoral studies in neurovirology and HIV/AIDS research at Johns Hopkins University and Dana-Farber Cancer Institute. Currently, Dr. Gabuzda is a Professor of Neurology with an affiliated appointment in Microbiology at Dana-Farber Cancer Institute and Harvard Medical School.

As a physician-scientist trained in neurology, molecular biology, and HIV/AIDS research, her major research interest is to understand how HIV causes disease in the nervous system. Her laboratory uses molecular, genetic, and bioinformatic approaches to study relationships between HIV genetic variants, viral replication in the brain, and development of HIV-related neurocognitive disorders. Recent studies from her laboratory identified genetic variants of HIV that increase macrophage tropism and neurotropism of HIV through enhancing interactions between the viral envelope and the viral receptor/co-receptor, CD4 and CCR5, respectively. Her laboratory is now using machine learning tools and large meta-datasets to identify viral genetic signatures in the brain and CSF predictive of neurocognitive outcomes in patients with ongoing viral replication. To facilitate this work and studies by others doing research on neuroAIDS, her laboratory recently built the HIV brain sequence database, now publicly available at http://www.HIVBrainseqDB.org (Holman *et al., AIDS Research and Therapy*, 2010).

The HIV Brain Sequence Database currently contains more than 2517 envelope *(env)* sequences from over 90 patients, with at least 1272 sequences from the brain. As newly identified HIV *env* sequences are reported, the database continues to grow. This valuable resource links clinical data with genetic aspects of HIV macrophage tropism for analyses of viral evolution within the brain and other tissue reservoirs. Other recent studies from her group investigated plasma and CSF biomarkers associated with neurocognitive impairment in HIV patients in the era of combination antiretroviral therapy (cART) (Lyons *et al*, *JAIDS,* 2011). This work showed that increased plasma and CSF levels of soluble CD14, a biomarker of monocyte activation, are associated with impaired neurocognitive testing in viremic but not in aviremic patients on cART (Fig. 1). Her group is now following up on these studies to learn more about mechanisms driving the milder forms of neurocognitive impairment that are prevalent in patients who achieve sustainable virological suppression on long-term cART and the role of chronic inflammation versus other mechanisms in the pathogenic processes. Dr. Gabuzda's honors include a Barr Investigator Award in Cancer Research, a Howard Temin Award, an Elizabeth Glaser Scientist Award from the Pediatric AIDS Foundation, and an Avant Garde Award for HIV/AIDS research from NIDA. She has trained many graduate students and postdoctoral fellows in HIV research, including several investigators who are now leaders in neurovirology research (Drs. Paul Gorry and Johnny He), and previously served as Associate Head of the Harvard Ph.D. Program in Virology from 2006-2011.

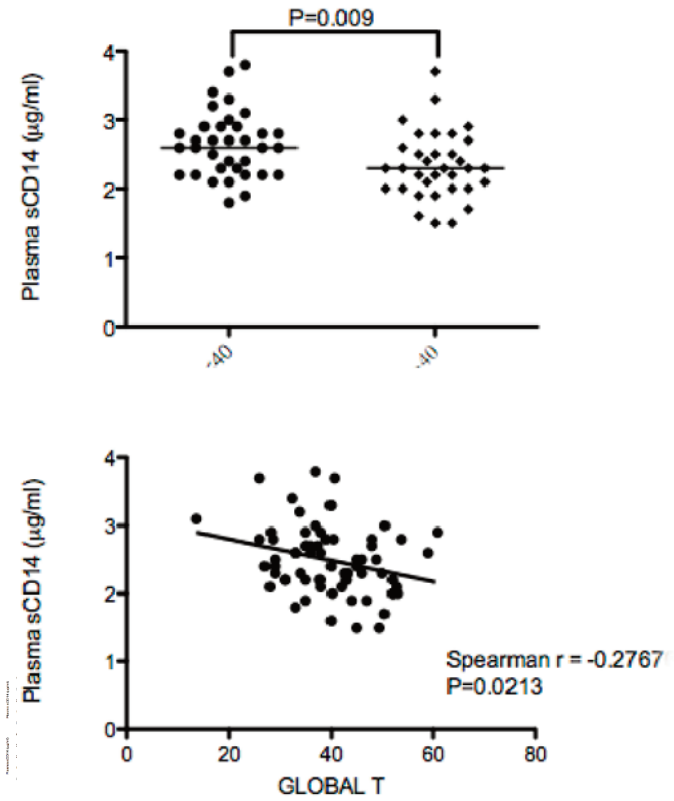


Figure 1. Higher plasma sCD14 levels were associated with global T scores indicating neurocognitive impairment. Subjects were grouped by global T scores of either < 40 or ≥ 40 to compare biomarker levels. Upper panel: Plasma sCD14 levels were significantly different between subjects with impaired global T scores vs. subjects without. Lower panel: Plasma sCD14 levels correlated negatively with global T scores. Statistical significance between groups was calculated using the 2-tailed Mann-Whitney test, and significance among continuous variables was calculated using Spearman rho correlation.

ISNV Highlights - Ken Williams, Ph.D.

Leslie Marshall

Dr. Williams' research group has a long-standing interest in monocyte/macrophage accumulation in the CNS as a driving force behind the neuropathology associated with AIDS. His group studies the hypothesis that expansion of activated, and sometimes HIV-infected monocyte/macrophages that migrate from bone marrow into the blood are required for lesion formation in the brain. Last year, his group showed in simian immunodeficiency virus (SIV)-infected monkeys that increased, intractable monocyte expansion, as measured by BrdU+ monocytes in blood, better correlates with development of AIDS than CD4+ T lymphocyte loss or viral load (Burdo, T. H., *et al., PLoS Pathog* 6:e1000842, 2010). Next, Dr. Williams and colleagues discovered that an increased rate of monocyte turnover, as measured by BrdU+ monocytes in blood, correlates with the rapid rate of development of AIDS, and the severity of SIVE (Hasegawa, A., H. *et al, Blood,* 2009). Moreover, their data demonstrated that sCD163 levels, a scavenger receptor released from monocyte/macrophages into the plasma of SIV infected animals, positively correlates with BrdU+ monocytes in the same animals, which suggests that sCD163 in the blood is a surrogate marker of monocyte expansion. Thus, these studies clearly demonstrated that both monocyte expansion and the magnitude of sCD163, which is detected as early as 20 days post infection, are predictors of how rapid animals will develop AIDS and the severity of SIVE.

More recently, Burdo *et al.*, found elevated levels of sCD163 in the plasma of chronically infected HIV+ patients (>1 year infection) compared to seronegative controls (Burdo, T. H., *et al., J Infect Dis.*, 2011). Effective antiretroviral therapy (ART) resulted in decreased levels of sCD163 and subsequent viral load in the plasma of these patients; however, levels did not return to baseline observed in seronegative controls. These findings suggest an incomplete reversal of monocyte/macrophage activation, even in the presence of undetectable virus in the plasma (Burdo, T. H., *et al., J Infect Dis.*, 2011). Examination of patients recently infected with HIV (< 1 year), revealed a lower level of sCD163 in plasma, which in combination with effective ART, returned to the same baseline levels of seronegative patients. Taken together, these studies suggest that HIV infection of at least 1 year is required for monocyte activation to persist despite of effective ART treatment. In addition, Williams and colleagues found a positive correlation between CD14+CD16+ monocytes and activated CD8+HLA-DR+CD38+ T lymphocytes, and an inverse correlation of CD163 expression on CD14+CD16+ monocytes in these patients, suggesting a relationship between CD14+CD16+ cells that are a significant source of sCD163 (Burdo, T. H., *et al., J Infect Dis.*, 2011). With ART interruption in the recently infected patient population, they observed a spike in elevated sCD163 that paralleled HIV RNA in plasma, and then rapidly returned to normal when ART was resumed. Finally, in a separate study, sCD163 levels in the plasma of HIV-infected patients with cardiac disease undetectable by classic markers (D-Dimer and C-reactive protein) was shown to be a marker for significant non-calcified plaques in the heart, which is consistent with macrophage accumulation in early lesions (Burdo, T. H., *et al., J Infect Dis.*, 2011).

Overall, these studies support an important role for 1) activated and/or infected monocytes in bone marrow and blood and 2) macrophages in the CNS and heart that contribute to SIV and HIV-mediated pathogenesis, even with effective ART in humans. In addition, these results underscore the utility of sCD163 levels in the plasma as a marker of such activation and a possible marker to monitor effective anti-HIV therapies.

Committee Reports

Fundraising Sub-Committee Update

Igor Koralnik

As we prepare for our 11th International Symposium next year in New York City, our committee has been diligently focused on obtaining support from some of the big pharmas such as Merck, Bristol-Myers Squibb (BMS), Gilead, Boehringer-Ingelheim, TEVA, and Biogen Idec, as well as the National Multiple Sclerosis Society. We are also utilizing our own contacts within various industries, including local vendors and our own institutions to secure funds in support of our upcoming meeting.

Some of our key strategies include talking directly to decision makers in the organizations, providing a detailed outline that includes various levels of conference sponsorship/advertising opportunities and enlisting the assistance of the ISNV membership in identifying sponsors and/or promoting their own programs to our captive audience.

We welcome your assistance in our efforts to raise funds and will gladly provide the necessary tools for marketing the ISNV to the outside community. For additional information or any questions, please contact the ISNV office at mail@isnv.org.

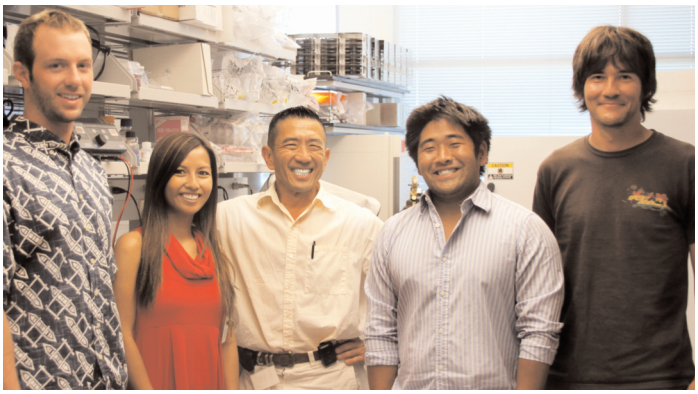
Fundraising Committee

Krzysztof Reiss — **Igor Koralnik (Chair)** — **Scott Letendre**

Avindra Nath — **Brian Wigdahl**

ISNV Highlights – Bruce Shiramizu, M.D.

Melissa Agsalda
Melissa is a 1st-year post-doctoral fellow in Dr. Shiramizu's laboratory.



(Left to Right: Patrick Kirkland, Melissa Agsalda, Ph.D., Bruce Shiramizu, M.D., Ian Nagata, and Iain MacPherson, Ph.D.).

Dr. Bruce Shiramizu is a Professor of Pediatrics and Medicine at the John A. Burns School of Medicine (JABSOM), University of Hawaii. He is a co-investigator at the Hawaii Center for AIDS. He also is the JABSOM RMATRIX Executive Director and serves on the graduate faculty in numerous Departments including Cell and Molecular Biology, Tropical Medicine, Physiology, and Neurosciences. Dr. Shiramizu received an M.S. in Bioengineering and an M.D. from the University of Utah in Salt Lake City. Following completion of a residency in Pediatrics at SUNY-Children's Hospital in Buffalo, New York, he completed a fellowship in Pediatric Hematology/Oncology at NCI at NIH. Prior to accepting a faculty position at the University of Hawaii in 1998, he served as an Associate Professor at the University of California, San Francisco in the Department of Pediatrics.

Translational research activities in his laboratory have focused on delineating the role that HIV DNA plays in the pathogenesis of neurocognitive disease. While antiretroviral therapy (ARV) can effectively control HIV RNA levels, monocyte reservoirs containing HIV DNA may remain relatively unaffected by ARV and are likely important in disease pathogenesis as patients live longer with therapy. As one of the initial groups involved with the Hawaii Aging with HIV Cohort in the Hawaii Center for AIDS, his group described an association between HIV DNA levels in peripheral blood mononuclear cells and severity of HIV-associated neurocognitive disorders (HAND) (Shiramizu *et al., J Neuropsychiatry Clin Neurosci,* 2009).

Recently, longitudinal data from HIV-infected patient cohorts followed over 5 years have confirmed that HIV DNA in activated monocytes remains high in patients with neurocognitive problems (Kusao *et al., J Neuropsychiatry and Clin Neurosci*, in press). At baseline, HIV DNA in activated monocytes is higher in patients with HIV-associated dementia compared to HIV-infected patients with either minor cognitive motor disorder or normal cognition. Annually, over the 5 years that the patients with high HIV DNA in activated monocytes were followed, lower NPZ8 scores were observed (Fig.1), (Kusao *et al., J Neuropsychiatry and Clin Neurosci,* in press). These data provided the background for a program project grant in collaboration with UCSF and Boston College to study new agents to treat HAND.

Other collaborations include ongoing work with the South East Research Group to study HAND in Thailand and with investigators from the University of Puerto Rico Medical Science Campus. Dr. Shiramizu's research is funded through NIH and supported in part through the UH JABSOM RMATRIX program funded by the National Center for Research Resources.

Dr. Shiramizu has mentored many graduate students in his laboratory and served on numerous graduate degree committees where he provides much guidance and support to help candidates achieve their goals. It is through his direction that his students are recognized in the scientific community. Dr. Shiramizu hopes that the knowledge obtained with his laboratory team will lead to a better understanding of HAND and more effective treatments for patients.

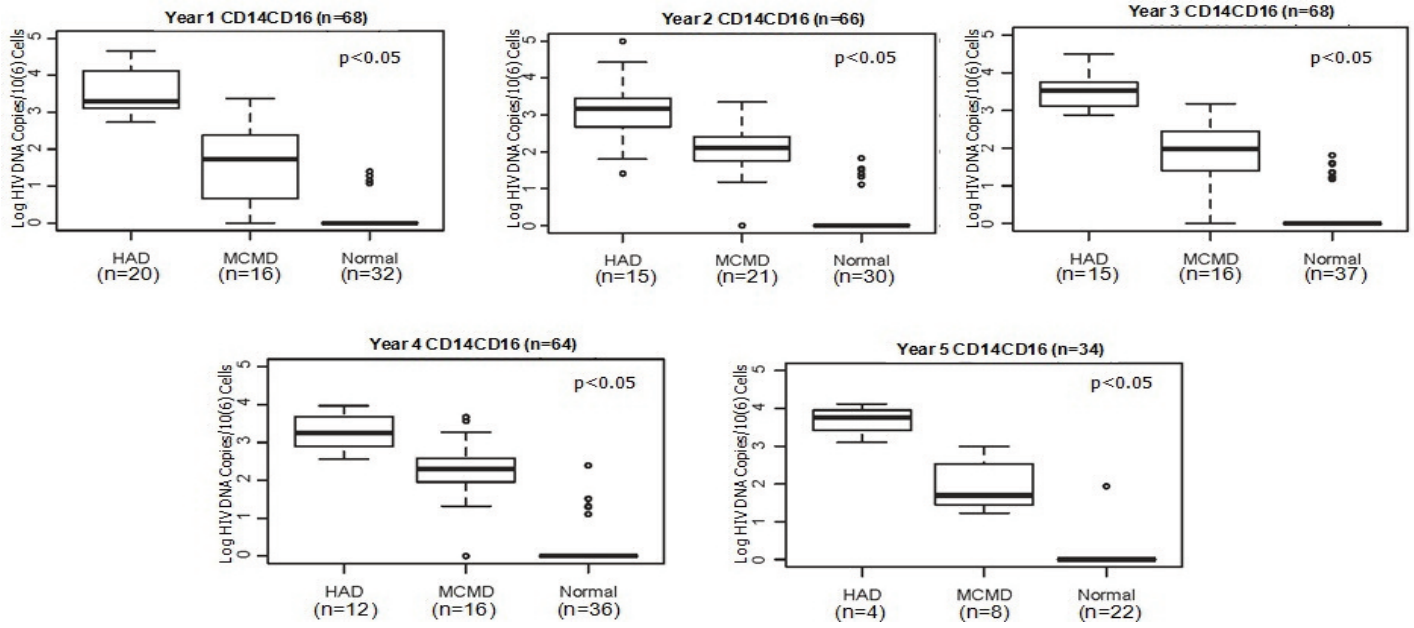


Figure 1. Longitudinal HIV DNA Copy Numbers in Activated Monocytes from the Hawai Aging with HIV Cohort (HAHC). Mean HIV DNA copy numbers per 10(6) activated monocytes (CD14CD16) from each year (Years 1-5) for subjects diagnosed with HIV-associated dementia (HAD), minor cognitive motor disorder (MCMD), and normal cognition.

Classifieds

LABORATORY MANAGER:

A laboratory manager position is immediately available. Candidates must have excellent organizational and technical skills. Duties include ordering and inventory of lab supplies and equipment, safety training and record keeping, general laboratory organization and technical support, computer analysis and graphic presentation of experimental data, assistance with experiments, responsibility for laboratory budget and projections, optimization of laboratory efficiency and maintaining moral of all research personnel. Requirements are Bachelor's degree in biology, molecular biology, or a related field, 3-5 years including managerial responsibilities, experience in the academic environment, excellent organizational skills, including the ability to efficiently evaluate, prioritize and handle multiple and changing projects and priorities, and excellent verbal and written communication skills as to afford effective operational coordination and some scientific writing. Salary is commensurate with credentials and experience. Please contact:

Dr. Johnny J. He
Graduate School of Biomedical Sciences
Department of Cell Biology and Anatomy
University of North Texas Health Science Center
Fort Worth, Texas 76106
Email: johnny.he@unthsc.edu

POSTDOCTORAL FELLOWSHIPS:

The Mount Sinai Institute for NeuroAIDS Disparities (MSINAD) in New York City is seeking applicants for the 2012 class. This 6-week NIMH funded opportunity offers intensive didactic and experiential training in the science and behavioral aspects of neuroAIDS disorders as they pertain to minority populations. All expenses for the summer are paid, and a generous pilot grant is awarded to all scholars. Please contact:

Applications are due Feb 1, 2012 for Summer 2012.
For details & application visit www.msinad.org
or email: desiree.byrd@mssm.edu.

POSTDOCTORAL FELLOWSHIPS:

Postdoctoral positions are available immediately to study virology and pathogenesis of HIV-1 and HCV in the central nervous system. The applicants should have completed their Ph.D., be highly motivated, and have a passion for science with solid laboratory skills in molecular biology, cell biology, and/or biochemistry, and with an English proficiency to communicate effectively. Ph.D. training in the fields of HIV-1/HCV is preferred for the positions. Applicants should email their curriculum vitae, a brief summary of research experience, and contact information for three references in one single PDF file to:

Dr. Johnny J. He
Graduate School of Biomedical Sciences
Department of Cell Biology and Anatomy
University of North Texas Health Science Center
Fort Worth, Texas 76106
Email: johnny.he@unthsc.edu

Contact the ISNV Administrative office at:

Tel: (215) 707-9788 **Fax: (215) 707-9838**

Email: mail@isnv.org **www.isnv.org**

IMM&ID Institute for Molecular Medicine & Infectious Disease

Center For Scientific Communication & Outreach

RCE

Drexel University College of Medicine

ISNV Newsletter printed by the Center for Scientific Communication and Outreach: Director, Michael Nonnemacher, Ph.D.