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Research Committee Report

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In This Issue

2021 Marshall Klaus Neonatal-Perinatal Medicine

On behalf of the American Academy of Pediatrics (AAP), Section on Neonatal-Perinatal Medicine (SoNPM) and this year's Klaus Neonatal-Perinatal Research Fund supporters, we would like to congratulate the recipients of the 2021 Marshall Klaus Neonatal-Perinatal Research Award! The Research Committee received 53 very strong applications and all applicants deserve recognition for their already strong bios, outstanding mentors, and exciting projects. This year's awards are sponsored by Mead Johnson Nutrition, Johnson & Johnson Pediatric Institute, Brodsky & Martin's Neonatology Review Series, Beth Israel Deaconess Medical Center, and the AAP SoNPM. Thanks to our sponsors, this year we are able to fund the top 9 ranked applications with the $5,000 research award. The money will be used towards supplies etc. to support the proposed research project.

Special thank you to the award sponsors

Klaus Bench or Clinical Research Award

SoNPM (AAP), Johnson & Johnson Pediatric Institute, Mead Johnson Nutrition

Neonatology Education Research Award

Brodsky & Martin's Neonatology Review Series

Health Services Research Award

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Klaus Bench or Clinical Research Awardees

Emily A. Callan, MD
Medical College of Wisconsin

Title: *Aberrant regulation of PGC-1α in persistent pulmonary hypertension of the newborn (PPHN)*

Mentor: G. Ganesh Konduri, MD

Personal Statement:
 My interest in persistent pulmonary hypertension of the newborn (PPHN) originated in my first year of medical school when I began working in the lab of Dr. Girija Konduri, one of the world's leading experts on this devastating condition. I found Dr. Konduri's enthusiasm and steadfast dedication to deciphering the pathophysiology of PPHN, and developing new treatment options, to be contagious. PPHN affects 1.8 in 1000 liveborn infants, with the highest incidence being in late preterm infants at 5.4 per 1000 live births. Although the use of inhaled nitric oxide (iNO), a potent pulmonary vasodilator, has led to a profound advancement in PPHN therapy, some infants fail to respond to iNO and require alternative therapies such as extracorporeal membrane oxygenation (ECMO) to

survive. Inconsistent results in the current therapeutic strategies suggest that targeting specific signaling pathways is required for successful therapy. Under the mentorship of Dr. Konduri, I have focused most of my investigation on the mitochondrial transcription co-activator, Peroxisome Proliferator-Activated Receptor Gamma Co-Activator-1α (PGC-1α), which we have shown is decreased in fetal lamb PPHN pulmonary artery endothelial cells (PAECs) and linked to decreased mitochondrial biogenesis and impaired angiogenesis function in pulmonary hypertension. I am deeply honored and humbled to be selected as a recipient of the prestigious Marshall Klaus Award. In addition to funding my research, this award will help me on my quest to become a successful physician-scientist by providing me with invaluable experience designing and managing my own studies, increasing my efficiency in the lab, sharpening my communication skills, expanding my substantive knowledge base, and making me a more effective and empathetic clinician. Hoping to have my own research lab one day, I crave opportunities to continue performing neonatal research and honing my skills; and, the Marshall Klaus Award is helping make my dream come true.

Abstract:

Persistent Pulmonary Hypertension of the Newborn (PPHN) is a clinical syndrome characterized by sustained elevation of pulmonary vascular resistance (PVR) after birth, resulting in extra-pulmonary right-to-left shunting and severe hypoxemia. PPHN is caused by abnormal metabolic regulation that disrupts homeostasis in pulmonary artery endothelial cells (PAECs), leading to increased apoptosis and impairment of angiogenesis function. Our lab uses a lamb model of PPHN in which ligation of the fetal ductus arteriosus creates elevated postnatal PVR. PAECs from these PPHN lambs show impaired angiogenesis function compared to control lamb PAECs. Peroxisome Proliferator-Activated Receptor Gamma Co-Activator-1α (PGC-1α), a mitochondrial transcription co-activator, is a key regulator of energy metabolism in PAECs via its downstream effects on mitochondrial transcription factors. We previously found that PGC-1α protein levels were decreased in PAECs that were isolated from fetal lambs with pulmonary hypertension in utero. Moreover, we discovered that when PGC-1α is decreased using silencing RNA (siRNA) in control lamb PAECs, there is reduced capillary tube formation, cell migration, and cell proliferation, indicating impaired angiogenesis function. Looking upstream, PGC-1α function can be increased via post-translational modifications by 5'-AMP-Activated Protein Kinase (AMPK) and Sirtuin-1, and its expression can be augmented through transcriptional modifications by Ca^{2+}/CaM-dependent protein kinase kinase β (CaMKKβ) and CaMKIV. However, whether aberrant regulation of PGC-1α occurs in PPHN remains unknown. Studying these upstream pathways may reveal if PGC-1α is a critical factor in the angiogenesis dysfunction in PPHN via its regulation of mitochondrial biogenesis. The overall goal of my project is to elucidate the exact molecular mechanism that decreases PGC-1α expression and function during times of stress, resulting in the PPHN phenotype. My proposal will test the specific hypothesis that aberrant upstream signaling results in decreased expression and

function of PGC-1α in PPHN, leading to reduced mitochondrial biogenesis and impaired angiogenesis function. Since PGC-1α increases the expression of a plethora of target genes and is simultaneously involved in multiple pathways, we believe that it may serve as an innovative target to treat PPHN resistant to conventional drugs.

Adrian A. Epstein, MD, PhD
Duke University Medical Center

Title: *Disruption of oligodendrogenesis following sepsis-induced injury to the postnatal subventricular zone*

Mentor: Eric J. Benner, MD, PhD

Personal Statement:
The overarching goal of my program of research is to improve neuroprotective care for preterm neonates. During my 3rd year of neonatology fellowship at Duke University my training will focus on neurobiology related to neonatal clinical problems. My research mentor, Eric Benner develops neural stem cell therapies using a neonatal mouse model of experimental peritonitis where systemic polymicrobial infection leads to diffuse white matter injury. We found that the germinal matrix at the subventricular zone (SVZ) displays a

degenerative phenotype with reduced activity of neural progenitor cells following severe sepsis in the animal model. Currently there are no therapies for preterm neonatal brain injuries however, with the support of the Marshall Klaus Neonatal-Perinatal Research Award we will investigate neural stem cell responses to systemic inflammation during the sensitive period of neural progenitor cell development. Discoveries of novel mechanisms of injury unique to premature infants will hopefully strengthen translational research to improve neurodevelopmental outcomes. To complete the aims of this project I will have the opportunity to work with influential collaborating scientists at Duke using advanced techniques in neuroimmunology and molecular neuroscience. I am also currently supported by the Unified Program for Therapeutics in Children, a T32 training program mentored by pediatric physician scientists across Duke University and University of North Carolina, Chapel Hill. My long-term goal is to practice as a neonatologist-neuroscientist – to improve developmental outcomes using neurorestorative therapies for infants in the NICU.

Abstract:
Preterm neonates are at high risk of brain injury leading to impairments in motor, cognitive and behavior development. Prematurity-specific mechanisms of neonatal neurological disease are not well defined and no therapies are available at this time. The subventricular zone (SVZ) is part of the germinal matrix and critical for postnatal brain development. The SVZ neural stem cell niche includes ciliated ependymal cells that form a barrier between the cerebral spinal fluid and neural stem cells, as well as paracrine

regulation of stem cell activity. Progenitor cells from the SVZ contribute to growth of white matter and the cerebral cortex beyond infancy and through childhood. This proposal aims to test the hypothesis that injury to cells in the SVZ interfere with brain development. Our laboratory has developed a clinically relevant rodent model where experimental bowel perforation leading to neonatal sepsis replicates brain injuries seen among preterm-born infants. These injuries include white matter injury as well as motor deficits. Preliminary evidence shows that systemic bacterial sepsis leads to neuroinflammation in the SVZ with robust microglial activation. Moreover, we have observed a novel injury to the ependymal layer of the SVZ along the lateral

ventricular wall. In this research proposal we will measure neural stem cell responses to neonatal experimental bowel perforation and sepsis in the animal model. We plan to combine cell isolation techniques with transcriptomics and immunofluorescence to measure specific changes in SVZ ependymal, glial, and neural stem cells in septic compared to littermate control animals. Next, we will determine the functional impact of SVZ injury on postnatal brain development using genetic lineage tracing of SVZ

progenitor cells to demonstrate and measure the outcome of SVZ injury on brain development compared to littermate controls. The results of this investigation into SVZ pathobiology will generate a novel mechanism of brain injury unique to premature birth that may impact how we implement neuroprotection and diagnose

early brain injury. Such discoveries may expand the repertoire of therapeutic possibilities and capacity for developmental recovery for babies born prematurely.

Meagan M. Goates, MD
Texas Children's Hospital

Title: *Deciphering the mechanistic role of regulatory T cells in experimental bronchopulmonary dysplasia*

Mentor: Binoy Shivanna, MD, DM, PhD

Personal Statement:
I am a first-year neonatology fellow training at Baylor College of Medicine/Texas Children's Hospital. I am interested in neonatal T-cell function specifically the balance of the T Helper 17/Regulatory T cell (TH17/Treg) pathway and how this contributes to neonatal inflammatory conditions such as bronchopulmonary dysplasia (BPD). I first developed an interest in neonatal T-cell development during my pediatric residency training at the University of Rochester Medical Center. In residency I explored neonatal helper T-cell cytokine profiles at different timepoints and gestational ages. The project also looked at neonatal oxygen exposure with the ultimate purpose of identifying predictive factors of respiratory morbidity at one year of life. It was during this investigation and reviewing the pertinent literature that I became particularly interested in TH17 and the proinflammatory effects seen in neonates. We discovered that IL-17 (the signature cytokine of TH17) positive T-cells at NICU

discharge along with neonatal 14-day supplemental oxygen exposure was predictive of respiratory morbidity at 1-year of life. My current research goal is to explore the role of neonatal regulatory helper T cells and TH17 cells in BPD and how hyperoxia affects the TH17/Treg balance. I am excited to uncover more knowledge regarding neonatal T-cell development and its impact on neonatal inflammatory diseases through the support of the Marshall Klaus Neonatal-Perinatal Research Award.

Abstract:
Bronchopulmonary dysplasia (BPD) is the most common chronic lung disease of preterm infants that is associated with life-long morbidities and lacks curative therapies. Regulatory T cells (Tregs) are crucial to maintain immune homeostasis and prevent tissue damage in several organs. Further, they promote the resolution of lung inflammation and injury in adults; however, their role in BPD pathogenesis is poorly studied. Addressing this unmet need will help us understand how inflammation resolves and identify therapeutic strategies. In contrast, Helper T 17 (TH17) cells are pro-inflammatory and thought to play a role in neonatal inflammatory diseases. Hyperoxia (HO) increases Th17 cells in the lungs of adult mice, resulting in airway and lung inflammation; however, the interactions between Tregs and Th17 cells in HO-exposed neonatal murine lungs are unclear. Thus, this project will examine the mechanistic and therapeutic role of Tregs as well as the role of the TH17/Treg balance in BPD by using transgenic mice and a well-established murine model of HO-induced neonatal lung injury. The study will test the central hypothesis that Treg/Th17 imbalance toward the TH17 phenotype potentiates HO-induced inflammation and injury in neonatal murine lungs and an increase in the Treg phenotype mitigates it. The long-term goal is to develop innovative therapies for severe BPD and other neonatal inflammatory conditions by targeting Treg and Th17 cells. This project will be an initial step toward this long-term goal.

Pearl D. Houghteling, MD
Stanford University

Title: *Esophageal atresia: a natural experiment of the effects of oral inoculation on the gut microbiome*

Mentor: David A. Relman, MD

Personal Statement:
I am honored to receive this award, and to be associated with Dr. Klaus, whose work on the care of the infant and their parents influences my practice of neonatology every day. My goal is to become a neonatologist-scientist, using translational research focused on the microbiota to improve infant nutrition and growth. I became fascinated with the gut microbiome in medical school and was fortunate to work with Dr. W. Allan Walker and Dr. Kate Gregory at Massachusetts General Hospital/Brigham and Women's. In working with them, and during my residency at Yale-New Haven

Hospital (thanks to Dr. Richard Ehrenkranz, Dr. Sarah Taylor, and others' nutritional expertise), I came to see the critical crosstalk between nutrition and the microbiota as central to the infant's developing immune system and overall health. Here at Stanford Lucile Packard Children's Hospital, I have been fortunate to work with Dr. David Relman and the Center for Human Microbiome Studies, whose ecological framework and sophisticated analytic techniques have dramatically broadened and deepened my understanding. This project, looking at the microbiota, nutrition, and immune system of neonates with esophageal atresia, will leverage this congenital anomaly as an experimental condition. In the long term, I plan to continue to work with infants with esophageal atresia to improve outcomes for this challenging disease. My other interest focuses on the interplay between iron deficiency and supplementation in the human host and the gut microbiota.

Abstract:

Infants with esophageal atresia (EA) may not have anastomosis of the upper and lower segments of the esophagus until they are several months old. Thus, the intestines in children with EA are not exposed to the oropharyngeal microbiota or other components of oral secretions prior to surgery, resulting in an extreme and potentially prolonged disruption to microbial community assembly in the gut. It is anticipated that the delay in oral inoculation of the gut contributes to the persistent comorbidities of esophageal atresia, especially poor growth, but there are no data to prove this.

Infants with EA and gestational-age matched controls in the neonatal intensive care unit will be evaluated for their microbiome (oral, gastric and stool using 16S rDNA sequencing), metabolomics (blood, urine, stool) and immune profiling (using CyTOF). Infants with EA will be administered their own saliva via G-tube with each feed for one week as an experimental condition to observe the effects of saliva on the microbiota, immune system, and nutrient utilization.

An adjunctive therapy for EA using pre-anastomosis gastric inoculation with oral secretions might result directly from this research. In addition, this project may further elucidate the role of the gut microbiota in altered growth and immune development in children.

Brooke A. Krbec, DO
Boston Children's Hospital

Title: *Novel noninvasive bedside arterial monitoring for infants in the NICU*

Mentor: P. Ellen Grant, MD, MSc

Personal Statement:
My career goal is to become a physician researcher and clinical liaison for the development and institution of novel innovations or devices that will advance bedside clinical care. My current research focus is neonatal neurocritical care; specifically focusing on validating non-invasive devices that can substitute current invasive

techniques to study cerebral autoregulation in the neonatal population. I have a strong background from an undergraduate degree in Neuroscience from Tulane University, Master's degree in Cellular and Molecular Biology from the University of North Carolina at Greensboro, as well as a research internship in Newborn Medicine through Boston Children's Hospital that have prepared me for a research career. Prior to medical school, I operated a horse training and sales business, and was a college instructor in Forensics, Biology, Anatomy and Physiology. I also worked as a traveling lab technician for a whole-body donation clinic where I refined my procedural skills and helped to innovate a latex injection protocol for cadavers. During medical school at Lake Erie College of Osteopathic Medicine, I lead a multi-disciplinary research study on how to reduce stress in first year medical students for which I was recognized on a National level. Together these experiences and accomplishments solidified my true passion and interest in clinical research, which is the perfect combination for my leadership and teaching skills, as well as my passion for innovation. With the support of the 2021 Marshall Klaus Neonatal-Perinatal Research Award and my mentorship team at the Fetal-Neonatal Neuroimaging and Developmental Science Center (FNNDSC) I will be able to build a solid foundation for a career in clinical neonatal neurocritical care research.

Abstract:
Continuous measurement of arterial blood pressure (ABP) is common in clinical practice where precise blood pressure regulation is necessary. Options for measuring ABP are limited in the neonatal population and requires invasive catheterization. There are significant complications associated with these invasive techniques. Despite inconsistencies and risk, invasive monitoring remains the gold standard for ABP measurement. The Finapres® (Finometer®, Finapres Medical Systems, Amsterdam, Netherlands) device is currently FDA approved for non-invasive continuous ABP monitoring in adults, but studies have been limited in infants. The goal of this project is to validate this non-invasive blood pressure device in a high-risk neonatal population to ultimately provide a safe, bedside alternative to invasive ABP monitoring over both invasive and standard oscillometric BP cuffs. Furthermore, we will aim to test the feasibility of integrating a novel technology: diffuse correlation spectroscopy (DCS), combined with advanced frequency-domain NIRS (FDNIRS), to simultaneously monitor cerebral blood flow to study cerebral autoregulation using the Finapres® device as a surrogate for cerebral perfusion pressure.

Jacquelyn D. Lajiness, MD, PhD
Indiana University School of Medicine

Title: *Maternal lipids regulate neonatal dendritic cells during development of allergic disease*

Mentor: Joan Cook-Mills, PhD

Personal Statement:
I am a second-year Neonatal-Perinatal Medicine fellow at Indiana University dedicated to contributing to medicine and science as a physician-scientist. My training has been driven by a passion for developmental biology and for understanding, on a cellular signaling level, the diseases affecting our youngest patients. My work caring for neonates in the Neonatal Intensive Care Unit has reinforced how critical the time period before and immediately after birth is for establishing a trajectory towards health or illness. Clinically, we have an innate sense that maternal factors affect the in-utero environment and the fetus, but many of these factors remain incompletely understood. We do know that children of mothers with allergic disease have an allergic predisposition themselves and that this begins in utero. This is important as allergic disease represents an increasingly significant morbidity and mortality for vulnerable pediatric populations including preterm infants. Since starting fellowship, I have worked in Dr. Joan Cook-Mills' lab to investigate allergic disease and how offspring susceptibility to the development of allergy is modulated in utero by maternal factors. My project focuses on how maternal lipids (both those the mom produces as a result of her allergic disease as well as those she consumes in her diet) affect her offspring and their susceptibility to allergic disease. I am incredibly honored to be one of the recipients of The Marshall Klaus Perinatal Research award this year and look forward to being a small part of Dr. Klaus' legacy of supporting physician-scientists.

Abstract:
The incidence of allergic disease and asthma have been rapidly increasing over the past several decades. Our lab is interested in the maternal factors that alter offspring responsiveness to allergen. Maternal lipids are of particular interest because lipids are altered during allergic inflammation, can regulate inflammation, and are transported across the placenta to the fetus. Previous studies have shown that dendritic cells (DCs) are sufficient to promote allergic predisposition in offspring of allergic mothers. We have identified glucosylceramide (GlcCer) as an important maternal lipid involved in the establishment of allergy in offspring of allergic mothers. This effect is modulated by tocopherol isoforms present in the maternal diet. It is not known whether lipids of allergic mothers regulate dendritic cell maturation and phenotype directly or which signaling pathways may be involved. We hypothesize that GlcCer increases the number of monocyte-derived DCs (mDCs). We further hypothesize that tocopherol isoforms modulate the effect of GlcCer and that signaling pathways involving Protein Kinase C (PKC) play an important role in this effect. We demonstrate in vitro that exposure

to GlcCer and γ-tocopherol increases mDCs while administration of α-tocopherol mitigated the GlcCer-induced increase in mDCs. These data shed light on mechanistic effects of environmental factors including maternal lipids on DC populations and how this may serve to promote allergic disease in offspring. Understanding these targets and the signaling pathways which mediate their effects on cell types such as DCs is critical to our understanding and management of allergic disease in our youngest patients.

Health Services Research Awardees

Daria C. Murosko, MD, MPH
Children's Hospital of Philadelphia

Title: *The COVID-19 pandemic and effect on infant rehospitalizations*

Mentors: Heather H. Burris, MD, MPH & Scott A. Lorch, MD, MSCE

Personal Statement:
I aim to become an academic physician-researcher whose clinical work informs health services research focused on racial and ethnic health disparities in neonates. My interest in this area was sparked during my master's degree in Public Health at the University of Pennsylvania, where my thesis demonstrated an association between the maternal experience of residential racial segregation and intraventricular hemorrhage. Throughout my residency and chief residency in the Boston Combined Residency Program, I saw how the legacy of systemic racism, ongoing oppression, and chronic disinvestment led to poor health outcomes in children. I also saw that a commitment to providing exceptional care without exception is the first step to creating health equity for children and their communities. This inspired my focus on discovering and eliminating drivers of neonatal health disparities, utilizing research as my tool for making change. Health services research allows investigators to untangle complex factors that lead to inequities, identify areas for potential solutions, and evaluate the impact of our healthcare policies.

While the COVID-19 pandemic represents the most devastating public health challenge of our lifetimes, it also provides an opportunity to invest in rigorous research to understand the unique interplay between healthcare and social determinants of health. With the support of my mentors, Dr. Heather Burris and Dr. Scott Lorch, I will evaluate the impact that the pandemic has had on infant rehospitalizations, a marker of infant wellbeing. Though my work will look broadly at a national sample, I will specifically assess outcomes for infants in communities of color and those living in poverty. In addition to enabling this investigation into critical questions about the consequences of the pandemic, the Marshall Klaus Award will serve as the launching point for my career as an academic neonatologist working to eliminate racial and ethnic disparities in neonates.

Abstract:
The coronavirus 2019 (COVID-19) pandemic has created unprecedented changes in the institutions that ensure pediatric health and wellbeing. Unlike older children, infants are also vulnerable to disruptions in perinatal care, both prenatally and during the birth hospitalization. Low-income communities and communities of color have been disproportionately affected with respect to infection, mortality, and economic hardships during the COVID-19 pandemic. Additionally, infection rates and public responses also vary across regions and over time. Infant hospitalization from home, or rehospitalization, is an indicator of poor health that may be modifiable by healthcare practices, reflecting not only the care provided during the birth hospitalization, but also outpatient services and community supports. Using data from a large, national sample, our project seeks to delineate how infant wellbeing has changed over the course of the COVID-19 pandemic as measured by infant rehospitalization in the first two weeks of life, and to identify sociodemographic and geographic factors that differentially affect risk of rehospitalization. As 3.7 million full-term infants are born annually in the United States, identifying processes that protect infant health and understanding outcome variation by group and location have implications for public health and policies, even after the COVID-19 pandemic is controlled.

Genevieve G. Guyol, MD
Boston Children's Hospital

Title: *Determinants of school readiness among low birthweight children*

Mentor: Margaret (Meg) Parker, MD, MPH

Personal Statement:
I am interested in a career as an academic neonatologist who uses health services research methodology to characterize racial and socioeconomic disparities in the educational outcomes of preterm infants and designs evidence-based interventions that improve health and educational equity. I bring a unique perspective to this work as a former early elementary special education teacher with a master's degree in special education. As a resident in the Urban Health and Advocacy Track of the Boston Combined Residency Program in Pediatrics, I received additional training on how to research health disparities and apply findings to inform programmatic and policy change. Now, as a fellow in neonatal-perinatal medicine, I will investigate determinants of school readiness in low birthweight infants and how family protective factors and community supports modify the effects of special healthcare needs and family social disadvantage on school readiness. This study represents an important first step toward development of effective community interventions aimed at improving school readiness among a highly vulnerable group, low birthweight children. The Marshall Klaus Award will support my career development by helping me gain foundational epidemiological and

biostatistical skills that I will need to become an independent health services researcher. In the future, I hope to build upon this work by designing and evaluating evidence-based interventions that promote early educational and health equity among at-risk newborns.

Abstract:
School readiness at kindergarten entry is a strong predictor of later academic achievement and therefore is linked to economic opportunities and subsequent risk of chronic disease in adulthood. Preterm and low birthweight infants represent a highly vulnerable pediatric population at risk for poor school readiness. First, these children are more likely to have special healthcare needs such as developmental and medical disabilities which have been associated with poor academic outcomes. Second, social characteristics associated disproportionately with prematurity and low birthweight, such as non-English-speaking or low-income households and non-Hispanic Black race, are also associated with decreased academic achievement. Conversely, family protective factors, such as family resilience, household routines, and parental emotional support and community supports, including the medical home and early intervention, are associated with increased school readiness in other vulnerable populations of children. The overarching goal of this study is to use a nationally representative sample to investigate determinants of school readiness among the medically and socially high-risk population of low birthweight children. First, I will determine the national prevalence of school readiness among low birthweight children 3- to 5-years old. Second, I will examine the extent to which special healthcare needs, family social factors, community supports, and family protective factors are associated with school readiness in low birthweight children. Finally, I will explore the extent to which community supports and family protective factors modify the effects of special healthcare needs and family social disadvantage on the school readiness of low birthweight children.

Newborn Medicine Education Awardee

Kirsti E. Martin, MD
University of Massachusetts Medical School

Title: *Assessing podcasts as an adjunct curricular tool for delivering a neonatology curriculum to pediatric residents*

Mentor: D. Reid Evans, PhD

Personal Statement:
I am currently a second year Neonatal-Perinatal Medicine fellow at the University of Massachusetts in Worcester, Massachusetts, where I also completed my residency in pediatrics. My career goal is to become an academic neonatologist with a focus in quality improvement and medical education, specifically in curriculum development and evaluation research. To help me in these

endeavors, I am currently pursuing a Master of Medical Education through the University of Cincinnati. Using the knowledge, I have learned thus far in the program, I have focused my scholarly efforts on creating learning tools to assist resident physicians in digesting the complex disease processes of neonates so they can translate physiology to bedside evaluation and treatment. I have become interested in podcasts as a tool in medical education due to their portability and brevity, allowing trainees to create time for learning where little extra time exists. My proposed project will compare the knowledge gain and retention between podcast lessons and traditional lectures used to teach topics in neonatology to pediatric residents. Under the guidance of my mentor Dr. Reid Evans, whose expertise is qualitative educational research, I will use semi-structured interviews to assess attitudes of pediatric residents towards the integration of podcasts into an official neonatology curriculum. The results of this project will inform the utility of a podcast neonatology curriculum to augment the existing curriculum. This project will give me valuable experience in instructional design as well as quantitative and qualitative evaluation of interventions in medical education. I am honored to be a recipient of the Marshall Klaus Award and am thankful to be a part of this incredibly supportive neonatology community.

Abstract:
The breadth of topics a pediatric trainee must be familiar with in order to successfully care for infants in the NICU is wide, and the topics they are formally taught during their rotations vary, leaving gaps in knowledge. As resident and educator time is limited during clinical rotations in the NICU, adding more didactic lecture time to the curriculum to better cover core topics is not feasible. Instead, we are creating an audio podcast curriculum to efficiently address curricular gaps. Medical education audio podcasts have become a popular means of extracurricular education for residents of varying specialties. However, there are few published studies assessing the effect of audio-only podcasts on knowledge gain. The goal of this project is to assess the utility of audio podcasts as an adjunct curricular tool for delivering a neonatology curriculum to pediatric residents. We aim to do this by assessing knowledge gain and retention after a podcast lesson and comparing this to knowledge gain and retention after a traditional didactic lecture. A secondary aim is to obtain learner insight regarding the feasibility and use of such a curriculum using qualitative research methods. We hypothesize that knowledge gain and knowledge retention from a podcast lesson will not be significantly different from that resulting from a traditional lecture. We also hypothesize that pediatric trainees will approve of the use of podcasts to supplement traditional didactic learning in an official curriculum. Results supporting our hypotheses would support the creation of supplemental podcast curricula for use in graduate medical education.