





# Program

**Continental Breakfast** 8:30 to 9:00 a.m.

**Introductory Remarks** 9:00 to 9:15 a.m.

**Stephen F. Lowry, MD, MBA**

Senior Associate Dean for Education, UMDNJ-RWJMS  
Professor and Chairman, Surgery, UMDNJ-RWJMS

**Terri Goss Kinzy, PhD**

Senior Associate Dean, Graduate School of Biomedical  
Sciences at UMDNJ-RWJMS

Director, RWJMS/Rutgers/Princeton MD/PhD Program

Professor, Department of Molecular Genetics,  
Microbiology, and Immunology, UMDNJ-RWJMS

**Student Presentations (Session 1)** 9:15 to 10:30 a.m.

**Break** 10:30 to 10:40 a.m.

**Student Presentations (Continued)** 10:40 to 11:40 a.m.

**Break** 11:40 to 11:50 p.m.

**Student Presentations (Session 2)** 11:50 to 12:50 p.m.

**Luncheon** 12:50 to 1:50 p.m.

**Dean's Welcome Remarks** 1:50 to 2:05 p.m.

**Peter Amenta, MD, PhD**

Dean, UMDNJ-RWJMS

Professor, Department of Pathology and Laboratory  
Medicine, UMDNJ-RWJMS

**Student Presentations (Session 3)** 2:05 to 3:35 p.m.

**Break** 3:35 to 3:50 p.m.

**Keynote Address** 3:50 to 4:50 p.m.

**Joan C. Marini, MD, PhD**

Chief, The Bone and Extracellular Matrix Branch of  
National Institute of Child Health and Human  
Development at the National Institutes of Health

**“Paradigms for Genetic Bone Disorders and a  
Career in Translational Research”**

**Concluding Remarks** 4:50 to 5:00 p.m.

## **Acknowledgments:**

The MD/PhD Symposium was made possible by the support of Dr. Arnold Rabson, Interim Senior Associate Dean for Research and Dr. Peter Amenta, Dean of UMDNJ-Robert Wood Johnson Medical School.

We would like to acknowledge Dr. Terri Goss Kinzy, Director of the MD/PhD program, and Perry Dominguez, the Program Administrator at RWJMS. We also want to thank Dr. Lori Covey, Rutgers Liason, and Dr. Elizabeth Gavis, Princeton Liason.

Thank you,

Eileen Hwang  
Gerard Limerick  
Peter Mazari  
Jean McGee  
Jay Oza



## **Order of Presentations**

### **Session 1- Part I: Neuroscience**

#### **Session Chair: Gerard Limerick**

**Richard Sun**, “Characterization of genetically-encoded calcium indicators for probing neural activity”

**Issa Bagayogo**, “Regulated release of BDNF by cortical oligodendrocytes is mediated through metabotropic glutamate receptors and the PLC pathway”

**Dakim Gaines**, “The effects of the pesticides maneb and paraquat on the developing dopaminergic system”

**Hilary Grosso**, “Regulation of synucleins by microRNA”

**Christopher Langhammer**, “A neuromuscular junction-based neural interface for neural signal acquisition”

## **Session 1- Part II: Neuroscience**

### **Session Chair: Gerard Limerick**

**Brian Barlow**, “Interactions of prenatal and adulthood pesticide exposures, gender, and ageing: implications for the fetal basis of adult disease hypothesis in a murine model of Parkinson’s disease”

**Jean-Paul Abboud**, “A comprehensive *in silico* model of retinal neurogenesis”

**Eli Levin**, “Neurons express vimentin in Alzheimer’s disease brain as part of a generalized dendritic damage-response mechanism”

**Desmond Brown**, “The role of the primary cilium in Shh signaling and CNS tumorigenesis”

## **Session 2: Virology and Biomedical Sciences**

### **Session Chair: Jean McGee**

**Sean Liu**, “A lipidomic view of Human Cytomegalovirus”

**Eileen Hwang**, “A peptide model of a 9-residue sequence interruption in type IV collagen forms amyloid-like fibrils”

**Peter Mazari**, “Characterization of the novel human-tropic FeLV Envs: CP & L1”

**Matthew Treiser**, “Cytoskeleton-based forecasting of stem cell lineage fates”

## **Session 3: Cancer Biology**

### **Session Chair: Peter Mazari**

**Erin Haley**, “Self-cannibalism and survival of quiescent fibroblasts”

**Jay Oza**, “Role of polycomb group genes in genomic integrity”

**Jean McGee**, “The role of Rif proteins in preferential elongation of short telomeres”

**Kevin Anton**, “Tumor-associated macrophages induce tumor growth and chemoresistant properties in glioblastoma: A role of interleukin-6”

**Nilay Sethi**, “The role of Notch signaling in breast cancer metastasis”

**Shannon Agner**, “Quantitative classification of triple negative breast cancer using DCE-MRI”

## **About the MD/PhD program at RWJMS...**

The University of Medicine and Dentistry of New Jersey- Robert Wood Johnson Medical School was established as a part of Rutgers, The State University of New Jersey in 1966 and is located on the Busch Campus of Rutgers University in Piscataway, New Jersey and adjacent to Robert Wood Johnson University Hospital (RWJUH) and several campuses of Rutgers University in New Brunswick, New Jersey. In 1986, the name of Rutgers Medical School was changed to Robert Wood Johnson Medical School, in honor of Robert Wood Johnson, a former member of the Board of Trustees of RWJUH. Rutgers, The State University of New Jersey was chartered in 1766. It has a unique history as a colonial college, a land-grant institution, and a state university. The MD/PhD program has historically been joint with Rutgers University, owing to the joint nature of all RWJMS-based graduate programs and the historic and physical links of the schools and campuses. Princeton University was chartered in 1746 as the College of New Jersey and renamed Princeton University in 1896 when university status was achieved. Princeton University joined RWJMS and Rutgers University in the MD/PhD program in the fall of 2005 through the Department of Molecular Biology.

The MD/PhD program has existed at RWJMS since its inception, and the MD/PhD program of RWJMS/Rutgers University/Princeton University is based on the strengths of the three universities to create a unique opportunity for trainees to select from among a wide variety of programs and mentors for the PhD portion of the dual degree. The missions of the RWJMS/Rutgers University/Princeton University MD/PhD Program are:

- To train the next generation of physician scientists to advance biomedical research and medical therapy and to provide service to our communities;
- To promote the interdisciplinary research training necessary to capitalize on growing scientific opportunities;
- To support the unique career paths and address the challenges of students in the MD/PhD program;
- To foster a community of researchers, educators, and clinical scientists conducive to the training of program students.

To this end, a core of faculty from the three institutions has been recruited to administrative, support and mentorship roles in the MD/PhD program. These individuals represent a diverse array of scientific disciplines. While each student chooses his or her own laboratory and mentor, all benefit from the support and expertise of the program faculty and the diversity of student interests. Most importantly, the program is designed to maintain integrated training in science and medicine through the first two years of medical school (M1 and M2), the PhD phase (P1-P3), and the final clinical years of medical school (M3 and M4).

The immediate proximity of faculty laboratories at RWJMS and Rutgers University and the close scientific ties to Princeton University build the essential relationships for the MD/PhD program:

- An intensive research experience where the student works closely with a faculty member who serves as a research advisor

- Formal course offerings from both the medical and graduate school curricula
- Seminars, journal clubs, research conferences, and symposia which are held on the campuses
- Attendance and presentations at local and national meetings
- Free and informal access to all training faculty as well as to other members of the academic community on this campus
- An annual symposium where trainees present their research results to the entire program
- Clinical exposure and individualized tutorials in clinical medicine during the PhD years

## **C<sup>2</sup>alc “Clinical Continuity a la carte”**

Once a student enters the PhD phase of the MD/PhD program, the question arises as to how to maintain the “clinical contact” and assure a smooth transition back to the M3 year. As such, the MD/PhD program offers an “a la carte” selection of opportunities to fit the unique interests, needs, and locations of our students. Students do not exercise ALL of these options, but select the best ones for their specific stage of the PhD. The C<sup>2</sup>alc options are below and we welcome student suggestions.

### **Cognitive Skills Tutoring**

- Contact Dr. Norma Saks at: [saks@umdnj.edu](mailto:saks@umdnj.edu)

### **Physical Diagnosis Tutoring**

- Contact Dr. Carol Terregino at: [terregca@umdnj.edu](mailto:terregca@umdnj.edu)

### **PCM Mentor Shadowing**

- Contact your M1/M2 mentors

### **Promise Clinic or Other Service Learning**

- Go to <http://rwjms.umdnj.edu/hiphop/>

### **Interim OSCEs**

- Contact Dr. Carol Terregino at: [terregca@umdnj.edu](mailto:terregca@umdnj.edu)

### **Local Physician Shadowing**

- Contact our clinical program faculty or Perry

## **Abstracts**



## Characterization of genetically-encoded calcium indicators for probing neural activity

Xiaonan Richard Sun, Bernd Kuhn, and Samuel S.-H. Wang

Department of Molecular Biology, Princeton University, Princeton, New Jersey

### Abstract:

Monitoring neural activity is a central problem in neuroscience. Since neural signals (action potentials) produce correlated transient intracellular calcium increases, a variety of methods have been developed for the measurement of calcium signals. While organic calcium indicators (i.e. BAPTA-based indicators) have been widely used in monitoring neural activity, there are numerous limitations in their applications for *in vivo* environments. Some of these limitations include the lack of cell-specificity, wash-out from cells, and difficulties in labeling small structures such as dendrites. In order to overcome these obstacles, it is of great interest to use a class of biosensors known as genetically-encoded calcium indicators (GECIs), which are fusion proteins comprised of GFP-based fluorophores and calcium-sensing peptides. While comparative studies have been performed for most published GECIs, the most recent modifications have not been assessed in standardized settings. Our goal is to use a high-throughput method to identify the GECI with the best overall performance in cultured cells. The GECIs that will be investigated include a troponin-based FRET indicator (TN-XXL) and several unreported versions of a calmodulin-based single-fluorophore indicator (GCaMP). Each GECI of interest was cloned into a mammalian expression vector and were subsequently transfected into HEK-293 cells. Immediately before the experiment, we labeled the transfected cells with the red synthetic calcium indicator X-Rhod-1, whose calcium binding and fluorescence properties have been widely reported. Calcium transients were elicited through ATP-induced release and recording of the fluorescence changes were performed with 2-photon laser scanning microscopy (2PLSM). By using the fluorescence change of X-Rhod-1 as a unit of measurement, we are able to make quantitative comparisons of the fluorescence and calcium binding properties among the tested GECIs. The completion of this screening process would allow us to choose optimal GECIs for further investigation in neural tissue both *in vitro* as well as in the intact animal.

## **Regulated release of BDNF by cortical oligodendrocytes is mediated through metabotropic glutamate receptors and the PLC pathway**

**Issa Papiss Bagayogo and Cheryl F. Dreyfus**

Department of Neuroscience and Cell Biology, UMDNJ-RWJMS, Piscataway, New Jersey

### **Abstract:**

A number of studies suggest that oligodendrocytes (OLGs), the myelinating cells of the central nervous system, are also a source of trophic molecules, such as neurotrophins that may influence survival of proximate neurons. What is less clear is how the release of these molecules may be regulated. My studies investigated effects of BDNF derived from cortical OLGs on proximate neurons as well as regulatory mechanisms mediating BDNF release. I determined that BDNF derived from cortical OLGs increased the numbers of VGLUT1 positive glutamatergic cortical neurons. Furthermore, glutamate acting through metabotropic, and not AMPA/Kainate or NMDA receptors, mediate BDNF secretion. The PLC-pathway is a key mediator of metabotropic actions to release BDNF. Treatment of OLGs with the PLC activator m-3M3FBS induced robust release of BDNF, while inhibitors of the pathway significantly inhibited release. Taken together, my studies suggest that OLG lineage cells release BDNF, a molecule trophic for proximate neurons. BDNF release is regulated by glutamate acting through metabotropic glutamate receptors and the PLC pathway. Thus, glutamate and BDNF may be molecules that support neuron-OLG interactions in the cortex.

## **The effects of the pesticides maneb and paraquat on the developing dopaminergic system**

**Dakim K. Gaines<sup>1,2</sup>, Brian K. Barlow<sup>2</sup>, and Mona Thiruchelvam<sup>2</sup>**

<sup>1</sup>St. Mary's College of Maryland and the RISE Program at Rutgers University and UMDNJ-RWJMS

<sup>2</sup>Environmental and Occupational Health Sciences Institute, UMDNJ-RWJMS, Piscataway, New Jersey

### **Abstract:**

Parkinson's disease (PD) is a neurodegenerative disorder characterized pathologically by the loss of nigral dopaminergic neurons and environmental factors have been widely implicated in the etiology of sporadic PD. Previous studies have shown that exposure to maneb (MB) and paraquat (PQ) (two agrichemicals used on various crops) in mice induces a PD phenotype. Additionally, recent data suggests that exposure to environmental factors early in life can predispose or cause nigral dopaminergic neurodegeneration. The mechanism(s) responsible for this increased risk remains unknown and thus, was the focus of this study. Pregnant dams were treated with either saline, 0.6 mg/kg PQ, 2 mg/kg MB or PQ+MB from gestational day (GD) 10 - 15. Effects of these pesticides on the developing brain were determined on GD16 and GD18 mouse embryos, 24hrs and 72hrs after the last exposure. The end-points that were analyzed include PQ toxicokinetics, proteasomal function, and levels of dopamine, the dopamine metabolites DOPAC and HVA, serotonin, and the serotonin metabolite HIAA. Paraquat was detected in brain of embryos from dams treated with either PQ or PQ+MB, both at GD16 and GD 18, with higher levels observed at the earlier age. Embryos from dams treated with MB showed an increase in proteasomal activity at GD18. A decrease in serotonin occurred in embryos from MB and PQ+MB treated dams at GD16 and GD 18, while a decrease in dopamine, DOPAC, and HIAA was observed only in GD18 embryos. It was also determined that there were no significant gender-related in effects. To investigate dose-dependent effects of MB, and to determine whether the effects of MB generalize to other ethylene-bis-dithiocarbamates (EBDCs) or might be due to a metabolite of EBDCs, additional studies used a similar dosing paradigm with 1,2,4, or 6 mg/kg MB, the EBDC nabam, or the metabolite ETU, at the GD18 timepoint. Results of those experiments for neurochemistry and proteasome function were consistent with previous results, suggested dose dependence, suggested generalization to other EBDCs, and suggested the parent EBDC rather than the metabolite to be the neurotoxic compound. The results of these studies give us additional mechanistic insight into how exposure to environmental factors can alter neurodevelopment, and subsequently increase the risk of PD onset later in life. Furthermore, future studies may identify specific targets, which can eventually be used or manipulated with specific interventions that can alter disease progression. Supported by NIH ES016277 (MT) and ES159792 (BKB). \*

\* adapted from Abstract 1694, The Toxicologist CD — An official Journal of the Society of Toxicology, Volume 102, Number S-1, March 2009

## **Regulation of synucleins by microRNA**

**Hilary Grosso and M. Maral Mouradian**

Department of Neuroscience, UMDNJ-RWJMS, Piscataway, New Jersey

### **Abstract:**

MicroRNA is a non-coding, conserved type of RNA that recognizes the 3' untranslated region (3'-UTR) of mRNA and regulates protein expression either by degrading the mRNA or interfering with translation. MicroRNA has been suggested to play an important role in biological functions through its regulation of protein expression. Our lab has recently demonstrated this point with microRNA-7 (miR-7) and  $\alpha$ -synuclein ( $\alpha$ -syn).  $\alpha$ -Syn is a protein of unknown function that plays an important role in Parkinson's disease (PD). This protein is a major component of Lewy bodies, the proteinaceous aggregates that are the pathological hallmark of PD. Additionally,  $\alpha$ -syn gene multiplication causes autosomal dominant PD in humans, and over-expression of human  $\alpha$ -syn in animal models is deleterious to dopaminergic neurons. These observations indicate that  $\alpha$ -syn is a toxic molecule and that gene dosage is an important factor in this toxicity. Our lab showed that miR-7 can inhibit  $\alpha$ -syn expression and that this down-regulation is protective against  $\alpha$ -syn-induced cytotoxicity.

b-Synuclein (b-syn) is a gene predicted to have a protective role in PD. Over-expression of b-syn in  $\alpha$ -syn over-expression models ameliorates  $\alpha$ -syn toxicity and prevents  $\alpha$ -syn aggregation. We hypothesized that microRNAs could regulate b-syn expression. Using publicly available prediction algorithms, we identified microRNAs that could potentially bind the 3'-UTR of b-syn, and thus could regulate its expression. We chose candidate microRNAs based on the conservation of their target sequences in the b-syn 3'-UTR across multiple species from humans to lizards, and their prediction being common to multiple algorithms. The aim of my work is to demonstrate in cultured cells that these microRNAs regulate b-syn expression, and to determine whether this regulation can impact  $\alpha$ -syn toxicity.

## **A neuromuscular junction-based neural interface for neural signal acquisition**

**Christopher Langhammer<sup>1</sup> and Bonnie Firestein<sup>1,2</sup>**

<sup>1</sup>Biomedical Engineering, Rutgers University, Piscataway, New Jersey

<sup>2</sup>Cell Biology and Neuroscience, Rutgers University, Piscataway, New Jersey

### **Abstract:**

The ideal neural interface is a bidirectional transducer that establishes contact between a technical device and neural structures within the body. The objective of such devices is to record bioelectrical signals from the nervous system or to implant such signals in order to restore motor and sensory function in disabled patients. Research in the fields of neural interfaces and neural prostheses focuses on restoring motor and sensory function in patients with limb amputations, spinal cord injury, stroke, and degenerative diseases. However, advances in these fields have thus far translated into only modest clinical improvements despite the technologies' tremendous potential. A modification of the known "cultured-probe" design, a neural interface in which neurons cultured directly onto an electrode surface prior to implantation facilitate incorporation into the host nervous system, may significantly improve the recording capabilities of current neural interfaces. By using myotubes rather than neurons as the electrogenic cell type cultured onto the electrode surface and by targeting the peripheral nervous system as the implantation site, we hope to overcome many of the critical barriers to progress in this field. In this project, we aim to develop a modified planar microelectrode array (MEA) designed to facilitate integration of muscle cells (myotubes) grown in culture. The MEA surface will be tailored specifically to improve the sealing between myotubes and electrodes for improvement upon the ability of current devices to distinguish electrical activity of individual cells. Preliminary results demonstrate the feasibility of using the device to record the activity in a single neural circuit connecting a spinal cord motoneuron and a myotube. We are using a highly reductionist approach to a complex clinical problem to demonstrate that recording motor intention along its final common pathway is an achievable goal.

## **Interactions of prenatal and adulthood pesticide exposures, gender, and ageing: implications for the fetal basis of adult disease hypothesis in a murine model of Parkinson's disease**

**Brian K. Barlow<sup>1,2</sup>, Jaspreet Kochar<sup>2</sup>, Mona Thiruchelvam<sup>2</sup>**

<sup>1</sup>MD/PhD Program, Graduate School of Biomedical Sciences at Robert Wood Johnson Medical School, Piscataway, New Jersey

<sup>2</sup>Department of Environmental and Occupational Medicine, Environmental & Occupational Health Sciences Institute, a joint institute of Rutgers University and the UMDNJ-RWJMS, Piscataway, New Jersey

### **Abstract:**

Environmental insults during periods of neurodevelopment have been hypothesized to initiate or cause susceptibility to neurodegenerative processes, such as the degeneration of the nigrostriatal dopamine system seen in Parkinson's disease (PD). Using two pesticides that are associated with PD in epidemiological studies and in animal models, we've previously shown that prenatal exposure to the fungicide maneb (MB) increases susceptibility to the neurotoxic effects of adulthood paraquat (PQ) exposure. This multiple hit paradigm was hypothesized to be a useful model for investigating a relationship between PD and the developmental origins of adult disease hypothesis. Pregnant C57Bl/6 mice were injected with saline or 2 mg/kg MB on gestation days 10-18. The large cohort of male and female offspring generated was divided into subgroups who were injected with saline or PQ (10 mg/kg) every-third-day from 7 to 13.5 weeks of age (total of 15 doses), and sacrificed at 3, 15, 30, or 45 weeks after the final injection (up to ~14 months of age); for one subset of animals, sacrificed at the latest time-point, open field activity assessments were made during the exposure period and coinciding with each of the other time-points. Late-appearing (not until 30-weeks post-PQ exposure) and persistent robust behavioral changes (e.g. ~30-40% reductions, relative to other groups, in ambulatory distance in a 1-hour session) were observed only in males with prenatal-MB + adulthood-PQ exposure, whereas females did not exhibit this phenotype. Analysis of striatal dopamine, the dopamine metabolites DOPAC and HVA, serotonin, and the serotonin metabolite HIAA did not reveal any significant effects of prenatal or adulthood exposure on levels of the parent neurotransmitters dopamine or serotonin; however, significant interactions of gender, prenatal exposure, adult exposure, and/or ageing were noted for DOPAC, HVA, dopamine turnover, HIAA, and serotonin turnover. The results suggest that whereas striatal neurochemical effects of adulthood PQ exposure in prenatal-saline exposed animals are transient (i.e. returning to normal within several weeks of cessation of exposure), males and females exposed to PQ in the context of prenatal-MB exposure show long-lasting disruptions (notable 45-weeks post-PQ). Activity of the 20S proteasome was also assessed in striatal tissue, and decreases were noted for prenatal-MB males at 3 weeks. At 45 weeks, however, prenatal-MB + adulthood-PQ males and females exhibited relative decreases in proteasome activity, with a greater effect seen in males, suggesting interactions of exposure history, gender, and ageing. The results of this study, combined with other results in our lab, suggest that prenatal MB exposure increases the neurotoxic effects of later PQ exposure, that gender modulates the effect, and that ageing exacerbates these interactions. The emergence of a behavioral phenotype, disruptions in striatal dopamine and serotonin homeostasis, and striatal proteasome dysfunction are suggestive of a neurodegenerative process that may model PD or act as a risk factor for the disorder in the context of neurodevelopmental and adulthood environmental "multiple hits." Further work will determine the applicability of this model to linking the FeBAD hypothesis with PD. Supported by NIH ES016277 and ES159792.

## A comprehensive *in silico* model of retinal neurogenesis

Jean-Paul J. Abboud<sup>1,2</sup>, Oliver Camand<sup>2</sup>, Zhenhong Bao<sup>2</sup>, Nancy L. Hayes<sup>2</sup>,  
and Richard S. Nowakowski<sup>1,2</sup>

<sup>1</sup>Biomedical Engineering, Rutgers University, Piscataway, New Jersey

<sup>2</sup>Neuroscience and Cell Biology, UMDNJ-RWJMS, Piscataway, New Jersey

### **Abstract:**

The retina is a self-contained outgrowth of the neural tube. Within the developing retina the only proliferative zone, the ventricular zone, produces all 7 retinal cell types (6 neuronal, 1 glial). In the mouse, the full complement of cells is produced over ~22 cell cycles in a specific order from embryonic day 10 until postnatal day 11. This work proposes a biologically realistic model of this cell production process based on the concept that neurogenesis is generally the result of the 3 independent decisions made by each progenitor cell: 1) cell cycle exit decision, 2) cell fate decision, and 3) cell death decision. While each decisional step has been studied independently, this is the first generalized model incorporating all 3 developmental steps to be constructed for any CNS structure. A comprehensive *in silico* model of mouse retinal neurogenesis that accounts for both cell number and cell class is proposed. The *in silico* model itself has 3 steps: Step 1 reflects the systematic changes in the proportion of daughter cells that remain vs. those that quit the cell cycle. Step 2 reflects a set of probability density functions that describe the cell fate per cell cycle for each of the retinal cell classes. Step 3 reflects the effect of apoptosis on overall cell survival. The model makes allowances for biological processes, such as the natural variations in the types of cell division (symmetric vs. asymmetric), the expansion of the cell population (~150), and proper retinal cell class distributions. The model can simulate the diversity of developmental processes in retinal neurogenesis in central vs. peripheral retina and in different species. Mouse knockouts are exploited to evaluate the biological realism of the model and to evaluate the biological functions of genes involved in retinal development.

## **Neurons express vimentin in Alzheimer's disease brain as part of a generalized dendritic damage-response mechanism**

**Eli C. Levin and Robert G. Nagele**

NJ Institute for Successful Aging, UMDNJ-School of Osteopathic Medicine,  
Stratford, New Jersey

### **Abstract:**

Early pathological features of Alzheimer's disease (AD) include synaptic loss, dendrite retraction and eventual neuronal loss. How neurons respond to evolving AD pathology remains elusive. In the present study, we used single- and double-label immunohistochemistry to investigate the relationship between neuronal vimentin expression and local brain pathology. Vimentin was localized to neuronal perikarya and especially dendrites in AD brain, with vimentin-immunopositive neurons prevalent in regions exhibiting intra- and extracellular beta-amyloid<sub>1-42</sub> (ab42) deposition. Co-localization of vimentin and ab42 in neurons was common in the cerebral cortex, cerebellum and hippocampus. Additionally, neurons in affected brain regions of AD transgenic (Tg2576) mice and in brain tissue subjected to mechanical injury expressed vimentin, while those in comparable regions of control mouse brain did not. Finally, we show that neurons in human fetal brain express vimentin concurrently with periods of rapid neurite extension. Overall, our results suggest that neurons express vimentin as part of an evolutionarily conserved, damage-response mechanism which recapitulates a developmental program used by differentiating neurons to establish dendrites and synaptic connections.

## **The role of the primary cilium in Shh signaling and CNS tumorigenesis**

**Desmond A. Brown and Jonathan Eggenschwiler**

Department of Molecular Biology, Princeton University, Princeton, New Jersey

### **Abstract:**

Sonic hedgehog (Shh) is an evolutionarily conserved protein with widespread functions in embryonic development. This function hinges at least in part on the ability of Shh to regulate the differentiation of stem cells. Uncontrolled activity of the Shh pathway has been implicated in roughly a quarter of all human tumors! This is particularly true of tumors of the central nervous system. Despite the central role that Shh signaling plays in human cancers, much of the mechanistic details remain elusive. There is abundant evidence suggesting that the primary cilium plays a critical role in Shh signaling. I will explore the role of this organelle in the Shh pathway. Electron microscopy will be used to investigate at high resolution, the spatial organization of Shh components that localize to the primary cilium. I will also explore the dynamics of the localized components using fluorescence recovery after photobleaching (FRAP). Finally, the relevance of the Shh signaling system in both medulloblastoma and glioblastoma multiforme will be investigated using both cell culture and animal models and will be based in part on employing both genetic and physical (laser microdissection) techniques to disrupt cilia formation in a variety of contexts. This work will contribute to our understanding of Shh signaling in mammals and specifically to its role in human tumors and, may eventually pave the way for the development of new therapeutic avenues in the ongoing battle against cancer.

## **A lipidomic view of Human Cytomegalovirus**

**Sean T. Liu<sup>1</sup>, Thomas E. Shenk<sup>1</sup>, Joshua Rabinowitz<sup>1</sup>, and Alex Brown<sup>2</sup>**

<sup>1</sup>Department of Molecular Biology, Princeton University, Princeton, New Jersey

<sup>2</sup>H. Alex Brown, Department of Pharmacology, Vanderbilt University School of Medicine, Nashville, Tennessee

### **Abstract:**

Human cytomegalovirus (HCMV) is an enveloped, double-stranded DNA virus belonging to the beta-herpesviridae family. HCMV is estimated to affect over 50% of the adult population and poses a serious threat to the immune-compromised community. By using cutting-edge liquid chromatography tandem mass spectrometry techniques, we have been able to monitor the levels of 146 glycerophospholipid species in human foreskin fibroblasts during infection with HCMV. Our results show that HCMV uniquely and significantly modulates the cellular lipidome. Finding the biological significance of these changes has led towards a lipidomic analysis of purified virions to see whether the observed changes directly correlate with the composition of the virion's outer envelope. Furthermore, inhibitor studies and knockdown studies targeting key cellular enzymes in lipid metabolic pathways hope to reveal essential players in viral replication that may potentially lead to novel drug targets.

## **A peptide model of a 9-residue sequence interruption in type IV collagen forms amyloid-like fibrils**

**Eileen Hwang and Barbara Brodsky**

Department of Biochemistry, UMDNJ-RWJMS, Piscataway, New Jersey

### **Abstract:**

Peptide models in the Gly-Xaa-Yaa repeating sequence of collagen: association to higher order structure. Eileen Hwang, Geetha Thiagarajan, Barbara Brodsky Close packing of three polyproline II-like helices to form a prototypic collagen triple-helix requires a perfect (Gly-Xaa-Yaa)<sub>n</sub> repeating sequence, yet all non-fibrillar collagens contain interruptions in this tripeptide repeating sequence. Interruptions can range in length from 1 residue to more than 30 residues, but more than 75% contain 9 residues or less between (Gly-Xaa-Yaa)<sub>n</sub> domains. Previous studies on peptides with 1-residue and 4-residue interruptions showed only a localized perturbation within the triple helix. These studies were extended to introduce interruptions of 3, 5, 6, 7, 8 and 9 residue lengths from collagen chains within a fixed peptide context (Gly-Pro-Hyp)<sub>5</sub>-INT-(Gly-Pro-Hyp)<sub>4</sub>. All peptides with interruptions appeared to form a triple-helix molecule with hydrodynamic properties similar to that of a standard triple-helix, but with decreased triple-helix content, stability, and calorimetric enthalpy. Surprisingly, the peptide with a 5-residue interruption, whose sequence is equivalent to Gly missense mutation as seen in genetic diseases of collagen, showed the least perturbation, with a CD spectrum and enthalpy similar to a typical triple-helix but a decreased thermal stability. The peptides containing 8- and 9-residue interruptions exhibited a strong propensity for self-association to form fibrous structures. A small peptide containing only the 9-residue sequence also aggregated into an amyloid-like structure. Interruptions within the Gly-Xaa-Yaa repeating pattern have been suggested to play a role in molecular flexibility, collagen degradation, development, and ligand binding. The results reported here suggest interruptions in the 8- to 9-residue size range may also play a role in the molecular association to higher order tissue structures of non-fibrillar collagens.

## **Characterization of the novel human-tropic FeLV Envs: CP & L1**

**Peter Mazari<sup>1</sup>, Daniela Linder-Basso<sup>1</sup>, Anindita Sarangi<sup>1</sup>, Takele Argaw<sup>2</sup>, Carolyn Wilson<sup>2</sup>, and Monica Roth<sup>1</sup>**

<sup>1</sup>Department of Biochemistry, Graduate School of Biomedical Sciences  
UMDNJ-RWJMS, Piscataway, New Jersey

<sup>2</sup>Division of Cellular and Gene Therapies, Center for Biologics Evaluation and  
Research, US Food and Drug Administration, Bethesda, Maryland

### **Abstract:**

Our laboratory has developed a system to retarget retroviral entry through the screening of random libraries generated within the receptor-binding domain of the Feline Leukemia Virus (FeLV) envelope protein (Env). This has led to the isolation of several novel Envs that utilize non-cognate receptors. Two of these Envs (CP and L1) produce high titers on human cell lines and are of particular interest for their potential role in gene delivery both in vitro and in vivo.

CP is a broad tropism, and produces extremely high titers on several human cell lines. This high efficiency makes it an excellent vehicle for gene delivery. Isolation and characterization of the CP receptor will be discussed.

L1 has a very narrow tropism, and is only capable of infecting 293T and the human osteosarcoma cell line 143B at appreciable levels. This selectivity suggests that L1 is utilizing a receptor that is tissue (bone) or even disease (osteosarcoma) specific. Preliminary work to characterize the L1 receptor will be discussed.

## Cytoskeleton-based forecasting of stem cell lineage fates

Matthew D. Treiser<sup>1</sup>, Eric Yang<sup>1</sup>, Simon Gordonov<sup>1</sup>, Daniel M. Cohen<sup>2</sup>, Ioannis Androulakis<sup>1,3</sup>, Joachim Kohn<sup>4</sup>, Christopher S. Chen<sup>2</sup>, and Prabhas V. Moghe<sup>1,3</sup>

<sup>1</sup>Biomedical Engineering, Rutgers University, Piscataway, New Jersey

<sup>2</sup>Bioengineering, University of Pennsylvania, Philadelphia, Pennsylvania

<sup>3</sup>Chemical Biochemical Engineering, Rutgers University, Piscataway, New Jersey

<sup>4</sup>NJ Center for Biomaterials, Rutgers University, New Jersey

### Abstract:

While the differentiation of stem cells in response to substrata and soluble factors is widely studied, the mechanisms by which these signals are propagated from stimulus to differentiation remain poorly understood. This study reports that changes in cell shape and cytoskeletal organization that occur within hours of stimulation forecast the lineage commitment fates of human mesenchymal stem cells (hMSCs). To capture these defining morphological changes, a single cell imaging approach was employed. This approach captures numerous early (24 h), quantitative features of actin fluororeporter shapes, intensities, textures and spatial distributions (collectively termed morphometrics). An established approach was used to reduce these morphometrics into “combinations” that then allowed the identification of distinct subpopulations of cells featuring unique combinations. We demonstrate that whereas hMSCs cultured on fibronectin-treated glass substrates under environments permissive to bone lineage induction could not be discerned within the first 24 hours from those cultured in basal- or fat-inductive conditions based on traditional cell shape analysis, they could be readily discerned by certain cytoskeletal feature combinations. We extend the utility of this approach to forecast stem cell lineage fates under conditions permissive to both bone and adipogenic lineages. Within the first 24 hours following stem cell seeding across a series of synthetic polymeric materials with a diverse range of physicochemical properties, we could successfully “profile” the substrate responsiveness prospectively in terms of the degree of bone versus fat predisposition (0.87 Pearson correlation coefficient for 2 week outcomes). The morphometric methodology also provided insights into how substrates may modulate the pace of lineage specification. Cells on glass substrates deficient in fibronectin showed a similar divergence of lineage fates, but delayed beyond 48 hours. In summary, this high content imaging and single cell modeling approach may be a valuable framework for elucidating and manipulating determinants of stem cell behaviors, as well as for accelerated screening of stem cell targeted regenerative materials and drugs.

## **Self-cannibalism and survival of quiescent fibroblasts**

**Erin M. Haley, Johanna M. Scarino, Noah W. Brown, and Hilary A. Collier**

Department of Molecular Biology, Princeton University, Princeton, New Jersey

### **Abstract:**

Many cells in the human body are able to exit the cell cycle and enter a state of reversible cell cycle exit termed quiescence. Lack of nutrients causes cells to enter quiescence and to activate a survival mechanism known as autophagy, in which cells sequester cytoplasm for degradation at the lysosome with reclamation of amino acids. Autophagy may represent one way that cancer cells survive in the harsh conditions present in a solid tumor, and quiescence may be a way that cancer stem cells avoid death by traditional chemotherapeutic agents. We sought to define the role for autophagy in quiescence in primary human fibroblasts induced into quiescence by contact inhibition. We found that contact-inhibited fibroblasts are surprisingly metabolically active, yet still induce autophagy. Autophagy induction in quiescence does not proceed through the classical pathway involving mammalian target of rapamycin kinase inhibition. We hypothesize that other molecules in the autophagy signaling cascade, like the sole autophagy kinase ULK1 or its binding partner Atg13, may regulate autophagy induction in quiescence. Fibroblasts may induce autophagy upon quiescence not to meet metabolic demands, but as protection against various damaging agents such as reactive oxygen species (ROS). Consistent with this hypothesis, quiescent fibroblasts maintain viability when treated with hydrogen peroxide at concentrations that induce rapid death in proliferating fibroblasts. Our studies have begun to suggest some approaches, such as induction of autophagy and increased expression of specific ROS detoxifying enzymes including manganese superoxide dismutase, that quiescent cells may use to ensure that cellular integrity is maintained. We anticipate that pathways used by quiescent cells to maintain viability may be co-opted by tumor cells to ensure survival during dormancy and to resist chemotherapy.

## **Role of polycomb group genes in genomic integrity**

**Jay Oza, Vasudeva Ginjala, and Shridar Ganesan**

The Cancer Institute of New Jersey, RWJMS, New Brunswick, New Jersey

### **Abstract:**

Genomic integrity is central to the maintenance of normal cells and numerous types of cancer are associated with some form of DNA damage - mutations, translocations, insertions, amplifications, deletions etc. Hence our cells have evolved to have numerous mechanisms to maintain genomic integrity. Disruptions in these pathways lead to genomic instability and are often associated with cancer. For example, mutations in BRCA1, BRCA2, ATM, p53, BLM, WRN, FANC – important DNA-repair genes, are associated with cancer-prone syndromes. Mechanistically, DNA-damage manifests its carcinogenic effects by either down-regulating the expression of tumor-suppressors or by overexpression of oncogenes. Another mechanism which can potentially alter gene expression, but without altering the DNA sequence itself, is through the epigenetic modification of chromatin. One of the major group of epigenetic regulators of gene expression is the polycomb group (PcG) proteins. PcG genes are thought to mediate transcriptional silencing and have been implicated in the control of embryogenesis, development, stem-cell self renewal, heritable epigenetic states, as well as cell proliferation and cancer.

It is the link between genomic integrity and these epigenetic factors that lay the context of the present work. It was observed in the Ganesan lab that polycomb factors such as CBX2 and BMI-1 localize to the sites of DNA-damage. We further found that other polycomb members such as CBX6, CBX7 and CBX8 also localize to sites of UV induced DNA-damage. High resolution time-lapse confocal imaging using CBX8-GFP reporter revealed that CBX8 localizes to sites of DNA-damage within 30 seconds. Our preliminary results suggest that CBX8 localization to sites of DNA-damage is independent of 53BP1 or H2AX since MEFs deficient in these important mediator and sensor proteins of DNA-damage, respectively, exhibit CBX8 localization similar to HeLa or wild-type MEFs. We further plan to investigate which structural features of these proteins are responsible for their localization to DNA-damage and what their function is at these sites.

## The role of Rif proteins in preferential elongation of short telomeres

Jean S. McGee and Virginia A. Zakian

Department of Molecular Biology, Princeton University, Princeton, New Jersey

### **Abstract:**

In both lower and higher eukaryotes, telomerase preferentially elongates short telomeres. Consistent with previous findings, I propose that the difference in the binding level and activity of Rif1p and Rif2p, negative regulators of telomerase, on short telomeres marks those telomeres for preferential telomerase recruitment. I hypothesize that the reduced Rif2p binding at short telomeres recruits Tel1p, an ATM/ATR-like kinase shown to be necessary for recruiting telomerase subunits to short telomeres. Tel1p subsequently phosphorylates Rif1p and this phosphorylation relieves the ability of Rif1p to inhibit telomerase in *cis*. According to this model, the inactivation rather than the displacement of Rif1p allows telomerase recruitment and activation. I propose to test this hypothesis with the following aims 1) confirm the reduced Rif2p and WT-level Rif1p binding to short telomeres using chromatin immunoprecipitation and quantitative PCR 2) determine if Rif1p is regulated by Tel1p-dependent phosphorylation by employing synthetic dosage lethality, and 3) assess if Rif1p phosphorylation affects telomerase action by performing site-directed mutagenesis of the Tel1p consensus sites on Rif1p, and then analyzing telomere lengths. As telomerase is active in more than 85% of all human cancers, the study of its regulation will provide important insights into cancer biology.

## **Tumor-associated macrophages induce tumor growth and chemoresistant properties in glioblastoma: A role for interleukin-6**

**Kevin Anton<sup>1</sup>, Debabrata Banerjee<sup>1,2</sup>, and John Glod<sup>1,3</sup>**

<sup>1</sup>Pharmacology, UMDNJ-RWJMS, Piscataway, New Jersey

<sup>2</sup>Medicine, The Cancer Institute of New Jersey, New Brunswick, New Jersey

<sup>3</sup>Pediatrics, Division of Hematology and Oncology, Cancer Institute of New Jersey, New Brunswick, New Jersey

### **Abstract:**

Interactions between a tumor and cells of the tumor microenvironment influence tumor growth, invasion, angiogenesis, and metastasis. In addition, the tumor microenvironment provides a level of chemoresistance to the tumor. Tumor-tumor microenvironment cross-talk can occur through cell-cell contact or the release of soluble factors, which bind to their respective receptors in an autocrine or paracrine manner.

The tumor microenvironment is composed of many different cell types, including tumor-associated macrophages (TAMs), fibroblasts, mesenchymal stromal cells (MSCs), and tumor cells. Some tumor types, such as glioblastoma multiforme and breast carcinoma, contain a stroma dominated by a large population of macrophages. Macrophages have the capacity to both support and impede tumor growth under various circumstances. The most well characterized role of macrophages in the tumor microenvironment is the production of angiogenic factors. Bone marrow-derived mesenchymal stem cells (MSCs) are pluripotent cells that localize to solid tumors when administered i.v. The interactions with the tumor microenvironment that promote homing of MSCs as well as their functional roles have not been well characterized.

We propose that the interaction between tumor cells, macrophages, and MSCs generates a microenvironment that promotes tumor growth and survival.

Using co-culture of macrophages, MSCs, and a glioma cell line in an attempt to partially recapitulate the tumor microenvironment leads to significant changes in the profile of secreted cytokines compared to each of these cell types cultured in isolation. Among these changes is an increase in interleukin-6 (IL-6) production. IL-6 functions in B-cell development, myeloid lineage maturation, bone absorption, hepatic function, and acute phase responses. In addition, multiple studies have shown IL-6 to be a potent growth factor for several cancers. We provide evidence that soluble factors produced by macrophages can provide chemoresistance to glioblastoma cells treated with Etoposide.

## The role of Notch signaling in breast cancer metastasis

Nilay Sethi and Yibin Kang

Department of Molecular Biology, Princeton University, Princeton, New Jersey

### Abstract:

The Notch signaling pathway regulates a broad spectrum of cell-fate decisions during development and postnatal life by facilitating communication between neighboring cells. In addition to its conventional role in embryonic development, the pathway is now being recognized for its aberrant activation in cancer. Although an oncogenic role for Notch was first discovered by uncovering activating mutations in the *notch1* gene locus of more than 50% of T-cell acute lymphoblastic leukemia patients, the mechanism underlying its contribution to solid tumor malignancies, such as breast cancer, remain unknown as mutations in the Notch receptors have not been revealed in patients.

Investigations in our laboratory have addressed this outstanding question by demonstrating that upregulation of the Notch ligand Jagged1 supports breast cancer metastasis to the bone by facilitating tumor-stroma interactions. We show that elevated expression of Jagged1 correlates with increased bone metastatic ability across a panel of breast cancer cell-lines, sublines, and progression series. We further demonstrate that Jagged1 expression is induced in breast cancer cells upon stimulation by TGF $\beta$ , an important mediator of breast cancer metastasis to the bone, providing a possible mechanism of aberrant Jagged1 expression in the primary tumor. Using our *in vivo* mouse xenograft model, we demonstrated that enforced expression of Jagged1 in a mildly metastatic breast cancer subline promotes metastasis to the bone by activating Notch signaling in the bone stromal microenvironment. Conversely, shRNA-mediated knockdown of Jagged1 in a highly bone metastatic subline reduces metastatic burden in mice. We next tested whether inhibition of Notch in the tumor microenvironment using a gamma-secretase inhibitor will reduce tumor burden in mice inoculated with a bone metastatic breast cancer subline with high endogenous expression of Jagged1. Consistent with our hypothesis, inhibition of Notch via gamma-secretase treatment prevented bone metastasis without affecting primary tumor growth and furthermore rescued the bone metastatic phenotype observed in mice inoculated with Jagged1-overexpressing breast cancer cells. Mechanistically, *in vitro* functional studies demonstrated that Jagged1-expressing tumor cells have a growth advantage and stimulate osteoclastogenesis when cocultured with bone-specific cells. Importantly, clinical evaluation of tumor samples demonstrated that elevated Jagged1 expression predated a poor survival rate in breast cancer patients.

Taken together, our investigation supports a novel mechanism of breast cancer metastasis in which TGF $\beta$ -mediated induction of Jagged1 in tumor cells facilitates colonization of the bone microenvironment by supporting tumor growth and osteoclastogenesis. Furthermore, we reveal that this ligand-dependent activation of Notch signaling in the bone microenvironment is subject to pharmacological inhibition, providing preclinical evidence for gamma-secretase inhibitors as potential therapy against metastasis in breast cancer patients with aberrant Notch ligand expression.

## Quantitative classification of triple negative breast cancer using DCE-MRI

Shannon C. Agner<sup>1</sup>, Jun Xu, Hussain Fatakawala<sup>1</sup>, Shridar Ganesan<sup>1</sup>, Sarah Englander<sup>2</sup>, Mark Rosen<sup>2</sup>, Kathleen Thomas<sup>2</sup>, Mitchell Schnall<sup>2</sup>, Michael Feldman<sup>2</sup>, John Tomaszewski<sup>2</sup>, and Anant Madabhushi<sup>1</sup>

<sup>1</sup>Biomedical Engineering, Rutgers University, Piscataway, New Jersey

<sup>2</sup>University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania

### **Abstract:**

Triple-negative (TN) breast cancer has been the subject of much research recently due to its lack of response to receptor-targeted therapies and its aggressive clinical nature. In this study, we evaluate the ability of a computer-aided diagnosis (CAD) system to not only distinguish benign from malignant lesions on dynamic contrast-enhanced magnetic resonance imaging (DCE-MRI), but also to quantitatively distinguish triple negative breast cancers from other molecular subtypes of breast cancer. 41 breast lesions (24 malignant, 17 benign) as imaged on DCE-MRI were included in the dataset. Of the 24 malignant cases, 13 were of the TN phenotype. Following quantitative morphological, textural, and kinetic feature extraction, a support vector machine classifier was employed to distinguish (a) benign from malignant lesions and (b) TN from non-TN cancers. In the former case, the classifier yielded an accuracy of 83%, sensitivity of 79%, and specificity of 88%. In distinguishing TN from non-TN cases, the classifier had an accuracy of 92%, sensitivity of 92%, and specificity of 91%. The results suggest that the TN phenotype has distinct and quantifiable signatures on DCE-MRI that will be instrumental in the early detection of this aggressive breast cancer subtype.



## **MD/PhD Student Biographies**



**Pre-MD/PhD Student**

## Dakim Gaines

- **Hometown:** Scotch Plains, NJ
- **College(s) Attended and Degree(s) Earned:** St. Mary's College of Maryland/ BA in Biology
- **Year in MD/PhD Program:** Currently applying to MD/PhD programs
- **PhD Thesis Laboratory:** N/A
- **Area of Interest in Clinical Practice:** Emergency Medicine/ Trauma
- **Area of Interest in Research:** Cell Physiology/ Molecular Biology
- **Personal Information:** I just graduated college and plan (hope) to become an MD/PhD student at Robert Wood Johnson in 2010. I have been interested in medicine since my senior year of high school after completing the mini-med school program at Robert Wood Johnson. I love to play and watch sports and I'm die-hard New York Yankee and Dallas Cowboy fan.

## **Incoming Students**

## Kathleen Capaccione

- **Hometown:** Bridgewater, NJ
- **College(s) Attended and Degree(s) Earned:** New York University, College of Arts and Science, 2009
- **Year in MD/PhD Program:** Medical School, Year 1
- **PhD Thesis Laboratory:** Currently rotating in Dr. Collier's laboratory at Princeton, undecided about PhD lab
- **Area of Interest in Clinical Practice:** Oncology
- **Area of Interest in Research:** Cell cycle control
- **Personal Information:** I am thrilled to be a part of the MD PhD program at RWJMS so that I can combine my passion for research with a career of service as a physician. My past research includes investigation of the evolutionary biology of the cavefish *Astyanax mexicanus*, research into the human visual system using functional MRI, and the use of bioimpedance to assess gastric motility. In my spare time, I enjoy reading, visiting friends and family, going to New York City, and learning to play the guitar. Formerly known as the New York University Mascot, the Bobcat, I hope to use my experience as a furry good-will ambassador to volunteer with pediatric cancer patients to bring them smiles and laughter.

## Jinesh Gheeya

- **Hometown:** Ahmedabad, Gujarat, India
- **College(s) Attended and Degree(s) Earned:** University of California at Davis; B.S. in Genetics with high honors
- **Year in MD/PhD Program:** MS1
- **PhD Thesis Laboratory:** N/A
- **Area of Interest in Clinical Practice:** Oncology
- **Area of Interest in Research:** Oncogenomics / Epigenetics
- **Personal Information:** Jinesh grew up in Ahmedabad, India. He immigrated to USA in 2002. He went to University of California at Davis and obtained a Bachelor in Science in Genetics with high honors. During undergraduate, he worked with Dr. Fred Chédin to generate DNA Methyltransferase 3B1 mutant. After graduation, he accepted Cancer Research Training Award at NIH with Dr. Javed Khan where he conducted drug screen to find more effective and less toxic chemotherapy for Neuroblastoma patients. Currently, he is starting an MD/PhD program at UMDNJ RWJMS. In his free time, he enjoys spending time with his family—especially his two little nephews, reading books, cooking food from different parts of the globe and running. He would like to run a marathon one day.

## Charles Miller

- **Hometown:** New York, NY
- **College(s) Attended and Degree(s) Earned:** University of Pennsylvania, BA in Political Science
- **Year in MD/PhD Program:** MS1
- **PhD Thesis Laboratory:** N/A
- **Area of Interest in Clinical Practice:** Surgery
- **Area of Interest in Research:** Tumor Microenvironment
- **Personal Information:** I finished college one year ago and have spent the past year working at a lab in Philadelphia. I am an avid sailor and snowboarder and also enjoy cycling, movies, and television. I am a big fan of classic rock, particularly the works of Bruce Springsteen. I have been waiting for medical school since I was 5 years old, wearing my Dad's hospital ID badge and receiving pages on my bubble gum beeper. I began research in oncology after my junior year of high school and have remained engaged in the field ever since.

## Mazell Tetrushvily

- **Hometown:** Marlton, NJ
- **College(s) Attended and Degree(s) Earned:** University of Pennsylvania, B.A. in Biochemistry, Biology and M.S. in Chemistry
- **Year in MD/PhD Program:** M1
- **PhD Thesis Laboratory:** Still rotating...
- **Area of Interest in Clinical Practice:** Oncology
- **Area of Interest in Research:** Cell signaling in tumors
- **Personal Information:** A bit about me: My main project outside of school is training and certifying my dog as a therapy pup to work with children in local hospitals. Other than my dog, I'm a big fan of hockey (Go Flyers!) and am currently attempting to read as many literary classics (Russian and English) as I can before the start of Medical school.



**MS- Year II**

## Christopher Kimes

- **Hometown:** Leawood, Kansas
- **College(s) Attended and Degree(s) Earned:** Attended Benedictine College, then transferred to Case Western Reserve University; earned Bachelors of Science in Chemistry at Case Western Reserve University
- **Year in MD/PhD Program:** MS2
- **PhD Thesis Laboratory:** Still searching for one
- **Area of Interest in Clinical Practice:** Undecided
- **Area of Interest in Research:** Something interesting
- **Personal Information:** I miss eating Kansas City barbecue. As an undergrad, I did research in a chemistry lab working with nanoparticles. Now I am doing a rotation involving the collaboration of two labs: a biology lab and a chemistry lab. It is an interesting arrangement that allows me to continue working with chemistry techniques while simultaneously giving me the opportunity to learn biology and biochemistry techniques I have never used before in lab.

## Gerard Limerick

- **Hometown:** Beltsville, MD
- **College(s) Attended and Degree(s) Earned:** Oakwood University, B.S. Biochemistry
- **Year in MD/PhD Program:** M2
- **PhD Thesis Laboratory:** Estela Jacinto, Physiology and Biophysics, RWJMS
- **Area of Interest in Clinical Practice:** Still open, frontrunners are cardiology, surgery or ENT
- **Area of Interest in Research:** intracellular signaling networks, molecular mechanisms which regulate cellular metabolism, kinase activity
- **Personal Information:** I'm a Christian and enjoy many types of music, basketball, reading (prior to med school) and traveling. My secret is that I sometimes watch WeTV. I think that they have high quality programming: Bridezillas, Secret Lives of Women, etc.

## Adriana Martin

- **Hometown:** Trinidad and Tobago
- **College(s) Attended and Degree(s) Earned:** Bloomfield College 2008, Chemistry and Biology
- **Year in MD/PhD Program:** MS II
- **PhD Thesis Laboratory:** Soon...
- **Area of Interest in Clinical Practice:** Pending...
- **Area of Interest in Research:** Drug design and synthesis, Medicinal and Computational Chemistry, Oncology and pain management.
- **Personal Information:** Whenever Adriana gets a chance to venture out of the world of cubicles “the airplane,” she tends to enjoy the sun on her face, cooking, painting, spending time with her little sister and anything that involves bodies of water... be it beach, river, lake or puddle.

## Lincoln Smith

- **Hometown:** Indianapolis, IN
- **College(s) Attended and Degree(s) Earned:** Wabash College, majored in Biology and Philosophy
- **Year in MD/PhD Program:** MII
- **PhD Thesis Laboratory:**
- **Area of Interest in Clinical Practice:**
- **Area of Interest in Research:** Genomics/Bioinformatics
- **Personal Information:**

## Jennifer Stundon

- **Hometown:** Lewistown, PA
- **College(s) Attended and Degree(s) Earned:** Bryn Mawr College, BA Biology
- **Year in MD/PhD Program:** 2<sup>nd</sup> yr
- **PhD Thesis Laboratory:**
- **Area of Interest in Clinical Practice:** peds, oncology
- **Area of Interest in Research:** cancer, genetics
- **Personal Information:**



**PhD- Year I**

## Timothy Arlow

- **Hometown:** Southampton, NJ
- **College(s) Attended and Degree(s) Earned:** Syracuse University, BS in Biomedical Engineering and Molecular Biology
- **Year in MD/PhD Program:** PhD 1
- **PhD Thesis Laboratory:** Murphy Lab, Princeton University
- **Area of Interest in Clinical Practice:** Pathology, Endocrinology
- **Area of Interest in Research:** Aging, Gene Therapy
- **Personal Information:**

## Eric Chen

- **Hometown:** Livingston, NJ
- **College(s) Attended and Degree(s) Earned:** Dartmouth College, 2006
- **Year in MD/PhD Program:** PhD-I
- **PhD Thesis Laboratory:** Berry Lab, Princeton University
- **Area of Interest in Clinical Practice:**
- **Area of Interest in Research:** Neuroscience
- **Personal Information:** When he is not studying or working in lab, Eric likes to duff it up on the golf course and occasionally enjoys a beer or three.

## Christiaan deVries

- **Hometown:** Milltown, NJ
- **College(s) Attended and Degree(s) Earned:** Rutgers University – New Brunswick, BA, 2004
- **Year in MD/PhD Program:** PhD-1
- **PhD Thesis Laboratory:** Dr. Edmund Lattime, CINJ
- **Area of Interest in Clinical Practice:** Medical Oncology
- **Area of Interest in Research:** cancer immunotherapy, tumor immunology
- **Personal Information:** Christiaan is excited to get back to the lab after 2 years of living, eating, and sleeping in the airplane.

## Clifton Fulmer

- **Hometown:** Closter, NJ
- **College(s) Attended and Degree(s) Earned:** The College of New Jersey, B.S. Biology 2006
- **Year in MD/PhD Program:** PhD1
- **PhD Thesis Laboratory:** Cheryl Dreyfus
- **Area of Interest in Clinical Practice:** Pathology, Oncology, Neurology
- **Area of Interest in Research:** Neuroscience
- **Personal Information:**

## Rebekah Gensure

- **Hometown:** Pittsburgh, PA
- **College(s) Attended and Degree(s) Earned:** Boston University, BS in Biomedical Engineering, 2005
- **Year in MD/PhD Program:** PhD-I
- **PhD Thesis Laboratory:** Dr. David Foran
- **Area of Interest in Clinical Practice:** Not sure yet; thinking about IM, radiology, anesthesiology, oncology, sports medicine, PM&R, surgery (basically anything)
- **Area of Interest in Research:** Imaging & image analysis, applications to cancer diagnosis and treatment
- **Personal Information:** My creativity is all gone for the moment...I'm just really looking forward to being done with Step 1 by the time this symposium takes place!



**PhD- Year II**

## Shannon Agner

- **Hometown:** Cherry Hill, NJ
- **College(s) Attended and Degree(s) Earned:** A.B., 2002, Dartmouth College; M.S., 2004, Thayer School of Engineering, Dartmouth College
- **Year in MD/PhD Program:** PhD II
- **PhD Thesis Laboratory:** Laboratory for Computational Imaging and Bioinformatics, Department of Biomedical Engineering, Rutgers University
- **Area of Interest in Clinical Practice:** Undecided
- **Area of Interest in Research:** Image analysis, computer-aided diagnosis of cancer
- **Personal Information:** Free time is not just an aspiration now that the qualifier is over (woohoo!), but Shannon is still spending her time trying to catch up on her leisure reading, traveling, hiking when she is around mountains, and hanging out at the beach when she is down at the Jersey Shore. Oh, and you'll probably find her in the lab during the week.

## Desmond Brown

- **Hometown:** Brown's Town, St. Ann, Jamaica
- **College(s) Attended and Degree(s) Earned:** Temple University School of Medicine, M.S. Biochemistry & Biophysics 2006; Temple University, B.S. Chemistry & Biochemistry 2003
- **Year in MD/PhD Program:** Entering my 4<sup>th</sup> year in the program, second year in the lab. Wow time really flies when you're having fun...
- **PhD Thesis Laboratory:** Eggenschwiler Lab, Department of Molecular Biology, Princeton University
- **Area of Interest in Clinical Practice:** Neurosurgery
- **Area of Interest in Research:** Brain Tumor Development & Tumor-Induced Brain Plasticity
- **Personal Information:**
  - I have...
  - One wife, April
  - One daughter, Tsyon
  - One dog, Faith
  - One immediate goal, to graduate
  - One real hobby, martial arts (currently **trying** to get back in shape for another fight this Fall [obviously, not working])
  - One real love, FOOD (thus the conflict with my hobby)

## Hilary Grosso

- **Hometown:** Delran, NJ
- **College(s) Attended and Degree(s) Earned:** New York University, Neuroscience and Psychology
- **Year in MD/PhD Program:** PhD 2
- **PhD Thesis Laboratory:** Dr. M. Maral Mouradian
- **Area of Interest in Clinical Practice:** Neurology
- **Area of Interest in Research:** Neuroscience
- **Personal Information:** Hilary has been in her lab for a year now, studying Parkinson's disease. When she's not in the lab she spends her time staying active, singing, and enjoying being well rested.

## Eileen Hwang

- **Hometown:** West Windsor, NJ
- **College(s) Attended and Degree(s) Earned:** Princeton University, AB in Physics, Biophysics Certificate
- **Year in MD/PhD Program:** PhD II
- **PhD Thesis Laboratory:** Barbara Brodsky, RWJMS Dept of Biochemistry
- **Area of Interest in Clinical Practice:** Undecided.
- **Area of Interest in Research:** Sequence dependent protein folding.
- **Personal Information:** Eileen loves being a PhD student because she has the freedom to explore some of the many unanswered questions in medicine! In her free time, she enjoys climbing, frisbee, and snowboarding. She also volunteers at least once a week as a 9-1-1 EMT in West Windsor. Her mother and one of her four brothers are also members of the first aid squad.

## Jay Oza

- **Hometown:** East Windsor, NJ
- **College(s) Attended and Degree(s) Earned:** BS, Rutgers University
- **Year in MD/PhD Program:** 4 (PhD year 2)
- **PhD Thesis Laboratory:** Shridar Ganesan, CINJ
- **Area of Interest in Clinical Practice:** Oncology
- **Area of Interest in Research:** DNA repair, Epigenetics, Molecular mechanisms of carcinogenesis
- **Personal Information:** 1 younger brother who is in college. In the enormous free time I have, I like playing sports (anything like volleyball, tennis, ping-pong, cricket, basketball, etc). Recently, I started to learn swimming. I thought it would be easy; but it seems that like the MD-PhD program it might also turn out to be a 7 year learning program!



**PhD- Year III**

## Erin Haley

- **Hometown:** Palmyra, NJ
- **College(s) Attended and Degree(s) Earned:** Loyola College in Maryland, B.S.
- **Year in MD/PhD Program:** Ph.D Year III
- **PhD Thesis Laboratory:** Hilary Coller, Princeton University
- **Area of Interest in Clinical Practice:** Oncology or Dermatology
- **Area of Interest in Research:** Cell Cycle Control, Quiescence, Autophagy
- **Personal Information:**

## Eli Levin

- **Hometown:** Haddonfield, NJ.
- **College(s) Attended and Degree(s) Earned:** The College of New Jersey, BS in Biology, 2005
- **Year in DO/PhD Program:** PhD III
- **PhD Thesis Laboratory:** Dr. Robert Nagele, UMDNJ: New Jersey Institute for Successful Aging.
- **Area of Interest in Clinical Practice:** Anesthesia
- **Area of Interest in Research:** Neurodegenerative disease
- **Personal Information:** Eli is far too serious and important to condescend to fill a “personal information” section. He looks forward to erasing the heading from his next symposium profile...or cultivating some interests in the interim.

## Sean Liu

- **Hometown:** Oceanside, NY
- **College(s) Attended and Degree(s) Earned:** MIT, B.S.
- **Year in MD/PhD Program:** 5<sup>th</sup> year
- **PhD Thesis Laboratory:** Thomas E. Shenk, Princeton University
- **Area of Interest in Clinical Practice:** Undecided.
- **Area of Interest in Research:** Virology
- **Personal Information:** I work at night, enjoy going to NYC on the weekends, and wish that there was a more linear correlation between productivity and effort in lab. Too bad it's more of a poker game.

## Peter Mazari

- **Hometown:** Wildwood Crest, NJ
- **College(s) Attended and Degree(s) Earned:** BA Rutgers University
- **Year in MD/PhD Program:** PhD Phase (3<sup>rd</sup> year)
- **PhD Thesis Laboratory:** Monica Roth
- **Area of Interest in Clinical Practice:** Hematology/Oncology & Pathology
- **Area of Interest in Research:** Retroviral Gene Therapy
- **Personal Information:** I grew up at the beach in Wildwood Crest, NJ and moved here in 2001 when I started at Rutgers and stayed here to start the MD/PhD program here at RWJ. I'm kind of a big music buff too and used to have a radio show in college. In my spare time (like there is so much of it) I try to get back home and enjoy the beach and the outdoors, and to get some sun to get rid of this "lab induced pallor".

## Jean McGee

- **Hometown:** Bridgewater, NJ
- **College(s) Attended and Degree(s) Earned:** Cornell University, BA 2003 and MS 2006 in Biological Sciences and Nutritional Biochemistry respectively.
- **Year in MD/PhD Program:** PhD-III
- **PhD Thesis Laboratory:** Dr. Virginia Zakian, Princeton University
- **Area of Interest in Clinical Practice:** Ummm, a good question.
- **Area of Interest in Research:** Currently telomere length regulation
- **Personal Information:** My husband and I are deciding on where to go for our annual summer vacation. Last year was Puerto Rico. This year, Cayman Islands or Key Biscayne? Any suggestions?

## Nilay Sethi

- **Hometown:** Cranbury, NJ
- **College(s) Attended and Degree(s) Earned:** The College of New Jersey (2004), BS in Biology
- **Year in MD/PhD Program:** PhD III
- **PhD Thesis Laboratory:** Yibin Kang, PhD, Molecular Biology, Princeton University
- **Area of Interest in Clinical Practice:** Oncology
- **Area of Interest in Research:** Mechanism underlying cancer metastasis
- **Personal Information:** Outside of lab, I enjoy playing sports such as basketball, football, and tennis. I also spend some of my time as a Residential Graduate Student in Whitman College on the Princeton University Campus. I have one older sister who is also pursuing a career in medicine and is currently in her internship year of a family practice residency.

## Richard Sun

- **Hometown:** Paramus, NJ
- **College(s) Attended and Degree(s) Earned:** Rutgers College, BA
- **Year in MD/PhD Program:** Third
- **PhD Thesis Laboratory:** Samuel S.-H. Wang, Ph.D
- **Area of Interest in Clinical Practice:** Neurosurgery
- **Area of Interest in Research:** Using bio-engineered calcium sensors for probing neural circuitry.
- **Personal Information:** I love my golden retriever!

## **PhD- Year IV and Beyond**

## Kevin Anton

- **Hometown:** Westfield, NJ
- **College(s) Attended and Degree(s) Earned:** Penn State University, BS in Pre-Medicine
- **Year in MD/PhD Program:** PhD IV
- **PhD Thesis Laboratory:** John Glod
- **Area of Interest in Clinical Practice:** Hematology/Oncology
- **Area of Interest in Research:** Oncology- Investigating the role of macrophages in the tumor microenvironment.
- **Personal Information:** The 2009-2010 year is already shaping up to be packed with new adventures, including a Ph.D. dissertation defense, re-entry into clinical medicine, and a publication or two (hopefully).

## Brian Barlow

- **Hometown:** Utica, NY
- **College(s) Attended and Degree(s) Earned:** University of Rochester, Neuroscience and Psychology
- **Year in MD/PhD Program:** PhD IV
- **PhD Thesis Laboratory:** Dr. Mona Thiruchelvam, Environmental & Occupational Health Sciences Institute
- **Area of Interest in Clinical Practice:** Keeping an open mind
- **Area of Interest in Research:** Developmental Origins of Disease / Parkinson's Disease / Toxicology
- **Personal Information:** In my free time, I really enjoy baking, riding my motorcycle, and standing ashamedly near the fence of the dog park while my sheepdog-werewolf hybrid, Baxter, gets all up in everyone's business.

## Christopher Langhammer

- **Hometown:** Washington, D.C.
- **College(s) Attended and Degree(s) Earned:** Princeton University, B.S.E
- **Year in MD/PhD Program:** PhD IV
- **PhD Thesis Laboratory:** Bonnie Firestein
- **Area of Interest in Clinical Practice:** Spinal Cord Injury
- **Area of Interest in Research:** Spinal Cord Injury
- **Personal Information:**

**MS- Year III**

## Jean-Paul J. Abboud

- **Hometown:** Zahleh, Lebanon
- **College(s) Attended and Degree(s) Earned:**  
Rutgers University (BS in Biological Sciences - 2002)  
UMDNJ – Graduate School of Biomedical Sciences (PhD in Biomedical Engineering – 2009)
- **Year in MD/PhD Program:** MS-III
- **PhD Thesis Laboratory:** Richard S. Nowakowski, PhD
- **Area of Interest in Clinical Practice:** Surgery
- **Area of Interest in Research:** Neural Development
- **Personal Information:**  
24 carat Lebanese. Naturalized American. Jack of a few trades. Master of none. Doctor of Philosophy. Student of Medicine. Aspiring surgeon. Proclaimed preppy. Seasonal athlete. Amateur photographer. Tourist in his own backyard. Scientist. Engineer. Thinker. Creator. Dreamer. Observer. Traveler. Adventurer. Poet. Artist. Brother. Son. Godfather. Grandson. Nephew. Cousin. Friend. Human being.

## Issa Bagayogo

- **Hometown:** Abidjan, Ivory Coast
- **College(s) Attended and Degree(s) Earned:** Hunter College (CUNY), BA. Robert Wood Johnson Medical School (GSBS), Ph.D.
- **Year in MD/PhD Program:** M-III
- **PhD Thesis Laboratory:** Dr. Cheryl F. Dreyfus
- **Area of Interest in Clinical Practice:** Open minded as of this moment, though recently I have been thinking a lot about neurology.
- **Area of Interest in Research:** Neuroscience
- **Personal Information:** Married to a beautiful lady and very excited and looking forward to being a dad sometimes this November. Had no idea that finding an interesting name would be such hard work, but we still have plenty of time. Hopefully, we'll do a good job.

## Matthew Treiser

- **Hometown:** North Brunswick, NJ
- **College(s) Attended and Degree(s) Earned:** B.S., Columbia University in the City of New York
- **Year in MD/PhD Program:** MSIII
- **PhD Thesis Laboratory:** Joachim Kohn and Prabhas Moghe
- **Area of Interest in Clinical Practice:** Surgery (Orthopedic or Plastic)
- **Area of Interest in Research:** Biomaterials and Tissue Engineering
- **Personal Information:**

**MD- IV**

## Bonnie Hall

- **Hometown:** Sayreville NJ
- **College(s) Attended and Degree(s) Earned:** UC California, Berkeley BS Chemistry
- **Year in MD/PhD Program:** Finished 3<sup>rd</sup> year Medical School. Taking year off with kids.
- **PhD Thesis Laboratory:** Foran Laboratory
- **Area of Interest in Clinical Practice:** Surgery, possibly ophthalmology
- **Area of Interest in Research:** imaging, using technology in clinical applications
- **Personal Information:** I came into medical school totally writing off surgery because of the lifestyle. Unfortunately, it was my favorite clerkship, next to OB. Currently, I'm taking a year off to learn to sew, write a children's book, dabble in ophthalmology research, and most of all take care of my 3 ½ year old boy and my newest baby born Feb 8, 2009.

## Marcelo Rocha

- **Hometown:** Rio de Janeiro (Brazil), Strasbourg (France) and Edison, NJ
- **College(s) Attended and Degree(s) Earned:** BA (UMBC) and PhD (UMDNJ/Rutgers)
- **Year in MD/PhD Program:** 8<sup>th</sup> year (now MSIV)
- **PhD Thesis Laboratory:** Patricia Sonsalla (RWJMS Neurology department)
- **Area of Interest in Clinical Practice:** neurology
- **Area of Interest in Research:** neurotransmission, neuroplasticity and neurodegeneration
- **Personal Information:** any questions? Let me know! (email rochama@umdnj.edu)

# NOTES

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