EASTERN-ATLANTIC
STUDENT RESEARCH FORUM

41ST ANNUAL MEETING
ABSTRACT BOOK
Program and Abstracts
February 25 – 28, 2015

Hosted by the University of Miami
Leonard M. Miller School of Medicine

Nicholas Cnossen, Editor
Marie Maloof, Editor
Stephanie Yahn, Editor

Eastern-Atlantic Student Research Forum
University of Miami Miller School of Medicine—P.O. Box 016960—Miami, FL 33101

http://uresearch.miami.edu/esrf
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WELCOME FROM THE DIRECTORS

To All Participants:

It is our distinguished pleasure to welcome you to the 41st annual Eastern-Atlantic Student Research Forum. This is an exciting time as we look to our 41st year of providing educational and networking opportunities for medical students, graduate students, and residents to present their research before a group of peers and faculty. We hope that the ESRF has and continues to promote the development of our future physician-scientists and researchers.

We are very lucky to have gathered a wonderful group of distinguished investigators and students to present at this year’s forum. We are pleased to announce that Pascal J. Goldschmidt, MD, Senior Vice President for Medical Affairs and Dean of the University of Miami Leonard M. Miller School of Medicine will provide the welcome address.

We are privileged to have award winning physician-scientist, Geoffrey Ginsburg, MD, PhD, director of the Center for Applied Genomics and Precision Medicine at Duke University and Advisory Council Member for the NIH’s National Center for Advancing Translational Science, deliver our keynote address. Dr. Ginsburg is a world renowned pioneer in translational genomics and aims to integrate genomic tools into direct patient care applications ranging from diagnosis to personalized therapeutics. Having worked with industry and in creation of infrastructure to explore implementation of personalized medicine, we are honored to have Dr. Ginsburg impart his wisdom.

In addition to student presentations and faculty addresses throughout the forum, we are also hosting interactive educational discussions with faculty and students. Our plenary session revolves around the recent Ebola outbreak and is entitled "Ebola: The Basics, Preparation, And How to Stop it." Our enthusiastic faculty will bring us up to speed on this evolving crisis and foster intriguing discussion. We have invited Dr. Jamie S. Rubin, PhD, to once again host her sensational career development workshop. Dr. Rubin is the Director for Research Development in the Department of Medicine at Columbia University Medical Center, and we are grateful for her wisdom and expertise.

Dr. Charles Nemeroff, MD, PhD, will conclude this year’s forum at our Awards Banquet. He is the Leonard M. Miller Professor and Chairman of the Department of Psychiatry and Behavioral Sciences, Director of the Center on Aging, Chief of Psychiatry at Jackson Memorial and University of Miami Hospitals, and Associate Director of the MD/PhD program at the University of Miami Miller School of Medicine. His work in Psychopharmacology is unparalleled, and his interactions with academia and industry grant him a unique perspective as a physician-scientist. We look forward to learning from his invaluable experiences.

We are honored to host ESRF for the 41st year, and it would not be possible without all of the wonderful presenters, committee chairs, faculty advisors, and support staff who contribute so much time to ensure a successful conference. We give special thanks to Isabel Perez, who works year round to make this conference perfect, and is a great advocate of student research. Without her there would be no ESRF. It has been a wonderful 41 years, and we hope students and researchers will continue to enjoy and benefit from the success of the forum for years to come.

Sincerely,

Zachary Most
UMMSM, Class of 2015
ESRF Co-Director

Holly Stradecki
UMMSM MD-PhD, GS2
ESRF Co-Director

Nikesh Shah
UMMSM, Class of 2016
ESRF Co-Director
WELCOME FROM THE DEAN

Dear Participant:

The University of Miami Miller School of Medicine is proud and pleased to welcome you to the 2015 Eastern-Atlantic Student Research Forum. This four-day international meeting is a unique opportunity to bring together some of the brightest young minds beginning their biomedical research careers. The investigations you will review at this 41st annual conference, and your future research, will lead to important discoveries for fighting and preventing diseases that take a terrible toll on our fellow humans.

It is my hope that this forum’s distinguished presenters -- medical, graduate, M.D./Ph.D. students and resident physicians from the United States and dozens of other countries around the world -- will challenge you to fully engage in basic science and clinical research.

Thank you for joining us in the vital pursuit of a deeper understanding of medical science. There is no more important mission than finding answers for the patients who depend on us for our knowledge and our compassion, both now and in the future.

With warmest regards,

Pascal J. Goldschmidt, M.D.
Senior Vice President for Medical Affairs and Dean,
University of Miami Leonard M. Miller School of Medicine
CEO, University of Miami Health System
Serving more than five million people as the only academic medical center in South Florida, UHealth – University of Miami Health System/ Miller School of Medicine has earned international acclaim for research, clinical care, and biomedical innovations. Founded in 1952 as Florida’s first accredited medical school, the University of Miami Leonard M. Miller School of Medicine provides medical staff for the nationally renowned University of Miami/Jackson Memorial Medical Center and University of Miami Hospital. University of Miami Hospital is the flagship facility of UHealth, which also includes two additional University-owned hospitals: Sylvester Comprehensive Cancer Center and Anne Bates Leach Eye Hospital, home to the top-ranked Bascom Palmer Eye Institute. Our affiliated hospitals on the medical campus include Jackson Memorial Hospital, Holtz Children’s Hospital, and the Miami VA Medical Center.

Each year the medical school’s more than 1,200 faculty physicians have more than a million patient encounters in primary care and more than 100 medical specialties and sub-specialties. UHealth also has more than 8,000 employees. In 2013, U.S. News & World Report listed Bascom Palmer Eye Institute as the number one hospital in the country for ophthalmology for the tenth year in a row. Two other UM Miller School of Medicine specialties were also listed among the nation’s best: ear, nose and throat and geriatrics.

Research is a top priority, with more than 1,500 ongoing projects funded by more than $200 million in external grants and contracts to UM faculty. The medical campus consists of nearly 68 acres within the 153-acre complex of the University of Miami/Jackson Memorial Medical Center, including more than 500,000 square feet of research space with plans underway to build the UM Life Science Park, which will add an additional two million square feet of space adjacent to the medical campus. The UM Life Science Park will bring together academia and industry for collaboration in bioscience research innovation. The medical campus is also home to the following acclaimed medical facilities:

- **Bascom Palmer Eye Institute** has been named the country’s number one eye hospital ten years in a row by *U.S. News & World Report* for its ongoing excellence in ophthalmic clinical care and research. The Anne Bates Leach Eye Hospital annually serves 160,000 outpatients of ophthalmology and other specialties, largely for microsurgery procedures.
- **The Diabetes Research Institute** is a recognized world leader in cure-focused research. The DRI has pioneered many of the techniques used worldwide in islet cell transplantation, including advances in cell biology, immunology and harnessing the power of stem cells as a reliable source of insulin-producing cells for transplantation.
- **The Sylvester Comprehensive Cancer Center** treats nearly 4,000 newly-diagnosed cancer patients each year, and treats thousands more in ongoing treatment from throughout the United States and Latin America. Approximately 200 clinical trials are underway, supported by more than $33 million in research grants.
- **Dedicated to finding a cure for paralysis resulting from spinal cord injury, researchers at the Miami Project to Cure Paralysis** found the first direct evidence of successful regeneration of adult human central nervous system tissue. The Miami Project, the world’s largest comprehensive spinal cord injury research center, conducts basic and
clinical research trials, as well as a program that permits spinal cord injured men to father children. The center is currently awaiting FDA approval for human trials on schwann cell transplantation.

- **The University of Miami Ear Institute** houses the nation’s second most active cochlear implant program, restoring hearing to adults and children with profound deafness. Over the years the ear, nose and throat program has steadily climbed up the *U.S. News & World Report* rankings.

- The nationally renowned research efforts of the **Department of Pediatrics** are housed in the magnificent Batchelor Children’s Research Institute. The Miller School’s Mailman Center for Child Development has a number of model programs that help children with developmental disabilities.

- **The Transplant Institute at the University of Miami/Jackson** is one of the nation’s best and busiest, responsible for half of the pediatric multi-visceral transplants in the world. University of Miami/Jackson has an active transplant program for bone marrow, heart, lungs, kidneys, liver, pancreas and intestines.

- Significant federal funding supports research at the **Comprehensive AIDS Program**, including HIV studies in pregnant women, pediatric AIDS clinical trials, various drug protocol studies, heterosexual transmission of AIDS, transfusion safety studies, and the national cooperative drug discovery group. The Miller School’s Developmental Center for AIDS Research (DCFAR), is one of the first of its kind in the state of Florida.

- **The John P. Hussman Institute for Human Genomics** is designed to discover the genetic influences on human health and apply the knowledge to the practice of medicine through improved diagnostics, treatments and medications. Under the stewardship of two of the most highly-acclaimed geneticists in the world, Margaret Pericak-Vance, Ph.D., and her husband Jeffery Vance, M.D., Ph.D., their work has uncovered critical clues to the origins of diseases such as Parkinson’s, Alzheimer’s and macular degeneration, and now they will work to integrate all of the School’s existing genetics research strengths into a single powerhouse program. The researchers and their collaborators at other medical centers have identified the first common genetic risk factor for autism spectrum disorder, nine genes that may increase susceptibility for Alzheimer’s disease and confirmed a region on chromosome 12q long believed to harbor an Alzheimer’s risk gene.

- **The Interdisciplinary Stem Cell Institute** is leading the way in the use of adult stem cells to repair malfunctioning human organs. Joshua M. Hare, M.D., director of the Interdisciplinary Stem Cell Institute, led the Transendocardial Autologous Cells in Ischemic Heart Failure Trial (TAC-HFT) study, using a novel catheter and is at the forefront of stem cell therapy research. The Institute’s goal is to find new treatments for heart disease, neurological disorders and other chronic and incurable diseases.

- The **new Biomedical Research Building**, a 182,000-square-foot facility houses the Interdisciplinary Stem Cell Institute, the John P. Hussman Institute for Human Genomics and will serve as a wet lab facility with office space for researchers. The facility is also LEED (Leadership in Energy and Environmental Design) certified, reducing the negative environmental impact of the building and improving occupant health and well-being.
EASTERN-ATLANTIC STUDENT RESEARCH FORUM - 2015

PROGRAM SCHEDULE

Wednesday, February 25, 2015

Noon-1:00 PM   Registration for UM Students               RMSB 2nd Floor Student Lounge
6:00 PM - 8:00 PM   Welcome Reception/Registration         SpringHill Suites at Marriott
                              1311 NW 10th Avenue
                                      Miami, FL 33135
8:00 PM –11:00 PM   Hospitality Suite Open                   Room 133

Thursday, February 26, 2015

8:30 AM– 9:30 AM   Breakfast/Late Registration             Café 20/20
                              (1st Floor below Jose Berrocal Auditorium)
9:30 AM – 9:45 AM   Welcome Address
                                           Bascom Palmer Eye Institute (BPEI)
                        Pascal J. Goldschmidt, MD
                            Dean & Senior Vice President
                                  University of Miami Miller School of Medicine
10:00 AM – 11:45 AM   Oral Presentations I  (Session 1)   BPEI Jose Berrocal Auditorium
12:00 PM - 1:00 PM   Keynote Address  (Session 2)          BPEI Jose Berrocal Auditorium
                        Geoffrey S. Ginsburg, MD, PhD
                                      Director, Center for Applied Genomics & Precision Medicine
                                                   Duke School of Medicine
1:00 PM - 2:00 PM    Keynote Luncheon                         Café 20/20
2:15 PM – 5:00 PM   Oral Presentations II  (Session 3)    BPEI Jose Berrocal Auditorium
8:00 PM –11:00 PM   Hospitality Suite Open                   SpringHill Suites at Marriott

Friday, February 27, 2015

7:00 AM- 8:00 AM   Poster set up                              Lois Pope Life Center (Shoninger Quadrangle)
7:00 AM- 8:00 AM   Breakfast                                   Café 20/20
8:30 AM- 11:00 AM  Oral Presentations III  (Session 4)     BPEI Jose Berrocal Auditorium
11:00 AM- 12:00 PM  Career Development Workshop (Session 5)
                                               BPEI Jose Berrocal Auditorium
                        Jaime S. Rubin, PhD
                                      Director for Research Development
                                          Columbia University, NY
12:00 PM– 1:00 PM   Lunch                                      Café 20/20
1:00 PM – 2:00 PM   Plenary Session   (Session 6)            BPEI Jose Berrocal Auditorium
2:15 PM – 4:30 PM   Poster Presentations  (Session 7)      Lois Pope Life Center (Shoninger Quadrangle)
3:00 PM – 4:30 PM   Wine & Cheese Reception                   Lois Pope Life Center (Shoninger Quadrangle)
8:00 PM –11:00 PM   Hospitality Suite                         SpringHill Suites at Marriott

Saturday, February 28, 2015

6:00 PM– 9:30 PM   Awards Banquet Address
                        Charles B. Nemeroff, MD PhD
                                      Professor and Chairman
                                             Department of Psychiatry & Behavioral Sciences
                                           Director, Center on Aging
                                                 University of Miami Miller School of Medicine
                        University of Miami Hospital
                              1400 NW 12 Ave. (Seminar A,B & C)
Pascal J. Goldschmidt, M.D., an internationally renowned cardiologist and cardiovascular researcher, is Senior Vice President for Medical Affairs and Dean of the University of Miami Leonard M. Miller School of Medicine. He also serves as Chief Executive Officer of the University of Miami Health System (UHealth), which includes six hospitals and more than two dozen outpatient facilities in Miami-Dade, Broward, Palm Beach, Monroe and Collier counties, with more than 1,200 physicians and 8,000 staff.

Since his arrival in April 2006, Dean Goldschmidt has overseen tremendous growth on the medical campus in Miami, with the November 2007 purchase of Cedars Medical Center, which is now University of Miami Hospital, the flagship hospital of UHealth. He also established the first allopathic internal medicine residency training program in Palm Beach County.

New global health clinical and research initiatives include the Global Institute for Community Health and Development and the International Medicine Institute. It was through the Global Institute for Community Health and Development that Dean Goldschmidt was able to launch and oversee the medical relief effort in Haiti after the January 2010 earthquake. A team of Miller School physicians, nurses and staff was the first to arrive in Port-au-Prince, and within nine days of the earthquake the University of Miami Hospital in Haiti was open and treating patients.

The research enterprise has also grown significantly since 2006 with the creation of the John P. Hussman Institute for Human Genomics and the Interdisciplinary Stem Cell Institute. Both institutes are headed by world-renowned researchers.

In October 2008, Dr. Goldschmidt received the inaugural Jay and Jeanie Schottenstein Prize in Cardiovascular Sciences from the Ohio State University Heart and Vascular Center. The prize is awarded biennially to an international leader in the clinical sciences of cardiovascular medicine, cardiothoracic surgery, or the basic sciences of molecular or cellular cardiology.

Dr. Goldschmidt, whose research applies genomics and cell therapy to the prevention, diagnosis and treatment of coronary artery disease, was previously chairman of the Department of Medicine at Duke University Medical Center. Before taking the chairman’s role, he served as chief of Duke’s Division of Cardiology.

Before joining the Duke faculty in 2000, he was director of cardiology at The Ohio State University College of Medicine and Public Health, where he built the Heart and Lung Research Institute and a heart hospital.

A native of Belgium, Dr. Goldschmidt received his medical degree from the Universite Libre de Bruxelles and completed residency and fellowship training in Brussels at Erasme Academic Hospital and in the United States at The Johns Hopkins University. Following his training at Hopkins, he served as an associate professor in the university’s Department of Cell Biology and Anatomy, Department of Pathology, and Division of Cardiology in the Department of Medicine until 1997.
KEYNOTE ADDRESS
Geoffrey S. Ginsburg, M.D., Ph.D.

Director, Center for Applied Genomics & Precision Medicine,
Duke University School of Medicine
Professor of Medicine and Pathology,
Duke University Medical Center
Professor of Biomedical Engineering,
Duke Pratt School of Engineering

Genomic and Precision Medicine:
The Future is Now

Dr. Geoffrey S. Ginsburg, M.D., Ph.D., founded the Center for Applied Genomics & Precision Medicine at Duke in 2014 resulting from his positions as director for Genomic Medicine the Duke Institute for Genome Sciences & Policy from 2004-2014 and as director of the Center for Personalized and Precision Medicine established in the Duke University Health System from 2010-2014.

While at Duke, Dr. Ginsburg has pioneered translational genomics, initiating programs in genome enabled biomarker discovery, longitudinal registries with linked molecular and clinical data, biomarker-informed clinical trials, and the development of novel practice models and implementation research for the integration of genomic tools in health care systems. With a strong commitment to interdisciplinary science he has led projects to develop predictive models for common complex diseases using high dimensional genomic data as well as collaborations with engineering groups to develop novel point of care sensors. His work spans oncology, infectious diseases, cardiovascular disease and metabolic disorders, and his research is addressing the challenges for translating genomic information into medical practice using new and innovative paradigms, and the integration of personalized medicine into health care. He is an internationally recognized expert in genomics and personalized medicine with over 200 published papers, and funding from NIH, DOD, Air Force, DARPA, the Gates Foundation, and industry.
In 1990, he joined the faculty of Harvard Medical School, where he was director of Preventive Cardiology at Beth Israel Hospital and led a laboratory in applied genetics of cardiovascular diseases at Children’s Hospital. In 1997 he joined Millennium Pharmaceuticals Inc., as senior program director for cardiovascular diseases and was eventually appointed vice president of Molecular and Personalized Medicine, where he was responsible for developing pharmacogenomic strategies for therapeutics, as well as biomarkers for disease and their implementation in the drug development process.

He has received a number of awards for his research accomplishments, including the Innovator in Medicine Award from Millennium in 2004, the Basic Research Achievement Award in Cardiovascular Medicine from Duke in 2005, and the ILCHUN Molecular Medicine Award from Korea in 2014. He is a founding member and former board member of the Personalized Medicine Coalition, a section editor for *The Journal of the American College of Cardiology* and an editorial advisor for *Science Translational Medicine*. In addition he is the editor of *Genomic and Personalized Medicine* (Elsevier) published in 2012. He is a member of the Faculty of 1000.

He has been a member of the Secretary of Veterans Affairs Advisory Council on Genomic Medicine, a member of the NIGMS External Scientific Panel for the Pharmacogenomics Research Network, and the National Advisory Council for Human Genome Research at NIH. He is currently an international expert panel member for Genome Canada, a member of the Board of External Experts for the National Heart, Lung and Blood Institute, and a member of the World Economic Forum’s Global Agenda Council on The Future of the Health Sector. He has recently been appointed to the Advisory Council for the National Center for Advancing Translational Sciences at NIH and is the Vice Chair for the Cures Acceleration Network Board. He is co-chair of the Institute of Medicine’s Roundtable on Translating Genome-Based Research for Health.

He received his MD and PhD in biophysics from Boston University and completed an internal medicine residency at Beth Israel Hospital in Boston, MA. Subsequently, he pursued postdoctoral training in clinical cardiovascular medicine at Beth Israel Hospital and in molecular biology at Children’s Hospital as a Bugher Foundation Fellow of the American Heart Association.
BANQUET ADDRESS

Charles B. Nemeroff, M.D., Ph.D.

Leonard M. Miller Professor and Chairman
Department of Psychiatry and Behavioral Sciences
Director, Center on Aging
Chief of Psychiatry, Jackson Memorial Hospital
Chief of Psychiatry, University of Miami Hospital
Associate Director - MD/PHD Program
Leonard M. Miller School of Medicine
University of Miami

The Rage to Know, the Rage to Teach, the Rage to Heal or Why We Are All Not On Wall Street

Charles B. Nemeroff, M.D., Ph.D. is the Leonard M. Miller Professor and Chairman of the Department of Psychiatry and Behavioral Sciences and Director of the Center on Aging at the University of Miami Miller School of Medicine in Miami, Florida. He received his MD and Ph.D. (Neurobiology) degrees from the University of North Carolina (UNC) School of Medicine in Chapel Hill, North Carolina. After psychiatry residency training at UNC and Duke University, he held faculty positions at Duke and at Emory University before relocating to the University of Miami in 2009. He has served as President of the American College of Psychiatrists (ACP) and the American College of Neuropsychopharmacology and sits on the Scientific Advisory Board and Board of Directors of the American Foundation for Suicide Prevention and the Anxiety and Depression Association of America. He has received a number of research and education awards including the Kempf Award in Psychobiology, the Samuel Hibbs Award, Research Mentoring Award, Judson Marmor Award and the Vestermark Award from the American Psychiatric Association, the Mood Disorders Award, Bowis Award and Dean Award from the ACP. He was elected to the Institute of Medicine of the National Academy of Sciences in 2002. His research has focused on the pathophysiology of mood and anxiety disorders with a focus on the role of child abuse and neglect as a major risk factor. He has also focused on the role of mood disorders as a risk factor for major medical disorders including heart disease, diabetes and cancer.

He has served on the Mental Health Advisory Council of NIMH and the Biomedical Research Council for NASA. He is the co-editor in chief of the Textbook of Psychopharmacology, published by the APA. His research is currently supported by grants from the NIH.
CAREER DEVELOPMENT WORKSHOP

Jaime S. Rubin, Ph.D.

Jaime Rubin, Ph.D. received a B.S. in physics *sigma pi sigma* in 1977 from The Cooper Union for the Advancement of Science and Art (New York, NY). She then received M.Sc. and Ph.D. degrees from the Ontario Cancer Institute/University of Toronto in 1980 and 1984, respectively. Her Ph.D. thesis, published in the journal, *Nature*, described the first molecular identification and characterization of a human DNA repair gene. Since 1985, she has held a number of senior level positions at Columbia University's Medical Center, including Acting Associate Dean for Graduate Affairs, having served as the founding Director of the Office of Graduate Affairs, and Acting Associate Vice President/Acting Associate Dean for Research Administration, having served as one of the founders of the Office of Research Administration. She is currently the Director for Research Development in the Department of Medicine. All of these positions have allowed for the teaching and mentoring of junior investigators, including medical and graduate students, postdoctoral fellows, and assistant professors. She founded the graduate-level course "Funding for Research Activities: Basic Issues in Obtaining Support" in 1996 and served as an Associate Program Director for the Doris Duke Clinical Research Fellowship Program, having helped initiate the program at Columbia in 2000. She started and continues to co-direct the Medical Center's course on "Responsible Conduct of Research." Other roles include serving as Associate Director for Career Development on a number of NIH-funded pre-doctoral and postdoctoral training grants as well as an advisory board member of Columbia’s Patient-Oriented Research (POR) Master of Science Program and CTSA (Education).
PLENARY SESSION

EBOLA: The Basics, Preparation, and How to Stop it

Thomas M. Hooton, M.D.

Dr. Thomas Hooton is Professor of Clinical Medicine at the Miller School of Medicine, Associate Chief of Staff of Medical Service at the Miami VA Healthcare System, and Medical Director for UHealth Infection Control, Occupational Health, and Workers Compensation. Dr. Hooton is one of the nation’s leading experts in the epidemiology, pathogenesis, and treatment of UTI. He was on the faculty of the University of Washington for 22 years before moving to the University of Miami in 2006. He also served for five years as an Epidemic Intelligence Officer and researcher for the Centers for Disease Control and Prevention in Atlanta. He has authored or co-authored over 140 peer-reviewed journal articles and over 200 other articles, book chapters and abstracts in the fields of UTI, antimicrobial stewardship, STDs, and HIV/AIDS.

Samita S. Andreansky, Ph.D.

Dr. Samita Andreansky is an Assistant Professor at the Department of Pediatrics at the University of Miami Miller School of Medicine. She received her graduate training at the University of Alabama at Birmingham followed by a fellowship in viral immunology at the Center of Excellence for Influenza Research and Surveillance at St. Jude Children’s Research Hospital. Her primary research interest is to understand how host modulates adaptive immune response against viruses and cancer.

Paola Natalia Lichtenberger, M.D.

Dr. Paola Lichtenberger is an infectious disease specialist in Miami, Florida and is affiliated with Miami Veterans Affairs Healthcare System. She received her medical degree from El Bosque University and completed a fellowship in infectious disease at Jackson Memorial Hospital in Miami.
FACULTY JUDGES

The directors and staff of the 2015 ESRF would like to express their gratitude to the following individuals for contributing their time and expertise in the evaluation of this year’s Forum presentations:

Chrisfouad Alabaid, MD
Morad Askari, MD
Sanjoy Bhattacharya, PhD
Laura Bianchi, PhD
Margaret Byrne, PhD
Alberto Caban-Martinez, DO, PhD
Kara Cauuto, MD
Kevin Curtis, PhD
Daftarian Pirouz, PhD
Gary Danton, MD, PhD
Kunjan Dave, PhD
Joseph De La Garza, MD
Juan Pablo de Rivero Vaccari, PhD
Amar Deshpande, MD
John Diaz, MD
Noella Dietz, PhD
Derek Dykxhoorn, PhD
Nagy Elsayyad, MD
Julia Escandon, MD
Christian Faul, PhD
Alessia Fornoni, MD, PhD
Enrique Ginzburg, MD
Guy Howard, PhD
Barry Hudson, PhD
Joaquin Jimenez, MD
Venkata Kakulavarapu, PhD

Robert Keane, PhD
Tulay Koru-Sengul, PhD
Sandra Lemmon, PhD
John Lew, MD
Robert Levy, PhD
Paola Lichtenberger, MD
Diana Lopez, PhD
Janice Maldonado, MD
Wilberto Nieves-Neira, MD
Joseph Panoff, MD
Anthony Panos, MD
Zubin Panthaki, MD
Irena Pastar, PhD
Carlos Perez-Stable, PhD
Vittorio Porciatti, D.Sc
Kottill Rammohan, MD
Ami Raval, PhD
Elena Roth, MD
Stephanie Sacharow, MD
Christopher Salgado, MD
Jason Salsamendi, MD
Adam Sise, MD
Brian Slomovitz, MD
Jorge Luis Sotelo, MD
Olivera Stojadinovic, MD

Mark Stoutenberg, PhD, MSPH
Leonardo Tamirez, MD
Akin Tekin, MD
Ashok Verma, MD
Katherine Walz, PhD
Liyong Wang, PhD
Medhi Wangpaitri, PhD
Juan Young, PhD
Mina Zarei, MD
Fangliang Zhang, MD

We thank the following individuals for contributing their time and expertise in the evaluation of presentations by students eligible to receive the prestigious Alving Award:

Maria Marin-Castano, MD, PhD
Sawsan Khuri, PhD
Micheline McCarthy, MD, PhD
Tatjana Rundek, MD, PhD
Thomas Van De Water, PhD

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ESRF SPONSORS

We appreciate the donation of time and resources from the following:

- Department of Biochemistry
- Department Cell Biology & Anatomy
- Department of Dermatology
- Department of Microbiology & Immunology
- Department of Molecular & Cellular Pharmacology
- Department of Neurology
- Department of Neurosurgery
- Department of Otolaryngology
- Department of Pathology
- Department of Physical Therapy
- Department of Physiology & Biophysics
- Department of Psychiatry & Behavioral Sciences
- Department of Radiology
- UM Office of Graduate Studies
- UM Office of Medical Education
- UM Office of Research
- University of Miami Leonard M. Miller School of Medicine
- Columbia University
ALVING AWARD
BARBARA & CARL ALVING, M.D.

The Alvings made a $100,000 gift to the Miller School of Medicine to endow the Dr. Carl and Barbara Alving Endowed Award. The award will be presented to the medical student who has had the most outstanding research achievement for the year. An award committee at the medical school will select the winning candidate based on a set of criteria established by the committee. The student would win a medal and also a substantial unrestricted personal monetary award to encourage the student to pursue medical research. The award is open to any medical student and not limited to one win during the course of the student’s medical school career.

Dr. Alving says, “Although I trained in internal medicine, I have actually dedicated my career to doing fundamental research rather than direct patient care. It naturally makes sense that I would want to inspire students who have an interest in research. My wife also has had an illustrious research career and is very deeply involved in medical research. We believe the promotion of research will benefit people very greatly because it provides the fundamental underpinning of medicine I would hope there might be a possibility that this would serve as inspiration for provision of additional research resources to the medical school by others and provide stimulation for medical students who are interested in engaging in a research career.”

Previous Alving Award Winners:
Lucas Cavallin (2014)
Nikesh Shah (2013)
Stavros Moysidis (2012)
Mircea Cristescu (2011)
Michael Gorin (2010)
Dominic Maggio (2009)
Seth Miller & Christine Dinh (2008)
2015 ESRF EXECUTIVE COMMITTEE

**Director**
Isabel Perez

**ESRF Co-Directors**
Zachary Most
Nikesh Shah
Holly Stradecki

**Hospitality Committee**
Sandy Jiang

**Judging Committee**
Mark Barton
Devon Cohen (Chair)
Saloni Mehta

**Plenary Session Committee**
Steven V. Lord
Chelsea Marcus (Chair)
Joshua Parker

**Publications Committee**
Nicholas Cnossen
Stephanie Yahn

**Registration Committee**
Sandy Jiang
Marie Maloof
Jeffrey Peterson
## ESRF Directors, 1975-Present

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<th>Year</th>
<th>Director(s)</th>
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<td>1975</td>
<td>Stephan Dresnick, MD</td>
<td>1994</td>
<td>Andrew Greenberg, MD PhD</td>
<td>2011</td>
<td>Michael Gorin, MD</td>
<td>Siddharth Mahure, MD</td>
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<td>Sandy Martin, MD</td>
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<td>Richard Lee, MD PhD</td>
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<td>1976</td>
<td>Wayne Schonfeld, MD</td>
<td>1995</td>
<td>Jeffrey A. Hertz, MD</td>
<td>2012</td>
<td>Jeremy Dennis, MD</td>
<td>Siddharth Mahure, MD</td>
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<td>Cliff Stamler, MD</td>
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<td>Melinda Merchant, MD PhD</td>
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- Ohio State University College of Medicine, Columbus, OH
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- Yale University School of Medicine, New Haven, CT
ACKNOWLEDGMENTS

The directors and staff of the 2015 ESRF would like to express their gratitude to the following individuals for their part in making this year’s forum a success through the contribution of their time, resources, and advice:

Dean Pascal Goldschmidt MD, Omaida Velazquez, MD, and Mark O’Connell MD for their generous support of the ESRF. With your help, we can ensure that the ESRF continues to have many more successful events in the future.

Our Faculty Judges, who donate their time and expertise to enhance our experience by providing valuable feedback for our presenters.

Isabel Perez, whose dedication throughout the years to the ESRF is unwavering. You truly make the ESRF a success year after year.
Oral Presentations I
February 26, 2015
Surgery

10:00 AM

TREATMENT OF TYPE II ENDOLEAK WITH ONYX GEL EMBOLIZATION: A LITERATURE REVIEW. Mohammad Elsayed BA and Manuel Garcia-Toca, MD. Department of Vascular Surgery, Rhode Island Hospital, Warren Alpert Medical School of Brown University, Providence, RI.

Type II Endoleak (T2E) is the most common complication after Endovascular Aneurysm Repair (EVAR) of Abdominal Aortic Aneurysms. Occurring in up to 25% of patients after EVAR, T2E develops from retrograde blood flow into the aneurysm sac, increasing luminal pressure and the risk of rupture. Management of T2E is controversial, as various technologies and approaches have been proposed. In order to improve an understanding of T2E treatment, a literature review of gel embolization using Onyx ethylene vinyl copolymer was conducted. A search with terms related to Onyx and T2E on PubMed and Google Scholar identified five relevant studies examining 150 patients. The mean age of patients receiving treatment was 76.6 years (range= 37-93). Available data show a mean follow up time of 58.9 months from two studies and median follow-up of 15 weeks from a third. Initial technical success, defined by elimination or reduction of the endoleak at the time of surgery, was encountered in 89.5% of 133 patients. Clinical success, defined by the reduction or maintenance of aneurysm sac size on follow-up was encountered in 77.90% of 95 patients. Endoleak persisted in 34.3% of 73 patients after Onyx embolization. Four new endoleaks and zero ruptures were recorded after treatment. This analysis demonstrates that technical and clinical success is achieved in a majority of patients treated for T2E with Onyx polymer. However, there is a considerable rate of failure and persistent endoleak. Such findings indicate that research is necessary to identify optimal surgical candidates and appropriate scenarios for using gel embolization. Since rupture due to T2E is a rare occurrence, surgeons should consider conservative therapy and surveillance in the long-term.

10:15 AM

TO LAP OR NOT TO LAP: COMPARING TWO TECHNIQUES USED IN THE REPAIR OF INCISIONAL HERNIAS. Yu-Wei Wayne Chang, MS. Sandy Fogel, MD FACS. Department of General Surgery, Virginia Tech Carilion School of Medicine, Roanoke, VA 24016.

Ventral hernias are a common problem in the United States, with approximately 380,000 ventral hernias being repaired annually. A majority of these repairs are attributed to prior incisions from abdominal procedures. While some hernias are asymptomatic, they can cause pain or present as medical emergencies. Repair is generally indicated. Our study goal was to examine patient outcomes between two techniques currently used to repair incisional hernias. We designed a retrospective cohort study of patients (≥18 years old) who underwent a laparoscopic or open repair of their hernia at Carilion Roanoke Memorial Hospital between 1/1/2008–12/31/2012. We excluded patients without a true incisional hernia and those who had hernias repaired in a contaminated field in which a biologic mesh was used. Complications related to the technique of repair were defined as the following: bowel injury, post-op infection within 30 days, or hernia recurrence within 2 years. We enrolled 322 patients: 150 had a laparoscopic repair and 172 had an open repair. Our study population was predominately white (90%) and female (70%) with an average age of 55 years (range 23 – 82) and an average BMI of 33.4 (range 18 – 63). Compared to patients with open repairs, those who underwent laparoscopic repair had a longer operative duration (2.3 vs 2 hrs, p <0.001), required more narcotic orders (8.3 vs 5.7, p <0.001), and stayed longer in the hospital (1.1 vs 0.7 days, p <0.001), but had a lower complication rate (8.7% vs 15.1%, p = 0.09). Controlling for sex, hernia dimension, and BMI revealed that patients who underwent laparoscopic repair of incisional hernias had 60% lower odds of suffering a complication compared to patients who had an open repair (p <0.03). In conclusion, our data demonstrate that laparoscopic repair of incisional hernias, although they had some drawbacks, is a safe and effective technique compared to the open repair.
10:30 AM


Acute fluid resuscitation is critical to survival of burn patients. Crystalloid at 2-4 cc/kg/% total body surface area (TBSA) burn for the first 24 hours post-injury, titrated to maintain urine output of 30-50 ml/h, is considered sufficient for acute resuscitation of burn patients. Complications of over-resuscitation include respiratory compromise, sepsis, and death. This study aims to evaluate the role of fluid resuscitation and clinical contributors to hospital mortality after burns. We reviewed the burn database at our American Burn Association verified burn center for all patients suffering from greater than 20% TBSA burn from 1/1/2010-12/31/2013 (n=101). Demographics, fluid resuscitation, vital signs, laboratory values, and outcomes were collected by chart review. Univariate and multivariate analysis using a logistic regression model were performed. Significant predictors of death during the first two weeks of admission included TBSA burn ≥40% (33.3% vs 6.2%, p<0.001) and need for mechanical ventilation (21.2% vs 5.7%, p=0.042). Significant predictors of death during total admission included TBSA burn ≥40% (50.0% vs 20.0%, p=0.002), age (57±19 vs 44±14, p=0.001), and mechanical ventilation (42.4% vs 8.6%, p=0.001). Patients receiving ≥ 4cc/kg/TBSA burn in the first 24 hours had higher mortality during admission (43.5% vs 21.9%, p=0.47); those receiving ≥ 4cc/kg/TBSA in the first 48 hours had higher mortality during admission (34.0% vs 12.5%, p=0.0.03), and during the first two weeks of treatment (14.0% vs 0%, p= 0.027). Fluid resuscitation ≥4cc/kg/TBSA in the first 24 hours and 48 hours was not associated with pneumonia in the first two weeks, acute kidney injury in the first 48 hours, or PaO2/FiO2 <200 at day 1, 7, or 14 of mechanical ventilation. A logistic regression model was created to determine if fluid resuscitation was an independent contributor to death during admission. Only TBSA burn ≥ 40%, age, and need for mechanical ventilation proved significant (area under receiver operator curve= 0.852). Independent predictors of hospital mortality include TBSA ≥ 40%, age, and need for mechanical ventilation but not volume of fluid resuscitation.

10:45 AM

INCREASED RATE OF INCIDENTAL PAPILLARY THYROID CANCER IN PATIENTS TREATED OPERATIVELY FOR BENIGN THYROID DISEASE. Andrea R. Marcadis, Z. Khan, S. Liu, M. Rodriguez, J. I. Lew; University of Miami Miller School of Medicine, Division of Endocrine Surgery, Miami, FL, USA.

Patients who undergo surgical resection for benign thyroid disease may have incidental papillary thyroid cancer (PTC) discovered on final pathology. This study determines if there are preoperative factors that predict a higher risk of incidental PTC in patients with benign thyroid disease. A retrospective review of prospectively collected data of 1822 consecutive patients who underwent thyroidectomy at a single institution was performed. Of these patients, 355 underwent surgical resection for benign thyroid disease, such as obstructive or compressive symptoms (n=142), hyperthyroidism (n=111), goiter size >4 cm (n=92), and substernal goiter (n=10). Patients with indeterminate or malignant preoperative FNA results were excluded. Incidental cancers were defined as PTC discovered only on final pathology. Age and gender of patients were evaluated to determine if there was an association with incidental thyroid cancer. Overall, 14% (50/355) of patients who underwent surgical resection for benign thyroid disease had incidental PTC on final pathology. Of all patients, 74% (n=263) underwent total thyroidectomy, and 26% (n=93) underwent thyroid lobectomy. Patients treated for obstructive symptoms had an incidental PTC rate of 19% (27/142), followed by goiters >4 cm at 15% (14/92), and hyperthyroidism at 8% (9/111). Benign final pathology included nontoxic multinodular goiter (MNG) (n=136), toxic MNG (n=75), nontoxic solitary nodule (n=67), toxic solitary nodule (n=21) and Graves’ disease (n=6). Patients <50 years of age with benign indications for surgical resection had incidental PTC rates of 18% (33/187), significantly higher than patients >50 years of age (10%, 17/169) (p=0.0394). There was no significant difference between incidental PTC rates in women (14%) vs men (12%). There is a higher than expected rate of incidental PTC in patients who undergo operations for benign thyroid disease, especially in those <50 years of age. Therefore, total thyroidectomy by an experienced surgeon should be strongly considered when managing benign thyroid disease in such patients.
Oncology

11:00 AM

SENTINEL LYMPH NODE BIOPSY IN ENDOMETRIAL AND CERVICAL CANCER. Daphne Papatheomas, BS1,2 Fausto Andrade, MD2, Ricardo Estape, MD2 Nillofar Nasseri-Nik, MD2 Eric Schroeder, MD2 Karissa Lopez, PA2 John P. Diaz, MD2 (1) University of Miami Miller School of Medicine, Miami, FL 33131. (2) South Miami Gynecologic Oncology Group, Miami, Florida 33134

Sentinel lymph node (SLN) biopsy is a new surgical technique for the staging of gynecological malignancies. The goal of the study is to describe our first experience with a modified surgical lymph node assessment of mapping and sentinel lymph node biopsy using minimally invasive approach for cervical and uterine malignancies. Since February 2013, our institution has been utilizing near-infrared (NIR) fluorescence imaging for robotic surgical cases. For this analysis, we identified all cases of newly diagnosed endometrial and cervical cancer undergoing a minimally invasive staging procedure with sentinel lymph node mapping. In all cases, SLN mapping was performed with an intra-cervical injection of indocyanine green (ICG) divided into the 3- and 9-o'clock positions before laparoscopic entry. A total of 32 patients were identified from February 2013 through July 2014. The median age of diagnosis was 56.5 (range 28-77) years old. The median BMI was 32.4 (21.6-46.2). SLN mapping was successful in 29 (91%) of 32 patients. A total of 75 SLN were identified, with a median of 2 SLN per patient (range, 0-6 SLN). The majority of SLN were located at two main sites, external iliac (66%) and obturator (60%). The median number of lymph nodes (LN) was 11 (range, 1-34 LN). The median estimated blood loss was 50 ml (range, 25-250 ml). Three patients experienced intra-operative or postoperative complications not related to the SLN mapping. In conclusion, SLN mapping with a minimally invasive approach is feasible in newly diagnosed gynecological malignancies. NIR fluorescence imaging with intra-cervical ICG injection using the robotic platform has a high bilateral SLN detection rate. SLN mapping was successful in 95% of the cases, which is comparable to larger studies using robotic and conventional laparoscopic techniques.

Surgery

11:15 AM

DETRIMENTAL EFFECT OF BLOOD-PRODUCT TRANSFUSION ON SURVIVAL FOR PATIENTS WITH BREAST CANCER IN FLORIDA (1996-2007). Hattan A. Alghamdi, Tulay-Koru Sengul, Feng Miao, Dido Franceschi, Margaret M. Byrne. Department of Public Health Sciences, Department of Surgery, Sylvester Comprehensive Cancer Center, University of Miami Miller School of Medicine, Miami, Florida.

There has been a large body of evidence discussing the immunological effects of blood transfusion on survival in patients with a number of different types of cancers. However, few studies have examined the effect of blood transfusion on survival in breast cancer patients and these have had inconsistent results. Data from Florida Cancer Data System, Agency for Health Care Administration (AHCA), and the US Census were linked for female breast cancer patients in Florida (1996-2007) to understand the association of blood transfusion with female breast cancer patients’ survival. Multivariate regression analyses for overall survival were adjusted for potential confounders related to blood transfusions, including age, gender, race, treatment received, and comorbidities. Among 120,940 patients identified, 17,686 (14.6%) received blood transfusion during the course of their treatment. The majority of patients were White (90.4%) and non-Hispanics (90.2%); most live in neighborhoods with middle-high (37.9%) and middle-low (29.2%) SES. Patients who had transfusion were older (mean=66 years), with more advanced SEER stages (24.6% metastasis vs. 20% regional, direct extension, lymph nodes). Transfusion rates were higher in Blacks (18.8%) than in Whites (14.3%) and other races (8.2%). Medicare patients (18.1%) received more blood transfusion compared to private insurance and non-insured population (12%, both). Patients who received chemotherapy (16.8%) had higher rates of blood transfusion compared to the non-chemotherapy group (13.8%). Transfused patients had a shorter overall median survival time (7.7 years) compared to non-transfusion group (12.7 years). Among patients who received transfusion, Blacks, non-Hispanics, and patients with lower SES categories had lower overall median survival. Multivariate analysis indicated that blood transfusion is a significant independent predictor for survival (HR=1.08;95%CI:1.03,1.13; p<0.001) after adjusting demographic, clinical, and comorbid factors. Our study concludes that blood transfusion is a significant independent risk factor for survival in female breast cancer patients.
Oral Presentations II

February 26, 2015
Cell Biology

2:15 PM
APPLICATION OF MODULAR THERAPY FOR RENOPROTECTION IN EXPERIMENTAL CHRONIC KIDNEY DISEASE. David Kepecs, Yanling Zhang, Kerri Thai, Suzanne L. Advani, Darren A. Yuen, Kim A. Connelly, Hari Kosanam, Eleftherios Diamandis, Michael V. Sefton and Richard E. Gilbert. Institute of Medical Science, University of Toronto, Toronto, ON.

Cell-based regenerative therapies, offer a new, alternative approach to the treatment of chronic disease. Specifically, studies by our laboratory and others have shown that a subpopulation of cells derived from the bone marrow, known as early outgrowth cells (EOCs), are able to attenuate the progression of chronic kidney disease (CKD). Here we examined the efficacy of a tissue engineering system, in which EOCs were embedded into submillimeter sized collagen cylinders. These small individual units are referred to as modules and together form a functional microtissue. Due to their resemblance to endothelial cells, late outgrowth cells (LOCs) were used to coat the module surface and hypothesized to promote vascularization and enhance engraftment of the encapsulated EOCs. These coated modules were transplanted subcutaneously into the subtotally nephrectomized rat model of CKD. While coated module therapy significantly improved both renal structure and function, non-coated modules with embedded EOCs were unable to reproduce these salutary effects on the kidney. Nevertheless, in both treatments, the embedded EOCs quickly degraded the modular environment and were seen to migrate to organs of the reticuloendothelial system as early as 6 days after transplantation. With the efflux of EOCs and, unexpectedly, no evidence of vascularization, we hypothesized that the LOCs did not enhance EOC engraftment, but rather augmented the renoprotection provided by EOCs by secretion of their own soluble and potent anti-fibrotic factors. To the best of our knowledge, this is the first study to document an effective subcutaneous approach for renoprotection.

2:30 PM
TNFA-INDUCED PODOCYTE INJURY IN FOCAL SEGMENTAL GLOMERULAR SCLEROSIS THROUGH AVB3 INTEGRIN ACTIVATION. Farah Leclercq 1,2, Christopher E. Pedigo 1,2, Alessia Fornoni 1,2, Sandra Merscher 1,2 1Division of Nephrology and Hypertension, University of Miami, Miami, FL. 2Katz Family Drug Discovery Center, University of Miami, Miami, FL.

Focal Segmental Glomerulosclerosis (FSGS) is a disease characterized by podocyte damage and scarring of the glomeruli. FSGS accounts for up to 20% of end stage renal disease (ESRD) in the United States. Current treatments for patients with steroid resistant FSGS include Cyclosporin A, a calcineurin inhibitor that suppresses the Nuclear Factor of Activated T cell (NFAT) pathway. Podocyte urokinase-receptor (UPAR) dependent αVβ3 integrin activation and decreased glomerular sphingomyelin-like phosphodiesterase 3b (SMPDL3b) expression was described in glomeruli of patients with FSGS and it was suggested that a circulating factor is involved in the pathogenesis of FSGS. However, the circulating factor(s) that regulate podocyte SMPDL3b expression and αVβ3 integrin activation in FSGS remain to be identified. Clinical data suggest that circulating TNFα is increased in some patients with FSGS and anti-TNFα antibody therapy proved beneficial in the treatment of a subset of patients with recurrent FSGS. Nonetheless, a potential link between TNFα, αVβ3 integrin activation and NFAT signaling has not been established. We tested the hypothesis that TNFα induces αVβ3 integrin activation in an SMPDL3b/NFAT dependent manner. Treatment of human podocytes with TNFα significantly reduced SMPDL3b mRNA expression (p<0.05), increased UPAR mRNA (p<0.05) and protein expression (P<0.05), and activated αVβ3 integrin (p<0.05). Inhibition of NFAT signaling with Cyclosporin A protected from TNFα induced SMPDL3b downregulation but did not protect from increased UPAR expression. Increased SMPDL3b expression prevented TNFα induced αVβ3 integrin activation (p<0.05). Injection of recombinant TNFα in mice caused significant albuminuria (p<0.05). In conclusion, our data suggests that circulating TNFα in FSGS contributes to podocyte injury by NFAT-mediated inhibition of SMPDL3b expression, which is necessary to allow for UPAR-mediated αVβ3 integrin activation and podocyte injury in FSGS.
2:45 PM

CHARACTERIZING CAMKII ISOFORM EXPRESSION IN FAILED ARTERIOVENOUS FISTULAS
Ravi Shah, Francis Jourd'heuil\(^1\), David Conti\(^2\), Arif Asif\(^3\), Harold Singer\(^1\), David Jourd’heuil\(^1\), Roman Ginnan\(^1\).
\(^1\)Center of Cardiovascular Sciences, \(^2\)AMC-Surgery and Transplant Center, \(^3\)AMC-Division of Nephrology, Albany Medical College, Albany, NY.

Arteriovenous fistula (AVF) surgeries are the current gold standard to maintain vascular access for patients requiring hemodialysis treatment for kidney failure. However, AVFs have a failure rate of 50%. Of the 50% of fistulas that do initially succeed, 35% fail after two years and require revision fistulas\(^1\). Neointimal hyperplasia, the primary cause of failure to these fistulas, has not been characterized molecularly. Calcium/calmodulin-dependent protein kinase II (CaMKII), a multifunctional serine/threonine kinase, has been identified to be important in vascular smooth muscle cell (VSMC) proliferation causing arterial hyperplastic injury. The overall purpose of this study is to characterize neointimal hyperplasia molecularly by determining the potential role of CaMKII in the development of the AVF venous neoplastic response. AVF samples were obtained from patients undergoing revision fistulas that failed due to neointimal hyperplasia and from placement fistulas that require initial vascular access. CaMKII isoform expression was compared in patient-derived samples, including revision AVF, placement AVF, and cultured de-differentiated venous VSMC by immunofluorescence, Western blotting, and qPCR with isoform specific antibodies. Immunofluorescence indicated a higher local expression level of CaMKIIδ2 and CaMKIIδ6 in the neointima of the venous samples from revision fistulas. No neointima was present in placement fistula samples. From western blot analysis and qPCR analysis, CaMKIIδ2, CaMKIIδ6, and CaMKIIγ isoforms were expressed at higher levels in the neoplastic revision AVF samples compared to the placement AVF samples or cultured VSMC. Since revision AVF had the highest expression of CaMKII isoforms, further investigation of the functional role of CaMKII is required in the context of neointimal hyperplasia. Interestingly, the cultured de-differentiated venous SMC had low expression of CaMKII isoforms, indicating that CaMKII expression seen in the revision AVF samples may not solely be derived from the phenotypic modulation of VSMC. Rather, it is speculated that the robust CaMKII expression in the revision AVF samples is derived from an inflammatory component.

3:00 PM

PROGRESSIVE MORPHOLOGICAL AND FUNCTIONAL DEGENERATION IN THE RETINA OF A MOUSE MODEL OF MAK-ASSOCIATED RETINITIS PIGMENTOSA . Christine L. Bokman\(^1\), Ning Wang\(^2\), Yiwen Li\(^3\), Ron Wen\(^3\), Tracey Topacio\(^3\), Byron L Lam\(^1\)\(^1\)Bascom Palmer Eye Institute, 900 NW 17th St, Miami, FL.

The purpose of this study was to measure the change in length of the rod outer segment over a 12-month period and to characterize the functional degeneration in a mouse model of male-associated kinase-knockout (Mak-KO) cause of retinitis pigmentosa (RP). In the study, Mak-KO mice for the autosomal were compared with control mice across a 12-month period at 6 times points: 20 days, 1 month, 3 months, 6 months, 9 months and 1 year. Light microscopy was used to characterize and measure the length of the rod outer segment. Electroretinography (ERG) was used to compare electrical responses of rods between Mak-KO and control mice. Mak-KO mice showed progressive rod outer segment shortening compared to the control mice at congruent time points; the control mice did not demonstrate any shortening in length. Similarly, the outer nuclear layer (ONL) of the retina in the Mak-KO mice progressively degenerated at a rate of 2 layers per time point compared to the ONL of the control mice. ERG electrical waveforms decreased in amplitude steadily overtime in Mak-KO mice and remained stable in the controls. The results demonstrate that the Mak gene plays a significant role in the maintenance of the rod outer segment. Mak-KO mice showed progressive degeneration of the rod outer segment, suggesting a role in the maintenance of its length. Mak-KO mice also had a significantly thinner ONL than the controls. Finally, we were able to measure the mice over a considerable period of time, which suggests Mak mutant causes progressive degeneration.
Ophthalmology

3:15 PM

HUMAN TEAR SEROTONIN LEVELS CORRELATE WITH SYMPTOMS AND SIGNS OF DRY EYE

1,2Priyanka Chhadva, 1Tinthu Lee, 1,3Constantine Sarantopoulos MD, 1Abigail Hackam, 3Allison McClellan, 1,3Elizabeth Felix PhD, 1,3Roy Levitt MD, 1,3Anat Galor MD. 1University of Miami Miller School of Medicine, Miami, Florida; 33136; 2Bascom Palmer Eye Institute, Miami, Florida, 33136; 3Miami Veterans Administration Medical Center, Miami, Florida, 33125

Serotonin, a neurotransmitter known to be involved in nociceptor sensitization, is present in human tears. This study evaluates whether tear levels of serotonin are associated with certain aspects of dry eye (DE), including symptoms, signs, and clinical descriptors of neuropathic ocular pain (NOP). Sixty-two patients with normal eyelid and corneal anatomy were prospectively recruited from a Veterans Administration Ophthalmology Clinic over 11 months. DE symptoms (Ocular Surface Disease Index [OSDI]), DE signs (tear break-up time [TBUT], corneal staining, and Schirmer’s score), and presence of clinical descriptors of neuropathic ocular pain (NOP) (sensitivity to light and/or sensitivity to wind) were assessed. For tear analysis, each patient’s tears were collected after instilling 50μl of sterile saline to the lower cul-de-sac of each eye and using capillary action microcaps to collect the ocular wash. Tear serotonin levels were measured using enzyme-linked immunosorbent assay. Serotonin concentrations negatively correlated with Schirmer’s scores (r=-0.28; p=0.01), but did not correlate with other DE parameters, such as OSDI scores, sensitivity to light or wind, TBUT, or staining. According to our hypothesis, we divided patients into groups based on both DE symptoms and aequous tear production; serotonin concentrations were found to be significantly higher in DE group 1 (OSDI≥6 and Schirmer’s<8) compared to both DE group 2 (OSDI≥6 and Schirmer’s≥8) and controls (OSDI<6 and Schirmer’s≥8). Patients in the DE group 2 more frequently complained of sensitivity to light (64%) and wind (67%) compared to the DE group 1 (40% and 60%, respectively) and more significantly than controls (8% and 17%, respectively) (p<0.0005 and p=0.001, respectively). Patients with DE symptoms and aqueous tear deficiency had higher tear serotonin levels suggestive of corneal peripheral sensitization compared to those with DE symptoms but normal tear production and those without DE symptoms nor tear deficiency.

3:30 PM

INVESTIGATING THE PROTECTIVE EFFECTS OF GRAPE DIETARY SUPPLEMENTATION IN RETINAL DEGENERATIONS. Maria Esperanza Rodriguez, Tinthu Lee, Ashley Davis, Amit Patel, and Abigail Hackam. Bascom Palmer Eye Institute, University of Miami Miller School of Medicine, Miami, FL, United States

Retinal degenerative diseases, such as AMD and retinitis pigmentosa, are a major cause of vision loss and affect over 5 million people in the USA. An effective nutritional therapy would permit disease prevention or treatment without the potential side-effects that occur with pharmaceutical interventions. The purpose of this study was to test whether a diet supplemented with grapes, in the form of chemically defined freeze-dried grape powder (FDGP), protected photoreceptors in two mouse models of retinal degeneration. The mouse rd10 genetic model of retinitis pigmentosa, and the induced oxidative stress mouse model of AMD, were fed a grape-supplemented diet, sugar-matched control diet, or normal chow control diet, from birth for the rd10 mice, and for 5 weeks prior to injury for the PQ-injured mice. The rd10 mice were analyzed at post-natal days (P)18 to P32, and the oxidative stress model was analyzed at 2 weeks post injury; the injury was subretinal injection of 1 mM paraquat (PQ) in C57Bl/6 mice. Photoreceptor function was analyzed using ERGs and retinal outer nuclear layer (ONL) thickness was measured using optical coherence tomography. Levels of specific proteins were analyzed by Western blots on whole retinas. We demonstrated that rd10 mice fed the grape-supplemented diet showed up to 3-fold higher rod and cone photoreceptor responses when compared with the control diets (N=7, p<0.001). Grape diet also had a 40% decrease in phospho-P65 NFKB and a 20% decrease in the microglia marker IBA1 (N=5), suggesting reduced inflammatory responses. The grape diet also significantly rescued the retina in the oxidative stress model, and showed up to 2-fold higher photoreceptor responses and 17% thicker ONL compared with mice on the control diet (N=8, p<0.05). Retinal lysates were analyzed by Western blot with antibodies against phosphorylated and total GSK3β. Our results showed that GSK3β was increasingly inactivated by phosphorylation (shown by ratio of pGSK3β to total GSK3β) in grape-fed mice. These findings suggest a role for GSK3β inactivation in retinal degeneration. The grape diet increased levels of inactive GSK3β compared with control diet in the PQ-injected mice (N=3, GPQ vs CPQ p=0.031). In conclusion, these results demonstrate that a diet supplemented with grapes significantly protected retinal structure and function in two different mouse models of retinal degeneration. The mechanism of protection by grapes may involve inhibition of GSK3β-mediated cell death pathways and decreased retinal immune response. Therefore, our study suggests the possibility of grape supplementation as an adjuvant nutritional therapy for the prevention of retinal degenerative diseases.
Immunology

3:45 PM

MIR-33 ANTAGONISM INHIBITS ATHEROSCLEROSIS PROGRESSION BY PROMOTING MACROPHAGE ALTERNATIVE ACTIVATION. Hasini Ediriweera1, Mireille Ouimet1, Frederick Sheedy1, Katey Rayner*, Christine Esau1, Morgan Fullerton*, Gregory Steinberg*, Kathryn Moore* 1Department of Medicine, NYU, New York, NY.. 2University of Ottawa Heart Institute, Ottawa, ON, Canada. 3Regulus Therapeutics, San Diego, CA.

MicroRNA-33 (miR-33) plays a central role in regulating HDL biogenesis and reverse cholesterol transport (RCT) via post-transcriptional repression of cholesterol efflux genes. Delivery of miR-33 inhibitors to mice and non-human primates fed a chow diet raises plasma HDL cholesterol and increases RCT, suggesting miR-33 as a promising therapeutic target for the treatment of atherosclerosis. However, recent studies in mice have shown that the HDL-raising effects of anti-miR33 are blunted during Western diet feeding, and its efficacy in reducing atherosclerosis progression under these conditions remains controversial. To further test the effects of miR-33 inhibition during atherogenesis, we treated Ldlr−/− mice with control or anti-miR33 oligonucleotides for 8 weeks coincident with Western diet feeding. Morphometric analyses revealed that anti-miR33 reduced atherosclerosis progression by 40% in both the aortic root and the aorta en face without altering plasma total or HDL cholesterol. Because anti-miR33 accumulates in plaque macrophages where it may directly alter gene expression, we performed Nanostring profiling of plaque macrophages isolated by laser capture microdissection. We found a notable increase in markers of alternatively activated M2 macrophages in anti-miR33-treated mice compared to controls. Moreover, in vitro transfection of peritoneal macrophages with anti-miR33 reduced the expression of M1 markers and increased the expression of M2 markers, suggesting that miR-33 directly controls the inflammatory polarization of macrophages. This regulation of macrophage polarization by miR-33 was found to be dependent on the targeting of AMP Kinase, a key integrator of cellular energy homeostasis. Notably, inhibitors of AMPK blocked the anti-inflammatory effects of miR-33 inhibition in vitro and in vivo. Collectively, these results identify a novel role for miR-33 in the regulation of macrophage inflammation and show that antagonism of miR-33 prevents atherogenesis, in part, by reducing plaque inflammation through the polarization of macrophages from a pro-inflammatory M1 to a reparative M2 state.

4:00 PM

ALLERGIC PULMONARY INFLAMMATION ACCELERATES METASTASIS: IS THERE A ROLE FOR IMMATURE LEUKOCYTE SUBPOPULATIONS? Stephanie Libreros1, Ramon Garcia-Areas1, Nathalia Gazaniga1, Patricia Keating2, Phillip Robinson1, and Vijaya Iragavarapu-Charyulu1. 1Charles E. Schmidt College of Medicine, Florida Atlantic University, Boca Raton, FL. 2Charles E. Schmidt College of Science, Florida Atlantic University, Boca Raton, FL.

It is well-established that disseminated metastasis accounts for a majority of cancer-related deaths and that breast cancer metastasizes to the lungs. It is known that immature myeloid cells play a role in tumor growth. Our previous work has demonstrated that myeloid cells are present in the pre-metastatic lungs of mammary tumor bearers and that these cells may contribute towards establishment metastatic foci by infiltrating tumor cells. Allergen sensitized mice implanted with 4T1 mammary tumors had a 5-fold increase in formation of metastatic foci in their lungs compared to control mammary tumor bearers. Further, allergic mice showed accelerated tumor growth and shorter disease-free survival. To understand the host responses that contributes to accelerated rate of metastasis to the lungs, we determined the alterations in leukocyte subpopulations that could occur during allergic pulmonary inflammatory response. We and others have observed increased numbers of myeloid suppressor-like subpopulations in mice sensitized with ragweed allergen and these numbers were further increased upon inoculation with mammary tumor cells. Mammary tumor-bearing mice with allergic pulmonary inflammation had higher levels of pro-inflammatory mediators, CHI3L1, CCL2, CXCL2 and MMP-9 but decreased levels of IFN-γ compared to mice with allergic pulmonary inflammation alone. We show that allergic pulmonary inflammatory microenvironment attracts leukocyte subpopulations and that this inflammatory milieu supports incoming mammary tumor cells for establishment of metastasis. Mammary tumor-bearing control mice and mice sensitized with ragweed allergen prior to tumor implantation were assessed for monocytic and myeloid lineages in the pulmonary microenvironment. We found that there were increased numbers of immature monocytic and myeloid subpopulations lungs of mammary tumor-bearing mice previously sensitized with ragweed allergen. These studies suggest that altered pulmonary microenvironment in allergen sensitized mice could create a niche for incoming mammary tumor cells and accelerate metastatic growth.
Microbiology and Infectious Diseases

4:15 PM

THE ANTI-TOXIN PROPERTIES OF GRAPE SEED PHENOLIC COMPOUNDS. Patrick Cherubin, Camilla Garcia, David Curtis, and Ken Teter. Burnett School of Biomedical Sciences, College of Medicine, University of Central Florida.

_Vibrio cholerae_, _Escherichia coli_, _Ricinus communis_, _Pseudomonas aeruginosa_, and _Corynebacterium diphtheriae_ produce AB toxins which share the same basic structural characteristics: a catalytic A subunit attached to a cell-binding B subunit. All AB toxins have cytotoxic targets despite an initial extracellular location. AB toxins use different methods to reach the cytosol and have different effects on the target cell. Broad-spectrum inhibitors against these toxins are therefore hard to develop because they use different surface receptors, entry mechanisms, enzyme activities, and cytotoxic targets. In many cultures, herbal remedies have been used for centuries to help alleviate diarrheal diseases like cholera. We have found grape seed extract provides resistance to five different AB toxins: cholera toxin (CT), Shiga toxin (ST), ricin, _P. aeruginosa_ exotoxin A, and diphtheria toxin. To identify individual compounds in grape seed extract that are capable of inhibiting the activities of these AB toxins, we screened twenty common phenolic compounds of grape seed extract for anti-toxin properties. Twelve compounds inhibited CT, one inhibited ricin, four inhibited exotoxin A, and three inhibited diphtheria toxin. No individual compound conferred resistance against ST. However, a cocktail of all 20 compounds conferred resistance against ST. Additional studies were performed to determine the mechanism of inhibition against CT. Two compounds inhibited CT binding to the cell surface and even stripped bound CT off the plasma membrane of a target cell. We have thus identified individual toxin inhibitors from grape seed extract and one of their mechanisms of inhibition against CT. This work will help formulate a defined mixture of phenolic compounds that could potentially be used as a therapeutic against a broad range of AB toxins.

4:30 PM

KAPOSI’S SARCOMA-ASSOCIATED HERPESVIRUS (KSHV) K-RTA PROTEIN IS ACETYLATED IN THE PEPTIDE AA515-534, TO POTENTIALLY AFFECT ITS TRANSCRIPTIVATION FUNCTIONS. Francisco Puerta Martinez¹,², Qiyi Tang² and Ana Maria Castejon¹ ¹College of Pharmacy Nova Southeastern University, University Drive, Fort Lauderdale, USA. ²Ponce School of Medicine and Health Sciences, Department of Microbiology, Zona Ind. Reparada 2, Ponce, PR.

KSHV is a herpesvirus that displays two different lifecycle phases, latency and lytic replication. The switch between latency and lytic replication is regulated by different factors, such as epigenetic factors, however the critical event in the viral reactivation is the expression of the KSHV RTA (replication and transcription activator), the major viral transcription factor of the lytic cycle. Many investigations have focused on the functions of this key KSHV protein, however their role in the lytic cycle and their functional mechanisms are not yet fully understood. This study has found that the interaction between K-RTA and the cellular co-activators P300/CBP, which have intrinsic KAT activity (i.e. histone acetylation activity), lead to direct acetylation of K-RTA to potentially regulate its transactivation functions. In order to identify potential putative acetylation motifs a predictive algorithm proposed by Ryan et. al in 2006, was used to analyze the K-RTA sequence. Based on the predictive analysis a series of deletional mutations across the K-RTA protein length were performed and the resulting K-RTA mutants, 12 in total, were then cloned into the pCR3.1 expression plasmid. These plasmids were transfected into HEK293 cells, and expression of the mutant K-RTA proteins was monitored by Western blotting using an anti-RTA antibody. Acetylation was detected by Western blotting using an anti-acetyl lysine antibody and mass spectrometry. Although the exact acetylation site of K-RTA could not be confirmed, the peptide aa 515-534 was identified as essential for K-RTA acetylation, since the mutant D4 lacking this sequence (deletion: ATTPKRKQRSKERSSKKRKRA) was found consistently unacetylated. As a conclusion, the finding that K-RTA is acetylated brings a new tool to dissect its role in the lytic cycle of KSHV and therefore a better understanding of its pathogenesis.
LEPTIN INDUCES SITE SPECIFIC TRANSACTIVATION OF VEGFR-2/NOTCH CROSSTALK IN ENDOTHELIAL CELLS. Viola Lanier, Merle Leffers, Johannes Waltenberger M.D., Ph.D., F.E.S.C., Leonard Anderson PhD, and Ruben R. Gonzalez-Perez PhD., Department of Microbiology, Immunology, and Biochemistry Morehouse School of Medicine, Atlanta GA.

Notch and leptin signaling play essential roles in breast cancer (BC) development and angiogenesis. We have shown that Notch is upregulated by leptin in several cancer types (i.e., breast, pancreatic and endometrial cancer) [1]. We have also found that leptin is a proliferation and angiogenic factor for breast cancer, which crosstalks to several oncogenic signaling pathways, stimulates cell proliferation, cell survival and migration. Additionally, we found that leptin induces the expression of the vascular endothelial growth factor (VEGF) and its receptor type 2 (VEGFR-2) [2,3]. We found that binding of leptin to its receptor (OB-R) in endothelial cells (EC) transactivates VEGFR-2, inducing angiogenic features [2] and increasing expression of Notch [4]. We hypothesize that Notch expression and development of angiogenic features in EC are dependent on leptin actions, and occurs through site-specific transactivation/phosphorylation of VEGFR-2. To assess the role of VEGFR-2 and Notch in leptin induction of angiogenic features, human umbilical vein (HUVEC), and porcine aortic endothelial cells (PAEC, that do not express VEGFR-2) were used. PAEC were stably transfected to overexpress VEGFR-2 and challenged with leptin (0.6, 1.2, 6.2 nM), and inhibitors of Notch (DAPT) and VEGFR-2 kinase (SU5416). Additionally, VEGFR-2 knockout via silencing RNA was used to determine VEGFR-2 role in leptin-mediated induction of Notch. Several antibodies were also used to determine whether leptin-induced specific phosphorylation/activation of VEGFR-2 (Y1175, Y1214, Y951, and Y966). The effects of leptin-induced Notch and VEGFR-2 on HUVEC’s tubule formation were assessed via matrigel assays. Our studies show for the first time that leptin is a potent inducer of site-specific phosphorylation/transactivation of VEGFR-2 that increases Notch expression, and contributes to the development of angiogenic features in EC. Leptin secreted either by breast cancer cells or adipose tissue could contribute to tumor angiogenesis by acting directly on endothelial cells; thus inducing VEGFR-2/Notch crosstalk. Combinatory therapies that target leptin, VEGFR-2 and Notch signaling could be a new strategy for breast cancer therapy, specifically in obese patients showing the highest levels of leptin and poor outcomes.
Oral Presentations III

February 27, 2015
Dermatology

LONG-TERM EMOTIONAL AND PHYSICAL COMPLICATIONS OF STEVENS-JOHNSON SYNDROME (SJS) AND TOXIC EPIDERMAL NECROLYSIS (TEN). Cristina Olteanu, Roni P. Dodiuk-Gad, Rena Hashimoto, Hall Chew, Sonia Whyte-Croasdile, Marjorie Burnett, Shachar Sade, Anthony Feinstein, Robert Cartotto, Marc Jeschke, Neil H. Shear. 1 University of Toronto, Toronto, ON, 2Sunnybrook Health Sciences Centre, Toronto, ON, 3SJS and TENS Group Canada-CAST International, Toronto, ON.

Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are life threatening mucocutaneous reactions resulting mainly from drugs. We aim to characterize the long-term emotional and physical complications of SJS/TEN. Patients ≥18 who survived SJS/TEN were assessed using various parameters. Emotional assessment was conducted by 3 validated questionnaires: Revised Impact of Events Scale, General Health Questionnaire, and Hospital Anxiety and Depression Scale. Health-Related Quality of Life was assessed by 3 validated questionnaires: Dermatology Life Quality Index, EQ-5D, Skindex-29 and one specially designed for the study. Medical assessment was conducted by an interview and by skin, oral mucous membrane and detailed ophthalmic exam. We herein report preliminary results gathered from 13 patients with mean 66.3±80.3 months (range 1-228) following SJS/TEN. Symptoms of post-traumatic stress were reported in 9/13 participants (Revised Impact of Events Scale, mean total score=25.5±21.4). Symptoms of psychological distress were reported in 11/13 participants (General Health Questionnaire, mean total score=18.6±7.4) and symptoms of moderate anxiety were present in 7/13 participants (Hospital Anxiety and Depression Scale, mean total score=10.8±4.8). In 10 out of 13 participants signs of moderate depression were demonstrated (Hospital Anxiety and Depression Scale, mean total score=11.2±2.6). Skindex-29 indicated a severely impaired Health-Related Quality of Life in 9/13 participants (mean total score=63.5±27.1). Participants rated their general health at a mean of 64.6/100±18.3 (EQ-5D Visual Analogue Scale). The Dermatology Life Quality Index indicated a moderate to an extremely large effect on the lives of 8/13 participants (mean total score=8.3±8.1). Eleven out of 13 participants expressed fear in using medication. Eleven out of 13 participants reported long-term physical complications, the most common being cutaneous and ophthalmic. Based on preliminary results, SJS/TEN leaves lasting negative emotional and physical complications, with most participants reporting post-traumatic stress symptoms. We hope that this study will lead to a better understanding of the issues facing SJS/TEN survivors and will provide strategies for the management of these patients.

PROGNOSTIC IMPLICATIONS OF ACQUIRED GENETIC MUTATIONS IN MALIGNANT MELANOMA. Christina Del Guzzo, Krystle Collins, Bret Taback, MD. Department of Surgery, Columbia University College of Physicians and Surgeons, New York, NY.

Acquired mutations in BRAF, NRAS and c-kit have important implications in melanoma. Mutations in these genes are used as therapeutic targets in patients with advanced stage disease, but their prognostic utility in advanced stage melanoma is unknown. In this study we assessed the prognostic utility of genetic mutation analysis in patients with high-risk primaries or those with recurrent disease presenting to our melanoma center. A retrospective study of tissue samples collected from patients with advanced stage melanoma at a large academic center was performed. Tissue samples were obtained prospectively and serially in patients diagnosed with melanoma presenting with a high-risk primary melanoma (>4mm), lymph node positive disease or disease recurrence. The samples were sent to Genoptix for evaluation of BRAF, NRAS and c-KIT mutations. Patient demographics, clinical pathologic factors and clinical outcomes were assessed. Tissue samples from 42 patients were submitted for testing, of which 38 resulted. Four patients had insufficient material for analysis. Among the 38 patients, 11 (28.9%) had BRAF mutations, 12 (31.5%) had NRAS mutations, no patients had c-KIT mutations, and 15 patients (39.4%) were wild type. Mean age was 73 in BRAF in comparison to 64 in NRAS, median tumor thickness was 2.6mm in BRAF compared to 2.9mm in NRAS, and 4 BRAF patients versus 2 NRAS patients had positive sentinel lymph nodes. 6 recurrences were noted in each. Median time from initial diagnosis to first recurrence was 11 months in NRAS patients in comparison to 15 months in BRAF patients. In this study of high-risk melanoma patients, a greater proportion harbored either NRAS or BRAF mutations than c-KIT. Furthermore, NRAS mutations were associated with an earlier age of presentation and earlier recurrence, whereas BRAF mutations were associated with a higher number of positive sentinel lymph nodes. These results suggest that NRAS and BRAF mutational analysis may have predictive utility in identifying high-risk patients in whom more aggressive treatment and surveillance strategies are indicated.
**Epidemiology**

9:00 AM

**INDIVIDUAL AND COMMUNITY FACTORS CONTRIBUTING TO ANEMIA AMONG INDIGENOUS WOMEN AND CHILDREN LIVING IN BAJA CALIFORNIA, MEXICO.** Molly Moor, Stephanie Brodine, Richard Garfein, Miguel Fraga, Hooman Rashidi, John Elder. Department of Family and Preventive Medicine, Joint Doctoral Program in Public Health, University of California, San Diego & San Diego State University, San Diego, CA.

Disproportionately high anemia prevalence exists among the families of indigenous laborers in Northern Mexico. A cross-sectional study was performed in October 2012 among 118 women (15-49 years) and 25 children (24-59 months) living in a rural, agricultural community in Baja California, Mexico to measure anemia prevalence and to identify the individual and community factors contributing to causes of anemia. Participant recruitment was performed by the random selection of community households and the random selection of attendees at a free, temporary medical clinic. All participants completed a demographic, socioeconomic (SES), and dietary survey and received anemia. A sample of venous blood was collected from anemic women and children and sent to a laboratory for analysis. Anemic individuals also received vitamins and nutritional counseling. Six community grocery stores were also visited to ascertain the types of foods available for purchase. Anemia prevalence was 22% among women and 20% among children. Blood tests revealed all cases of anemia were due to nutritional deficiencies, with iron deficiency being the primary culprit in 100% of children and 80.8% of women. Other causes of anemia in women were vitamin B-12 deficiency (11.5%) and combined iron and vitamin B-12 deficiency (7.7%). Among women, low SES was significantly associated with being anemic (p=0.008) and enrollment in the government assistance program Oportunidades was associated with being anemic (p=0.042). Vitamin supplement use was protective against being anemic (p=0.024). Dietary assessments showed limited consumption of iron absorption promoting foods. Grocery store assessments revealed at least one type of meat and citrus fruit available for purchase at each store; however, leafy green vegetables were only available for purchase at one store. The high prevalence of anemia among women and children due to nutritional deficiencies, in conjunction with their limited food variety, demonstrates a need for community interventions.

9:15 AM

**THE IMPORTANCE OF INTEGRATING CULTURAL COMPETENCY IN COMMUNITY-BASED INTERVENTIONS: AN EXAMPLE OF MEDICINAL FOODS.** Jiang Sandy, Sarah Messiah, Guillermo Prado, Michelle Lampf, Cassandra Quave, Peter Brown, Michelle Parsons. 1University of Miami Miller School of Medicine, Miami, FL. 2Emory University, Atlanta, GA.

Cultural competency is an integral public health component and strategy of community-based interventions by understanding health beliefs of diverse populations. Medicinal foods are used by some cultures to treat specific illnesses, due to their secondary metabolites that have therapeutic values. The examination of medicinal food usage allows for better information about potential food/drug interactions and insight into future pharmaceutical research, which can lead to more optimal healthcare and intervention efforts. This study was conducted to (1) better understand the use of medicinal foods among Asian immigrants to the southern US; and (2) explore generational differences in beliefs and usage patterns. Randomized semistructured interviews were conducted with Taiwanese (n=60) and Chinese (n=60) immigrants. Both Taiwanese and Chinese groups had a total of 20 subjects each who immigrated to the US in the 1970’s, 1980’s, or 1990’s. A total of 30 plant specimens were collected and stored at the Emory University Herbarium for analysis. Data were explored for group differences in medicinal/plant food beliefs and usage. The 1970’s and 1990’s showed no significant differences between Taiwanese and Chinese immigrants in 6 different categories, but in the 1980’s showed significant differences with p values ranging from 0.0011-0.0471 on 7 health categories. Taiwanese reported higher preferences for TCM. Medicinal plants listed were high in antioxidant, antibacterial, and anti-inflammatory properties. Immigrants differed on their medicinal and health perspectives based on the decade they immigrated to the US. A decrease in medicinal food usage over time was found, as immigrants’ children acculturate to Western medicinal health systems. Understanding cultural health beliefs such as those captured here is crucial for informing both community-based health and wellness intervention efforts and healthcare professionals about patient behaviors and practices. This work will be translated into work with Familias Unidas in which EBI will be used to assess Latino adolescents and their parents’ perspectives and views on medicine through nutrition and exercise evaluations and programs.
THE IMPACT OF INTIMATE PARTNER VIOLENCE SCREENING AT MEDICAL STUDENT RUN COMMUNITY HEALTH FAIRS. Ishna Sharma, Dr. Pat Caralis. Department of Student Activities. University of Miami School of Medicine, Miami, FL.

Intimate partner violence continues to be a health problem in communities. Preventative measures in the form of IPV screening in health care settings have shown to be effective means to reduce the violence. However, less than half of healthcare professionals use any form of IPV screening in their daily practice. The University of Miami has seven, student-run community health fairs annually which provide screening and care for an estimated 1500 underserved patients. HEAL (Health Empowerment Achievement Life) is a student group at UMMSM dedicated to working with victims of IPV and educating other students and health professionals about the problem. We initiated an IPV screening program as a routine part of the health fair. The IPV screening is done by administering a short questionnaire at the “female health” station. We have partnered with a local women’s shelter to refer those who screen positive for help. We quantified the results of this program from 2014-2015’s series of health fairs to assess the quality and impact of this preventative measure. At one such health fair, 35 female patients were screened, of which 3, or 8.5%, were positive for domestic abuse without any immediate danger, and were referred to the appropriate resources. 31% of the total sample did not know how to contact the police in case of an imminent threat. 100% did not know about any local domestic violence assistance resources. Data is pending from the remaining 6 health fairs, it will have been collected and analyzed by the time of ESRF 2014.

Radiation Oncology

SUB-TOTAL LYMPHOID IRRADIATION IN THE TREATMENT AND PREVENTION OF PEDIATRIC CARDIAC ALLOGRAFT REJECTION—A 17-YEAR EXPERIENCE. Sherry X. Yan¹, A.K. Jain², S. Crockford¹, D. P. Horowitz², L.J. Addonizio³, S. K. Cheng². ¹Columbia University College of Physicians and Surgeons, New York, NY. ²Department of Radiation Oncology, New York Presbyterian Hospital Columbia Campus, New York, NY. ³Division of Pediatric Cardiology, Department of Pediatrics, Columbia University College of Physicians and Surgeons, New York, NY.

Total lymphoid irradiation (TLI) has been used to treat recurrent rejections in pediatric cardiac allograft recipients, albeit not without complications. We report the long-term outcomes of 21 pediatric heart transplant patients treated with sub-total lymphoid irradiation (STLI) either for the treatment of allograft rejection or as prophylactic therapy for prevention of recurrent rejection in a second heart transplant. Between 1996 and 2011, 21 pediatric heart transplant patients received STLI for treatment of recurrent rejections (n=10) or prophylactically for rejection prevention (n=11). Treatment consisted of 800 cGy in 10 fractions delivered twice a week to mantle and paraaortic/spleen fields. Rejection was defined as ≥ Grade 1B on endomyocardial biopsy by ISHLT criteria. Median follow-up was 9.1 years, ranging from 6 months to 17 years. Twenty patients completed treatment of 800 cGy over a mean of 36 days despite relative leukopenia during STLI; treatment was truncated in one patient due to cholangitis. In patients who received STLI for treatment of rejection, number of rejections dropped from 3.72 before treatment to 1.90 after treatment (p=0.04). Three patients were rejection-free after STLI. Percentage of patients experiencing rejection one year before treatment was 81.8 compared to 54.5% one year after STLI. Mean time to rejection was delayed from 121.3 days from transplant to 789.9 days from STLI completion (p=0.07). In patients who received STLI prophylactically, number of rejections was 2.4 during the first graft versus 2.1 during the second graft (p=0.72). Mean time to first rejection was 566.7 days compared to 962.8 days (p=0.73). Graft survival increased from 6.5 years in the first graft to 8.1 years in the second graft (p=0.53). Five-year graft survival rose from 50.0% to 85.7%, and ten-year graft survival rose from 10% to 66.7%. In all patients, there was no incident of hematological malignancies or blood dyscrasias during long-term follow-up as noted by other groups. The findings suggest that sub-total lymphoid irradiation is effective for treating recurrent rejections in pediatric cardiac allograft recipients. It also may be efficacious in prolonging second graft survival if given prophylactically. It is well tolerated and not associated with secondary hematological malignancies with long-term follow-up.
Radiology

10:00 AM

RESTING STATE fMRI AND PET CORRELATIONS IN THE DOPAMINE REWARD PATHWAY IN OBESE ADULTS WITH TYPE 2 DIABETES MELLITUS. Le Zhong, B.S., Waqas Majeed, Ph.D., Heidi Silver, Ph.D., Kevin Niswender, M.D., Ph.D., Malcolm J. Avison, Ph.D. 
1University of Miami Miller School of Medicine, Miami, FL. 2Lahore University of Management Sciences, Lahore, Pakistan. 3Vanderbilt University Medical Center, Nashville, TN. 4Vanderbilt University Institute of Imaging Science, Nashville, TN.

The dopamine (DA) reward pathway, whose dysfunction is a hallmark of drug addiction, has been hypothesized to exhibit similar alterations in obesity. A recent model of the DA reward pathway suggests that the nucleus accumbens (NAcc) integrates inputs from salience attribution areas including the amygdala, which drive motivated drug and food seeking behaviors, and frontal lobe areas such as medial orbitofrontal cortex (MOFC) that exert cognitive control over this motivational drive. Thus signals from the amygdala have been hypothesized to upregulate the reward pathway, while signals from MOFC have been hypothesized to suppress the cue-driven reward seeking. Using resting state functional magnetic resonance imaging (rsfMRI) and 18F-Fallypride positron emission tomography (PET) data combined with fat-water imaging, biochemical, and demographic data obtained from the 2nd and 6th weeks of a longitudinal study of the impact of insulin therapy on brain DA systems, we examined the impact of adiposity, insulin resistance, NAcc DA D2 receptor availability, and leptin on the relative strengths of the NAcc-Amygdala and NAcc-MOFC connections determined from the strength of the inter-regional temporal correlation (r2) of the resting state fMRI time course. We show that in obese adults with moderate type 2 diabetes mellitus, the ratio of visceral adipose tissue to lean tissue, a direct measure of adiposity, correlated negatively with the non-displaceable binding potential obtained from PET in several brain regions. Treatment with amphetamine (AMPH) led to a significant negative NAcc-MOFC r2 relationship with the total adipose tissue to lean tissue ratio (TAT/LT), but no significant change in the dependence of the NAcc-Amygdala r2 on TAT/LT. Treatment with insulin detemir led to decreased dependence of the NAcc-MOFC r2 and increased dependence of the NAcc-Amygdala r2 on TAT/LT. AMPH administration alone did not significantly change the NAcc-Amygdala r2, while it increased the NAcc-MOFC r2. There were significant correlations between fasting leptin levels, an indirect measure of adiposity, and the NAcc-Amygdala r2 but not the NAcc-MOFC r2. The NAcc-Amygdala r2 correlation with leptin levels was unaffected by AMPH, but the direction of the correlation was affected by insulin detemir treatment, suggesting an alternate mechanism through which leptin may mediate NAcc-Amygdala connections with modulation from insulin. Our studies provide evidence that adiposity and its correlates play both direct and indirect roles in modulating the DA reward pathway and affecting adjacent cortical regions that govern it.

Orthopedic Surgery

10:15 AM

RISK AND COST OF REOPERATION AFTER SINGLE LEVEL POSTERIOR CERVICAL FORAMINOTOMY: A LARGE DATABASE STUDY. Arash J. Sayari, Alexander Tuchman, Jeremiah R. Cohen, Zorica Buser, Jeffrey C. Wang, Department of Orthopaedic Surgery, University of Southern California, CA

Nonfusino methods of relieving cervical radiculopathy have grown in popularity, in hopes of preserving kinematics of the spine. Recent literature, however, has noted that posterior cervical foraminotomy (PCF) and other decompression methods may actually not be as beneficial as once thought. To better address these concerns, we aimed to examine the risk of undergoing another cervical operation after single level PCF, and the costs of such reoperations. We used the PearlDiver Database, made up of over 48 million Medicare and private payer insurance billing records between 2005 and 2012. We created cohorts of patients using Current Procedural Terminology (CPT) and International Classification of Diseases, ninth edition (ICD-9) procedure codes. We incorporated coding commands to identify patients who underwent single-level PCF, and also had various reoperations of interest, within 1, 2, and 4 years of follow-up. There were 2905 PCFs in the Medicare group with 1 year follow-up, 2287 with 2 year follow-up, and 1335 with 4 year follow-up. The incidence of reoperation was 8.3% at 1 year follow-up, 9.8% at 2 year follow-up, and 10.5% at 4 year follow-up. There were 3299 PCFs in the private insurance group with 1 year follow-up, 2346 with 2 year follow-up, and 818 with 4 year follow-up. The incidence of reoperation was 13.6% at 1 year follow-up, 16.7% at 2 year follow-up, and 17.0% at 4 year follow-up. In the Medicare group, posterior decompression and fusion was the most expensive reoperation, with a per patient average charge (PPAC) of $77,976. In the private insurance group, anterior fusion was the most expensive reoperation, with a PPAC of
CaM KINASES ARE CRITICAL FOR ACTIVITY-DEPENDENT GENE EXPRESSION IN PV+ INHIBITORY INTERNEURONS. Samuel M. Cohen and Richard W. Tsien. NYU Neuroscience Institute, NYU Langone Medical Center, New York, NY.

Excitation-transcription (E-T) coupling is essential for the long-term adaptive changes that occur during brain development, learning and memory, and drug addiction. Recent work has demonstrated that experience induces plastic changes in parvalbumin (PV) positive interneurons to regulate adult learning. Different behavioral paradigms modulate PV protein expression level, with direct implications for memory consolidation and structural synaptic plasticity. We study E-T coupling in PV+ interneurons. We employ pharmacological, imaging, and immunocytochemical techniques on cultured rat cortical neurons. Depolarization of PV+ neurons for 2 min with a 40mM K⁺ solution results in a marked increase in PV, GAD67, and c-fos expression in PV+ cells. This increase is blocked by Ca₂⁺/Calmodulin dependent Kinase (CaMK) blocker KN93. To understand the mechanism underlying this activity dependent increase in PV, we focused on the steps leading to Ser133 phosphorylation of the transcription factor CREB (pCREB). Regulation of this key signaling event remains relatively unexplored in inhibitory interneurons. We show that PV+ cells can employ CaMKs to trigger nuclear CREB phosphorylation. Nimodipine, KN93 and CaMKK inhibitor Sto609 prevent increases in pCREB after 30 seconds of K⁺-depolarization. Depolarization induced Ca²⁺ entry via CaV1 channels triggered increases in the nuclear CaM in a manner correlated with increases in pCREB. However, interneurons displayed little immunoreactivity for CaMKIV (CREB kinase in excitatory neurons). Our preliminary results suggest that CaMKI, a CaMK family member with a high degree of homology to CaMKIV, may translocate to the nucleus. CaMKI may serve as a CaM shuttle, or may itself phosphorylate CREB. Interestingly, evidence suggests the dysfunction of GABAergic transmission and of proteins involved in E-T coupling (CaV1, γCaMKII, βCaMKII, and Calcineurin) in numerous neuropsychiatric diseases. Specifically, disrupted expression of GAD67 and PV are hallmarks of schizophrenia. Understanding mechanisms of E-T coupling in PV+ cells will likely shed light on the cell biology underlying the experience regulated network plasticity important for learning, as well as the pathophysiology of neuropsychiatric disease.

RAPID MITOCHONDRIAL DYSFUNCTION MEDIATES TNF-ALPHA INDUCED NEUROTOXICITY. Danielle N. Doll, Stephanie L. Rellick, Taura L. Barr, Xuefang Ren, and James W. Simpkins. West Virginia University, Morgantown, West Virigina.

Tumor necrosis factor alpha (TNF-α) is known to exacerbate ischemic brain injury; however, the mechanism of action is unknown. Previous studies have evaluated the effects of TNF-α on neurons with long exposures to high doses of TNF-α, which is not pathophysiologically relevant. We characterized the rapid effects of TNF-α on basal respiration, ATP production, and maximal respiration using pathophysiologically relevant post-stroke concentrations of TNF-α. We observed a reduction in mitochondrial function as early as 1.5 hours after exposure to low doses of TNF-α, followed by a decrease in cell viability in HT-22 cells and primary cortical neurons. Subsequently, we used the HT-22 cell line to determine the mechanism by which TNF-α causes a rapid and profound reduction in mitochondrial function. Pre-treating with TNF-R1 antibody, but not TNF-R2 antibody, ameliorated the neurotoxic effects of TNF-α indicating TNF-α exerts its neurotoxic effects through TNF-R1. We observed an increase in caspase 8 activity and a decrease in mitochondrial membrane potential after exposure to TNF-α which resulted in a release of cytochrome c from the mitochondria into the cytosol. These novel findings indicate for the first time that an acute exposure to pathophysiologically relevant concentrations of TNF-α has neurotoxic effects mediated by a rapid impairment of mitochondrial function.
Poster Presentations
Cell Biology

Poster #1
CHARACTERIZATION OF HEPATOMA-DERIVED GROWTH FACTOR RELATED PROTEIN-3 (HRP-3) AS A PROLIFERATIVE ENDOTHELIAL LIGAND. Michelle E. LeBlanc 1,2, Weiwen Wang 1, Feiyu Guo 1, Chen Shen 1, Robert Chen 3, Feng Wang 3 and Wei Li 1. 1Bascom Palmer, Department of Ophthalmology, University of Miami School of Medicine, Miami, FL. 2Department of Pharmacology, University of Miami School of Medicine, Miami, FL. 3Dept. of Molecular & Human Genetics, Baylor College of Medicine, Houston, TX.

Endothelial cell (EC) dysfunction plays a critical role in the onset and progression of vascular disease. The mechanisms underlying endothelial dysfunction remain unclear, but may be partially explained through alterations in EC ligand binding activity. Systematic identification of novel EC ligands can present new targets for vascular disease therapy. Previously EC ligands have been identified on a case-by-case basis. To overcome this barrier, our lab utilizes a novel technology to identify novel EC ligands in vivo. Using this tool we identified hepatoma-derived growth factor related protein-3 (HRP-3). HRP-3 is a neurotrophic factor and homologous to HDGF, a well-established EC growth factor. We hypothesize that HRP-3 is a novel EC growth factor, capable of promoting EC growth and migration. To determine the function of HRP-3, human umbilical vein endothelial cells (HUVEC) and human aorta endothelial cells (HAEC) were incubated with HRP-3 for 24 and 48 hours. Results show HRP-3 significantly increases EC growth and proliferation at both 24 hours and 48 hours in a dose dependent manner. In a wound-healing assay HRP-3 significantly promotes the migration of HUVECs and HAECs into the denuded area. The angiogenic activity of HRP-3 was assessed using an in vitro endothelial spheroid sprouting assay. Results indicate HRP-3 significantly promotes HUVEC spheroid sprouts. Western blot analysis in HUVECs and HAECs showed a significant increase in phosphorylated ERK1/2 after 10 min, when treated with increasing concentration of HRP-3. In summary, our findings demonstrate that HRP-3 is a novel EC growth factor. The pro-growth effect of HRP-3 is at least partially mediated through activation of ERK1/2 signaling pathway. Our study suggests HRP-3 may be a potential therapeutic target for EC dysfunction in vascular disease.

Poster #2
INITIAL CHARACTERIZATION OF TETRASPANIN-2 IN VASCULAR SMOOTH MUSCLE CELLS. Emiley Tou, Angeline Richards, Wei Zhang, JinJing Zhao and Xiaochun Long, Center for Cardiovascular Sciences, Albany Medical College, Albany, NY.

Unlike other muscle cell types, vascular smooth muscle cells (VSMC) are not terminally differentiated and can switch between a contractile and synthetic phenotype depending on pathological and physiological cues. Upon vascular injury, VSMC undergo a transient phenotypic adaptation, switching from a non-proliferative, contractile phenotype to a synthetic phenotype. During vascular disease, this phenotypic adaptation is not transient and contributes to the formation of neointima, a major cause of vascular disease. This process of phenotype switching is regulated by a network of signal molecules, noncoding RNAs and transcription factors such as serum response factor (SRF) and a VSMC-enriched SRF cofactor Myocardin (MYOCD). The contractile phenotype has been shown to be characterized by high expression of a battery of genes with conserved CArG box(s) and the synthetic phenotype is defined by the downregulation of these contractile genes. Our RNA-seq of HCASM treated with TGFβ, a potent activator of VSMC differentiation, revealed Tetraspanin-2 (TSPAN2) to be drastically induced by TGFβ. TSPAN2 is a transmembrane protein that has been shown to have a role in cell development, proliferation, and motility. Further studies demonstrated that TSPAN2 is a VSMC-enriched gene and closely associated with VSMC contractile phenotype in several VSMC phenotypic modulation models such as aneurysm and rat balloon injury models. While MYOCD greatly induced TSPAN2 expression, knockdown of SRF by siRNA failed to decrease TSPAN2 expression; Moreover, luciferase assay showed that the TSPAN2 promoter is responsive to MYOCD rather than SRF, indicating a MYOCD-dependent and SRF-independent pathway for regulating TSPAN2 transcription. Our data also showed that knockdown of TSPAN2 in VSMC attenuated VSMC contractile gene expression, suggesting TSPAN2 is a potential positive regulator of VSMC contractile phenotype. Taken together, our data reveals that TSPAN2 is selectively expressed in SMC containing tissues, is induced by MYOCD in an SRF-independent pathway, and positively affects contractile gene expression. This initial characterization suggests that TSPAN2 contributes to VSMC contractile phenotype and may antagonize neointima formation in vascular disease.
**Poster #3**

**P2X7 RECEPTOR PATHWAY MAY CONTROL MELANOMA DIFFERENTIATION AND MODULATE THE SWITCH BETWEEN STEM CELL-LIKE CELLS AND PROLIFERATING/DIFFERENTIATED CELLS.** Jenna Bordelon, Margaret Sanchez, Samantha L. Schneider, Andrew L. Ross, James M. Grichnik

1 Department of Dermatology and Cutaneous Surgery, 2 Sylvester Comprehensive Cancer Center, 3 Interdisciplinary Stem Cell Institute, University of Miami Miller School of Medicine, Miami, Florida, USA

The processes that control the dynamic equilibrium between proliferating/differentiated tumor cells and more quiescent stem-like tumor cells have not been fully defined. There is increasing interest in the role that the P2X7 receptor plays in inflammatory and immune diseases, as well as in cancer. It has been shown that P2X7 expression is upregulated in melanoma. P2X7 is an ATP-regulated, plasma membrane channel that acts synergistically with PANX1 to regulate inflammasomal activation. P2X7 activation also has a number of downstream effects. Lastly, MITF is a transcription factor that promotes melanocyte differentiation and proliferation, and Fascin is an actin cross-linking protein that may be down-regulated in more advanced metastatic melanoma tumors. Brilliant Blue G (BBG) and Probenecid to inhibit the P2X7 and PANX1 receptors respectively, western blot (WB) and cell counting resulted in decreased MITF expression and decreased proliferation with treatment. Flow cytometry experiments and immunofluorescence microscopy after BBG/Probenecid treatment also showed a decrease in Ki67 and MITF expression, suggesting a transition to a less proliferative/differentiated state in vitro. WB also showed increased Fascin expression, which may indicate decreased melanoma progression. To determine the in vivo tendencies of receptor expression, we went to an RNA seq database of 289 tumors and showed that P2RX7 levels correlate directly with MITF and inversely with Fascin. These findings suggest that P2X7 regulated pathways may have the capacity to control the regulatory switch between the phenotypic states of quiescent and differentiating tumor cells. Despite targeted therapies, advanced melamomas often recur and remain lethal for the majority of patients. Stem-like cells in the melanoma tumor may be driving tumor resistance, and activation of their growth may be responsible for tumor recurrence. As such, P2X7 inactivation warrants further investigation as potential importance for the future of melanoma treatment.

**Poster #4**

**MALIGNANT MELANOMA: INCIDENCE INCLUDING SEX, BODY LOCATION, AND AGE AMONG PATIENTS SEEN IN A NATIONWIDE GROUP PRACTICE.** Renee Domozych, MD, James A. Solomon, MD PhD, Morgan Stines, University of Central Florida College of Medicine, Orlando, FL

Malignant melanoma (MM) is the most lethal form of skin cancer that carries an excellent prognosis if detected and treated early. Previous studies suggest that the incidence of MM is increasing, it is more common in men, and it primarily affects the trunk in men and lower extremities in women. This study analyzed the incidence of MM diagnoses from over 500,000 patients seen at Advanced Dermatology and Cosmetic Surgery (ADCS) from 2006 to 2013. All patients seen between 2006 and 2013 with ICD9 codes 172.0-172.9 were included. MM diagnoses data were extracted and categorized to determine the incidence each year per patient seen and the distribution among males and females, body location, and age. The overall rate of change of MM incidence from 2006 to 2013 was 135%. Significantly more males (n=4,492) were diagnosed with MM than females (n=3,033) ($\chi^2(1) = 865.1$, p < 0.000). The trunk and upper extremities were the two most common body locations in both sexes followed by face/ears in males and lower extremities in females. Ages were analyzed by creating five-year age groups, and the most common age group for both sexes was 65-69 years. Although significantly more males were diagnosed with MM overall, the majority of these cases were in men >45 years. More females were diagnosed for all ages <45 years. These results show that the incidence of MM among patients presenting to ACDS over the past eight years has increased with an age and body location distribution specific for each sex. In contrast to current literature, the two most common body locations in females were the trunk and upper extremities. Additionally, younger females and older males were disproportionately affected. These data can be used to target groups to increase awareness and primary prevention as well as to aid in sex-specific screening guidelines for MM.
DETERMINATION OF WHETHER THE INCREASE IN BIOPSY RATE AMONG DERMATOLOGISTS AS REPORTED BY THE US GOVERNMENT ACCOUNTING OFFICE (GAO) IS ASSOCIATED WITH AN INCREASING RATE OF BIOPSIES WITH NORMAL PATHOLOGY.  Julie Glener, James A. Solomon, MD, PhD; Robert Dellavalle, MD; April Armstrong, MD, MPH; Ameriderm Research, Ormond Beach, FL; University of Central Florida College of Medicine, Orlando, FL; Advanced Dermatology Cosmetic Surgery, Maitland, FL; University of Illinois College of Medicine, Urbana, IL; Department of Dermatology, University of Colorado School of Medicine, Anschutz Campus, Aurora, CO; Dermatology Service, Department of Veterans Affairs Medical Center, Denver, CO; Department of Epidemiology, Colorado School of Public Health, Aurora, CO.

The US GAO has suggested that dermatologists are performing increasing numbers of biopsies without clinical justification to increase clinical revenue. This study aims to discern whether there is an increased rate of biopsies performed by dermatologist providers (MD, DO, PA, RNP) that is associated with an increased rate of normal pathology. If the rate of normal biopsies decreased or remained constant, the increasing rate of biopsies will be deemed justifiable. Billing data from a large dermatology group practice identified 466,525 biopsies performed by dermatology providers from 2006 to 2013 wherein the ICD9 code, was 238.2 (neoplasm of uncertain biological behavior) and the CPT code is 11100 or 11101 (one or more skin biopsies). ICD9 codes were used to determine whether the biopsied lesion was normal or pathological. If any descriptive diagnosis under a given ICD9 code included a benign process, that ICD9 code was classified as normal. The data demonstrate that from 2006 to 2013 there was an increasing rate of 92.98% for the number of biopsies per dermatology provider. Furthermore, the rate of normal biopsies taken by each dermatological provider decreased from 27.5% to 18%. Thus, these data suggest that the increased rate of biopsies performed by dermatology providers is associated with increased disease diagnosis and thus justifiable.

ICAM-1 EXPRESSION IN TISSUE SPECIMENS FROM PATIENTS WITH ACTIVE VITILIGO
Eric Maranda, Janelle Vega, Mariya I. Miteva and Paolo Romanelli. Department of Dermatology & Cutaneous Surgery at the University of Miami Leonard M. Miller School of Medicine, Miami, FL 33101.

Vitiligo is an autoimmune phenomenon in which depigmentation occurs due to selective melanocyte destruction. While many theories exist, the precise pathophysiology underlying this disease is still unclear. Various studies have been done to characterize the cellular and molecular changes that occur in vitiligo. There is background literature suggesting that ICAM-1 (intercellular adhesion molecule-1), a member of the integrin family, may play an important role in vitiligo. ICAM-1 is involved in cell-cell interactions of leukocytes and plays an important role in the induction of immunologic and inflammatory reactions. Once melanocytes express an excess of adhesion molecules, there is an increased susceptibility of the melanocytes to immune cytotoxicity. Studies have found there is a significant increase of focal epidermal expression of ICAM-1 at the site of interaction between the immune infiltrates and the disappearing melanocytes in perilesional skin. It is currently unknown if ICAM-1 levels are in fact normal in non-lesional skin of vitiligo patients. We hypothesized that ICAM-1 is significantly increased in lesional as well as perilesional skin of patients with active vitiligo versus nonlesional skin from the same patient. To evaluate this, we used immunohistochemistry with antibodies targeting ICAM-1 and MART-1, a marker for differentiated melanocytes, followed by Hematoxylin and Eosin staining of perilesional, lesional, and non-lesional skin biopsies from patients with active vitiligo. We found that ICAM-1 expression was increased in lesional skin, specifically in the perivascular dermis, of patients with active vitiligo. We hypothesized that ICAM-1 is significantly increased in lesional as well as perilesional skin of patients with active vitiligo versus nonlesional skin from the same patient. To evaluate this, we used immunohistochemistry with antibodies targeting ICAM-1 and MART-1, a marker for differentiated melanocytes, followed by Hematoxylin and Eosin staining of perilesional, lesional, and non-lesional skin biopsies from patients with active vitiligo. We found that ICAM-1 expression was increased in lesional skin, specifically in the perivascular dermis, of patients with active vitiligo. Focal expression of ICAM-1 correlated with the focal loss of melanocytes in the leading edge and within the lesion. Additionally, ICAM-1 expression was increased in normal appearing skin of vitiligo patients compared to control, suggesting that subclinical inflammation may be occurring throughout the total skin surface of vitiligo patients. These findings suggest that ICAM-1 is implicated in the pathogenesis of vitiligo and may be useful in the development of future target-oriented therapeutics for the treatment of vitiligo.
RESULTS FROM A GLOBAL SURVEY OF PATIENT PERSPECTIVES AMONG FEMALES WHO DEVELOPED MELANOMA IN ASSOCIATION WITH INDOOR TANNING. Jennifer Nergard, Chauncey Caldwell, Morgan Stines, James A Solomon, MD PhD 1University of Central Florida, College of Medicine, Orlando, FL. 2Ameriderm Research d/b/a Advanced Dermatology and Plastic Surgery, Ormond Beach, FL.

A new U.S. FDA regulation categorizes tanning beds as category II, and similar global regulatory action require informing users of the “risk of skin cancer” as methods to reverse the growing trend of indoor tanning. However, little is known from the patient’s perspective on whether or not knowledge of risk of cancer is a deterrent to indoor tanning. A worldwide survey was launched questioning those who are at least 18 years old and have been diagnosed with melanoma after indoor tanning. The survey was made available to university and hospital dermatology departments, private dermatology practices, patient advocacy groups, and social media. A total of 178 females from nine countries responded to the survey. Among female responders, a positive relationship was identified between the frequency of indoor tanning and the perceived safety of indoor tanning (r = 0.247, p < 0.05). The warnings about skin cancer showed no influence on the frequency of indoor tanning. A significant relationship exists between the age of onset of indoor tanning and the age at melanoma diagnosis (r = 0.591, p < 0.01). Furthermore, there is a positive relationship between the age of onset of indoor tanning and the Breslow level (r = 0.211, p < 0.01). Those who more frequently tan indoors perceive this method as a safer alternative to outdoor tanning. Knowledge of the risk of skin cancer with tanning results in no decline in the frequency of indoor tanning. Neither the frequency of indoor tanning nor the age when the patient started indoor tanning has an effect on the time frame in which melanoma is diagnosed. However, those who started indoor tanning at a later age correlated with a diagnosis of higher Breslow levels.

INCREASED LOCAL CORTISOL SYNTHESIS AND ACTIVATION OF GLUCOCORTICOID RECEPTOR CONTRIBUTES TO PATHOGENESIS OF VENOUS LEG ULCERS. Ashley Rosa, Elizabeth Lebrun, Ivan Jozic, Olivera Stojudinovic and Marjana Tomic-Canic, Wound Healing and Regenerative Medicine Research Program, Department of Dermatology and Cutaneous Surgery, University of Miami Miller School of Medicine, Miami FL.

Venous leg ulcers (VLUs) represent a major burden for patients and healthcare professionals due to lack of knowledge regarding molecular mechanisms that contribute to inhibition of healing resulting in scarce treatments. Glucocorticoids (GCs) are potent inhibitors of wound healing. They mediate their function by binding to glucocorticoid receptor (GR). However, the role of GCs in pathogenesis of VLUs has not been studied. We have previously shown that epidermis serves as extra-adrenal site of cortisol synthesis and expresses steroidogenic enzymes including 11β-hydroxylase (CYP11B1) and 11-β-hydroxysteroid dehydrogenases (11βHSD1/2). We have also shown that cortisol synthesis is activated by tissue injury and can be induced by pro-inflammatory signals. Therefore, we postulated that activation of GCs synthesis contributes to inhibition of healing in VLUs. To test our hypothesis we obtained edge biopsies from patients VLUs, and measured cortisol levels by ELISA, and CYP11B1, 11βHSD1/2 and presence/activation of the GR by qPCR, immunohistochemistry and western blot. Skin biopsies from patients undergoing routine surgery served as controls. We found increased cortisol levels in VLUs as compared to normal skin controls suggesting increased local tissue synthesis. Furthermore, we found increased expression of CYP11B1, an enzyme responsible for the execution of the last step of cortisol synthesis, supporting the notion of increased synthesis of cortisol. As a result, we also found increased nuclear localization of phosphorylated form of GR in the non-healing edges of the VLUs, indicated hormone-activated GR. Interestingly, we found down-regulation of both 11-β-hydroxysteroid dehydrogenase enzymes that catalyze the inter-conversion of inactive cortisone to cortisol and vice versa. Decreased expression of 11βHSD enzymes in VLUs suggests loss of important feedback mechanisms that serve to regulate tissue cortisol levels. In conclusion, we demonstrate that skin derived from VLUs exhibits increased synthesis of cortisol, increased activation of GR and loss of feedback mechanisms, all of which contributes to impaired healing and pathogenesis of VLUs. We conclude that modulation of cortisol synthesis may represent an important regulatory mechanism during wound healing and that its deregulation may play a role in VLU patho-mechanism. Future studies will assess if targeting of cortisol synthesis pathway can serve as a potential new therapeutic avenue for the treatment of wound healing disorders.
AN UPDATE ON PHOTODYNAMIC THERAPIES IN THE TREATMENT OF ONYCHOMYCOSIS. Brian J. Simmons, BS, Leyre A. Falto-Aizpurua, MD, Robert D. Griffith, MD, Keyvan Nouri MD. University of Miami Miller School of Medicine Department of Dermatology and Cutaneous Surgery 1475 NW 12th Ave. Suite 2175, Miami, FL.

Onychomycosis is a common fungal infection of the nails that is increasing in prevalence in the old, diabetics and immunocompromised. Onychomycosis presents a therapeutic challenge that can lead to significant reductions in quality of life leading to both physical and psychological consequences. Current treatment modalities are difficult to implement due to the poor penetration of topical treatments to the nail bed, the slow growing nature of nails and the need for prolonged use of topical and/or oral medications. Standard of care medications have cure rates of 63 to 76% that leads to a high propensity of treatment failures and recurrences. Photodynamic therapy (PDT) offers an alternative treatment for onychomycosis. Methylene blue dye, methyl-aminolevulinate (MAL) and aminolevulinic acid (ALA) have been used as photosensitizers with approximately 630nm light. These modalities are combined with pretreatment of urea and/or microabrasion for better penetration. PDT treatments are well tolerated with only mild transient pain, burning and erythema. In addition, significant cure rates for patients who have contraindications to oral medications or failed standard medications can be treated. With further enhancements in photosensitizer permeability, decreased pretreatment and photosensitizer incubation times, PDT can be a more efficient and cost effective in office based treatment for onychomycosis.

THE EFFECT OF PERSONAL SKIN CANCER EXPERIENCE ON SUN PROTECTIVE BEHAVIORS, Rachel Wheatley, BS, Jerri Johnson, MD. University of Central Florida College of Medicine, Orlando, FL.

Skin cancer is one of the leading causes of cancer related deaths in the United States, despite the fact that it is a preventable disease through the use of sun protective behaviors, such as wearing sunscreen. Studies have shown that persons who have skin cancer (directly affected) are not more likely to protect themselves or their family members from the sun than the average population. That is, persons with skin cancer do not practice in sun protective behaviors. Lacking in the literature is an understanding about the sun protective behaviors of people that are indirectly affected by cancer (i.e., know “someone” with cancer). This study aims to gain an understanding of people that are indirectly affected by skin cancer. Four-hundred and fifty-two participants (70% female, Mean age = 20, SD = 5.0) responded to a 31-item questionnaire about sun protective behaviors, sun sensitivity factors, and skin cancer history. Participants that reported an indirect experience with cancer, responded to the Unidimensional Relationship Closeness scale (12-items; Dibble, JL 2012). An Independent sample t-test comparing differences in sun protective behaviors among those that know someone with skin cancer (n = 122) or had a personal experience with cancer (n= 2) with no personal cancer experience (n= 314) revealed no statistical differences in sun protective behaviors (Ms = 14.14, 13.56 and SDs = 5.87, 5.93, respectively; t (434) = .922, p = .357). Further analysis revealed that when controlling for closeness of the relationship, the results do not change. To the best of our knowledge, we are first to investigate the sun protective behaviors of persons that are indirectly affected by cancer. Concurrent with previous findings about people with cancer, results here demonstrate that knowing someone who has had skin cancer does not encourage sun protective behaviors. Results here suggest that more efforts are needed to educate young adults about the protective factors of skin cancer.
Epidemiology

THE EFFECT OF LANGUAGE DISCORDANCE ON THE SOURCE OF PRIMARY CARE IN NORTH MIAMI DADE. Jennifer Chen, Michelle M Abou-Jaoude, Grettel Castro, and Marcia Varella, MD, PhD, MHS. Department of Medical and Health Sciences Research, Herbert Wertheim College of Medicine at Florida International University, Miami, FL.

Emergency department (ED) overutilization is inefficient and hampers continuity of care. Language discordance between a patient and health care provider, defined as when the two do not speak the same language, has detrimental effects on selected health outcomes. Little is known about the effect of discordance on the usual source of primary care. We aim to test the association between language discordance and ED utilization patterns. We performed secondary analysis of cross-sectional data from households who were randomly selected to participate in the NeighborhoodHelp Benchmark Survey, in 2009-2010. Households were excluded if their primary language was English or if data was missing on language spoken. Language discordance was defined based on the question “Thinking about your most recent care, how often have you had a hard time speaking with or understanding a doctor, nurse or other health care provider because you and the doctor spoke different languages?” and was considered present if households responded any answer other than “never.” Usual source of care was defined as either ED or outpatient primary care. The independent association between discordance was tested using multivariate logistic regression and significance was considered for p-values ≤0.05. SPSS was used for analyses. A total of 535 households were included. About 14% of households reported language discordance. Before adjustment, discordance was significantly associated with ED usage [Odds Ratio (OR) =2.2, 95% Confidence Interval (CI) = 1.1-4.6, p=0.032]. After adjusting for language spoken, household income, employment status, household health, and level of education, the association between discordance and use of ED was stronger (adjusted OR=3.4, 95% CI=1.2-9.5, p=0.023). Language discordance is strongly associated with ED utilization in the North Miami Dade population. Further study on interventions to improve communication such as translation services in outpatient facilities is warranted to reduce ED overutilization in favor of outpatient primary care.

HPV INFECTION AND CIRCUMCISION STATUS IN MEN: NHANES 2009-2012. Erin Dunn BA1,2, Kevin Moore BA1,2, Tulay Koru-Sengul PhD2,3, 1Medical Education, 2Department of Public Health Sciences, 3Sylvester Comprehensive Cancer Center University of Miami Miller School of Medicine, Miami, FL.

In men, persistent Human Papillomaviruses (HPVs) can lead to penile, anal, and oropharyngeal cancers. Studies show circumcision to be a protective factor against genital HPV infection. A 2010 National Center for Health Statistics report states that US circumcision rates have declined to 58.3%. National population-based surveys provide estimates of population-specific prevalence, trend, and determinants to identify the burden of HPV in the male oropharynx and a possible association with circumcision status. We looked at circumcision status and HPV infection prevalence by HPV oral testing in the US from the 2009-2012 National Health and Nutrition Examination Survey (NHANES) to obtain a representative sample of the US non-institutionalized civilian population. We provided epidemiology of HPV infection for men as well as women for comparison. Analysis was performed by SAS v9.3 by taking into account the complex sampling design. Among oral-HPV positive persons, men had greater HPV prevalence (74.4%; 95%CI=68.6, 80.2) than women (25.6%; 19.768, 31.389). Among men, Whites had greatest overall (62.5%; 54.1, 70.9) and high-risk (66.6%; 59.2, 74.0) oral-HPV burden; whereas, “other race” had the lowest burden of overall (5.3%; 2.8, 7.7) and high-risk (4.7%; 2.1, 7.4) infection. Of oral-HPV infected men, 82.7% (76.4, 89.0) were circumcised. The circumcised subgroup had a greater prevalence of HPV infection for 18 of 37 types tested, 7 of which were high-risk, when compared to prevalence of those types in the general male population. Despite uncircumcised men having higher prevalence for 11 of the high-risk HPV types than the general population, circumcised men held a greater prevalence (95%CI=75.8, 90.3) of the total high-risk HPV burden (uncircumcised men: 16.9%; 9.7, 24.2). Using a large population-based survey, our results show increased prevalence of oral-HPV infection in men compared to women and increased high-risk infection in circumcised men compared to uncircumcised men. Revealing circumcision status and oral-HPV prevalence could lead to better appreciation of demographic disparities and sexual health determinants of men infected with HPV. Perhaps sexual or cultural practices associated with circumcision could elucidate this discrepancy in HPV transmission to the oropharynx. Thus, this study may assist in creating culturally competent, gender-targeted oropharyngeal HPV screening and prevention programs.
HIV PREVENTION INTERVENTION FOR SUBSTANCE USERS: A REVIEW OF THE LITERATURE.
Adel Elkbuli MD, MPH (C)1, Antonio Bustillo BSc1, Tulay Koru-Sengul PhD1,2 1Department of Public Health Sciences, 2Sylvester Comprehensive Cancer Center, University of Miami Miller School of Medicine, Miami, Florida, USA.

Substance use, including intravenous (IV) and non-IV drug use, is associated with higher risk for contracting HIV through unprotected sex or syringe-based exposure. Approximately 81% HIV-positive individuals report illicit drug use, 16.6% reporting via injection. We reviewed primary HIV interventions targeted to IV drug users (IDUs) and non-IV drug users (NIDUs). Our aim was to identify which type of prevention intervention was the most effective in reducing HIV transmission risk among IDUs and NIDUs. We conducted a PubMed literature review (1998-2013), limiting studies to HIV primary prevention interventions targeting adult HIV-negative substance. Out of 44 articles, we included 14 targeted IDUs (n=5) and NIDUs (n=9). Interventions were compared descriptively across sample sizes, sociodemographic, intervention setting, study design, use of theoretical models, and intervention effects. IDU studies had a smaller sample size than NIDU (range: 226-3,742 vs. 16-1,686). Compared to NIDU studies, IDU had less ethnic minority sample (78% vs. 60%). No IDU study targeted men who have sex with men, compared with 33% NIDU studies. Both IDU and NIDU studies were conducted in substance abuse treatment centers and included both group- (80% vs. 78%) and individual-based (20% vs. 11%) methods; only 1 (11%) IDU study was couple-based interventions. Quasi-experimental design was used more in NIDU (56%) than IDU (20%) studies while experimental design was used more in IDU (80%) than NIDU (44%). All IDU and 89% NIDU studies used explanatory and behavior-change theoretical models to guide selection of intervention components. Effectively reducing frequency of risky sexual behaviors was seen in 78% NIDU and 20% IDU studies. HIV testing and counseling were conducted 28% of all studies where 75% IDU studies effectively increased drug use-related hygiene and 50% examined decreased frequency of injections. However, out of 14 studies, 4 (28%) did start-of-study HIV testing and 3 (21%) examined HIV seroconversion. Overall, the interventions reviewed demonstrate promising results for decreasing risky sexual practices for NIDUs and reducing drug practices for IDUs, thereby reducing HIV transmission risk. Future studies should include HIV testing and measure HIV seroconversion to fully elucidate intervention effects.

TIME TRENDS OF WEIGHT MANAGEMENT TACTICS AND FACTORS ASSOCIATED WITH TACTICS AMONG US ADULTS: NATIONAL HEALTH AND NUTRITION EXAMINATION SURVEY (NHANES) 1999-2012 Ji-Young Lee BSc1, Junghye Sung PhD2, Tulay Koru-Sengul PhD1,3 1Department of Public Health Sciences at University of Miami Miller School of Medicine, 2Department of Biostatistics and Epidemiology at Jackson State University, 3Sylvester Comprehensive Cancer Center at University of Miami Miller School of Medicine.

Obese individuals are encouraged to lose their body weight due to its adverse effect on chronic disease, but the efficacy of the strategies for weight management remains controversial. This study aims to determine trends of weight management strategies (losing vs. maintaining weight) and to identify factors associated with preferred tactics. The analyses utilized the data from the nationally representative NHANES 1999-2012 which includes a valid sample of 33,826 adults. Weight management tactics included weight loss (tried to lose weight during the past year) and weight maintenance (tried not to gain). The analyses were performed by using Survey procedures to take into account the complex sampling design by incorporating the stratification, clustering, and sample weights. The prevalence of weight loss(33.5% in 1999 and 36.7% in 2011; p=0.0117) and weight maintenance(10.4% in 1999 and 12.9% in 2011; p=0.0559) significantly increased. A greater increased rate was found among African-Americans than non-African-Americans(p=0.065 for weight loss; p=0.0058 for weight maintenance), but not among subgroups of age and gender. A higher prevalence of weight loss was found among those aged 40-60 years, Whites, females, highly-educated, obese, non-smokers, diabetes patients, non-CVD history, metabolic syndrome, non-CKD, and insurance-covered(all p<0.05). A higher frequency of weight maintenance was reported among those 40 years and older, Whites, males, highly-educated, normal/over-weighted, non-smokers, non-diabetes, non-CVD history, and insurance-covered(all p<0.05). Our study found that US adults increasingly tried to manage their weight and preferred weight management tactics differed by socio-demographic characteristics and disease status. The findings may be useful for health planners and health promotion activities to prepare weight management tactics for at-risk populations by taking into account specific socio-demographic subgroups. Further study is needed to determine the characteristics of goal-achievers for weight management.

The composition of the human gut microbiome is determined in part by diet and has an impact on human health and metabolism. Controlled studies show that both dietary fiber intake and obesity are associated with changes in fecal microbial diversity. However, it is unknown how diet and obesity interact to influence the composition of the fecal microbiome. Therefore, we performed a cross-sectional study of ambulatory adults to assess associations between diet, obesity, and fecal microbial composition. We recruited 12 healthy adult volunteers with no exposure to antibiotics for at least one year. Diet was assessed during a single visit using the National Health Interview Survey’s Multifactor Screener. Stool samples were collected from each subject and analyzed by sequencing the V4 hypervariable region of the bacterial 16S rRNA gene. We measured microbial diversity within each sample using the Shannon index and also assessed relative abundance of specific phyla. We compared the microbiome of obese vs. non-obese subjects and high vs. low fiber diets, and we assessed for potential interaction between these two exposures. Dietary fiber intake was not significantly associated with fecal microbial diversity, but high fiber intake was associated with high relative abundance of Actinobacteria (P=0.04) and low relative abundance of Verrucomicrobia (P<0.01). Obesity was associated with high relative abundance of Firmicutes (P=0.02) and low relative abundance of Bacteroidetes (P=0.02). Dietary fiber intake significantly modified the relationship between obesity (classified by WHR) and the relative abundance of Firmicutes, with higher relative abundance of Firmicutes in subjects with low fiber intake (P=0.05). Dietary fiber intake did not significantly modify the relationship between obesity and relative abundance of Bacteroidetes (P=0.07) or the relationship between obesity and Shannon diversity (P=0.33). These findings suggest an interaction between fiber intake and obesity on phylum-level fecal microbial composition.

COMPUTER GAMES FOR OBESITY AND DIABETES PREVENTION: INTERFACING BEHAVIOR CHANGE TECHNIQUES TO GAME ELEMENTS. Brian Mayrsohn, MS1, William Butler1, Christopher Gates1, Georges Khalil MPH2, David Metcalf PhD1. 1University of Central Florida, Orlando FL. 2MD Anderson Houston, TX.

With the increasing burden of chronic diseases such as obesity and diabetes, there is a strong need for a low cost solution that can be scaled for the masses. Games for health is touted as that solution. A major benefit is that they can improve the delivery of care across the healthcare continuum while also increasing reach and patients buy in. Successful health games are based on theoretical frameworks, which require the successful implementation of game elements. However, terminology used by researchers is either asynchronous or totally lacking making it difficult for others to refer to a consistent approach. The purpose of this study is to bridge the gap between game designers and behavioral scientists to enable them to create games based on behavior change game-elements (BCGEE) that are optimally designed not only to be fun and engaging but also transformative. To merge these two fields a tool was developed to create a common language that could assist researchers and game designers in the identification of BCGEE necessary to create more efficacious games that can create behavioral changes. The tool was constructed using research obtained during a systematic review of the literature, the expert guidance of one game designer and three behavioral scientists that develop games for change. The tool was then tested through one-on-one interviews with game designers and behavioral scientists to validate. A mixed method research approach was taken by utilizing thematic analysis and likert scales to objectively and subjectively evaluate the transcripts obtained from the interviews as they contrast against the tool. The analysis of the transcripts led to redefining the game element definitions and links between the two fields. By including all stakeholders, we developed a common language that both fields can utilize to develop games for change.
BURDEN OF HUMAN PAPILLOMAVIRUS (HPV) ASSOCIATED CANCERS IN FLORIDA FROM 1981-2009. Feng Miao MSc1,2, Erin Dunn BA1,2, Kevin J. Moore BA1,2, Tulay Korus-Sengul PhD1,3 1Department of Public Health Sciences, 2Medical Education, 3Sylvester Comprehensive Cancer Center, University of Miami Miller School of Medicine, Miami, FL.

Human papillomavirus (HPV) has been associated with several types of cancer: cervical, vulvar, vaginal, penile, anal, oropharyngeal and bladder. We report the trend of HPV-associated cancer incidence from 1981 to 2009 in the Florida adult population by analyzing data from Florida Cancer Data System (FCDS). For adult patients (≥18 yrs) with multiple HPV-associated cancers, the earliest reported cancer was set as the primary cancer site. From 1981 to 2009, 109,165 men and 99,923 women residing in Florida were diagnosed with HPV-associated cancer. Incidence per 100,000 people for each gender was age-adjusted by using the 2000 US standard population. HPV-associated cancers in Floridian men included bladder (82.5%), tongue (9.1%), tonsil (5.4%) and anal (2.9%), while cancers of the cervix (45.5%), bladder (29.3%), vulva (11.3%), anus (4.9%), tongue (4.5%), vagina (2.6%) and tonsil (2.1%) were reported in Floridian women. There were no significant changes in incidence for cancers of the tongue, tonsil and vagina for women from 1981 to 2009. In contrast, the incidence of female vulvar and anal cancers and male tongue, tonsil, anal cancers increased significantly from 1981 to 2009. In 2000, bladder cancer incidence began to decline significantly for men and women: incidence in 2009 was only 2/3 of the rate in 2000. For women, the incidence of cervical cancer remained at 35-40 per 100,000 until 1989, and then hovered around 45-50 per 100,000 from 1990 to 1995. However, by 2009 the incidence had decreased to 12 per 100,000. HPV screening may be associated with the increase in HPV-associated cancer incidence from 1990 to 1995, while HPV prevention efforts may be associated with the overall decline in incidence beginning in 1996. Elucidating patterns of cancer incidence over time can lead to implementation of gender-targeted medical and public health interventions as well as assessment of past HPV-associated cancer screening and prevention efforts.

DECREASED ANEMIA PREVALENCE AMONG WOMEN AND CHILDREN IN BAJA CALIFORNIA, MEXICO: A SIX YEAR COMPARATIVE STUDY. Molly Moor, Stephanie Brodine, Richard Garfein, Miguel Fraga, Hooman Rashidi, John Elder, Department of Family and Preventive Medicine, Joint Doctoral Program in Public Health, University of California, San Diego & San Diego State University, San Diego, CA.

Limited information exists about the health of Mexican indigenous laborers and their families. This study sought to determine the prevalence and correlates of anemia among indigenous women and children by conducting a series of cross-sectional studies in 2004/2005(Wave 1) and in 2011/2012(Wave 2) among women(15-49 years) and their children(6-59 months) in a rural community in Baja California, Mexico. Participants were randomly recruited from a temporary medical clinic and by random household sampling. Participants included 201 women and 99 children in Wave 1, and Wave 2 comprised 146 women and 77 children. Demographic, health, and dietary data were collected via in-person interviews. Anemia testing was performed and blood smears were obtained from anemic participants. Individuals diagnosed with anemia received vitamins and nutritional counseling. Furthermore, in the years between Waves 1&2, biannual free health clinics and various public health interventions were offered to community members. Anemia prevalence among women decreased significantly from 42.3% in Wave 1 to 23.3% in Wave 2(p<0.001). Anemia prevalence in children age 24-59 months decreased from 46.5% to 30.2%(p=0.066); anemia prevalence among children 6-23 months decreased from 71.4% to 45.8%(p=0.061). Among women in Waves 1&2, the consumption of iron promoting foods within 48 hours prior to testing was protective against being anemic (p=0.018). Furthermore, among a subset of women tested in Wave 2, those who ate four or more servings of vegetables per week were significantly less likely to be anemic than women who consumed fewer than four servings of vegetables per week (p=0.034). Having diarrhea in the past two weeks was significantly associated with being anemic among children 25-59 months tested in Waves 1&2(p=0.007). Microscopic examination of blood smears from anemic individuals revealed microcytic, hypochromic red blood cells in 90% of anemic children and 68.8% of anemic women, which suggests iron deficiency anemia.
Female Breast Cancer Survival is Associated with Primary-Payer Status: A Florida Population-Based Study (1996-2007). Kevin J. Moore BA, Erin Dunn BA, Feng Miao MSc, Stacey L. Tannenbaum PhD, Margaret M. Byrne PhD, Tulay Koru-Sengul PhD, 1Medical Education, 2Department of Public Health Sciences, 3Sylvester Comprehensive Cancer Center, University of Miami Miller School of Medicine, Miami, FL.

Survival disparities for female breast cancer may be affected by primary payer status at diagnosis. We evaluated the association of having Medicaid at diagnosis with breast cancer survival among patients diagnosed in Florida. Data from the Florida Cancer Data System (1996-2007) was linked with the Agency for Health Care Administration and US census (n=120,940) to explore median survival time (MST) and 1-, 3-, and 5-year survival rates. Survival was compared by primary-payer at diagnosis (private insurance [referent], Medicare, Medicaid, Indian Health Services, Defense/Military/Veteran, uninsured, and insurance not otherwise specified). Cox proportional hazards regression models were used to obtain unadjusted and adjusted hazard ratios (HR) and 95% confidence intervals (95%CI). Among patients with Medicaid (n=3,888; 3.2%), most were White (72.0%) and non-Hispanic (73.8%). Compared to all other payers at diagnosis, having Medicaid imparted the smallest MST (7.6 months) and lowest survival rates at one (91.0%), three (73.4%) and five (60.5%) years. In unadjusted analysis, compared to those with private insurance, patients with Medicaid had nearly 2.5-fold higher risk (95%CI=2.30-2.60). In the fully adjusted model controlling for socio-demographics, tumor characteristics, treatment factors, and 31 comorbidities, survival remained significantly worse for patients with Medicaid ([1.66]; 1.54-1.79). Patients receiving Medicaid had the worst survival compared with private insurance. Worse survival in those receiving Medicaid may be due to diminished access to care; Medicaid may be a surrogate marker for chronic poverty, which is linked to treatment non-compliance. Further research is necessary to reduce the disparities among breast cancer patients with Medicaid.

Association Between Parental Country of Birth and Patterns of ED Utilization in Children in the United States. Christian Nagel, Kenneth Maskell, Brian Salzverg, Juan Acuna, Grettel Castro, Marcia Varella. Department of Medical and Health Sciences Research, Herbert Wertheim College of Medicine, FIU, Miami, FL, 33199.

Emergency department (ED) utilization for conditions manageable in primary care settings or as a primary source for health care is not only inefficient but hampers continuity of care. Inappropriate ED utilization relates to diverse socio-economic factors. In the USA, little is known about the association between immigration and ED utilization. The present study evaluates whether there is an association between parental immigration and the ED utilization for health-related care, and to test whether the race/ethnicity modifies that association. We used data from participants of the 2011/2012 National Survey of Children’s Health, which included randomly selected households with at least one resident child aged 0-17 years. Outcome was the self-reported choice of usual place of health care (ED versus any other place). Exposure was immigrant status defined as a child for which both parents were born outside the US. Independent association was tested using logistic regression. Lastly, regressions stratified by ethnicity were performed. Stata 12 was used for analysis. Children from immigrant families were more likely to be of Hispanic origin, having non-English as primary family language, dwelling in household with larger numbers of children, having less educated mothers, lower income, and were less likely to be insured. After adjusting for these characteristics, the immigration status was not associated with ED utilization [adjusted Odds Ratio (aOR)= 0.57, 95% Confidence Interval (CI)= 0.29 – 1.09]. However, when stratified by ethnicity, white immigrants (compared to white non-immigrants) were more likely to use the ED as their usual place of care (aOR= 3.4, 95% CI 1.41 – 8.2) and in the groups with ethnicity reported as other, immigrants were 4 times less likely to use the ED (aOR= 0.25, 95% CI 0.09 – 0.74). The groups with ethnicity self-reported as Hispanics or Blacks did not show significant associations between immigration status and ED utilization (OR=0.51, 95% CI 0.19-1.40 and OR=0.49, 95% CI=0.17-1.35, respectively). We found evidence for disparities in health care utilization according to immigration status in selected ethnic groups. These results support the need of tailored interventions that could help immigrants from selected ethnicities to improve their patterns of healthcare utilization.
EVALUATING THE EFFECTIVENESS OF A FRONT WINDSHIELD STICKER REMINDER IN REDUCING TEXTING WHILE DRIVING IN YOUNG ADULTS. Austin Rohl, BS1, David Metcalf, PhD2, 1University of Central Florida, Orlando, FL. 2University of Central Florida, Orlando, FL.

It is clear that cell phone use, and more specifically texting, while driving is a dangerous activity that is on the rise in the U.S. States. From 2011 to 2012 alone, there was a 9% increase in the number of people injured in a motor vehicle crash involving a distracted driver. Bans have already taken place in 46 states in the U.S., but studies suggest that these bans have a negligible effect, not reducing nor increasing crashes linked to texting while driving. What can be done about this? One simple method of reducing texting while driving that has not been specifically looked into is sticker reminders. Sticker reminders have already been proven to be an effective intervention in the realm of driver safety; one study found that a “Buckle-Up” dashboard sticker doubled the use of safety belts by front seat passengers. The purpose of this study was to initiate a simple sticker reminder intervention in young drivers and measure to see whether or not it is effective at reducing rates of texting while driving over a three week period. 104 medical students aged 18-29 from the UCF College of Medicine were recruited for participation. Participants were randomly divided into two groups, the treatment group receiving an interventional “Drive in the Moment” windshield sticker to be placed in a visible location to the driver, and a control group not receiving the sticker. Both groups took a pre and post survey that recorded self-reported texting and driving frequency. Participant’s mindfulness was also measured pre and post intervention using the CAMS-R mindfulness scale, as we believe the sticker may be modulating an effect through enhancing mindfulness. If this sticker intervention is effective, it could empower physicians, law makers, car manufacturers, insurance companies, and proactive drivers alike to take action and do something simple to reduce texting while driving, a dangerous epidemic.

DIABETES AND PREHOSPITAL DELAY TIME OF PATIENTS WITH ACUTE MYOCARDIAL INFARCTION IN PUERTO RICO. Stanley Tiu, Guillermo Ortega, Grettel Castro, Juan Zevallos, Marcia Varella, Department of Medical and Health Sciences Research, Herbert Wertheim College of Medicine, Florida International University.

Prehospital delay time -PHDT- (time from symptoms onset to hospital presentation) is potentially modifiable major determinant of acute myocardial infarction (AMI) prognosis. A factor shown in other populations to be associated with increased prehospital delay time of AMI is diabetes Mellitus (DM). Diabetes is highly prevalent among Puerto Ricans. Whether DM in Puerto Rico is also associated with increased PHDT is not known. To determine whether diabetes is associated with increased PHDT among patients with acute myocardial infarction in Puerto Rico. We performed secondary analysis of data from the cross-sectional Puerto Rico Cardiovascular Disease Surveillance (PRCDS). The PRCDS was based on abstraction of data from medical records of patients who were hospitalized in 23 acute care facilities in Puerto Rico with an incident myocardial infarction in 2007, 2009 and 2011.Variables: The main independent variable was presence of DM as recorded at admission to hospital. The outcome was PHDT categorized as short (≤4 hours) and long (> 4 hours). Potential confounders measured were gender, age, smoking, hypertension, angina, and mode of transportation. Analysis: Independent associations were assessed using multivariate logistic regression using SPSS version 20. Significance was considered for alpha ≤ 0.05. After adjustment for age, gender, smoking history, hypertension, angina, and mode of transportation, DM was not significantly associated with an increased frequency of long PHDT: OR=1.16, 95% CI=0.97-1.40. Only age was independently associated with PHDT. As compared with subjects younger than 55 years-old, the OR for having a long PHDT was 1.78 (95% CI=1.32-2.42) for those between 75 and 84 years old and 2.36 (95% CI=1.66-3.37) for those others over 84 years. Diabetes was not significantly associated with increased PHDT in the Puerto Rican population. Further research is needed to determine whether lack of significance of DM in the Puerto Rican population could be due to socioeconomic differences.
Genetics

Poster #23
AN AMINO ACID DELETION IN SZT2 IN A FAMILY WITH NON-SYNDROMIC INTELLECTUAL DISABILITY.  Michelle Falcone\textsuperscript{1}, Kemal O. Yariz\textsuperscript{1}, David B. Ross\textsuperscript{2}, Joseph Foster II\textsuperscript{1}, Ibis Menendez\textsuperscript{1}, Mustafa Tekin\textsuperscript{1}.  \textsuperscript{1}Dr. John T. Macdonald Department of Human Genetics and John P. Hussman Institute for Human Genomics, Miller School of Medicine, University of Miami, Miami, Florida, \textsuperscript{2}Comprehensive Neurobehavioral Institute, Plantation, Florida.

Autosomal recessive intellectual disability (ID) is characterized by extensive genetic heterogeneity. Recently, three mutations in \textit{SZT2} were reported in two unrelated children with unexplained infantile epileptic encephalopathy with severe ID. Here we report a European American family with three children having non-syndromic mild or moderate ID without seizures. Whole-exome sequencing of three affected siblings revealed a three base pair deletion (c.4202_4204delTTC) located in a 19 mb autozygous region on chromosome 1, leading to an amino acid deletion (p.Phe1401del) in \textit{SZT2}. All three children were homozygous for the deletion and their parents were heterozygous as expected in autosomal recessive inheritance. \textit{SZT2} is highly expressed in neuronal tissues and regulates seizure threshold and neuronal excitation in mice. We conclude that the disruption of \textit{SZT2} with some residual function might lead to mild or moderate ID without seizures.

Poster #24
REMODELING OF THE HEART IN HYPERTROPHY IN ANIMAL MODELS WITH MYOSIN ESSENTIAL LIGHT CHAIN MUTATIONS.  Hanyao Foong, Kazmierczak K, Yuan CC, Liang J, Huang W, Rojas AI, Szczesna-Cordary Department of Molecular and Cellular Pharmacology, University of Miami Miller School of Medicine Miami, FL, USA.

Hypertrophic remodeling is a highly debilitating phenomenon; however, the molecular mechanisms and signaling pathways underlying these processes are not yet fully understood. Recognition of functional, structural, metabolic, and molecular differences between pathological and non-pathological hypertrophy may potentially be helpful for therapeutic advancement. Pathological hypertrophy occurs in response to genetic mutations in sarcomere proteins including the myosin essential light chain (ELC). ELC is proven important for myosin contractile function in heart muscles. Our study aims to analyze heart performance via echocardiography in different phenotypes of cardiac hypertrophy of transgenic (Tg) mice carrying mutations in myosin ELC using two animal models of HCM: Tg-A57G and Tg-Δ43 mice. The A57G (alanine to glycine) mutation has been associated with pathologic HCM in humans (clinical phenotype of asymmetric septal hypertrophy and sudden cardiac death). In the Tg-Δ43 mouse model, the endogenous ventricular ELC is partially replaced with the 43-amino-acid N-terminal truncated human ventricular ELC protein. and data obtained indicated that when the mice grew older, the hearts of Tg-Δ43 mice hypertrophied, but the ventricles did not show any pathologic phenotype. Aged matched mice (4-5 months old) were separated into two groups: 1) sedentary and 2) subjected to strenuous exercise by swimming. Based on previous studies, we hypothesized that the A57G mutation will induce hypertrophic cardiomyopathy and/or heart failure, while the Δ43–dependent pathway hinted toward a non-pathological cardiac phenotype. Echocardiography was performed for both groups with the heart rates used as an indicator of anesthetic drug exposure. We compared parameters (in systole and diastole) such as intra ventricular septum dimension (IVS), Left ventricular internal dimension (LVID), Left ventricular posterior wall (LVPW) and endocardial volumes. Furthermore, parameters such as, stroke volume, cardiac output, ejection fraction, body weight and heart weight were also determined. We observed a consistent increase of the wall thickness (higher IVS and LVPW) and a decrease in LVID in Tg-Δ43 and Tg-A57G for the sedentary group against control-WT mice, indicating hypertrophic heart remodeling. Work is still underway to elicit results from the exercised(swimming) Tg-Δ43 and Tg-A57G mice .We anticipate that cardiac phenotypes will be augmented by strenuous exercise in these two animal models displaying two distinct phenotypes of a healthy \textit{versus} diseased heart.
Poster #25

GENOMIC SEQUENCING AS A NOVEL MECHANISM FOR THE COMPREHENSIVE ANALYSIS OF POLYMORPHISMS IN PRIMARY IMMUNODEFICIENCY DISEASE. Kristina Gemayel1, R. Haire2, P. Sriaroon2, J. Cannon2, J. Sleasman2, and G. Litman2. 1Nova Southeastern College of Osteopathic Medicine, 3301 College Ave. Fort Lauderdale, FL, 2Health Children's Research Institute, University of South Florida, 601 4th Street South, St. Petersburg, FL.

Genomic sequencing is quickly proving itself as a revolutionary tool in medicine and clinical diagnosis. In the present research, exome sequencing has been carried out using Illumina® technology to further study the preliminary genetic workup of patient CM10, presenting with an absence of B cells and profound agammaglobulinemia. A total of $5.5 \times 10^9$ total bases have been acquired from $55.2 \times 10^6$ reads, which represents a 53-fold average genome coverage (Otogenetics Corp.). DNA sequencing of a series of candidate genes identified an aberrant form of $\lambda_5$, the surrogate light chain that is required for the initial transport and expression of the Ig heavy chain on the cell surface, and normal B cell development. Preliminary analyses of the $\lambda_5$ sequences in CM10 using DNANexus confirm the same four variant alleles that were identified by conventional exon priming and sequencing in CM10. The observed genetic lesions seen in both the comprehensive screen of gene amplicons and the exome sequence of CM10 suggest a gene conversion event is occurring. Further studies has revealed a highly complex pattern of mutations involving $\lambda_5$ and a $\lambda_5$ pseudogenes that produces multiple aberrant alleles which give the impression of being introduced through gene conversion and indicate a novel mechanism of primary immunodeficiency disease.

Poster #26

VALIDATION AND OPTIMIZATION OF A BLOOD-BASED GENE EXPRESSION SIGNATURE FOR AUTISM. Jordan A. Spaw1, Dalibor Nakladal3, Stephen G. Grant2, and Ana M. Castejon1. 1College of Pharmacy, 2Public Health Program, College of Osteopathic Medicine, Nova Southeastern University, Fort Lauderdale, FL. 3Faculty of Pharmacy, Comenius University, Bratislava, Slovakia.

Autism is a complex neurodevelopmental disorder whose prevalence has increased 30% within the past two years. Since there are no biological tests for autism, it is usually diagnosed with a variety of behavioral assessments. Recently, genetic and/ or environmental predispositions to the disorder have been suggested by abnormalities in peripheral blood leukocytes of those with autism. Significant gene expression differences were found in 11 common genes (log-ratio $>$ 1.5; $p < 0.05$; $q \leq 0.05$) from children with autism and autism spectrum disorder (ASD) as compared with neurotypical controls; these genes were predominantly related to the natural killer-mediated cell cytotoxicity pathway (Gregg et al., 2008, Genomics 91, 22–29). We found that this signature was able to distinguish autistic and non-autistic children with a sensitivity (0.54) and specificity (0.92) in the original Gregg data. In the present study, this 11-gene “signature” was further analyzed in the aforementioned database and in a second, larger database originally contributed by Alter et al., (2011, PLoS ONE 6: e16715), consisting of 82 children with autism and 64 unaffected controls. We found that, individually, 7 out of the 11 signature genes were significantly differentially expressed in the Alter et al. database. However, this signature had a different sensitivity (0.57) and specificity (0.48) when applied to the Alter data. These results partially validate the findings of Gregg et al. Reducing the signature to just the 7 probes significant in both data sets had little effect on the concordances. Our next step will be to expand the signature probe set to include all known probes (n=24) coding for the original 11 genes. The validation and optimization of this group of genes across two independent sets of patients suggests that there is a shared gene expression signature for autism that could potentially provide the basis of a biological test for this disorder.
Geriatrics

**NATIONAL TRENDS IN THE ELDERLY (65-84) AND THE SUPRA-ELDERLY (>85) TRAUMA: 1997-2012.** Omeed Sizar¹, Abid Farooq¹, Fahim Habib². ¹Nova Southeastern University, Ft Lauderdale, ²Broward Health Medical Center, Trauma, Ft Lauderdale, FL.

Trends in incidence and outcomes of traumatic injury among the elderly (age 65-84) and the supra-elderly (age > 85) are unknown. This information has the potential to offer insight into informed trauma system planning and improve outcomes in this highly vulnerable population. The Healthcare Cost and Utilization Project Nationwide Inpatient Sample (HCUP-NIS) database was queried to identify patients with ICD codes for a traumatic injury. Data, stratified by age group was then abstracted for incidence, lengths of stay, charges, mortality and discharge status for patients for the period 1997-2012. The study period was divided into four periods of 4-years each. Statistical analysis was performed using the ANOVA, t test, and chi square test as appropriate. A p value of <0.05 was used to determine significance. Over the 16-year study period, traumatic events in the elderly have increased by 6.8% (p=0.0005) and by 29% in the supra elderly (p<0.001). In contrast, admissions for injury decreased in both adults and children (6%, and 29.5% respectively, p=0.0005). A decrease in length of stay was seen with decrease from 6.0 to 5.2 days (p<0.0001) in the elderly and 6.2 to 5.0 days (p<0.0001) in the supra-elderly. Length of stay for adults on the other hand has increased from 4.83 to 5.1 (p=0.06). Pediatric patient in-hospital mortality has decreased significantly (p=0.001) with concurrent increases in discharge to home (p=0.003). Adult in-hospital mortality rates and discharges home have remained stable (p=0.83, p=0.24 respectively). Elderly patients have shown stable in-hospital mortality rates (p=0.149) with decreased discharges home (p=0.0003). The supra-elderly have shown the worst trend in outcomes, with significant increases in in-hospital mortality (p=0.0003) and significantly fewer patients being discharged home (p=0.0004). Costs have risen for patients of all age groups over the study period (p<0.0001). Geriatric trauma is rising at an exponential rate, with the elderly and supra-elderly patients forming an increasing proportion of the trauma population. These elderly and supra-elderly patients have been shown to have poorer outcomes, as demonstrated by in-hospital mortality and discharge status. Geriatric specific trauma programs are urgently needed to address this evolving epidemic.

Immunology

**RELATIONSHIPS AMONG ANXIETY, DEPRESSION, SLEEP DISTURBANCE, FIBROMYALGIA, OBESITY, AND GASTROESOPHAGEAL DISEASE IN PATIENTS WITH RHEUMATIC DISEASE.** Ali H. Ahmed and Shazia Beg. Department of Clinical Sciences, University of Central Florida College of Medicine, Orlando, FL.

Patients with rheumatic diseases are at increased risk for the development of comorbidities and those with comorbidities are more likely to experience exacerbations of existing disease. Our goal was to determine the prevalence of anxiety, depression, sleep disturbance, fibromyalgia, obesity, and gastroesophageal reflux disease (GERD) among patients with rheumatoid arthritis (RA), Sjogren’s syndrome (SS), systemic lupus erythematosus (SLE), psoriatic arthritis (PsA) and/or fibromyalgia and to determine the relationship of the aforementioned outcomes with each other, patient demographics, and other clinical characteristics. This was accomplished by designing a cross-sectional, retrospective study with a random selection of 200 patients with any of the aforementioned rheumatic diseases. RAPID3 forms and electronic health records were utilized to collect clinical information including RAPID3 score, sleep quality, anxiety, depression, functional status, pain level, patient demographics, BMI, presence of GERD, and presence of fibromyalgia. Our preliminary data show that RA, SS, SLE, PsA, GERD, and fibromyalgia were 60.3%, 20.5%, 19.6%, 10.3%, 17%, and 19.2% respectively among the study subjects. Patients with PsA were found to have the highest average RAPID3 score of 12.98 while those with SLE were found to have the lowest (9.78). Among the comorbidities, sleep disturbance was found to have the highest correlation (.623) with RAPID3 score and those with fibromyalgia were found to have the highest absolute RAPID3 score (16.30). Interestingly, patients with PsA were found to have among the lowest prevalence of fibromyalgia as well. Our preliminary findings indicate that comorbidities commonly associated with rheumatoid diseases are important prognostic risk factors in predicting disease progression and severity and therefore be clinically addressed as a contributory factor to underlying rheumatic disease.
DEPLETION OF MELANOMA FN14 BY OLIGONUCLEOTIDE MEDIATED EXON SKIPPING. Kristen M. Beck, Sitharam Ramaswami and Matthew S. Hayden. Department of Dermatology and Department of Microbiology and Immunology, Columbia University College of Physicians & Surgeons, New York, NY.

Fibroblast growth factor inductive 14 (Fn14) is the receptor for the TNF family cytokine TNF-like weak inducer of apoptosis (TWEAK). TWEAK binding triggers multiple signaling pathways downstream of Fn14, including activation of the MAPK and NF-κB signaling pathways. Activation of these pathways can promote cell survival, proliferation, and angiogenesis. When Fn14 is highly expressed, it activates similar signaling pathways in a TWEAK-independent manner. Recently it has been shown that Fn14 is highly expressed in melanoma, as well as in several additional other malignancies. Overexpression of Fn14 may promote cancer cell survival and can correlate with poor outcomes. Consequently it is thought that the Fn14 pathway could be a therapeutic target in melanoma. Given that Fn14 can signal in both a TWEAK-dependent and -independent manner, we have developed a strategy to block both signaling mechanisms. Here we report an exon-skipping approach to ablate cell surface expression of Fn14 in melanoma cells. We designed an antisense nucleic acid analog oligonucleotide, morpholino, targeting the predicted exonic splicing enhancer sites in exon 3 of Fn14 pre-mRNA. Here we show that transfection of this morpholino yields efficient skipping of Fn14 exon 3 in human melanoma cell lines. Reverse transcription and cDNA sequencing confirmed that the morpholino prevented splicing of exon 3 and produced mRNA with the predicted sequence. The resulting mRNA, with in frame splicing of exon 2 to exon 4, encodes a truncated protein lacking a portion of the extracellular domain and entire transmembrane domain of the full-length Fn14 protein. Transfection of the morpholino oligonucleotide consequently leads to downregulation of Fn14 protein and loss of TWEAK induced NF-κB activation. The truncated Fn14 encoded by mRNA lacking exon 3 is predicted to be secreted and may be capable of functioning as a decoy receptor by binding to TWEAK. These data suggest a novel means of blocking Fn14 signaling in melanoma.

INHIBITION OF MELANOMA PROPAGATION BY INDUCED EXPRESSION OF AN IMMUNOGENIC MOLECULE IN A MURINE MODEL. Nicholas Cnossen, Geoffrey Stone, Jimmy Termini. Department of Microbiology and Immunology, University of Miami School of Medicine, Miami, FL.

Melanoma is believed to be an immunomodulated entity, as evidenced by documented spontaneous regressions in the absence of medical intervention. In addition, recent immunomodulatory melanoma treatment modalities such as high dose IL-2 and ipilimumab have shown efficacy in treating metastatic disease. However, these treatments cause significant toxicity due to lack of specificity, prompting investigations into the induction of tumor-specific immune responses. One promising method of local immune induction is viral transfection of malignant cells with genes coding for known immunogenic molecules. The current study was designed to characterize the local immune response and tumor growth pattern of melanoma transfected with an inducible immunogenic construct, SPD-CD40L, in a murine model. Expression of SPD-CD40L has previously been shown to activate cellular immune responses in vivo by clustering CD40 on the surface of dendritic cells and CD8+ T-cells. To accomplish this, a murine melanoma cell line was virally transfected with plasmids containing a tetracycline-inducible SPD-CD40L allele, and successfully transfected cells were obtained by antibiotic selection. Mice were subcutaneously inoculated with the cells and randomized to receive either distilled water or water with tetracycline (control or SPD-CD40L+, respectively). Tumor length and width in both groups was measured over time until the tumor area exceeded 225 mm² or the mouse passed away. Rate of tumor growth was significantly slower in the SPD-CD40L+ mice until approximately day 10, at which point tumor growth rates equalized. Histology of harvested tumor sections demonstrated markedly increased lymphocytic invasion in the SPD-CD40L+ mice as compared to the control mice, indicating successful induction of a localized immune response. The proportion of mice reaching endpoint criteria did not differ between the groups, most likely due to loss of plasmid during the monitoring period. Given our successful induction of a localized immune response with slowing of tumor growth rate, further studies with more secure plasmid retention are indicated.
Microbiology

Poster #31
REAL-TIME PCR SEROTYPING OF GROUP B STREPTOCOCCUS. Kathleen Breeding, MSPH, Tara M. Randis, MD, Adam J. Ratner, MD, MPH. Department of Pediatrics, Columbia University College of Physicians & Surgeons, New York, NY.

Screening for Streptococcus agalactiae (Group B Streptococcus [GBS]) rectovaginal carriage in late pregnancy with targeted intrapartum antibiotic prophylaxis has been effective for prevention of early-onset GBS disease. However, it is a resource-intensive strategy that is subject to missed opportunities for prevention and has not decreased the rate of late-onset disease. Because of these limitations, GBS remains a major cause of perinatal disease, and new strategies for prevention are urgently needed. Strategies currently under consideration include polysaccharide-based conjugate vaccines. However, vaccine candidates currently in evaluation target only a subset of GBS serotypes, raising the possibility of serotype replacement. To better understand the serotype distribution of GBS colonization, we developed a TaqMan-based real-time PCR serotyping method. The PCR method identifies unique regions in the capsular polysaccharide sequences (cps) within each of the 10 GBS serotypes. The PCR is sensitive to 25 picograms of GBS genomic DNA at a 30 cycle threshold and is robust to interference by non-targeted strains. Results were obtained within 2 hours and tested against latex agglutination (the gold standard). Real-time PCR using our designed primers and probes is a highly sensitive method for identifying serotypes of GBS and allows for accurate assessment of serotype prevalence in colonization. Importantly, this method allows detection of GBS serotypes directly from clinical specimens without the need for culture, which facilitates identification of co-colonization with multiple strains. This new method makes understanding GBS serotype prevalence to inform vaccine development more accessible and efficient.

Poster #32
PHYSICIAN AWARENESS AND UTILIZATION OF ANTIBIOTIC STEWARDSHIP IN THE STATE OF FLORIDA. Rachel L. Johnson and Leslie Beitsch, MD, JD. Behavioral Sciences and Social Medicine, Florida State University College of Medicine, Tallahassee, FL.

The Center for Disease Control’s 2013 Threat Report indicated an imminent need to address the growing problem of antibiotic resistance worldwide. Hospital-acquired infections (HAIs) have generated significant attention, and as a result research and stewardship programs have been implemented. There remains, however, a need to address the issue at the outpatient level. Studies suggest physicians are lacking tools to navigate the complexities of antibiotic prescribing, especially locally. As the problem intensifies and becomes more intricate, healthcare professionals of tomorrow will need information and resources that keep pace with the rapid evolution of treatment standards. With this in mind, a thirteen-question online survey was sent to board-certified practicing pediatricians and family practice physicians to assess antibiotic prescribing behaviors and knowledge concerning antibiotic stewardship. The ongoing survey was made available beginning in August of 2014, and responses will be collected through the end of December, 2014. Of the responses from pediatricians surveyed to date, about 45% have never been approached by an antibiotic stewardship organization. Most pediatricians, whether affiliated with a hospital or not, used national sources of information (Journals, CDC, etc.) for keeping up with antibiotic resistance concerns. Furthermore, our data suggests there is no association between how frequently physicians check local resistance patterns and whether or not they are affiliated with a hospital. This is contrary to what one would expect, considering many hospitals release monthly or quarterly antibiograms. These preliminary results suggest there may be a need for more widely promoted antibiotic stewardship programs in the state of Florida. Furthermore, there may be a need for an integrated resource concerning resistance issues in our state.
ROLE OF HYPOXIA INDUCIBLE FACTOR 1α IN GAMMAHERPES VIRUS REPLICATION AND PATHOGENESIS. Darlah M. Lopez, Samita Andreansky and Enrique Mesri. Department of Microbiology and Immunology, University of Miami Miller School of Medicine, Miami FL.

Kaposi’s Sarcoma Associated Herpes Virus (KSHV) causes Kaposi’s sarcoma (KS), an angiogenic spindle-cell sarcoma, associated with AIDS. KSHV has evolved genes that encode proteins that target the regulation of the host transcription factor, hypoxia inducible factor 1 alpha (HIF1α). Stabilization of HIF1α is essential for adaptation of cells and tissues to low oxygen and inflammation during microbial infection. In fact, HIF1α activation by KSHV is critical for viral induced-sarcomagenesis and persistence of the viral latent life cycle within the host cell. However, the role of HIF1α during acute lytic infection and latency establishment in the context of a natural host infection has not been explored. In this study we employed the murine gammaherpes virus 68 (MHV68), a biological and genetically murine pathogen similar to KSHV that infects laboratory mice, as a valuable small animal model. MHV68 is relevant to KHSV as it shares homologous viral genes and establishes a replicative program (lytic and latency) in the same cell types (B cells and endothelial cells). Also, Polcicova et al. has documented a role for HIF1α in MHV68 lytic reactivation to latency in hypoxia. To investigate the role of HIF1α in gammaherpes virus replication and pathogenesis we induced deletion of HIF1α in MHV68 virus-specific cells in vivo using the Cre recombinase-LoxP technology. Following intranasal infection, we assessed virus production by plaque assay from lungs, the primary site of acute infection for this model. We found that at day 3-post infection virus production does not change in the absence of HIF1α. However, during peak acute lytic infection (day 5) MHV68 virus production was significantly reduced upon virus-specific HIF1α deletion compared to wild type mice. Moreover, we assessed the frequency of latently infected splenocytes at day 16 in an infectious center assay and found that reactivation from explanted latently infected splenocytes was significantly impaired upon HIF1α deletion. Based on these preliminary results, we hypothesize that HIF1α plays a critical role in the virus infection and latency establishment of the murine gammaherpes virus MHV68. Our experimental system allows us to further understand the interaction between viral and cellular genes leading to gammaherpes virus pathogenesis, a crucial step towards the development of therapies for KS and other infectious diseases.

DETERMINATION OF THE BACTERIAL ETIOLOGY OF POST INFECTIOUS HYDROCEPHALUS IN HAITIAN CHILDREN. Michael Ragheb, University of Miami, Masters of Public Health, Clinical Research Building, 1120 NW 14th St. Miami, FL.

Most hydrocephalus in the developing world, including Haiti, is caused by a prior perinatal central nervous system infection. The cause(s) and source of these infections is unknown. Amplification and characterization of 16S rDNA in cerebrospinal fluid (CSF) samples taken during the treatment of children with clinically and radiographically suspected post-infectious hydrocephalus (PIH) can identify the causative organism even once the infection has subsided and cultures are sterile. We propose to use this strategy to study the children treated for hydrocephalus at the Project Medishare Hospital in Port au Prince, Haiti, in a three phase project. The initial phase will determine if rDNA can be identified in sterile CSF samples collected during surgery in a small sample size of 10-15 children. The subsequent phases will expand the initial collections to a larger sample to identify the predominant organisms that cause PIH. The final phase will be an epidemiologic assessment of the possible environmental sources of the most common causes of infection in the hope of developing a strategy to prevent the initial infections.
Neurology

Poster #35

LONGITUDINAL INVESTIGATION OF VISUAL ACUITY IN THE EARLIEST STAGES OF PARKINSONISM. Sarah A. Avila, Melissa K. Geary, Charles G. Maitland, Juliana J. Matthews, Nathan C. Nowalk and Shawn W. Adams. College of Medicine, Florida State University, Tallahassee, FL.

Parkinson’s Disease is characterized by the loss of dopaminergic neurons in the nigrostriatal system. Similarly, cells in the inner layers of the retina such as the interplexiform and amacrine cells also utilize dopamine for their ocular functions. A cohort of patients in the earliest clinical stages of Parkinsonism were studied and found to have statistically significant deficiency in contrast sensitivity vision compared to age matched controls. After 12-16 months we re-evaluated the cohort measuring changes in disease status using the Unified Parkinson's Disease Rating Scale (UPDRS) and the H&Y scale versus changes in contrast sensitivity visual acuity. Twenty-five early stage patients Stage I-II H&Y (15 Stage I) versus 25 controls were assessed and eighteen participants were reassessed after 12 months. Testing included UPDRS, contrast sensitivity acuities (SLOAN chart) and Optical Coherence Tomography (Zeiss). Visual acuities <20/50 and known ophthalmologic pathologies were excluded. Statistical data was analyzed via student’s t-test and analysis of variance. Seventy five percent (75%) of the original group reoccurred at 12 and 16 months. General visual examinations demonstrated no significant intervening pathologies; however contrast sensitivity visual acuities had declined in 11 patients, remained the same in four, and improved in three cases. No patient approached the baseline levels of the control group. Motor Scoring (part III UPDRS) demonstrated a decline in function on average by 4 points in 9 patients; 2 scores remained unchanged and 6 patients improved (average= 2.5 points). A correlation between loss of contrast visual acuity and UPDRS scoring was not established. This longitudinal study confirms that a deficiency in contrast sensitivity is present in patients with early signs of Parkinsonism. This deficiency continues to progress irrespective of motoric status. Given the ease with which contrast sensitivity visual acuity can be tested, it seems plausible that such testing may be used to monitor disease progression and potentially the efficacy of drug therapy.

Poster #36

DOES Admission to TEACHING VS. NON-TEACHING HOSPITALS INFLUENCE the association between day of HOSPITAL ADMISSION and in-hospital mortality AMONG STROKE PATIENTS IN FLORIDA? Jason I. Liounakos, Luv Hajirawala, Grettel Castro, Juan M. Acuña, Juan-Carlos Zevallos. Department of Medical and Health Sciences Research - Herbert Wertheim College of Medicine - FIU, Miami, Florida, 33199.

The “weekend effect” is a phenomenon where patients admitted to hospitals on weekends have poorer prognosis as compared to those admitted on weekdays. The weekend effect has been described for multiple illnesses, including stroke. However, evidence for such an effect in stroke patients is conflicting, and the underlying causes are unknown. This study aims to explore whether differences in in-hospital mortality exist between weekend and weekday hospital admissions for stroke in Florida and to evaluate whether the association is modified by teaching vs. non-teaching hospital status. We performed a retrospective cohort study using data from the Florida Stroke Registry database from 2008 to 2012. This registry is maintained by the Florida Agency for Health Care Administration. Exclusion criteria included the diagnosis of transient ischemic attack and patients less than 18 years of age. Independent association was tested using multivariate logistic regression. The primary independent variable was the day of admission (weekend or weekday) and the outcome was defined as in-hospital mortality. We performed a retrospective cohort study using data from the Florida Stroke Registry database from 2008 to 2012. This registry is maintained by the Florida Agency for Health Care Administration. Exclusion criteria included the diagnosis of transient ischemic attack and patients less than 18 years of age. Independent association was tested using multivariate logistic regression. The primary independent variable was the day of admission (weekend or weekday) and the outcome was defined as in-hospital mortality. We performed a retrospective cohort study using data from the Florida Stroke Registry database from 2008 to 2012. This registry is maintained by the Florida Agency for Health Care Administration. Exclusion criteria included the diagnosis of transient ischemic attack and patients less than 18 years of age. Independent association was tested using multivariate logistic regression. The primary independent variable was the day of admission (weekend or weekday) and the outcome was defined as in-hospital mortality. This analysis was also stratified by teaching hospital status. Our final sample size totaled 253,851 patients. Those admitted on the weekend for stroke were more likely to die compared to those admitted on weekdays, and the adjusted odds of in-hospital mortality were similar regardless of teaching hospital status. Compared to patients admitted on weekdays, those admitted on weekends to teaching hospitals had 33% higher odds of in-hospital mortality [Odds ratio (OR) = 1.33; 99% confidence interval (CI) 1.22 - 1.45] after adjusting for age, gender, ethnicity, race, diabetes mellitus, hypertension, atrial fibrillation, and length of stay. In non-teaching hospitals, weekend admissions were associated with 32% higher odds of in-hospital mortality (OR = 1.32; 99% CI 1.22 - 1.43) after the same adjustments. We found evidence that the risk of in-hospital mortality is higher in patients admitted on weekends compared to weekdays. We found no evidence that the association is modified by hospital teaching status.
Neuroscience

**POTENTIAL PROTECTIVE ROLE OF OXYTOCIN AGAINST APOPTOSIS IN ASTROCYTES AND NEURONS.** Mohammed M Alanazi, Jan Bakos, Zuzana Bacova, and Ana Maria Castejon. College of Pharmacy Nova Southeastern University, University Drive, Fort Lauderdale, FL. 1Institute of Experimental Endocrinology, Slovak Academy of Sciences, Vlarska 3 Bratislava, Slovakia.

The neuropeptide oxytocin that is released from the posterior pituitary into the systemic circulation has been implicated in several vital physiological processes, ranging from reproduction to social and non-social behaviors. Understanding its precise nature and functions is crucial both, within the theoretical context of neurobiology and related fields, but also within the context of clinical disorders such as anxiety, schizophrenia, or autism. Oxytocin has been recently also associated with cell protection against apoptosis. This finding might be relevant since certain neurological disorders have been linked to low systemic levels of oxytocin. In the present study, we evaluate the protective role of oxytocin against apoptosis and cell death in two different cell lines of astrocytes and neurons. In order to test our hypothesis, we developed two Oxytocin Receptor Knockdown cell lines, 1) U87-MG_KD OXT-R (astrocytes) and 2) SH-5H5Y_KD OXT-R (neurons), to further analyze apoptosis rates both, in absence and presence of pro-apoptotic agents such as Camptothecin. Analysis of apoptosis and cell death are being conducted by flow cytometry in conjunction with Annexin-V/7-AAD reagents. Preliminary observations have shown a potential correlation between oxytocin and apoptosis/cell death rates, being significantly higher in those knockdown cell lines in which oxytocin pathway has been impaired, comparing with the controls (regular U87-MG and SH-5H5Y cells lines). The outcomes of this study will contribute not just to a better understanding of the key role that oxytocin plays in multiple physiological processes, but also to a new approach for studying some current relevant neurological disorders such as autism.

**BRANCH SPECIFIC DENDRITIC Ca^{2+} SPIKES INDUCE PERSISTENT SYNAPTIC PLASTICITY.** Joseph Cichon & Wen-Biao Gan. Molecular Neurobiology Program, Skirball Institute, Department of Neuroscience and Physiology, New York University School of Medicine, New York, N.Y.

The brain has an extraordinary capacity for memory storage, but how it stores new information without disrupting previously-acquired memories remains unknown. We show that different motor learning tasks induce dendritic Ca^{2+} spikes on different apical tuft branches of individual layer V pyramidal neurons in mouse motor cortex. These task-related, branch-specific Ca^{2+} spikes cause long-lasting potentiation of postsynaptic dendritic spines active at the time of spike generation. Notably, when somatostatin-expressing interneurons are inactivated, different motor tasks frequently induce Ca^{2+} spikes on the same branches. On those branches, spines potentiated during one task are depotentiated when they are active seconds prior to Ca^{2+} spikes induced by another task. Concomitantly, increased neuronal activity and performance improvement after learning one task are disrupted when another task is learned. These findings indicate that dendritic branch-specific generation of Ca^{2+} spikes is critical for establishing long-lasting synaptic plasticity, thereby facilitating information storage associated with different learning experiences.
IDENTIFYING NUCLEAR AND CYTOPLASMIC EFFECTORS OF ONCOGENE SET-β IN CNS NEURONS. Xiongfei Liu1, Ephraim F. Trakhtenberg1, and Jeffrey L. Goldberg1,2 1Bascom Palmer Eye Institute, University of Miami School of Medicine, Miami, FL. 2Shiley Eye Center, University of California San Diego, La Jolla, CA.

Central nervous system (CNS) axon regeneration capacity declines after birth and is partially controlled by transcriptional regulators, oncogenes, growth factors, and inhibitory extracellular environment. Oncoprotein Set-β, an inhibitor of protein phosphatase PP2A (I2PP2A), is one of the factors that regulate axon growth. Predominately a nuclear protein that affects transcription, Set-β also functions at cellular membranes. Recently our lab found that nuclear Set-β suppressed axon elongation, whereas membrane-bound Set-β promoted axon growth. The goal of this project is to investigate the nuclear or cytoplasmic binding partners of Set-β in primary CNS neurons. Acutely dissected cortices or hippocampi from embryonic day 18 Sprague-Dawley rats were immunoprecipitated with anti-SET and normal rabbit IgG antibody. The immunoprecipitate was then further processed with Western Blot, gel staining, and Mass Spectrometry (MS) to assess Set-β pull down optimization and it associative binding partner in the nuclear and cytoplasmic fractions. PP2A (protein phosphatase 2A) was indeed pulled down with Set-β antibody, though PP2C (protein phosphatase 2C), KLFs (Krüppel-like family of transcription factors), and Rac-1 (ras-related C3 botulinum toxin substrate 1) proteins were not found. Moreover, MS of the immunoprecipitate from purified hippocampal neurons’ nuclear and cytoplasmic fractions recovered the regulatory subunit PPP2R2D of PP2A in the cytoplasmic fraction, with limited recovery of nuclear protein as only importin-a6 was reliably identified in the nuclear fraction. This was in line with importin-a being previously shown to translocate with Set-β into the nucleus. In addition, the observation that PP2A overexpression in postnatal RGCs (retinal ganglion cells) suppressed neurite growth was consistent with PP2A’ overall suppressive effect on neurite growth. Taken together, data suggested that cytoplasmic Set-β may stimulate axon growth by locally inhibiting PP2A. In conclusion, PP2A, not Rac1 or KLFs, is a cytoplasmic binding partner for Set-β. The endogenous Set-β recruitment to cellular membranes may promote axon growth through local inhibition of PP2A.

THE EFFECT OF MINDFULNESS MEDITATION ON BRAIN DYNAMICS: A PILOT STUDY. Michelle Shnayder, Department of Neuroscience, Brown University, Providence, RI.

Mindfulness meditation has been reported to improve both physical and mental health. Understanding the cortical brain rhythms involved in meditation training could provide insight into how the brain processes information and techniques to improve focus and attention. Somatosensory (SI) 7-14 Hz alpha rhythms are thought to function as a filter for the processing of irrelevant sensory inputs in primary sensory cortex. This filter creates an inverse relation between alpha rhythm and spatial attention, where alpha power is decreased in areas of SI that are attended to and increased in unattended locations. Meditation has been shown to enhance this top-down regulation of 7-14 Hz cortical oscillations, but only for brief periods of control. This study looked at the ability of three people in various degrees of meditation experience (a highly experienced meditator (~40 years), an intermediate meditator, and a non-meditator) to maintain alpha rhythm modulation over 120 sec trials, as recorded by magnetoencephalography (MEG), while attending to their right or left hand. We suspected the most experienced meditator would be able to both raise his alpha power in SI and maintain this elevation more consistently than the other two participants. While this subject demonstrated an increased ability to transiently raise his SI alpha power, he was no better at consistently maintaining that level than the other two subjects. These results suggest that alpha power alone and not temporal consistency is important in the attentional improvements arising from meditation training. This study is an important first step in understanding the neurologic implications of meditation.
ASSESSMENT OF SPASTICITY AND PAIN DURING THE PERFORMANCE OF A FUNCTIONAL TASK IN PERSONS WITH SPINAL CORD INJURY. Jacqueline Tibbett, Dr. Edelle Field-Fote, and Dr. Eva Widerstrom-Noga. Department of Physiology, University of Miami Miller School of Medicine, Miami, FL.

Spasticity (involuntary muscle spasms) and persistent neuropathic pain are two common consequences of spinal cord injury (SCI) that significantly decrease quality of life. The mechanistic basis for both conditions includes spinal neuronal hyperexcitability. No measure of spasticity during daily activities exists for persons who are unable to walk, thus, a quantitative assessment of spasticity and its relationship with pain during a common task may be useful for determining functional impact of interventions for spasticity. Participants with SCI enrolled in a two-day crossover study to capture spasticity evoked by seating transfers. Electromyographic (EMG) activity was recorded from three leg muscles, and spasticity and pain levels were reported immediately before and after the transfer(s) using a 10-point numerical rating scale. EMG data was analyzed for spasms using an algorithm-based approach. Participants also answered questions regarding persistent spasticity and pain using the SCI Spasticity Evaluation Tool and the International SCI Pain Basic Data Set. A majority of participants had persistent pain and also reported that spasticity itself was a source of pain. Seating transfers evoked spasms, and the duration and number could be quantified by EMG activity. Spasm duration captured through EMG during transfer correlated with self-report measures of spasticity. While both spasticity and pain levels increased during the transfer, only the increase in spasticity was significantly elevated compared to pre-transfer levels. Notably, when three transfers were performed consecutively, spasm duration decreased after the first transfer. Seating transfer evoked spasms that could be captured through EMG and that correlated with self-report. These findings suggest that seating transfers may be a reliable way to assess the functional impact of an intervention on spasticity. However, spasm duration will decrease with consecutive transfers regardless of intervention. Finally, the large proportion of participants who reported that spasticity increased their pain severity warrants further investigation into interventions that target both problems.

Neurosurgery

FDA-APPROVED HUMAN STEM CELL TRANSPLANTS AS A TREATMENT OF PENETRATING TRAUMATIC BRAIN INJURY. Lee Onn Chieng¹, Markus Spurlock¹, Shyam Gajavelli¹, Aminul Ahmed¹, Sam Pingdewinde¹, Ross Bullock¹. ¹Miami Project to Cure Paralysis, Department of Neurosurgery, University of Miami Miller School of Medicine, Miami, FL 33136.

Penetrating traumatic brain injuries (PTBI) are associated with the worst outcome and high mortality. Currently there is no FDA-approved intervention to mitigate consequences following PTBI. Stem cell transplantations have emerged as putative therapeutic approaches. Previously different cell types have been used to repair penetrating ballistic penetrating injury (PBBI), a rat model of PTBI, which yielded unsatisfactory results. In this rodent study, we evaluated the potential usage of FDA-approved human neural stem cells (hNSCs) in PBBI. Sprague-Dawley rats underwent unilateral PBBI. Immunosuppression was established before stereotactic injection of control or hNSCs into PBBI penumbra. Animals were sacrificed at defined time points post-transplantation. Brains were sectioned for assessment of engraftment and differentiation and integration with immunostaining. 8 weeks after transplantation, robust engraftment of ~15% of cells were observed in rat brains. A 2mm wide transplant could be seen rostral to PBBI lesion. GFP-labeled transplanted cells were positive immature neuronal marker, doublecortin, and mature neuronal marker, NeuN. Additionally, the hNSCs were also stained with synaptophysin, a presynaptic marker. In this preliminary study, the unprecedented engraftment of FDA-approved hNSCs was observed up to 8 weeks. Furthermore, the transplanted hNSCs were capable of differentiate and mature into neurons. These matured neurons were also shown to form synapse, which indicates transplanted cells integrated with host neuronal circuits. Before the use of these cells in clinic additional studies that assess whether transplants improve function are needed.
**Obstetrics and Gynecology**

**THE ASSOCIATION BETWEEN THE KOTELCHUCK INDEX AND BREASTFEEDING INITIATION AND DURATION.** Lori Marcu, Juan Acuna, Gretel Castro, Pura de La Vega, Marcia Varella.

Only 47% of infants in the United States in 2012 received the recommended exclusive breastfeeding at six months. Optimal prenatal care utilization could provide more opportunities for education about breastfeeding and ultimately improve breastfeeding practices. However, it is unknown whether measures of adequacy of prenatal care utilization index, such as the Kotelchuck Index (KI), are associated with breastfeeding practices. This study aims to explore whether there is an association between the KI and breastfeeding initiation and duration. Cross-sectional study of nationwide data from mothers who had a recent delivery and were randomly selected to participate at the Pregnancy Risk Assessment Monitoring System (PRAMS) in 2009. Only mothers of healthy singletons were included. The KI was the categorized as low (Inadequate or Intermediate) or high (Adequate or Plus Adequate). Breastfeeding initiation and duration (< or ≥ 3 months) were the study outcomes. Multivariate logistic regressions were used to calculate the independent odds of initiating breastfeeding and the odds of breastfeeding longer than 3 months. STATA 12 was used accounting for the complex survey design. Significance was considered for p-value ≤ 0.01. After adjustments for confounders, the KI was not independently associated with breastfeeding initiation [Odds Ratio (OR): 1.00, 99% Confidence Interval (CI): 0.89 - 1.12] or duration (OR: 0.92, 99% CI: 0.83 - 1.02). Other variables independently associated with higher odds to initiate and breastfeeding longer than 3 months were being of “other” race, being Hispanic, married, and having more years of education. Being a teen mother, obese pre-pregnancy, and participating in WIC were associated with lower odds for initiation or for breastfeeding for more than 3 months. Higher income and lower parity were associated with higher breastfeeding initiation. Lastly, higher parity was associated higher breastfeeding duration. The Kotelchuck Index was not associated with breastfeeding initiation. However, we found weak evidence that High Kotelchuck Index (Adequate or Plus Adequate) decreased the odds of breastfeeding for more than 3 months. Further research is needed to assess whether the association between high KI and lower breastfeeding duration is due to differences in maternal health status during pregnancy.

**Oncology**

**NILCO AS A NOVEL BIOMARKER FOR TYPE II ENDOMETRIAL CANCER.** Viola Lanier, D. Daley-Brown, G Oprea-Iliies, R Pattillo, J Lillard, RR Gonzalez-Perez. Morehouse School of Medicine, Atlanta, GA 30310 (DDB, VL, RP, JL, RRGP); Emory University, Atlanta, GA 30322(GOI).

Obesity is a major risk factor for endometrial cancer and a growing epidemic in the United States. African-American women have been shown to have a higher incidence of obesity and are more likely to die from endometrial cancer (EnCa) than Caucasian women. EnCa Type I is estrogen dependent, and has a better prognosis. In contrast, EnCa Type II is estrogen independent, usually associated with endometrial atrophy, more aggressive, has poor prognosis and higher recurrence. Increased expression of leptin correlates to obesity. Notch, IL-1 and leptin crosstalk outcome (NILCO) has been shown to lead to cell proliferation, metastasis, and poor cancer prognosis. We hypothesize NILCO expression correlates to EnCa Type II that could be more evident in obese patients. To validate this hypothesis, EnCa tissues from obese and lean African American patients and Chinese patients were interrogated for NILCO expression via western blot (WB), immunohistochemistry (IHC), and real-time PCR (RT-PCR) analyses. In addition, the effects of leptin and a novel inhibitor of leptin receptor (nanoparticles bound to LPrA2; IONP-LPrA2) on NILCO expression was investigated in human EnCa Type II cells lines. Our results show higher expression of NILCO in obese patients of Type II EnCa, and at a more advanced stage of the disease. IONP-LPrA2 decreased NILCO expression and consequently decreased cell proliferation. Our studies show that NILCO could be a novel biomarker for Type II EnCa, and may also be a possible chemotherapeutic target. Our observations are especially relevant for obese EnCa patients.

Studies suggest that stimulation of the β-adrenergic receptor may promote tumor proliferation and invasion, angiogenesis, and resistance to chemotherapy. We investigated whether β-adrenergic inhibition with beta-blockers (BBs) resulted in increased radiation sensitivities in vitro and differential treatment response and overall survival (OS) in patients treated with chemoradiation for locally advanced non-small cell lung cancer (LA-NSCLC).

To assess the efficacy of β-adrenergic blockade as a radiosensitizer in NSCLC, we analyzed with clonogenic survival assay in human adenocarcinoma cell lines (PC9 and A549) treated with and without propranolol (non-selective β-adrenergic receptor antagonist, 1 to 50 μM) and radiation (0 to 10 Gy). We also retrospectively evaluated 77 patients with Stage IIIA NSCLC who received neoadjuvant chemoradiation followed by surgery. A Cox proportional hazard model was used to determine associations between BB intake and treatment response and outcomes. Propranolol combined with radiation decreased clonogenic survivability of PC9 and A549 cells in vitro (propranolol at 50 μM, p < 0.01) compared with irradiation alone. In our clinical cohort, patients who took BBs (n =16) were compared with patients (n =61) who did not. BBs use was associated with decreased distant metastases (risk ratio [RR] 0.19; P=0.03). There was no association between BBs use and primary tumor response (P=0.40) or pathological mediastinal nodal clearance (P=0.68). In univariate analysis, patients taking BBs demonstrated a trend to improved OS at 1year (81.3% vs 57.4%, P=0.08) and median distant metastasis-free survival (DMFS) (2.6 years vs 1.3 years, P=0.16) compared with patients not taking BBs. β-adrenergic blockade enhanced the radiation sensitivity of lung cancer cells in vitro. BB use was associated with decreased distant metastases rate and potentially improved OS, DMFS, and PFS in patients with LA-NSCLC. Additional studies are warranted to further evaluate the benefits of β-adrenergic blockade on radiation treatment response and lung cancer recurrence.

Ophthalmology

THE IMPACT OF CONJUNCTIVOCHALASIS ON DRY EYE SYMPTOMS. Abigail Alexander1, Priyanka Chhadva1, Allison McClellan1, Katherine T McManus1, Benjamin Seiden1, Anat Galor2,3. 1University of Miami Miller School of Medicine, Miami, Florida, USA. 2Bascom Palmer Eye Institute, University of Miami, Miami, Florida, USA. 3Miami Veterans Administration Medical Center, Miami, FL.

The purpose of this project was to study the relationship between conjunctivochalasis (Cch) and ocular signs and symptoms of dry eye. Ninety-six patients with normal eyelid and corneal anatomy were prospectively recruited from a Veterans Administration Hospital over 12 months. Participants were classified into 3 groups (nasal chalasis [NCch; n=31], non-nasal chalasis [non-NCch; n=41], and no-Cch [n=24]). Symptoms and signs of dry eye were assessed along with quality of life implications. Statistical analyses comparing the above metrics among the 3 groups included chi-square, analysis of variance, and linear regression tests. Patients with NCch had more dry eye symptoms (DEQ5): NCch=13.8±5.0, non- NCch=10.2±5.0, no-Cch=11.6±5.8, p=0.014; ocular pain (numerical rating scale [NRS]): NCch=4.5±3.0, non-NCch=2.3±2.8, no-Cch=3.3±2.6, p=0.008; worse dry eye signs (Schirmer score): NCch=14.5±6.9, non-NCch=16.8±8.2, no-Cch=19.9±6.4, p=0.039; meibomian gland dropout: NCch 1.8±0.9, non- NCch=1.4±1.0, no-Cch=1.0±1.0, p=0.020; eyelid vascularity: NCch=0.84±0.8, non-NCch=0.74±0.7, no- Cch=0.33±0.6, p=0.019), a negative impact on quality of life (percent with moderate to severe impact): NCch=87%, non-NCch=51%, no-Cch=58%, p=0.005; and increased artificial tear use: NCch=83.9%, non-NCch=56.1%, no-Cch=66.7%, p=0.044 compared to those with non-NCch and no-Cch. The presence of NCch associates with dry eye symptoms and abnormal tear parameters and impacts quality of life compared to non-NCch and no-Cch.
ANDROGEN DEFICIENCY AND DRY EYE SYNDROME IN THE AGING MALE. Patrick M. Azcarate,1,2 Vincent D. Venincasa,1,2, William Feuer1, Frank Stanczyk1, Andrew V. Schally4 and Anat Galor1,2. 1Miami Veterans Administration Medical Center, Miami, FL, 2Bascom Palmer Eye Institute, University of Miami, Miami, FL, 3Departments of Obstetrics and Gynecology and Preventive Medicine, University of Southern California, Los Angeles, CA, 4Department of Pathology Department of Medicine Divisions of Oncology and Endocrinology, University of Miami, Miami, FL.

Dry eye (DE) is a group of eye diseases that causes ocular discomfort and can significantly decrease the quality of life. Normally, a tear film composed of three layers (lipid, aqueous, and mucous) protects the eye. The lipid and aqueous layers are primarily maintained by the meibomian and lacrimal glands, respectively. A disruption in any one of these layers can lead to DE. Various androgen-depleted states have shown meibomian gland dysfunction and abnormal tear film parameters. Our study evaluated the relationship between androgen levels and subjective and objective measures of DE. A total of 263 male patients from the Miami VA Medical Center eye clinic aged ≥50 were recruited for this prospective cross-sectional study. Patients completed Dry Eye Questionnaire 5, underwent tear film evaluation, and had serum androgen levels measured. The correlations between androgen levels, DES composite scores, DES symptoms, and global, lipid, and aqueous tear film parameters were evaluated. 263 patients with a mean age of 69 (50-95) were examined. There was no linear association between composite DES scores (generated using latent class analysis) and androgen levels. However, eyes with high DES scores (0.95-1.0) had higher levels of sex hormone-binding globulin (P = 0.03) and lower levels of dehydroepiandrosterone sulfate (DHEAS) (P = 0.02), androstenedione (A) (P = 0.02), and androstane-3α,17β-diol glucuronide (P = 0.03) compared to eyes with intermediate (0.05-0.95) or low (0-0.05) scores. There were no strong correlations between tear film measures and androgen levels. Regarding global parameters, a weak inverse correlation was found between corneal staining and A (r = -0.17, P = 0.009). For lipid parameters, a weak correlation existed between tear breakup time (TBUT) and A (r = 0.15, P = 0.02). When considering aqueous and lipid deficiency independently, the association between TBUT and A existed only with aqueous tear deficiency (r = 0.66, P = 0.002). Regarding aqueous parameters, a weak correlation existed between Schirmer test and DHEAS (r = 0.13, P = 0.047) and A (r = 0.21, P = 0.001). In conclusion, there was a weak correlation between higher levels of androstenedione and healthier global, lipid, and aqueous tear film parameters.

MANAGEMENT TRENDS IN PEDATRIC GLAUCOMA SUSPECT AND OCULAR HYPERTENSION. Matthew Greenberg, Kara M. Cavuoto M.D., Ta C. Chang M.D. Bascom Palmer Eye Institute, University of Miami Miller School of Medicine, Miami, FL.

Little consensus exists in the management and progression of pediatric glaucoma suspects and ocular hypertension (OHTN) patients. A retrospective chart review was done to ascertain treatment patterns in these patient populations. Charts were identified using the ICD-9 codes for OHTN and glaucoma suspect in patients younger than 10 years old. Patients were excluded if followed for less than two months, diagnosed with glaucoma on the first visit, or had undergone prior ophthalmic surgery. Glaucoma was diagnosed on subsequent visits as defined by the World Glaucoma Association Consensus criteria. Seventy-four charts were reviewed. Of these patients, 12.2% of patients were diagnosed with glaucoma, 25.7% presented with OHTN without glaucoma, and 18.9% had neither glaucoma nor OHTN, but presented with a high-risk syndromes. There was no significant difference in age amongst the groups. 93% of high-risk syndrome patients were referred by a pediatric ophthalmologist, compared to just 44% of glaucoma and 68% of OHTN patients. Between glaucoma and OHTN patients, average IOP, maximum IOP, corneal diameters, axial lengths, central corneal thickness (CCT), retinal nerve fiber layer thickness, and C/D ratios were similar. 28.6% of high risk patients were started on medication to lower IOP, compared to 36.9% of OHTN patients and 100% of glaucoma patients. Those treated had higher maximum IOPs, but insignificantly so (p=.09). Treated patients also had significantly higher CDTs (p=.008). All glaucoma patients presented with ocular hypertension, however, no initial biometric findings proved to be an accurate predictor of later glaucoma progression. More data is needed to ascertain glaucoma progression and treatment patterns in high-risk individuals without initial presentation of glaucoma or OHTN. These results suggest that pediatric OHTN patients and those with syndromes highly associated with glaucoma need to be closely monitored in regards to glaucoma development.
Orthopedic Surgery

Poster #49


Because healthy bone tissue involves the balance of bone formation by osteoblasts and bone resorption by osteoclasts, it is important to understand the interactions of these cells. In bacterial infection, the immune system exhibits overactive osteoclastogenesis resulting in significant bone loss. *S. aureus* is known to cause bone diseases like septic osteoarthritis and osteomyelitis. The difficulty in treating *S. aureus* infection stems from the ability of the bacteria to form abscesses within bone, evade the host’s immune system by secreting toxins, and develop antibiotic resistance. In addition, *S. aureus* can invade host cells like neutrophils and osteoblasts leading to cell lysis or apoptosis. It is known that osteoblasts and osteoblast precursor cells produce a number of pro-inflammatory cytokines like RANKL, TNF, and IL-6 which promote osteoclastogenesis, inhibit osteoblastogenesis, and trigger different inflammatory cascades. However, little is known about the effect of vancomycin treatment on the inflammatory response of osteoblast cells to *S. aureus* infection. The objective of this study was to better characterize the pro-inflammatory cytokine production of the *S. aureus*-treated and vancomycin-treated MC3T3 murine osteoblast precursor cell line. In order to study this, we treated MC3T3 cells with the bacteria and/or vancomycin for 0, 3, 6, and 9 hours, then extracted the bacteria and analyzed the MC3T3 mRNA levels of RANKL, TNF, IL-6, and GAPDH using qPCR. As expected, infection with *S. aureus* dramatically increased expression of inflammatory and pro-osteoclastogenic cytokines in murine osteoblasts. It appears that vancomycin treatment abrogates this response at all time points. While further work is necessary to elucidate the molecular pathways involved, particularly in studying vancomycin’s anti-inflammatory potential, our data appear to confirm that part of the antibiotic’s effectiveness in controlling the sequelae of infection is in inhibiting the destructive host response mechanisms. This opens a new possible treatment paradigm in which the clinician may consider not only bactericidal regimens but also those which reduce the inflammatory response so that unnecessary destruction caused by the host response is brought under control.

Poster #50

ONLINE REVIEWS OF ORTHOPAEDIC SURGEONS: AN EMERGING TREND. Chelsea D. Frost¹, Addisu Mesfin, MD². ¹University of South Florida Morsani College of Medicine. ²Department of Orthopaedic Surgery, University of Rochester School of Medicine and Dentistry.

Physician review websites have been developed due to the demand by patients for easily accessible information about physician quality. Many online physician-rating sites provide patients with information about physician quality and allow patients to rate physicians. These sites concern some physicians who argue that ratings can be misleading. In this study, we describe the landscape of online reviews of orthopaedic surgeons by looking at a sample of ratings from popular physician review websites. A public database of 2,570 United States orthopaedic surgeons was obtained that included the physician’s name, NPI number, mailing address, and gender. The 30 most populated cities in the United States, based on 2012 census bureau data, were selected for analysis and then the orthopaedics in each state were organized based on NPI number. The total number of physicians analyzed was 557 orthopaedic surgeons. The membership database for the J. Robert Gladden society was also used in order to analyze their ratings for a comparison to those that are non-minorities in the specialty. Numerical ratings from 7 popular PRWs were collected for each physician. Written reviews from 1 PRW were collected and categorized. Our representative sample included 509 (91.38%) male and 48 (8.62%) female orthopaedic surgeons from 30 of the most populated cities in the United States. Of those, 37 (88.10%) male and 5 (11.90%) female were members of the J. Robert Gladden Society. Overall, 76.12% of physicians had positive ratings (60% or greater). Statistically higher ratings for J Robert Gladden members (p=0.0066) and academic physicians (p=0.0066) were noted. A statistically higher number of ratings for private practice physicians (p=0.03) were also found. Most orthopaedic surgeons are rated on at least 1 PRW and most ratings and reviews are positive. However, composite scores are typically based on a small number and can be volatile.
PEDIATRICS

Poster#51

PARENTAL ATTITUDES TOWARDS INFLUENZA VACCINATION FOR CHILDREN IN INDIA. Chethan Ramprasad1, Rajeev Zachariah2, Mark Steinhoff3, Anna Simon2. 1 University of Miami Miller School of Medicine, Miami, FL. 2 Christian Medical College, Vellore, Tamil Nadu, India. 3 Cincinnati Children’s Hospital Medical Center, Cincinnati, OH.

The rate of influenza vaccination is low for children in India despite evidence from North America and elsewhere of its effectiveness. The purpose of this study is to assess parental attitudes towards influenza vaccination in South India. Participants were parents who brought their children to the Well Baby Clinic of Christian Medical College Hospital, Vellore, India for routine immunization. Participants answered questions by written survey while waiting for their child’s vaccination. A total of 456 surveys were completed (403 parents did not opt for trivalent influenza vaccination and 53 opted for influenza vaccination). The majority (58.56%) of those parents who did not accept influenza vaccination identified the lack of a doctor’s recommendation as the main reason. When asked separately, many non-acceptors (49.88%) indicated that they did not believe or were not sure that the influenza vaccine was effective. Nearly all non-acceptors (92.63%) stated that they would opt for influenza vaccination if a doctor recommended it. The most common reason cited by parents for not opting for influenza vaccination for their children was the lack of recommendation by a doctor. The results of this study suggest that recommendation by a doctor is a more important factor than belief in efficacy, cost, or convenience in parental decision-making regarding childhood influenza vaccination in India, unlike the United States where parents are less likely to follow recommendations.

PHARMACOLOGY

Poster #52

ASYMMETRIC SYNTHESIS OF AMITIFADINE: A NEW APPROACH VIA METALLORADICAL CATALYSIS. Jennifer Le, Theresa Schwitalla, Xin Cui, and X. Peter Zhang. Department of Chemistry, University of South Florida, Tampa, FL.

Azabicyclo[3.1.0]hexanes represent a diverse group of bioactive molecules that have been investigated for their antitumor and psychoactive effects. Among these compounds is Amitifadine, a triple reuptake inhibitor of serotonin, norepinephrine, and dopamine that is currently in clinical trials as a potential treatment for major depressive disorder. Preliminary results indicate that Amitifadine is well-tolerated and may decrease symptoms to a greater extent than currently available selective serotonin reuptake inhibitors. An efficient asymmetric synthetic method to access this drug and its analogs would be highly desirable from a pharmaceutical and research standpoint. The current synthesis of this drug developed by Merck relies on chiral starting materials and still suffers stereoselectivity issues. Utilizing intermolecular cobalt(II) D2-symmetric amidoporphyrin catalyzed cyclopropanation of α-arylacrylonitrile with a diazo reagent, we have developed a four-step asymmetric synthesis for Amitifadine which starts with readily available achiral starting materials. With this method, we can achieve up to 95% enantiomeric excess and 97.3 diastereomeric ratio for the key cyclopropane intermediate, surpassing the current synthesis. In addition, this work is the first report of asymmetric cyclopropanation of an α-arylacrylonitrile with diazoacetates to form highly polarized 2-cyanocyclopropanecarboxylates. The cyanocyclopropanecarboxylate can be subsequently reduced to the corresponding amino alcohol, followed by chlorination and cyclodehydration to afford the target compound with retention of stereochemistry. The high catalytic selectivity afforded by the Co(II) porphyrin catalyst, even at a sterically hindered position, demonstrates the power of the emerging field of metalloradical catalysis and its potential use in drug development.
STATINS CAN MODULATE IN VITRO INFECTION WITH VESICULAR STOMATITIS VIRUS (VSV) IN TRANSFORMED AND NORMAL CELLS. Varun Soti, Dayron Miranda, Paula Waziri, Luigi Cubeddu and Ana Maria Castejon. College of Pharmacy, Nova Southeastern University, Fort Lauderdale, FL.

Vesicular Stomatitis Virus (VSV), is an RNA virus that has been extensively used as a laboratory tool for the study of cellular physiology. During the last two decades VSV has been gaining attention as an oncolytic virus due to its relatively selective capacity to target tumor cells. Thus VSV has emerged as a relatively innocuous agent that can be engineered to target a wide spectrum of cancer cells without affecting normal cells. However, there is always a risk associated with the use of infectious agents as therapeutics, especially in vulnerable patients, such as cancer patients whose immune systems are found very often compromised. This study has found that in vitro VSV infection of both, normal and cancer cells can be modulate by using statins, the cholesterol-lowering drugs that have been reported to have anti-viral activities. In order to evaluate such effect of statins on VSV infection, HeLa cells and normal human fibroblasts BJ cells were pre-treated (2-24 hours) with different concentrations of Simvastatin and Pravastatin ranging from 0.1 to 4.0 µM per mL, prior to infection with a GFP-tagged Vesicular Stomatitis Virus (VSV-GFP) at different multiplicity of infection (MOI) ranging from 0.0001 to 0.1 MOI. After 16 hours postinfection, cells were analyzed by flow Cytometry for detecting 1) infection rate (GFP expression), 2) apoptosis rate (Annexin V staining) and 3) necrosis (7-AAD staining). The results showed that, at a lower concentration (~0.125 µM), Simvastatin not pravastatin, enhanced viral infection (high viral infection rates, increased cellular apoptosis/necrosis). In contrast, at higher concentrations (~0.25 µM or higher) Simvastatin not pravastatin, inhibited viral replication. Since treatment with statins or viral infection themselves can induce apoptosis/necrosis, a dose-response curves were performed in order to determine at what extent apoptosis/necrosis rates were due just to the treatment. The results of present study open new routes for the treatment of human cancers using viruses, such as VSV.

Radiation Oncology

STEREOTACTIC RADIOSURGERY FOR LARGE BRAIN METASTASES: IMPACT OF PATIENT AND TREATMENT VARIABLES ON OUTCOMES. Daniel Ebner, Paul Rava, Deus Cielo, Jaroslaw T. Hepel. 1Alpert Medical School of Brown University, Providence, RI. 2Department of Radiation Oncology, University of Massachusetts Memorial Medical Center, Worcester, MA. 3Department of Neurosurgery, Rhode Island Hospital, Providence, RI. 4Department of Radiation Oncology, Rhode Island Hospital, Providence, RI.

Radiosurgery is an established and effective treatment modality for limited brain metastases. However, large tumors are at increased risk for failure as well as treatment related complications with this approach. The aim of the present study is to evaluate the outcomes of stereotactic radiosurgery (SRS) for patients with large brain metastases. Over 500 patients with brain metastases were treated with SRS at our institution from 2001 to 2012. Only patients with brain metastases ≥3 cm, Karnofsky Performance Status (KPS) ≥70%, and follow-up of at least 3 months were considered for analysis. SRS was performed using the Gamma Knife model 4C. Local control, survival, and rate of radiation necrosis were evaluated. Univariate analysis was performed to determine the influence of tumor, patient, and treatment variables on outcomes. Eighty-five patients, consisting of 52 females and 33 males, were identified for analysis. The most common primary diagnoses were non-small cell lung cancer (48%), breast cancer (18%), and melanoma (13%). Tumor size was 3 to <3.5cm, 3.5 to <4cm, and ≥4cm in 29%, 32%, and 39% of patients, respectively. Surgery was combined with SRS in 56 patients. With a median follow up time of 22 months, the actuarial local control at 1 year was 72%. Univariate analysis did not demonstrate a correlation between local failure and surgery (p=0.887), tumor size (p=0.797), histology (p=0.969), or radiation dose (p=0.237). Overall survival at 1 year was 45%. Prolonged survival was seen in patients with age < 65 years (median: 16 vs. 8 months, p=0.019), primary treatment vs inclusion of WBRT salvage (median: 10 vs. 7 months, p=0.044), radiation dose >16 Gy (median: 11 vs. 7 months, p=0.016), and controlled primary tumor (median: 23 vs. 8 months, p=0.024). Eleven patients developed radiation necrosis. For a subset of patients with large brain metastases, long-term local disease control and survival can be achieved with SRS. Local failure, however, still remains suboptimal. Strategies to improved outcomes in this subgroup of patients are needed.
**Radiology**

**DEVELOPMENT OF TECHNIQUES TO NAVIGATE THE HUMAN CERVICAL SPINAL CORD.** Adam Cadotte, David Cadotte, Julien Cohen-Adad, Micha Livne, David Fleet, Jefferson R. Wilson, David Mikulis, Natalia Nugaeva and Michael G. Fehlings. CREMS Research Program, University of Toronto, Toronto Western Hospital, Division of Neurosurgery, Toronto, ON.

Current methods to analyze spinal cord MRI data use vertebral body (VB) locations to infer the position of nerve rootlets (NR). This assumes that the relative distance between the NR and the VB is consistent across individuals; recent studies suggest this is not the case. This research aims to create the tools required to measure distances along the spinal cord axis, to build a predictive model for NR location, and to measure spinal cord volume/area. Using T-2 weighted MRI images from 20 healthy volunteers (3T GE MR), two Neurosurgery residents identified the C3 to T1 VB and C3 to C8 NR positions using 3D Slicer. Data from 4 individuals was manually segmented to identify the characteristics of the spinal cord/CSF boundary. Using these ground-truth data, a template matching algorithm was designed to locate the spinal cord / CSF boundary for non-segmented images. A ‘spinal cord center line’ was generated to measure the arc-length distance to each of the VBs and NRs. The variability of these distances was determined and used to create a predictive model to estimate NR locations based on VB location, neck length, height, and a constant. We determined that using standard procedures, if one were to assume that a given spinal cord segment (eg, C5) is 1 VB length rostral to the corresponding VB (eg, C5), then one would capture only 44% of the corresponding spinal segment. In contrast, using linear regression techniques, we found that the spinal cord segments could be predicted with an average root mean square error of 3.39 mm. The tools created to measure distance along the cervical spinal cord have demonstrated that previous methods of identifying spinal cord segments are not accurate. We have shown that building a predictive model based on accurate measurements can be used to inform future MRI studies with a significantly increased degree of accuracy. This work will set the stage to develop an atlas of the human spinal cord to enhance assessments by high resolution MRI.

**Surgery**

**MINIMALIST APPROACH TO AORTIC VALVE REPLACEMENT FOR SEVERE AORTIC STENOSIS IN 68 EXTREME RISK PATIENTS: EARLY LEARNING PHASE OF COREVALVE TAVR.** Erin R. Cohen, Sydney Pomenti, Shawn Simek, Eduardo J. de Marchena. International Medicine Institute, University of Miami Miller School of Medicine, Miami, FL 33101.

Transcatheter aortic valve replacement (TAVR) is emerging as a novel therapy for severe aortic stenosis (AS). Prognosis of untreated symptomatic AS is poor, with a one-year mortality up to 50%. Prior management was limited to surgical aortic valve replacement (SAVR); however, patients often faced increased perioperative risks and did not qualify for surgical intervention. We report our single-center data of the first 68 extreme risk patients who underwent TAVR with the Medtronic CoreValve at University of Miami. Extreme risk patients include those with an estimated mortality of less than one year based on previous survival of demographics. Through a retrospective analysis, this study aims to explore the efficacy and outcomes of TAVR at a center in its early learning phase. In this cohort, with an average age of 84.6 ± 8.0, one-month mortality was 2.9%, and one-year mortality was 17.9% (n=56). The average procedural time was 1:15:36 ± 40:07 with an average use of 148 ± 78.2 mL of contrast. Length of hospital stay post-procedure prior to discharge was 6.8 ± 3.2 days. In contrast to SAVR where the majority of patients undergo general anesthesia, 82.4% of this group had conscious sedation during the procedure. The transthoracic echocardiogram (TTE) mean aortic gradient dropped from 49.2 ± 10.3 mmHg at screening to 8.6 ± 5.6 mmHg at one-month post TAVR. Serious adverse events included implantation of permanent pacemaker in 36.8%, conversion to SAVR in 1.5%, stroke in 8.8%, major bleeding in 5.9%, major vascular complications 1.5%, and acute kidney injury in 4.4%. Even in an extreme risk population, TAVR can be accomplished at a newly trained single-center that is well equipped for the management of serious complications. The data presented here provides support for expansion of TAVR as an important treatment option, and further evaluation should be given to patients with lower surgical risk.
Poster #57

CLINICAL UTILITY OF SERUM VERSUS CSF TUMOR MARKERS IN PATIENTS WITH CNS GERM CELL TUMORS. Arvind Krishnan BA1, Edward Melamed BA1, Mark D. Krieger MD1 Division of Neurosurgery, Children’s Hospital Los Angeles, Los Angeles, CA. Department of Neurological Surgery, Keck School of Medicine, University of Southern California, Los Angeles, CA.

The use of Alpha-fetoprotein (AFP) and Beta Human-chorionic gonadotropin (B-HCG) is well established in the management of patients with CNS germ cell tumors. This study aims to further characterize the predictive value of these tests in determining CNS germ cell tumor (GCT) histology. Retrospective review of 56 pediatric patients with CNS GCTs treated at the Children’s Hospital Los Angeles from 1993-2014. All patients were diagnosed via histologic examination upon biopsy. Serum and CSF AFP and B-HCG tumor marker results were recorded and analyzed. 100% (n = 10, p < 0.0001) of patients who had a serum AFP result above 4 ng/mL were diagnosed with non-germinomatous GCTs. For 100% (n = 3, p < 0.0001) of patients, serum B-HCG values above 150 mlu/mL predicted a non-germinoma diagnosis. 100% (n = 12, p < 0.0001) of patients who had a CSF AFP result above 5 ng/mL were diagnosed with non-germinomatous GCTs. For patients with a CSF B-HCG value above 150 ng/mL, 89% (n = 9, p = 0.0013) were found to have non-germinomatous GCTs. Serum and CSF AFP tumor marker tests are equally sensitive in predicting a non-germinatous GCT diagnosis. Similarly, serum and CSF B-HCG are also equally sensitive above the same threshold value of 150 mlu/mL. In the cases where values within these ranges are obtained from a blood test, CSF draws can be eliminated. Additionally, these threshold values can be implemented to help clinicians better interpret lab results and subsequently expedite care for patients with CNS germ cell tumors.

Poster #58

A DECADE OF WOMEN IN GENERAL SURGERY: A SINGLE INSTITUTION’S EXPERIENCE. Andrea R. Marcadis, Zahra F. Khan, Tanya Spencer, Laura F. Teisch, Nicole S. Mandel, John I. Lew, M.D., F.A.C.S Department of Surgery, University of Miami Leonard M. Miller School of Medicine, Miami, FL.

Although General Surgery has historically been a male-dominated field, a growing proportion of women in medical school are choosing to pursue a career in Surgery. This study determines the current status of the University of Miami Miller School of Medicine (UMMSM) in regards to the rates of women in medical school pursuing careers in Surgery, and in this institution’s General Surgery residency program. A retrospective review of the National Resident Matching Program match lists from UMMSM over the past 10 years (2005-2014) was undertaken. The percentage of women that matched into General Surgery residencies was calculated for each year. Only students that matched into categorical General Surgery positions were included. Additionally, housestaff rosters from UMMSM over the past 10 years were analyzed, and the percentage of General Surgery residents that were women was calculated for each year and compared to the national average. Over the past decade, 45.3% of UMMSM medical students that matched into categorical General Surgery residencies were women. In 5 of 10 years, the number of women who matched into General Surgery exceeded that of men (55.6% women in 2006, 57.1% in 2007, 70.0% in 2008, 54.5% in 2010, and 54.5% in 2014). While UMMSM’s General Surgery residency program fell below the national average with regards to women enrolled (22.9% vs 33.0%), the latter half of the decade had a statistically significant increase in percentage of women residents as compared to the earlier half-decade (25.9% vs 19.4%, p<0.05). Both UMMSM medical school graduating classes and its General Surgery residency program have paralleled the national trend of increasing rates of women pursuing careers in General Surgery. UMMSM encourages other institutions to evaluate their undergraduate and graduate Surgery programs to ensure that they are attracting the best and the brightest, regardless of gender.
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