2011

1st Pediatric Scientific Research Retreat

"Pediatric Research: From Bench to Bedside"

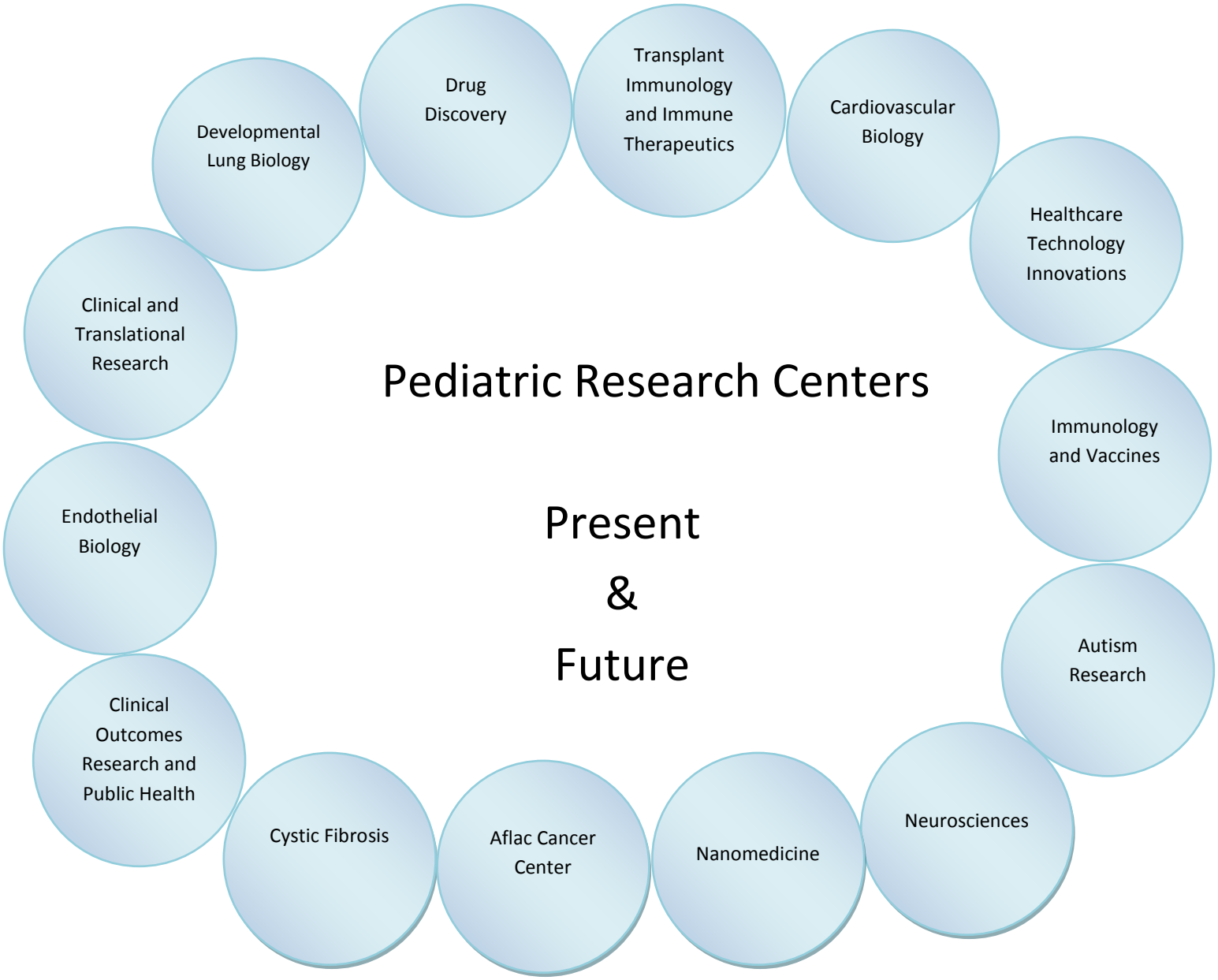
Friday, January 28, 2011

8:00 AM–5:30 PM

The Loudermilk Center

Atlanta Conference Center

40 Courtland Street NE, Atlanta, Georgia 30303



Research is a cornerstone of our mission to enhance the lives of children. Children's Healthcare of Atlanta, Emory University School of Medicine, Georgia Institute of Technology and Morehouse, seeks answers to the most challenging childhood medical conditions through teaching and research. By constantly developing new techniques, treatments and cures, we are committed to advancing pediatric research and medicine through the Pediatric Research Centers.

Children's Healthcare of Atlanta | EMORY ROBERT W. WOODRUFF HEALTH SCIENCES CENTER | Georgia Institute of Technology | MOREHOUSE SCHOOL OF MEDICINE

Agenda

8:00 – 9:00	*Registration open (Continental Breakfast available)*
9:00 – 9:15	Welcome and Overview of the Retreat (Barbara Stoll, Donna Hyland)
9:15 – 9:30	Update on Children's Pediatric Research Strategic Plan, Vision for future (Paul Spearman)
9:30 – 11:00	Introduction to the Children's Research Centers: Aflac Cancer Center & Blood Disorders Service Center for Cystic Fibrosis Research Center for Developmental Lung Biology Center for Drug Discovery Center for Endothelial Biology Center for Immunology and Vaccines Center for Cardiovascular Biology ***Coffee Break (15 minutes)*** Center for Pediatric Healthcare Technology Innovation Children's Transplant Immunology and Immune Therapeutics Center Center for Pediatric Nanotechnology Center for Autism Research Center for Clinical Outcomes Research & Public Health *Coming soon*: Center for Clinical and Translational Research
11:00 – 11:45	Scientific Focus I: Cystic Fibrosis
11:45 – 1:30	Lunch, Roundtable Discussions*, and Poster Session ***Lunch will run concurrent with the roundtable discussions and poster session.***
1:30 – 2:00	Awards ceremony for best posters: faculty and/or physician, postdoc/fellow, and student categories
2:00 – 2:30	Selected brief talks (chosen from submitted abstracts)
2:30 – 2:45	***Coffee break***
2:45 – 3:30	Scientific Focus II: Aflac Hematology/Oncology
3:30 – 4:00	Panel discussion on plans for the Children's Pediatric Research Strategic Plan, including the new building, areas of growth, need for new cores, ways to better connect institutions, etc. **Panel: Paul Spearman, Barbara Stoll, Beth Howell (CHOA), Beatrice Gee (Morehouse), Steve Cross (GA Tech)**
4:00	Wrap-up and adjournment
4:00 – 5:30	*Research networking, informal discussions*

**Based on registrants' collective interests, the selected Lunchtime Roundtable Discussion topics are 1) Genetics/Genomics; 2) Mucosal Immunology & GI Disease; 3) Nanotechnology. Those who indicated these topics as their top choice have already been notified of their invitation to participate. For those who did not receive an e-mail, we apologize for not being able to accommodate all discussions during the retreat. All other topics will, however, be scheduled for future Pediatric Research Center brainstorming sessions so stay tuned for more details on that. There may be space available for the three designated sessions. If you are interested in reserving a space, please contact Stacy Heilman. If you have questions or comments about the retreat's Lunchtime Roundtable Discussions, please contact stacy.heilman@emory.edu*

Loudermilk Center Floor Plan

Genetics/Genomics Roundtable

Nanotechnology Roundtable

GI & Mucosal Immunology Roundtable

Abstracts

1

SYMPTOMATIC AND ASYMPTOMATIC INFECTIONS OF ROTAVIRUS, NOROVIRUS, AND ADENOVIRUS AMONG HOSPITALIZED CHILDREN IN XI'AN, CHINA

Pengbo Liu[a, b], Tsun-Hsuan Chen[a,], Juan Wang[c], Changxing Dong[a], Shuwan Zhang[b], Jinjin Pan[b], Lihong Yang[b], Helen Tang[a], Wei Chen[b], Marina Fernandez[a], Christine Moe[a]
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Rotavirus (RV), norovirus (NoV), and adenovirus (AdV) have been reported as common viral pathogens of acute gastroenteritis in children. To determine the prevalence and epidemiological characteristics of RV, NoV, and AdV infections among hospitalized children with and without symptoms of acute gastroenteritis, fecal specimens and data on clinical symptoms were collected from 201 children with diarrhea and 53 children without diarrhea admitted to the Xi'an Children's Hospital in Xi'an, China between March 2009 and May 2010. RV, NoV, and AdV were identified in 68.7% (138/201), 20.4% (41/201), and 5.0% (10/201), respectively, of children with diarrhea. These three viruses were also detected in 13.2% (7/53), 35.9% (19/53), and 9.4% (6/53), respectively, of children without diarrhea (asymptomatic infections). Symptomatic children infected with RV alone had an average severity score of 6.5 that was significantly higher than the average score of 5.3 in children with no detected viruses ($p<0.05$). GII.3 and GII.4 were the only two NoV genotypes identified, and the GII.4 sequences were genetically close to GII.4 2006b cluster. These findings highlight the continued importance of rotavirus as a cause of severe pediatric gastroenteritis in China and the need to initiate rotavirus vaccination. The high norovirus infection rates, especially among asymptomatic children, indicate that this is an important pediatric pathogen and that children may play an important role in spreading this virus.

2

Treatment With Anti-CD137 (41BB) mAb Enhances Peptide Vaccination Against Respiratory Syncytial Virus in Aged Mice

Sujin Lee[1,2], Robert Mittler[3], and Martin Moore[1,2]

[1]Department of Pediatrics, Emory University, [2]Children's Healthcare of Atlanta, Atlanta, [3]Department of Surgery, Emory Vaccine Center, GA,

Respiratory syncytial virus (RSV) is the most important pathogen for lower respiratory tract illness (LRI) in infants and also a major cause of morbidity and mortality in the elderly. Using an aged mouse model of RSV pathogenesis, we found that aged mice had impaired antigen-specific CD8 T cell responses and delayed RSV clearance compared to young mice. We generated a peptide vaccine approach, TriVax, which is a co-mixture of a peptide representing an immunodominant RSV CD8 T cell epitope, a Toll-like receptor agonist, and a costimulatory anti-CD40 antibody. TriVax vaccination generated robust CD8 T cell responses and had a protective effect against RSV challenge in young BALB/c mice but not in aged BALB/c mice. We hypothesized that treatment of aged mice with agonistic CD137 (41BB) monoclonal antibody would stimulate T cells and enhance TriVax efficacy to RSV challenge in aged mice. We immunized 18-month old BALB/c mice twice with TriVax + anti-CD137 mAb. We found that co-administration of anti-CD137 mAb with TriVax enhanced antigen-specific CD8 T cell responses as well as showed a protective effect after RSV challenge. Thus, anti-CD137 mAb treatment rescued the deficient response to RSV TriVax vaccination in aged mice. The 41BB co-stimulatory pathway may be a target for enhancing T cell responses in the elderly.

3

ELEVATED NGAL LEVELS CONTRIBUTE TO DYSFUNCTIONAL INNATE IMMUNITY IN CYSTIC FIBROSIS

Susu M. Zughaier, Dimple Dhakal, Vin Tangpricha, Nael A. McCarty

Children's Healthcare of Atlanta Center for Cystic Fibrosis Research, *Emory University School of Medicine, Atlanta GA*

Background: Cystic fibrosis (CF) patients develop chronic bacterial infections reflecting a dysfunctional innate immunity. Neutrophil gelatinase-associated lipocalin (NGAL) functions as a cell growth and differentiation factor by donating iron to cells. NGAL also mediates an innate immune response to bacterial infection by scavenging bacterial iron-chelating siderophores. The aim of this study was to determine NGAL levels in serum from CF subjects compared to healthy donors and the effect of circulating NGAL on macrophage innate immune responses. **Methods:** NGAL levels were measured by ELISA method in sera from 33 CF subjects, 33 septic patients and 21 healthy controls. **Results**: Serum NGAL levels were highly elevated in CF patients when compared to healthy controls (p<0.0001). The mean serum NGAL level measured in CF was found to be 79.7 ± 24.6 ng/ml (range 40 – 135 ng/ml), compared to septic patients 130.7 ± 34.68 ng/ml (range 68 – 211 ng/ml), or healthy donors controls 47.93 ± 11.5 ng/ml (range 22 - 66 ng/ml). We then investigated the effect of elevated NGAL on macrophage innate immune function using THP-1 human macrophage-like cells treated with recombinant NGAL protein (100 ng/ml) overnight prior to stimulation with *Pseudomonas aeruginosa* LPS (10 pmole/ml). We found that NGAL suppressed LPS-induced cytokine production in THP-1 macrophages as demonstrated by decreased production of the inflammatory cytokine TNF□ and inhibition of the pro-inflammatory chemokine IP-10 (CXCL10) release. **Conclusions:** Elevated NGAL levels contribute to dysfunctional innate immunity in CF and NGAL could be a biomarker of progressive CF disease.

4

A FAILURE TO SCAVENGE CIRCULATING HEME CAUSES ACUTE LUNG INJURY AND SUDDEN DEATH

Solomon F. Ofori-Acquah, PhD. Center for Endothelial Biology, Aflac Cancer Center and Blood Disorders Service.

Heme is an iron-containing porphyrin that serves as prosthetic group for the respiratory protein hemoglobin, multiple enzymes and transcription factors that regulate circadian rhythm. It is rapidly removed from the circulation by plasma proteins consequently the blood is normally clear of protein-free heme. There are currently no human diseases described that are due to the body's failure to scavenge circulating heme. The plasma concentration of hemopexin the major heme scavenger is reduced in children with hemolytic disorders such as malaria, sickle cell anemia and porphyria cutanea tarda, however this relative deficiency has not previously been associated with life threatening conditions. In this study we tested the hypothesis that modest elevation of circulating heme would trigger acute lung injury (ALI) in hemolytic disorders. Here, we characterized the pulmonary responses induced by intravenously administered heme in anemic, and non-anemic mice, and in mice given recombinant hemopexin. Two models of transgenic sickle cell anemia mice developed ALI and succumbed to a low-dose of heme while multiple strains of non-anemic mice were protected from a medium-dose. Heme-induced ALI correlated with an increase in the concentration of protein-free plasma heme but not total-plasma heme highlighting a receptor mediated mechanism in this disease process. Recombinant hemopexin protected transgenic sickle cell anemia mice from ALI revealing a previously unknown risk associated with low-plasma hemopexin level. This study has identified a novel disease mechanism involving protein-free heme that may explain ALI secondary to severe hemolytic disorders. Initial preclinical data support the use of hemopexin to prevent heme-induced ALI and reduce mortality due to hemolytic crises.

5

Selling Sprinkles Reduces Anemia and Iron Deficiency among Young Children in Western Kenya

Parminder S. Suchdev[1,2] , Laird Ruth[2], Bradley A. Woodruff[2], Charles F. Mbakaya[3], Usha Mandava[2], Rafael Flores[2,] Maria Elena D. Jefferds[2,] Robert Quick[4]

1. Emory University, Department of Pediatrics, School of Medicine, Atlanta, GA
2. Nutrition Branch, Centers for Disease Control and Prevention, Atlanta, GA
3. Kenya Medical Research Institute, Nairobi, Kenya
4. Waterborne Diseases Prevention Branch, CDC, Atlanta, GA, USA

Background: Despite the established efficacy of micronutrient powders (e.g., Sprinkles™) for anemia reduction, few effectiveness studies have been performed to assess the impact of Sprinkles in real-world programs. In 2007, CDC and local Kenyan institutions evaluated the marketing and community-based distribution of Sprinkles.

Methods: We conducted a cluster-randomized trial in children aged 6-35 months in western Kenya. Sixty villages were randomly assigned to either intervention or control groups. In intervention villages, community vendors marketed and sold health products, including Sprinkles. In control communities, Sprinkles were neither promoted nor marketed. Biweekly household visits monitored Sprinkles use. Hemoglobin, iron indicators, and anthropometry were measured at baseline (n=1063) and at 12-month follow-up (n=862 children 18-47 months). Data were analyzed intention-to-treat, using generalized linear mixed models, accounting for cluster sampling. This trial is registered with ClinicalTrials.gov, number NCT01088958.

Findings: On average 33% of households in intervention villages purchased Sprinkles. The average weekly intake per child was 0.9 sachets (~11.3 mg iron). Compared to controls, intervention children had a 0.3 g/dL greater rise in hemoglobin and a 14% point greater decline in iron deficiency ($p<.05$). Anemia was cured in 53% of children in intervention villages. With adjustment for age, sex, socioeconomic status and maternal education, children who consumed more sachets had higher hemoglobin and better iron status ($p<.05$). No change in malaria prevalence, wasting or stunting was observed in either group.

Interpretation: Even with low and infrequent use, Sprinkles sales through community vendors are effective in reducing anemia and iron deficiency in a resource-poor setting.

6

Acceptance and Commitment Therapy for Pediatric Sickle Cell Disease

Lindsey L. Cohen, Ph.D., Aki Masuda, & Alcuin Johnson

Pediatric sickle cell disease (SCD) is a genetic chronic condition involving a number of debilitating symptoms. Patients report that the most frequent and interfering aspects of SCD are the acute pain episodes and chronic pain. SCD pain is related to impairments in academic, social, family, emotional, and physical realms. Adolescents with SCD have additional stressors related to growing independence and the transition of medical care from their parents to themselves. Parents of these adolescents report high stress and adjustment issues related to parenting in general and especially around SCD issues. Acceptance and Commitment Therapy (ACT) – a behavioral treatment encouraging willing acceptance of unchangeable experiences (e.g., pain, fears) when doing so facilitates committing to values-based action (e.g., attending school, interacting with peers) – can help both adolescents with SCD and their parents live more vital and fulfilling lives. Adolescents with SCD and their parents received ACT and completed measures of pain, acceptance, and quality of life prior to and following therapy. Results indicate that ACT is a viable intervention for adolescents with SCD-related difficulties and their parents. In addition, findings suggest that ACT might be effective for other adolescents struggling with chronic medical conditions and their parents.

7

Rare Sequence Variants at the X-linked *FMR1/AFF2* Contribute to Male Autism Susceptibility

Kajari Mondal, Katie Hagen, Amol. C. Shetty, Viren Patel, and Michael. E. Zwick

Department of Human Genetics, Emory University School of Medicine, Atlanta, GA

Autism spectrum disorders (ASDs) exhibit a high heritability and affect four times as many males as females. To test the hypothesis that X-linked rare variants contribute to this pattern, we comprehensively sequenced the *FMR1* and *AFF2* genomic region in 127 male ASD probands from the Autism Genetic Resource Exchange (AGRE) collection. Probands were selected from multiplex families with two or more affected males who share the Xq chromosomal arm, which includes FMR1 and AFF2. A total of 607 variants were observed, including 449 single base variants (SBVs) and 158 insertions/deletions (Indels). While the average level of variation (theta = 0.00059) agreed with the chr X expectation, an excess of rare variants was observed (Tajima's D = -1.44). Of 449 SBVs, 2 are exonic replacements in *AFF2*, 6 are exonic silent variants, 18 are in the untranslated regions (UTRs) and 423 are intronic. All of the 158 indels lie in introns. The AFF2 missense mutations (D714N, R927H) each affect a single proband and occur at highly conserved sites predicted to be highly damaging by Panther and SIFT. A third AFF2 misssense mutation (R837C) that is also predicted to be deleterious was identified in an ASD proband from the Simons Simplex Collection. A recent study that sequenced the chr X exome of 208 individuals with X-linked intellectual disability (Tarpey et al, Nat Gen, 41:535-43, 2009) identified two additional AFF2 missense mutations (H612D, P886A). In total, these data suggest that up to 1.5% (5/339) of male developmental delay (ASD or intellectual disability) could result from coding sequence mutations at the *AFF2* locus. Of the 18 3'UTR variants at *FMR1* and *AFF2,* 6 are found at highly conserved sites, do not exist in dbSNP, and could also act as ASD susceptibility loci. To interrogate the functional effects of these variants, we will present the results of ChIP-Seq experiments for the AFF2 missense mutations and luciferase assays for the UTR variants. Our main finding, that rare DNA sequence in *FMR1/AFF2* genomic region may contribute to ASD susceptibility, can help explain a portion of the ASD male excess and helps elucidate the research paradigm for assessing the common disease-rare variant model for complex human disorders.

8

Role of inflammation and oxidant stress in the development of cystic fibrosis related diabetes

Yue Luo, Arlene Stecenko and Greg Gibson

Cystic Fibrosis Related Diabetes (CFRD) is a common non-pulmonary complication that afflicts up to half of all CF patients, across the United States, by the age of 20. Genetic variation, oxidative stress, and inflammation are all thought to contribute to the exacerbated decline in lung function in CFRD relative to CF patients, but the relationship of these factors to the onset of diabetes is unclear.
We hypothesize that the accumulation of oxidative stress related to hyperglycemia and cystic fibrosis contributes to the onset of CFRD, and have initiated functional genomic approaches to test this. Neutrophils have been extracted from peripheral blood and induced sputum samples for 26 participants to date, and whole transcriptome gene expression profiles will be generated using Illumina HT12 microarrays. We will evaluate (i) whether CF neutrophils show evidence of oxidative stress that may contribute to the altered immunological competence, and (ii) whether there is an additional signature of a transition to glucose intolerance that is characteristic of type 2 diabetes patients. In parallel, measurements of systemic and respiratory tract redox status and inflammatory measures will be correlated with the degree of glucose intolerance in CF, CFRD, and type 2 diabetes patients as well as healthy controls.

Understanding the gene expression profiles of CFRD and CF patients will provide insights into the mechanism of CFRD and potentially inform treatment options for CF patients with glucose intolerance.

9

Determining Changes in Contractility and Myofilament Expression in the Developing Human Ventricle

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As pediatric cardiac surgery is increasingly performed in the first year of life, understanding contractility regulation in the developing human heart is increasingly important. We examined contractility and myofilament changes in ventricular biopsies removed as part of the surgical correction of congenital heart defects from newborns (hypoplastic left heart syndrom, < 1 wk old) and infants (tetralogy of Fallot, 3-12 mo old). Isometric developed force was measured in ventricular strips in response to forskolin which maximizes contractility. In newborn, maximum contractility was significantly smaller in the newborn (224±28%) than in the infant (404±77%, $p<0.05$). We investigated isoform changes in myofilament associated proteins. Cardiac TroponinI (cTnI) and slow skeletal TnI (ssTnI) were differentially expressed. Total TnI (cTnI+ssTnI) was not different between the ages (263±5a.u., newborns vs. 247±23a.u., infants). We found that the newborns had a significantly lower level of cTnI compared to the infants (116±8 a.u. vs. 218±17 a.u., $p<0.001$). The percent ssTnI in the newborn group was significantly greater than in the infant group (56.2±2.8% vs 11.0±4.6%, $p<0.05$). Analysis of RT-PCR revealed nearly significant lower levels of TnT isoform 1 in the infant group than the newborn group ($p= 0.078$), no significant difference in the levels of TnT isoform 2 ($p= 0.536$), and significantly higher levels of TnT isoform 3 in the infant group than the newborn group ($p= 0.039$). These results collectively suggest that contractility and myofilament changes, or the lack of changes, may serve as targets for clarifying elements associated with cardiac dysfunction in the developing human ventricle.

10

Psychosocial predictors of opposition to transition to adult care in adolescent renal transplant recipients

Jordan Gilleland, PhD[1], Megan McCormick, MS[3], Laura Mee, PhD[1], Sandra Amaral, MD, MHS[1], and Rochelle Schmidt, PharmD[2]

Emory University School of Medicine and Children's Healthcare of Atlanta[1]; Children's Healthcare of Atlanta[2]; University of Georgia[3]

Pediatric transplant patients are living longer due to improved allograft survival. As a result, these patients are maturing into young adults who enter adult healthcare systems. The transition of care from pediatric to adult care providers is a growing focus in research. Participants included 58 renal transplant recipients ages 14-21 who were taking part in a transition-focused clinic. Teens reported on medication adherence, opposition to transition, regimen responsibility, and psychological functioning.

Associations among adolescent opposition to transition, demographic and medical factors, self-reported medication adherence, and psychological functioning were examined using correlational analyses. Opposition to transition was not significantly related to age, renal function, or self reported adherence. Opposition to transition was significantly related to decreased teen medication regimen responsibility ($r= -.30$, $p=.021$), poorer psychological functioning ($r= -.28$, $p=.028$), and living with more than one parent or guardian ($r=.29$, $p=.022$). Using a hierarchical regression model, teen medication regimen responsibility accounted for 10.3% of the variance in opposition to transition, with psychological functioning and multiple care providers adding significant 6.7% and 9.2% increments, respectively. Taken together, these psychosocial factors accounted for 26.3% of the variance in teen opposition to transition.

Overall, teens who took greater responsibility for their medication taking, reported greater levels of psychological adjustment, and lived with a single care provider reported significantly less opposition to transitioning care. The data suggest that transition programming aimed at increasing adolescent healthcare responsibility, decreasing parental over-involvement in medication taking, and encouraging psychological well-being are important to ensure successful transition to adult focused care.

Plemper Laboratory Research Projects

Richard K Plemper

Structure-function analysis of native paramyxovirus envelope glycoprotein complexes

Protein-mediated membrane fusion at neutral pH is employed by a variety of viral families to gain cell entry and is essential for eukaryotic cell organization. Members of the paramyxovirus family typically rely on the concerted action of two envelope glycoproteins to achieve membrane fusion for viral entry. Despite their major clinical importance, many of the mechanistic principles that govern the organization of metastable paramyxovirus fusion complexes and their coordinated refolding remain poorly understood. Towards the overall goal of elucidating these principles, the Plemper lab studies the envelope proteins of measles virus, an archetype of the paramyxovirus family, to address three fundamental questions: What is the spatial organization of viral glycoprotein hetero-oligomer complexes in the native, metastable prefusion conformation displayed on infectious particles? How does receptor binding affect this organization? What is the nature of the intermolecular contacts that link receptor binding with refolding of the complexes into the stable postfusion conformation? The lab applies molecular virology, reverse genetics biochemistry, microchemistry, imaging and molecular modeling techniques in a comprehensive approach.

Small-molecule inhibitors of the paramyxovirus RNA-dependent RNA-polymerase

It is the overarching goal of this project to develop small-molecule blockers of the paramyxovirus RNA-dependent RNA-polymerase (RdRp) complex. Through structure-guided drug design and high-throughput screening, we have identified a first-in-class non-nucleoside inhibitor of the paramyxovirus RdRp. The lead compound shows good oral bioavailability and *in vivo* half-life in pilot pK analysis. The lab is currently advancing a subset of therapeutic lead analogs towards formal GLP development. In parallel, we employ molecular virology, microchemistry and molecular modeling technologies to elucidate the binding pose of this scaffold to RdRp.

Exploring host-directed antivirals to counteract myxovirus infection

To address the problem of viral escape from antiviral treatment conceptually, we pursue in this project an antiviral strategy that targets host cell components exploited by the pathogen for replication. In addition to a heightened barrier against resistance, this strategy has high potential to yield therapeutics with a broadened target range, moving beyond conventional "one-drug, one-bug" approaches. In a proof-of-concept project, we have identified a lead class of host-directed myxovirus inhibitors that show exquisite activity against different ortho- and paramyxoviruses and, thus, provides a solid platform to evaluate the strategy conceptually. Current research focuses on characterizing the mechanism of antiviral activity, improving potency and drug-like properties, determining biotoxicity and efficacy, and exploring whether viral resistance can eventually be induced.

12

Perceived Barriers as Prospective Predictors of Adherence and Clinical Outcomes among Adolescent Transplant Recipients

Megan McCormick, MS[1], Ronald Blount, PhD[1], Katie Devine, PhD[1], Laura Mee, PhD[2], Laura Simons, PhD[3]
[1]University of Georgia, [2]Children's Healthcare of Atlanta & Emory University, [3]Children's Hospital Boston & Harvard Medical School

The Parent and Adolescent Medication Barriers Scales (Simons & Blount, 2007) are two factor analytically derived, multidimensional screening tools for examining barriers to medication adherence. Nonadherence in pediatric organ transplant is prevalent and can result in multiple negative health outcomes including medical complications, rejection, allograft loss, and death. The current study sought to longitudinally investigate associations between barriers endorsed at the original assessment and medical adherence and clinical outcomes 18 months later. Both overall subscales scores and individual, face valid items were examined in relation to outcomes. Of the 82 adolescent recipients (ages 11-20) enrolled in the initial cohort, 66 families participated in the follow-up. Rejection episodes, hospitalizations, and patient death were most strongly associated with parent and adolescent Ingestion Issues subscale scores (r = .25, .26, and .26 respectively) and specific items such as not liking the taste of the medication, believing the medication has too many side effects, and having a hard time swallowing the medication. Among adolescent reports, higher Disease Frustration/Adolescent Issues subscale scores and items (e.g., not wanting to take the medication at school, being tired of living with a medical condition) were associated with taking doses late and erratic immunosuppressant drug levels (r = .32 and .29 respectively for subscale scores). Additionally, parent reported higher Regimen Adaptation/Cognitive Issues subscale scores and items (e.g., adolescent forgetfulness or lack of organization) were associated with missed doses of medication (r = .33 for subscale score). Perceived barriers to adherence are central to efforts to ameliorate adherence difficulties and related health complications. The PMBS and AMBS demonstrate prospective strength and specific items provide a means of identifying needed targets for intervention.

13

FATTY ACID ETHYL ESTERS IMPAIR THE FUNCTION AND VIABILITY OF ALVEOLAR MACROPHAGES

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Purpose of Study: Fetal alcohol exposure is thought to predispose the newborn to infections which can significantly impact morbidity. Fatty acid ethyl esters (FAEEs), non-oxidative metabolites of alcohol, are potential biomarkers of fetal alcohol exposure and are linked to disruption of cellular functions. Since it is unknown whether biomarkers of fetal alcohol exposure contribute to alveolar macrophage dysfunction, the aim of this study was to evaluate the effect of the oleic acid form of FAEEs on the function of AM and to determine if GSH can modulate these effects

Methods Used: The NR8383 cell line of rat AM was exposed to: control conditions, + 25 μM ethyl oleate, or + 50 μM ethyl oleate (60 min). After FAEE exposure, some cells were treated with GSH (500□M) for 4 hrs. Using fluorescent microscopy, phagocytosis of FITC-labeled *Staphylococcus Aureus* was evaluated and mitochondrial reactive oxygen species (mROS) was determined using MitoTracker Red CM-H_2X Ros. mROS and phagocytosis was quantified as relative fluorescent units (RFU)/cell.

Summary of Results: FAEE exposure impaired the phagocytic function of AM and these affects were attenuated by the presence of GSH. There was a decrease of mROS and an increase in phagocytosis in the cells treated with glutathione after exposure to oleic acid in comparison to cells exposed only to oleic acid.

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Role of GLP-1R agonists in decreasing hepatic steatosis and apoptosis in *ob/ob* mice undergoing ischemia reperfusion injury

Nitika Arora Gupta, Vasantha Kolachala, Jean Kwun, Rene Romero, Frank Anania, Allan Kirk, Stuart Knechtle

Introduction: Glucagon like peptide 1 (GLP-1) is secreted from the *L* cells of the small intestine and acts as an incretin hormone mediating glucose release in response to an oral glucose load. Its cognate receptor is present in the liver and we have shown that the receptor agonists decrease hepatic steatosis by a direct action. It is also well known that a steatotic liver is vulnerable to ischemic injury resulting in increased morbidity and mortality in an increasingly vulnerable population with non alcoholic fatty liver disease. **Aim**: The purpose of this study was to identify the role of GLP-1R agonist (Exendin-4) in decreasing ischemia reperfusion injury of steatotic livers of *ob/ob* mice. **Methods**: *ob/ob* mice were divided into two groups. Both groups underwent ischemia reperfusion (I-R) injury per protocol. Briefly, the abdomen was exposed and a pediatric clamp was applied across the hepatic artery, portal vein and bile duct inducing ischemia for 20m. Following this, reperfusion was allowed till the liver returned to normal color. One group was treated with Exendin-4 pre and post surgery and the other group was treated with saline. Sera and livers were harvested 12 hours post surgery for estimation of serum ALT, oil red o staining, triglyceride quantification and tunel assay. **Results:** As compared to non operated *ob/ob* mice, mean serum ALT was elevated in *ob/ob* mice undergoing I-R injury, and was significantly improved in *ob/ob* mice with IR injury who received pretreatment with Exendin-4 (144 vs 777 vs 280 IU/l ;$p<0.05$). Oil Red O staining showed a significant decrease in fat globules in the pretreated *ob/ob* mice with I-R injury which was confirmed by a quantitative triglyceride measurements which were markedly less in *ob/ob* mice pretreated with Exendin-4 ($p<0.05$). Tunel assay showed the presence of apoptotic bodies in livers of *ob/ob* mice undergoing IR injury and which were decreased with treatment with Exendin-4. **Conclusion**: These data indicate that GLP-1R agonists have a role in improving liver enzymes and decreasing apoptosis in livers of *ob/ob* mice undergoing IR injury. We speculate that this is either a direct action of the receptor or an indirect action mediated through loss of hepatic steatosis.

15

STAT5 has tumor suppressor activity in myc-initiated acute B-cell lymphoblastic leukemia/lymphoma

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Signal transducer and activator of transcription 5 (STAT5) is a critical regulator of normal and leukemic lympho-myeloid development through activation downstream of early-acting cytokines, their receptors, and JAKs. Despite identification of upstream activating mutations driving JAK-STAT5 phosphorylation in B-ALL, the majority of cases lack these mutations. Instead, Burkitt and Follicular lymphoma are characterized by immunoglobulin rearrangements driving myc and bcl-2 expression respectively, both of which are known STAT5 target genes. We used a transgenic mouse approach to first determine whether constitutive expression of bcl-2 in STAT5-deficient hematopoietic cells could restore normal hematopoiesis or promote B-lymphoid leukemogenesis. Transgenic H2K/bcl-2 expression in hypomorphic STAT5ab$^{\Delta N/\Delta N}$ mice largely rescued peripheral B and T lymphocyte numbers but could only rescue about 10% of myeloid cell number. Complete deletion of the entire STAT5ab locus resulted in severely blocked B and T cell development following transplantation of STAT5ab$^{null/null}$ fetal liver cells into irradiated wild type or $\gamma C^{-/-}$ recipients and peripheral B-cell development could not be restored by transgenic bcl-2 alone. Interestingly, in the complete absence of STAT5, E□/myc combined with H2K/bcl-2 induced B-ALL with elevated B cell counts and reduced latency compared to wild-type control in the fetal liver transplanted mice. Adult conditional knockout of STAT5 gave the same result accelerated disease phenotype. Therefore, STAT5 can play a tumor suppressor role in subsets of B-ALL not characterized by JAK kinase driven hematopoiesis.

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Genome- wide association study on Down syndrome associated congenital Heart defects

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Trisomy 21, the chromosomal abnormality responsible for Down syndrome (DS), is a complex condition with many characteristic symptoms as well as an increased risk for numerous congenital anomalies. Heart defects are among the most common congenital abnormalities associated with Down syndrome (DS), affecting nearly half of all people with DS. Of those with a congenital heart defect, nearly 20% have an atrioventricular septal defect (AVSD), representing a nearly 2000-fold increased risk of AVSD compared to the general population. Through a multi-site recruitment effort, we have ascertained individuals with DS who have a complete balanced AVSD (cases) and those who have structurally normal hearts (controls) and their parents. Using this carefully selected sample, we test several different hypotheses aimed toward identifying the genetic variation underlying susceptibility to AVSD in people with DS. First, we test the common disease/common variant hypothesis in 97 trios and 113 controls by checking more than 900,000 SNPs throughout the genome for association with AVSD. We further extend the common disease/common variant hypothesis genome-wide by identifying and testing deletions for association with AVSD. We did not observe any SNPs with genome-wide significant allele frequency differences. However, we did observe 87 autosomal deletions with frequencies ranging from 1% up to >20% for common loci. The project is still ongoing with 300 more cases and controls to be analyzed. Follow up studies will be done for all the interesting candidate loci observed in this study.

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A novel MHC-defined rhesus macaque model of GvHD identifies $CD28^-$ $CD8^+$ T cells as a reservoir of breakthrough T-cell proliferation during costimulation blockade and sirolimus-based immunosuppression

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Treatment of hematologic malignancies in the pediatric population remains a challenge, and haplo-identical hematopoietic stem cell transplantation (HSCT) is one treatment option that offers a potential cure. However, graft-versus-host-disease (GvHD) remains a substantial complication of HSCT, with current prophylaxis relying on nonspecific immunosuppression. We have developed an MHC-defined primate model of GvHD and determined the effect of CD28/CD40 directed T-Cell costimulation blockade and sirolimus (COBS) on this disease. Recipients received MHC haplo-identical HSCT after 8Gy TBI. Without immunosuppression (n=3), an exuberant alloresponse occurred, with rapid T cell proliferation, severe GI and liver infiltration, and 100% mortality within 7 days. Flow cytometry revealed CD8 predominant expansion and activation, including CD127 downregulation, CD95 upregulation, and skewing towards a $CD95^+/CD28^-$ Teffector/effector memory (Te/em) phenotype. The vast majority of both Te/em and $CD95^+/CD28^+$ (Tcm) $CD8^+$ cells showed signs of clonal expansion (increased Ki-67 expression), and acquisition of terminal effector function (downregulation of BCl-2). A cytokine storm also occurred, with GvHD-specific secretion of IL-1Ra, IL-18, and CCL4. The addition of COBS (n=5) resulted in striking protection against GvHD. At the 30-day endpoint, COBS treated recipients showed 100% survival compared to no survival in untreated recipients. Some treated animals did eventually develop clinical and histopathologic evidence of smoldering GvHD. Treatment resistant breakthrough immune activation included secretion of interferon-γ, IL-2, monocyte chemotactic protein-1, and IL-12/IL-23, and proliferation of costimulation blockade resistant $CD28^-$ $CD8^+$ T cells, suggesting adjuvant treatments targeting this subpopulation will be needed for full disease control.

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Left Ventricular Function During Exercise in Siblings with Phenotypically-Variable, Gene-Positive Hypertrophic Cardiomyopathy

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Background: Left ventricular diastolic function is abnormal in hypertrophic cardiomyopathy (HCM). tissue Doppler imaging (TDI) may help identify genetic mutation carriers, and exercise testing may unmask myocardial dysfunction. We attempted to determine the feasibility of assessing TDI velocity and LV strain during exercise in children with HCM, and to compare phenotypic expression among these patients.

Methods:Three siblings (2 females, ages 9-11 years) underwent exercise cycle ergometry with simultaneous echocardiography. Commercially-available post-processing software (QLAB, Phillips Company, Netherlands) was used to determine TDI velocities and strain.

Results: The 9-year-old brother and 10-year-old sister had dual identical mutations in the *MHY7* gene, Arg-723-Cys and Gly-1057-Ser*. The 11-year-old sister carried Gly-1057-Ser. The 9 y.o. brother demonstrated severe disease at rest. Exercise elicited a blunted BP response, ST changes on ECG, mitral valve systolic anterior motion (SAM) by echocardiography, and decreased LVOT gradient. The 10 y.o. sister demonstrated mild disease at rest. Exercise elicited mild T wave changes, mild SAM, and decreased lateral mitral E' and increased lateral and septal mitral S' velocities. The clinically unaffected sibling had a normal resting echo and normal exercise test parameters. She demonstrated decreased lateral mitral E' and increased lateral and septal mitral S' velocities with exercise.
During exercise, strain reproducibility was poor due to significant translational motion.

Conclusions: It is possible to obtain reliable data on TDI during cycle ergometry. Currently, strain analysis during exercise is challenging to consistently reproduce. Despite the presence of genetically-identical HCM-causing mutations in multiple siblings, there can be great phenotypic variability present.

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Hyposecretion of mucus in response to Substance P in Human Cystic Fibrosis airways.

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Tachykinins such as Substance P (SubP) are released from sensory neurons and have been shown to produce antimicrobial rich mucus secretion by airway glands. Here, we used optical imaging methods (Joo, AJP Lung, 2001) to measure mucus secretion from individual glands in response to serosal application of SubP in human airways. Tissues were obtained following lung transplants from 7 CF, 19 non-CF disease control (DC) and 19 normal control (HN) subjects. The average secretion rates to 10 µM SubP were: HN: 240 ± 60 pl/min/gl, n=14, (118 glands); DC: 200± 20 pl/min/gl from n =13 (115 glands), and CF: 10 ± 1 pl/min/gl n=7 (58 glands), p <0.01. Viable glands were defined as those responsive to carbachol (~100% of anatomically verified glands). SubP produced secretion in the following proportion of glands: HN: 208/271 glands (76%); DC: 155/258 (60%); CF: 5/175 (3%), p <0.01. Responses to SubP were additive with forskolin/VIP, but not with carbachol. After 1µM VIP or 10µM forskolin, 10µM SubP increased the secretion rate from 100 ± 30 pl/min/gl to 270 ± 100 pl/min/gl in 4 HN subjects and from 190± 30 pl/min/gl to 460 ± 90 pl/min/gl in 4 DC subjects, but in 5 CF subjects it failed to cause additional increases in secretion rates: 6 ± 1 pl/min/gl to 8 ± 2 pl/min/gl, p <0.01. Hyposecretion to SubP and absence of additivity of SubP with Forskolin/VIP in CF glands indicate that human gland secretion mediated by SubP requires CFTR. Supported by Nash Fellowships to MS by CFF, and by NIH RO1-51817 (JJW).

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KNOWLEDGE AND RISK PERCEPTION OF LATE EFFECTS AMONG CHILDHOOD CANCER SURVIVORS BEFORE AND AFTER VISITING A CHILDHOOD CANCER SURVIVOR CLINIC

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Background*:* Childhood cancer survivors are at risk for treatment-related medical problems and education about these risks is crucial to ensure appropriate surveillance and early intervention. In this study we explored survivors' baseline knowledge regarding late effects and measured change in knowledge after visiting a multi-disciplinary childhood cancer survivor clinic.

Methods*:* Participants over a recruitment period of one year completed a baseline questionnaire immediately before their first survivor clinic visit. During the visit survivors/parents received individualized education including a treatment summary, risk profile and surveillance plan using the Children's Oncology Group Long-term Follow-up Guidelines. A follow-up questionnaire (identical to the baseline) was distributed one month after the visit. Participants ≥16 years of age completed the questionnaires; the parent/guardian completed questionnaires for participants < 16 years old. 65 participants completed the baseline survey and 35 completed the follow-up survey.

Results*:* Overall survivors were less knowledgeable about their personal risk for late effects compared to parents. The baseline survey identified knowledge gaps among survivors ≥16 years old regarding risk for infertility, secondary cancers and cardiac late effects. Parents scored higher than survivors at baseline with 60% correctly identifying if their child is at risk for a secondary cancer. 80% of survivors at risk for secondary malignancies, and 43% at risk for cardiac problems, did not identify themselves at risk. Survey questions were converted into a knowledge score ranging from 0-100 with a high score being over 60 and a low score 60 and under. Overall survivors scored lower at baseline and follow-up and had a smaller knowledge score increase from baseline to follow-up compared to parents.

Conclusion: Receiving individualized education during a survivor clinic visit increased participant knowledge regarding risk for late effects. Additional research is needed to further improve overall knowledge of late effects in this vulnerable population.

Implications: Education is a vital component of the survivor clinic visit. This study provides evidence that current education practices are somewhat effective, but more research should be done to identify further knowledge gaps and possible efficacy of tailored educational interventions.

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Todd C. McDevitt Study

Todd C. McDevitt, Ph.D.**,** Petit Faculty Fellow, The Parker H. Petit Institute for Bioengineering & Bioscience, Director, Stem Cell Engineering Center at Georgia Institute of Technology, Associate Professor, The Wallace H. Coulter Department of Biomedical Engineering at Georgia Institute of Technology & Emory University, Human Pluripotent Stem Cells & Technologies: Novel Opportunities for Pediatric Research

The direct study of human development is limited by the lack of an ethically palatable, tangible and tractable experimental system. Human pluripotent stem cell (PSC) lines, derived from discarded IVF blastocysts or reprogrammed from somatic cells, can serve as a unique basis of in vitro systems to systematically probe mechanisms of normal and abnormal human development. This requires the development of robust and reproducible methods to differentiate human PSCs to specific lineages and mimic morphogenic aspects of embryogenesis. In vitro differentiated cells from human pluripotent sources recapitulate the phenotypes of immature cells analogous to fetal development and typically fail to acquire mature characteristics of adult cells, making PSCs perhaps more relevant to pediatric research issues than other areas of human biology and disease. Among several possibilities are opportunities to (1) examine the effects of dietary supplements and other environmental factors thought to benefit or disrupt embryological development in dose- and temporally-dependent manners, (2) perturb specific molecules and signaling pathways in order to definitively identify critical regulators of global development or differentiation of specific cell phenotypes, and (3) study processes of congenital, familial or genetic defects and disorders that can arise in utero. In addition to studying aspects of human developmental biology, the therapeutic utility of human cells derived from PSC lines may be advantageous for engineering of tissues that can grow and mature appropriately during post-natal development. For the aforementioned reasons, human PSC lines are a potent, yet untapped resource for a plethora of different pediatric research topics of interest.

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Catch-Up Immunization Scheduling For Children and Adolescents

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Pediatric Health Issue: Recommended immunization schedules for children and adolescents are published annually by ACIP. These schedules contain specific rules regarding the timing of each vaccine, as well as gaps between different doses of the same vaccine. These rules must be followed when constructing a catch-up vaccination schedule for someone who has fallen behind on one or more vaccinations – a task which is challenging and time consuming. Inappropriately constructed schedules may prevent timely administration of vaccination, potentially increasing the risk for contracting a vaccine-preventable disease.

Methods: The Catch-Up Immunization Scheduler was developed to alleviate the problem of untimely vaccination in children through age 6. The scheduler accepts input from the user including date of birth, the dates of administration for each dose and the number of doses of each vaccine that have been administered. The tool then determines the recommended immunization schedule for the child using a dynamic programming (DP) algorithm, the details of which have been published (Operations Research 2009; 57:1307-19). A companion tool for adolescents through age 18 was also developed.

Discussion: The problem of constructing catch-up immunization schedules is faced regularly by health-care professionals. The tools developed provide a means of educating individuals regarding vaccine recommendations and construct reliable immunization schedules quickly. The tool targeting children through age 6 is freely available (http://www.cdc.gov/vaccines/recs/scheduler/catchup.htm) and has been downloaded over 67,000 times since June 2008. The online version of the tool is planned for deployment in Spring 2011. The tool for adolescents will be released early this year.

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SurvivorLink as an on-line tool to improve patient knowledge and access to care beyond cancer

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Background: Survivorship care aims to educate and empower survivors to seek lifelong individualized surveillance and treatment for late effects to improve quality and length of life. High quality care is challenging due to lack of knowledge among both health care providers (HCP) and patients/families about the complex health care needs of survivors.

Methods: SurvivorLink is a web-based tool designed to improve awareness and provide information on best practices in survivor care. The Healthcare Provider portal, providing evidence-based education material on survivor care best practices has been successfully launched. Development of the patient/parent portal, providing educational material and serving as a repository for a personalized Survivor Healthcare Plan (SHP), is underway. The goal of this portal is to improve the exchange of clinical information at key care transitions and provide patients easy access to individualized educational materials.

Results: To inform design of the patient/parent portal, focus groups were conducted with two key users: parents of pediatric cancer survivors, and young adult (YA) survivors of childhood cancer. Overarching themes ascertained in focus groups were: 1) Parents are more concerned than YAs about effective strategies to prepare pediatric cancer survivors on managing their health and advocate for themselves. Both groups welcomed tools/resources to help with this transition; 2) Parents expressed difficulty finding resources for HCPs knowledgeable about survivor's special healthcare needs with experience and willing to treat survivors; 3) Both YA and parents had misconceptions about the privacy and security of storing the SHP online.

Conclusion: YAs and parents have different needs and wants, which are addressed in the design of the portal. Targeted education is directed to reassure privacy/security of the SHP. An evaluation plan will examine utilization, user feedback, and the effect of SurvivorLink use on quality of care and patient outcomes. This portal is scheduled for completion in June, 2010.

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Hyperoxia promotes epithelial-to-mesenchymal transition of alveolar epithelial cells

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Although oxygen is life-saving for patients, prolonged hyperoxia leads to alveolar epithelial cell (AEC) injury and lung fibrosis. Hyperoxia increases reactive oxygen species (ROS) and ROS activate transforming growth factor-β1 (TGF-β1). TGF-β1 induces alveolar epithelial-mesenchymal transition (EMT), a process that increases myofibroblasts and matrix deposition. We hypothesized that hyperoxia promotes TGF-□1 signaling and alveolar EMT. RLE-6TN, an AEC line, was treated with hyperoxia (85%) or control conditions and EMT was assessed. To determine the role of ROS, control and hydrogen peroxide-treated (H_2O_2; 10-50 μM) AEC were evaluated for EMT and TGF-β1 activation. A marker of TGF-β1 signaling, phosphorylated Smad3 (p-SMAD3) was quantified by densitometric analyses of immunoblots from hyperoxia-, H_2O_2-, and control-treated cells. A TGF-β1 neutralizing antibody (TGFnAb; 8 μg/mL) was then added to one group and EMT and p-SMAD3 was evaluated. Treatment with both hyperoxia and H_2O_2 increased expression of myofibroblast markers, α-smooth muscle actin (α-SMA) and vimentin, and depleted the epithelial markers, cytokeratin, lamellar protein, and E-cadherin by immunofluorescence. Immunoblot quantifications showed that hyperoxia increased α-SMA 8-fold and vimentin 1.8-fold, and that H_2O_2 increased α-SMA, N-cadherin and vimentin 2-fold over controls. H2O2-exposure doubled active TGF-β1 by ELISA and both hyperoxia and H2O2 increased p-SMAD3 expression 6.6 and 3.2-fold, respectively. Treatment with TGFnAb dramatically decreased immunofluorescence for α-SMA and vimentin and reduced p-SMAD3 30% below levels seen with H_2O_2 alone. Hyperoxia induces alveolar EMT through TGF-β1 activation and Smad3 signaling. A better mechanistic understanding of the role of oxidative stress in lung fibrosis may prevent complications of oxygen therapy.

Conclusions: Acute exposure to FAEE(specifically ethyl oleate), increased AM mROS and impaired phagocytosis. Treatment with GSH attenuated these effects suggesting that FAEE exposure alone promoted oxidative stress. We hypothesize that FAEE due to prenatal alcohol exposure can contribute to the dysfunction of developing AM and that these effects may be modulated by GSH.

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Building Ecological Technology Tools to Improve Asthma Management in Pediatric Patients

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The goal of the Pediatric Asthma Initiative is to develop technologies to automate current treatment, monitoring and management practices for this condition. Web and mobile technologies are a central part of our focus since they are becoming ubiquitous regardless of socio-economic status. Our research efforts mediate communication between physicians and patients between regularly scheduled visits because this is central to adherence and improved patient outcomes. We have built a variety of technologies these include a mobile based software solution to lung function analysis. We created the proof of concept and are working toward a reliable prototype. We have also designed the Georgia Asthma Resource website which is meant to be a repository of all relevant information about asthma in Georgia. This website has a number of applications (air quality/ exercise SMS service, asthma computer game) that are meant to provide information to help facilitate asthma management for a variety of stakeholders while at the same time provide a stream of research data to better understand technology usage in the pediatric asthma population (usage of the technology will be contingent on our ability to access the data of those that download our apps). We have also implemented a SMS/physician dashboard application that will investigate the role that data gathered from pediatric patients (concerning asthma symptoms and general asthma content) in the interim between scheduled visits effects the interactions between patients and physicians during face to face communication.

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CANCER SURVIVOR CLINIC ATTENDANCE: PATIENT CHARACTERISTICS

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Background: Childhood cancer survivors are at increased risk for late-effects for which they need survivor–focused care. At Children's Healthcare of Atlanta (CHOA) only 48% (433/909) of eligible patients participate in the Cancer Survivor Program (CSP). The objective of this study was to determine barriers to survivorship care.

Methods: Demographic information (age at diagnosis, gender, race, insurance status, zip code and diagnosis) was abstracted from the CHOA Tumor Registry and Cancer Survivor Database. Brain tumor patients, followed in a different clinic, were not included. Data were compared for 433 CSP patients (attended the CSP from 2003-2007) and 909 CSP-eligible patients (diagnosed from 1998-2002; alive in 2003). Insurance data were unavailable prior to 2001 for the CSP-eligible patients; therefore newly diagnosed patients from 2003-2007 were utilized as a comparison group to analyze insurance data.

Results: When compared to the CSP-eligible population, CSP patients were more likely to be younger at diagnosis (less that 13 years) (OR 3.53; 95% CI 3.14-3.92), diagnosed with a liquid versus solid tumor (OR 1.41, 95% CI 1.18-1.64), and non-Hispanic white compared with other races (OR 1.25, 95% CI 1.00-1.50). Geographic location inside versus outside the Metro Atlanta area was not a significant factor in CSP attendance. A shift in insurance coverage was evident between the newly diagnosed population (52% private) and the CSP patients (72% private). Those with private insurance were over twice as likely to attend the CSP (OR 2.44, 95% CI 2.20-2.68).

Conclusion: Low CSP attendance rates can be addressed by ensuring that all survivors are educated about the importance of survivorship-care upon completion of cancer therapy. Contact should be maintained with patients from the end of therapy through the first CSP visit. Attention should be directed to at-risk populations including those with solid tumors, older at diagnosis, and with Medicaid or no insurance.

CLINICAL AND MOLECULAR EPIDEMIOLOGY OF PEDIATRIC NOROVIRUS IN METROPOLITAN ATLANTA.

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Background: Universal rotavirus immunization has resulted in a decline in the burden of sporadic acute gastroenteritis (AGE) in children. Improvement in molecular diagnostics has resulted in increased detection of norovirus from children with AGE.

Objective: We assessed the molecular epidemiology of norovirus among a cohort of children < 21 years of age with AGE seeking care in metropolitan Atlanta.

Design/Methods: Samples of prospectively collected stool from symptomatic children with AGE were tested for norovirus by reverse transcription polymerase chain reaction and rotavirus with enzyme-linked immunosorbent assay. A retrospective review of the corresponding medical records was performed to assess clinical characteristics. The 20-point Vesikari score was used to determine disease severity.

Results: Between December 2009 and September 2010 norovirus was detected in 26 (22.6%) and rotavirus in 7 (6.1%) stool specimens of 115 children with AGE. Among 20 children ≤5 years of age, the mean age of those with stools containing norovirus (n=14) was 20.9 months (standard deviation (SD)14.3 months) and rotavirus (n=6) was 42.7 months (SD 15.7 months), p=0.02. Completion of a series of rotavirus immunization was 78.5% among those with a norovirus infection and 16.7% among those with a rotavirus infection (p=0.01). The majority of norovirus infections, 24 (92.3%), occurred between December and March, and accounted for 24 (40%) of AGE during this period. The mean Vesikari scores were similar as were the percentages of children with Vesikari scores ≥11. Nineteen (73%) children with norovirus required hospitalization. All norovirus isolates were genogroup II.

Conclusions: Noroviruses are an important etiology of moderate to severe pediatric AGE in metropolitan Atlanta with a seasonal prevalence. These results will contribute to efforts to develop rapid diagnostics and an effective vaccine.

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Epidemiology of *Staphylococcus aureus* bacteremia and meningitis among very low birth weight infants from the NICHD Neonatal Research Network, 2006 -- 2008

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BACKGROUND: The impact of methicillin resistant *Staphylococcus aureus* (MRSA) emergence on staphylococcal (SA) meningitis and bacteremia among very low birth weight (VLBW) infants is uncharacterized.

METHODS: VLBW infants (birth weight 401-1500 g) born 1/1/2006 --12/31/2008 who received care at centers of the NICHD Neonatal Research Network were studied. Early onset (< 72 hours) and late onset (> 72 hours) infections were defined by blood or cerebrospinal fluid cultures and any antibiotic treatment for ≥ 5 days (or death < 5 days with intent to treat).

RESULTS: Among 8444 infants who survived >3 days, culture-proven SA infection occurred in 316 (3.7%). Eighty-eight infants had MRSA (1% of all infants, 28% of infants with SA); 228 had MSSA (2.7% of all infants, 72% of infants with SA). No infants had both MRSA and MSSA infections. Ninety-nine percent of MRSA infections were late onset. The percent of infants with MRSA varied by center, $p<0.001$, with no cases identified at 9/20 centers. Risk of MRSA infection was inversely related to gestational age. Need for mechanical ventilation, diagnosis of respiratory distress syndrome, necrotizing enterocolitis and other morbidities did not differ between infants with MRSA and MSSA. Approximately 25% of infants with SA infections died, 26% MRSA and 24% MSSA.

CONCLUSIONS: Few VLBW infants had SA meningitis or bacteremia. The 1% with MRSA had morbidity and mortality rates similar to infants with MSSA. Prevention and treatment practices should provide equal focus on reducing the burden of both MRSA and MSSA infections.

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Imaging Tools for the Study of RNA Regulation and RNA Viruses

The Santangelo lab in the Wallace H. Coulter Department of Biomedical Engineering at Georgia Institute of Technology and Emory University is focused on the development of tools for the study of RNA regulation and RNA viruses, many relevant to many pediatric pathologies. Our work focuses on the RNA of human Respiratory Syncytial Virus (with the Crowe lab at Vanderbilt University and Moore lab at Emory University) and Influenza, both important pathogens in children, as well as the effects of alcohol on posttranscriptional regulation of RNA in alveolar epithelial cells (with the Brown lab at Emory University), all at the subcellular level. *In vivo*, in order to aid in the development and study of highly active antiretroviral therapy (HAART), we are developing an immuno-PET agent against SIV in the rhesus macaque model (with Villinger and Hunter labs at Emory University and the Yerkes Primate Center). In addition to antiviral study, we feel this agent and derivatives will have other applications, such as the study of the transmission of HIV to humans, and possibly to children. Presented are data depicting the time course of an RSV infection, highlighting the induction of stress granule formation, detection and trafficking of influenza genomic RNA during virus assembly, and the detection of stress granule formation in mouse alveolar epithelial cells after alcohol exposure. In addition, preliminary PET data in SIV infected and non-infected macaques is also included.

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Machine Learning Framework for Classification in Medicine and Biology

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Systems modeling and quantitative analysis of large amounts of complex clinical and biological data may help to identify discriminatory patterns that can uncover health risks, early disease formation, monitoring treatment and prognosis, and predicting treatment outcome. In this talk, we describe a machine-learning framework for classification in medicine and biology. It consists of a pattern recognition module, a feature selection module, and a classification modeler and solver. The pattern recognition module involves automatic image analysis, genomic pattern recognition, and spectrum pattern extractions. The feature selection module consists of a combinatorial selection algorithm where discriminatory patterns are extracted from among a large set of pattern attributes. These modules are wrapped around the classification modeler and solver into a machine learning framework. The classification modeler and solver consist of novel optimization-based predictive models which maximize the correct classification while constraining the inter-group misclassifications. The classification/predictive models 1) have the ability to classify any number of distinct groups; 2)allow incorporation of heterogeneous, and continuous/time-dependent types of attributes as input; 3)utilize a high-dimensional data transformation that minimizes noise and errors in biological and clinical data; 4)incorporate a reserved-judgment region that provides a safeguard against over-training; and 5)have successive multi-stage classification capability. Successful applications of our model to developing rules for epigenetics in early cancer prediction, predicting the immunity of vaccines, identifying the cognitive status of individuals, and predicting metabolite concentrations in humans will be discussed.

We acknowledge our clinical/biological collaborators: Dr. Vertino (Winship Cancer Institute, Emory), Drs. Pulendran, Ahmed (Emory Vaccine Center), Dr. Levey (Neurodegenerative Disease and Alzheimer's Disease), and Dr. Jones (Emory Clinical Biomarkers).

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Notes

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