National Institute of Environmental Health Sciences

NIEHS 50 YEARS 1966-2016

Discovering how the environment affects people in order to promote healthier lives.

Awardee Symposium

May 17-18, 2016

Tuesday, May 17 • 8:30 a.m. – 5:30 p.m.

Wednesday, May 18 • 8:30 a.m. – 1:00 p.m.

NIEHS Building 101, Rodbell Auditorium

National Institutes of Health • U.S. Department of Health and Human Services

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Table of Contents

Agenda 1

Presentation Abstracts 3

Biographies 15

Participants 24

National Institute of Environmental Health Sciences

ONES
Outstanding New Environmental Scientist

Awardee Symposium

May 17-18, 2016

NIEHS Building 101, Rodbell Auditorium • Research Triangle Park, N.C.

AGENDA

Tuesday, May 17, 2016

8:00 a.m. Registration

8:30 a.m. **Welcome**
Linda Birnbaum, Director, National Institute of Environmental Health Sciences (NIEHS)

8:40 a.m. **NIEHS 101: Introduction to the National Institute of Environmental Health Sciences**
Gwen Collman, Director, Division of Extramural Research and Training, NIEHS

9:00 a.m. **SESSION ONE: Moderator, Fred Tyson**, Genes, Environment, and Health Branch, NIEHS

9:05 a.m. **Genetic and Developmental Susceptibility to Mitochondrial Toxicity**
Joel Meyer, Duke University

9:25 a.m. **Role of DNA Methyltransferases in Toxicant-induced Alterations in DNA Methylation**
Neelakanteswar Aluru, Woods Hole Oceanographic Institution

9:40 a.m. **Protecting Children Around the Globe From the Harms of Toxic Metals**
Rebecca Fry, University of North Carolina at Chapel Hill

10:00 a.m. **The Impact of Arsenic on the Gut Microbiome: From 16S Sequencing to Functional Characterization**
Kun Lu, University of Georgia

10:15 a.m. Break

10:45 a.m. **Pathogenic Alterations of Manganese Neurobiology and Neurotoxicity in Huntington's Disease**
Aaron Bowman, Vanderbilt University

11:05 a.m. **The Mechanism Underlying Epigenetic Effects of Exposure to Hydrogen Sulfide**
Dana Miller, University of Washington

11:25 a.m. Discussion

11:45 a.m. Lunch (Executive Conference Room)

1:00 p.m. **SESSION TWO: Moderator, Janice Allen**, Scientific Review Branch, NIEHS

1:05 p.m. **Role of Autophagy Pathways in UVA-induced Skin Cancer**
Yu-Ying He, University of Chicago

1:25 p.m. **The Role of Nrf2 in Bisphenol-induced NAFLD**
Angela Slitt, University of Rhode Island

1:45 p.m. **The Role of Nrf2 in Immune Cell Polarization and Allergy**
Cheryl Rockwell, Michigan State University

2:00 p.m. **A Flame Retardant Story: Bridging Science With Policy**
Heather Stapleton, Duke University

2:20 p.m. Discussion

2:40 p.m. Break

2:55 p.m. **SESSION THREE: Moderator, Patrick Mastin**, Deputy Director, Division of Extramural Research and Training, NIEHS

3:00 p.m. **Investigating Aryl Hydrocarbon Receptor-Cofactor Interactions That Mediate Toxicity**
Daniel Gorelick, University of Alabama at Birmingham

3:15 p.m. **Pattern Recognition Receptors Mediate the Lung Injury-Bone Loss Inflammatory Axis to Organic Dust Exposure(s)**
Jill Poole, University of Nebraska Medical Center

3:30 p.m. **Role of Bio-coronas in the Enzymatic Oxidation of Single-walled Carbon Nanotubes**
Alexander Star, University of Pittsburgh

3:50 p.m. **Integrative Nanotoxicology: Linking Rapid Assays and Informatics to Understand Nanomaterial-Biological Interactions**
Stacey Harper, Oregon State University

4:10 p.m. **Maternal Nanomaterial Inhalation During Gestation: Fetal Microvascular Consequences and Transcriptomic Dysregulations**
Timothy Nurkiewicz, West Virginia University School of Medicine

4:30 p.m. Discussion

5:00 p.m. Adjourn

Wednesday, May 18, 2016

8:00 a.m. Registration

8:30 a.m. **SESSION FOUR: Moderator, Janice Allen**, Scientific Review Branch, NIEHS

8:35 a.m. **Developmental Exposure to DDT Impairs Energy Expenditure Leading to Insulin Resistance**
Michele La Merrill, University of California, Davis

8:50 a.m. **Periconceptional Biomonitoring**
Christina Porucznik, University of Utah School of Medicine

9:10 a.m. **Utilizing Mouse Genetic Diversity to Identify Mechanisms of Environmentally-induced Airway Disease**
Samir Kelada, University of North Carolina at Chapel Hill

9:25 a.m. Discussion

9:45 a.m. Break

10:05 a.m. **SESSION FIVE: Moderator, Jonathan Hollander**, Genes, Environment, and Health Branch, NIEHS

10:10 a.m. **Does Arsenic Increase Risk of Neural Tube Defects Among a Highly Exposed Population**
Maitreyi Mazumdar, Harvard Medical School

10:25 a.m. **Neurotoxicity of Airborne Particles in the Setting of Stroke and Cerebral Hypoperfusion**
William Mack, University of Southern California

10:40 a.m. **Hereditary Parkinsonism Due to Loss of Function Mutations in a Manganese Efflux Transporter**
Somshuvra Mukhopadhyay, University of Texas at Austin

10:55 a.m. **Exposure to Welding Fumes in a Local Cohort: A Longitudinal Neuroimaging Study**
Ulrike Dydak, Purdue University

11:15 a.m. **Developmental Pesticide Exposure Causes Long-term Decreases in Sodium Channels and Glutamate Release in the Hippocampus**
Jason Richardson, Northeast Ohio Medical University

11:35 a.m. Discussion

12:05 p.m. **Closing Remarks, Carol Shreffler**, Exposure, Response, and Technology Branch, NIEHS

12:15 p.m. Adjourn

Presentation Abstracts

Neelakanteswar Aluru
Woods Hole Oceanographic Institution
Role of DNA Methyltransferases in Toxicant-induced Alterations in DNA Methylation

Epigenetic changes, such as DNA methylation, have been implicated in the persistent effects of developmental exposure to toxicants, but the mechanisms by which toxicants alter methylation processes are not well understood. We are testing the hypothesis that toxicants alter patterns of de novo DNA methylation through their effects on the function of DNMT3s. We are conducting these studies using zebrafish, a well-established developmental and toxicological model. Due to a whole-genome duplication in the teleost fish lineage, zebrafish have multiple dnmt3a and dnmt3b genes, providing a unique opportunity for dissecting multiple functions of these DNMTs, which in humans would be accomplished by a single ortholog. In vitro episomal methylation assay results suggest that dnmt3a paralogs show sequence specific DNA methylation. Using dnmt3aa and dnmt3ab-null zebrafish, we determined their target genes and DNA methylation patterns using RNAseq and RRBS, respectively. Studies are currently underway to determine toxicant-induced changes in DNA methylation.

Aaron Bowman
Vanderbilt University
Pathogenic Alterations of Manganese Neurobiology and Neurotoxicity in Huntington's Disease

Manganese (Mn) is an essential metal that is both critical for neuronal function and toxic in excess. Altered Mn homeostasis has been implicated in the pathophysiology of some neurological diseases, including Huntington's disease (HD). Expansion of the polyglutamine tract in Huntingtin (HTT) leads to selective neurodegeneration of the striatum in HD. Expression of mutant HTT is also associated with a Mn accumulation deficit in the striatum of YAC128Q mice, a HD model. This deficit in neuronal Mn accumulation is associated with protection from Mn neurotoxicity. Thus, impaired neuronal Mn handling may contribute to HD pathophysiology, as well as provide a novel tool for understanding Mn neurotoxicity. Results from two studies will be presented. First, HD is associated with alterations in urea cycle metabolism and genetic models exhibit deficient manganese (Mn) handling. We find reduced ex vivo striatal arginase activity at a prodromal stage in an HD mouse model that is reversed by in vivo exposure to the arginase cofactor Mn, coinciding with post-transcriptional increases in ARG2 protein. The data support a hypothesis that lack of bioavailable Mn contributes to HD striatal pathophysiology. Second, we present evidence that cross-talk between the mTOR, phosphotidylinsolitol three-kinase, and p53 signaling networks regulate Mn transport and toxicity via an autophagy-mediated mechanism. Together, these studies implicate a novel pathway in neuronal Mn homeostasis altered in HD. In future studies, we plan to examine these pathways in other cell models, including patient hiPSC-derived striatal and cortical neuroprogenitors and neurons. human aging and potentially aid in the amelioration of age-related disease, extending the number of healthy and active years of life.

Ulrike Dydak
Purdue University
Exposure to Welding Fumes in a Local Cohort: A Longitudinal Neuroimaging Study

Chronic high exposure to welding fumes containing the metal manganese (Mn) is a risk factor to develop a Parkinsonian disorder. But how high is high? Having been intrigued by significantly altered GABA levels in the brains of Chinese smelters exposed to Mn, we embarked to establish a *local* cohort of welders, working under typical U.S. exposure conditions, and follow them longitudinally – The Wabash Study. This talk will give an overview of the results four years into this study, in which we follow the welders with personal air sampling, exposure modeling, measures of biomarkers (toenails), cognitive and motor testing, and novel neuroimaging techniques to assess brain Mn and brain iron levels, changes in brain volume, as well as metabolic and neurotransmitter changes in the brain – in particular basal ganglia GABA levels and dopamine function. We have found strong relations between imaging markers, such as brain Mn and GABA levels to exposure, but also to effects such as motor and cognitive impairment. Further, we could show that GABA levels only rise above a certain threshold of exposure, and are reversible with decreasing exposure levels. A population of Idiopathic Parkinson Disease (IPD) patients, undergoing all the same study procedures, has been compared to the welders as a positive control group to learn more about the similarities and differences of Mn toxicity to IPD.

Rebecca Fry
University of North Carolina at Chapel Hill

Protecting Children Around the Globe From the Harms of Toxic Metals

Children worldwide are exposed to harmful levels of toxic metals potentially through their drinking water or from food. Exposure to metals during key developmental periods has been associated with a variety of adverse health effects, including both cancer and non-cancer endpoints. Many of these health effects present during adulthood support the developmental origins of health and disease hypothesis. There is considerable interest in identifying the underlying molecular mechanisms that relate early-life exposure to toxic metals with both early and later life disease. Molecular epidemiologic studies from Mexico have elucidated a potential molecular basis for these latent life health impacts such as altered inflammatory signaling at the gene and protein level. Evidence supports a role for epigenetic (e.g. CpG methylation, miRNAs) processes as transcriptional mediators that underlie these altered inflammatory-responses. Research from our prospective arsenic-exposed cohort in Mexico highlights the complex factors that influence children's health. These factors include maternal metabolism of arsenic, maternal genotype related to arsenic metabolism, and epigenetic modification with functional impact on gene expression. An increasing number of studies are highlighting the harms of exposure to metal mixtures on children's health, including populations in the U.S. and abroad. Taken together, these ongoing studies identify a role for epigenetic reprogramming, the impact of metal mixtures, and the identification of developmental windows of vulnerability.

Daniel Gorelick
University of Alabama at Birmingham

Investigating Aryl Hydrocarbon Receptor-Cofactor Interactions That Mediate Toxicity

Exposure to byproducts of industrial combustion, such as dioxins and polycyclic aromatic hydrocarbons (PAH), causes multi-organ phenotypes, including cardiotoxicity. Understanding the signaling pathways by which such compounds cause toxicity is crucial for predicting acceptable exposure levels and for reversing the effects of adverse exposure. Dioxins and PAHs activate aryl hydrocarbon receptors (AHR) and ligand-dependent transcription factors that recruit cofactors to DNA and directly regulate gene expression. However, our knowledge of AHR cofactor recruitment and regulation is limited. The goal of this proposal is to identify the transcriptional cofactors by which AHR mediates cardiotoxicity and test the hypothesis that AHR ligands modulate cofactor recruitment. Structurally distinct AHR ligands induce differential transcriptional responses, suggesting that different ligands bind AHR and recruit different cofactors, leading to differential gene expression. Consistent with this hypothesis, our preliminary data in zebrafish embryos, an established model of dioxin and PAH toxicity, indicates that AHR-mediated cardiotoxicity is ligand dependent. Acute exposure to the AHR ligand 2,3,7,8-tetrachlorodibenzodioxin (TCDD), but not the AHR ligands 3-methylcholanthrene (3MC) or benzo(a)pyrene (BaP), caused cardiac edema and heart malformation. While the idea of ligand-dependent cofactor recruitment has been established for the estrogen receptor family of transcription factors, whether AHRs act in a similar manner is less well understood. Approximately 20 AHR cofactors have been identified, yet aside from the ARNT protein, it is not known which cofactors are required for cardiotoxicity or whether cofactors are recruited in a ligand-dependent manner.

We propose that TCDD-AHR recruits a different complex of transcriptional cofactors than either 3MC-AHR or BaP-AHR and that differential cofactor recruitment underlies ligand-dependent gene expression and cardiotoxicity. In Aim 1, we will identify AHR cofactors required for TCDD-dependent cardiotoxicity. Using genetic approaches, we will test whether candidate cofactors are required for TCDD cardiotoxicity. We will also use unbiased biochemical and proteomic approaches to purify TCDD-AHR from the developing heart and identify interacting proteins. In Aim 2, we will identify ligand-dependent AHR cofactors from whole embryos and select tissues. Using zebrafish 'knock-in' strains with epitope tags inserted into endogenous AHR signaling genes, we will test whether known cofactors differentially interact with endogenous AHR depending on the AHR ligand. We will also purify AHR from zebrafish embryos and use mass spectrometry to identify cofactors bound to AHR following exposure to TCDD, 3MC, or BaP. Results from this proposal will identify a mechanism of TCDD cardiotoxicity and will provide a foundation for elucidating how the structure of AHR changes in response to differential ligand binding. Furthermore, our results will increase our understanding of the role of cofactors in endogenous AHR signaling, which may lead to improved treatment for diseases where AHR signaling is abnormal.

Stacey Harper
Oregon State University

Integrative Nanotoxicology: Linking Rapid Assays and Informatics to Understand Nanomaterial-Biological Interactions

Understanding the inherent and conditional factors associated with nanomaterial toxicity is critical to the development nanotechnologies that pose minimal threats to humans and the environment over the life cycle of the nanomaterial. Currently, vast toxicological data gaps exist regarding the risks associated with nanomaterial exposure, and the principal characteristics that may be predictive of nanomaterial interactions with biological systems have yet to be identified due of this lack of information. Thus, rapid testing strategies are immediately necessary to identify the specific features of nanomaterials that result in toxicity in order to mitigate risks from exposure and define structure-property relationships that can be used to predict nanomaterial hazard *in lieu* of empirical data. We have compared the results of several different models built to predict toxicity from the open-source data on nanomaterial toxicity to embryonic zebrafish *(Danio rerio)* found in the Nanomaterial-Biological Interactions (NBI) knowledgebase at Oregon State University. Model comparisons included the ABMiner predictive models, MATLAB clustering analysis and the use of Self-Organizing Map (SOM) based consensus clustering conducted on the data in the NBI knowledgebase (nbi.oregonstate.edu).

Overall results suggest that exposure concentration and outermost surface chemistry (and thus surface charge) both should be considered in conjunction with the core composition of nanomaterials when trying to develop predictive models for developing zebrafish. Thus, classification of nanomaterials by simple descriptors such as core composition may not be sufficient for predicting nanomaterial toxicity or managing nanomaterial risks. Computational analysis of the data from OSU's Nanomaterial-Biological Interactions knowledgebase has revealed that surface chemistry is one of the key features that determine nanomaterial toxicity.

Yu-Ying He
University of Chicago

Role of Autophagy Pathways in UVA-induced Skin Cancer

Skin cancer is the most common type of cancer in the US, with an estimated 3.5 million cases diagnosed each year. Exposure to ultraviolet (UV) radiation, namely UVA (320-400 nm) and UVB (290-320 nm), is the major risk factor for the development of skin cancer. UVA is 20-fold more abundant in sunlight than UVB and is the major component of tanning beds. However, the mechanism of UVA's contribution to skin cancer remains unclear. One putative effector of UVA in skin cancer is p62, a selective autophagy cargo protein and substrate. We have found that UVA upregulates p62 expression, independent of autophagy, by inducing transcription of p62. p62 is upregulated in many types of cancer, including squamous cell carcinoma (SCC) and melanoma, and can facilitate the activation of a number of pathways to promote cell proliferation, invasion, inflammation, and survival. We have previously shown that p62 stabilizes Twist1 to promote invasion and proliferation, indicating p62 can function independent of autophagy to promote tumor progression. However, the function of p62 in UVA-induced tumor progression is unknown. Using a candidate gene approach, we identified a novel relationship between p62 and cyclooxygenase-2 (COX-2), which suggests a putative function for p62 in UVA-induced skin cancer. COX-2 is a prostaglandin synthase often overexpressed in cancer, where it correlates with poor prognosis. COX-2 promotes cell proliferation and survival, and inhibition of COX-2 prevents skin cancer development. COX-2 protein expression is induced concomitantly with p62 in response to UVA and knockdown of p62 prevents induction of COX-2 by UVA.

Therefore, we hypothesized that p62 transcription is induced by UVA to promote stability of COX-2 and, consequently, tumor progression. We show here that p62 stabilizes COX-2 protein and consequently increases PGE^2 levels in vitro. In a syngeneic mouse model of SCC, knockdown of p62 significantly slows tumor growth. As both p62 and COX-2 are critical for tumor progression in the skin and other epithelial cancers, understanding the link between these proteins will unravel a signaling axis central to cancer progression and may provide new opportunities for developing improved prevention and therapies by targeting p62/COX-2.

Samir Kelada
University of North Carolina at Chapel Hill

Utilizing Mouse Genetic Diversity to Identify Mechanisms of Environmentally-induced Airway Disease

Identifying the genetic determinants of variation in response to exposure to environmental health hazards provides insight into sources of inter-individual variation and toxicologic mechanisms. However, identifying such gene-environment interactions is a difficult task in epidemiologic studies due to a number of vexing methodological and statistical issues; thus, complementary approaches are clearly needed. We aim to identify genetic loci that control response to exposures associated with the development of allergic airway disease, namely allergens and traffic-related air pollution (TRAP), using a new mouse genetic reference population known the Collaborative Cross (CC). The CC is a panel of recombinant inbred strains of mice derived from eight-way crosses using five classical inbred strains (C57BL/6J, 129S1/SvImJ, A/J, NOD/ShiLtJ, and NZO/H1LtJ) and three wild-derived inbred strains (WSB/EiJ, PWK/PhJ, and CAST/EiJ). The abundant genetic variation present in CC lines makes this a unique and powerful tool for detection of gene-environment interactions.

As a proof of concept, we examined responses to a single exposure, house dust mite allergen. We phenotyped 150 nascent CC lines for airway inflammation (cell counts in lavage fluid), mucin protein levels, and also measured gene expression in whole lung RNA using microarrays. After high density, genome-wide genotyping, we identified genetic loci that control inflammation phenotypes and gene expression, using quantitative trait locus (QTL) mapping. We identified QTL for important disease phenotypes, including inflammation and mucus production. Using gene expression data in conjunction with bioinformatic tools and resources, we identified a small number of promising candidate genes for these loci. These novel candidate genes suggest new mechanisms by which allergen exposure affects lung immunobiology. We are now examining responses to exposure to both the ozone and house dust mite allergen to identify loci that affect the development of allergic inflammation as a function of exposure to TRAP.

Our preliminary results indicate that when ozone exposure alters the lung such allergic inflammation is enhanced. These results begin to address the important issue about how TRAP exposure can influence the development of asthma and set the stage for a comprehensive genetic analysis to identify genes that mediate this process. More broadly, our results demonstrate that the CC is a powerful mouse genetics resource that can be used to identify the novel mechanisms by which exposures to environmental health hazards can affect health.

Michele La Merrill
University of California, Davis

Developmental Exposure to DDT Impairs Energy Expenditure Leading to Insulin Resistance

Dichlorodiphenyltrichloroethane (DDT) has been used extensively to control malaria, typhus, body lice and bubonic plague worldwide, until countries began restricting its use in the 1970s. Its use in malaria control continues in some countries according to recommendation by the World Health Organization. Individuals exposed to elevated levels of DDT and its metabolite dichlorodiphenyldichloroethylene (DDE) have an increased prevalence of diabetes and insulin resistance. Here we hypothesize that perinatal exposure to DDT disrupts metabolic programming leading to impaired metabolism in adult offspring. To test this, we administered DDT to C57BL/6J mice from gestational day 11.5 to postnatal day 5 and studied their metabolic phenotype at several ages up to nine months. Perinatal DDT exposure reduced core body temperature, impaired cold tolerance, decreased energy expenditure, and produced a transient early-life increase in body fat in female offspring. When challenged with a high fat diet for 12 weeks in adulthood, female offspring perinatally exposed to DDT developed glucose intolerance, hyperinsulinemia, dyslipidemia, and altered bile acid metabolism. Perinatal DDT exposure combined with high fat feeding in adulthood further impaired thermogenesis as evidenced by reductions in core temperature and in the expression of numerous RNA that promote thermogenesis and substrate utilization in the brown adipose tissue of adult female mice. These observations suggest that perinatal DDT exposure impairs thermogenesis and the metabolism of carbohydrates and lipids which may increase susceptibility to the metabolic syndrome in adult female offspring.

Kun Lu
University of Georgia

The Impact of Arsenic on the Gut Microbiome: From 16S Sequencing to Functional Characterization

The gut microbiota has important functions in metabolic processing, energy production, immune cell development, food digestion, epithelial homeostasis, and so forth. Mounting evidence indicates that dysregulated gut microflora contributes in a significant way to a variety of diseases, including diabetes, obesity, cardiovascular diseases, allergies, inflammatory bowel disease, and others. The composition of the gut microbiome is highly diverse, and this diversity can be readily affected by external factors, such as environmental exposures. Arsenic exposure affects large human populations worldwide, with contamination of drinking water by geological sources of inorganic arsenic being the primary route of exposure. Hundreds of millions of people around the world, especially in South and East Asia, drink water with arsenic levels that far exceed the 10-µg/L guideline established or accepted by the World Health Organization and the U.S. EPA. However, the functional impact of arsenic exposure on the gut microbiome is poorly understood. We have demonstrated that arsenic exposure not only induced dose-dependent gut microbiome composition changes, but also altered functional metagenome in C57BL/6 mice. Moreover, we discovered that arsenic caused differential gut microbiome responses in males and females, which may provide important mechanistic insight into sex-specific susceptibility of human diseases arising from exposure to arsenic and beyond.

William Mack
University of Southern California

Neurotoxicity of Airborne Particles in the Setting of Stroke and Cerebral Hypoperfusion

Inflammation is believed to play an important role in the progression of cerebral ischemia. Air pollution is a potent environmental source of inflammation and oxidative stress. This program examines the joint effects of inhalation of airborne particulate matter (PM) derived from urban traffic on the progression of brain injury following acute stroke and/ or Chronic Cerebral Hypoperfusion (CCH). Urban PM is collected with a particle sampler situated near the CA-110 Freeway in Los Angeles. Collected aerosols represented a mix of fresh PM, predominantly from vehicular traffic. These samples are then distilled to pollutant nanoparticles (<200 nm) and re-aerosolized for administration to mice through exposure chambers for five hours a day, three times per week. In the first study, mice were exposed to aerosolized PM (n=12) or filtered air (n=12) for forty-five cumulative hours and then underwent middle cerebral artery ischemia (35 minutes)/ reperfusion (I/R). Infarct volumes and neurological function were compared between mice exposed to PM and filtered air on postoperative day 1. Following cerebral I/R, mice exposed to PM demonstrated larger infarct volumes ($p<0.05$) and less favorable neurological scores ($p<0.05$) when compared to mice exposed to filtered air. Mortality was higher for the PM cohort (8.3% vs. 0%, p=ns). This data establishes a detrimental effect of particulate matter in the setting of acute stroke and suggests a potential inflammatory mechanism of action. The second study establishes baseline PM exposure data to utilize in the assessment of the joint effects of PM and CCH on white matter toxicity and neurocogntitive decline. Mice were exposed to aerosolized PM (n=18) or filtered air (n=18) for one hundred-fifty cumulative hours. White matter toxicity (Kluver Barrera stain), inflammation (reactive astrocytes/ GFAP and microglia/ IBA-1), and neurobehavior (8 arm radial maze/ novel object recognition) were assessed and compared between groups. The nPM exposed mice demonstrated significant increases in IBA-1 ($p<0.05$) in the region of the corpus callosum (but not GFAP). Further, subtle white matter injury in the corpus callosum ($p<0.05$) and neurocognitive deficits in working memory (p=ns) were evident. These data establish an inflammatory response and early white matter – neurocognitive changes following nPM exposure. Ongoing studies will assess the joint effects of this nPM exposure in the setting of CCH.

Maitreyi Mazumdar
Harvard Medical School

Does Arsenic Increase Risk of Neural Tube Defects Among a Highly Exposed Population

Neural tube defects are debilitating birth defects that occur when the developing neural plate fails to close in early gestation. In humans, neural tube defects are characterized by high rates of mortality and lifelong disabilities, including paralysis, hydrocephalus, and epilepsy. Arsenic induces neural tube defects in animal models, but whether environmental arsenic exposure increases risk of neural tube defects in humans is unknown.

Studies investigating arsenic's role in human neural tube defects have been hampered by the relative rarity of these birth defects in the United States and the lack of individual measures of exposure. Though difficult to study, it is critical to understand the role of arsenic exposure in neural tube defects, as exposure to arsenic is common in the United States and throughout the world, and reduction of arsenic exposure and mitigation of arsenic toxicity may present new opportunities for prevention.

Our long-range goals are to develop novel screening strategies to identify populations at high risk of neural tube defects and to direct the development of more effective preventive interventions. In this application, we present preliminary data that suggest that folic acid supplementation, currently the primary strategy for neural tube defect prevention, may be less effective in preventing neural tube defects in areas with high arsenic exposure.

We now propose a comprehensive set of studies that leverage a unique case ascertainment program in rural Bangladesh, a country that is currently experiencing an epidemic of arsenic poisoning through contaminated drinking water. We plan to understand how arsenic influences risk of neural tube defects in humans through mechanism that include maternal glucose and folate metabolism, as well as epigenetic effects. We will also test whether sweat chloride concentration, a potential new biomarker for arsenic toxicity, can be used to identify women at higher risk for having a child affected by neural tube defect. Finally, in an exploratory aim, we propose to collect and study dural tissue, obtained at the time of surgical closure of the defect, to provide insight into the epigenetic mechanisms by which prenatal arsenic exposure affects the developing nervous system.

These studies explore mechanisms by which arsenic may increase risk of neural tube defects in humans, and use a unique population with high arsenic exposure in order to test hypotheses. We expect these studies to identify populations at high risk of neural tube defects due to environmental exposures as well as direct development of novel screening strategies for maternal risk. In addition, results from these studies will provide fundamental new knowledge regarding the effect of arsenic on the developing nervous system and the mechanisms that affect developmental neurotoxicity.

Joel Meyer
Duke University

Genetic and Developmental Susceptibility to Mitochondrial Toxicity

The long-range goal of this research is to elucidate the role of persistent mitochondrial DNA damage in neurodegeneration. There is now strong evidence that neurodegeneration in the majority of Parkinson's Disease cases is the result of the interplay of genetic differences with environmental exposures (gene-environment interactions), but neither the genes nor the environmental exposures involved are well understood. We are investigating the novel hypothesis that some important environmental toxins contribute to neurodegeneration by causing persistent mitochondrial DNA damage, and that genetic deficiencies in the processes that handle such damage lead to greater susceptibility. Mitochondrial DNA is more sensitive than nuclear DNA to many insults, and there is no apparent repair pathway for handling mitochondrial DNA damage caused by important environmental genotoxins such as polycyclic aromatic hydrocarbons and ultraviolet radiation. We will test the role of such damage in causing neurodegeneration as a result of exposure during key developmental time periods. The specificity of this effect will be tested using innovative new transgenic strains of Caenorhabditis elegans. We will also test the hypothesis that specific genes involved in mitochondrial fusion and autophagy protect against such damage, taking advantage of the genetic and molecular tools available in Caenorhabditis elegans.

There is now strong evidence that neurodegeneration in most Parkinson's disease cases is the result of the combined effects of genetic differences and environmental exposures (gene- environment interactions), but neither the genes nor the environmental exposures involved are well understood. We will test the hypothesis that important, common environmental toxins contribute to neurodegeneration act by causing persistent mitochondrial DNA damage during vulnerable periods of development, and that genetic deficiencies in the processes that handle such damage lead to greater susceptibility. If this is the case, better regulation of such chemicals could greatly reduce the incidence of Parkinson's disease and other neurodegenerative diseases as well.

Dana Miller
University of Washington

The Mechanism Underlying Epigenetic Effects of Exposure to Hydrogen Sulfide

We have found that exposure to low levels of hydrogen sulfide establishes an epigenetic bookmark in *C. elegans*. Animals transiently exposed to low sulfide remember this exposure after long periods in the absence of sulfide, and are able to survive subsequent exposure to high sulfide conditions that are lethal to untreated, naive controls. This bookmark of sulfide exposure can be formed at any developmental stage, and persists through embryonic and post-embryonic development. However, the bookmark is erased by fasting. We have identified several epigenetic factors that are required to maintain the hydrogen sulfide bookmark, including histone methyl transferase and demthylase enzymes and the SWI/SNF chromatin remodeling complex. We are using this system to delineate mechanisms by which exposure to environmental toxins are translated into the epigenetic landscape in animals.

Somshuvra Mukhopadhyay
University of Texas at Austin

Hereditary Parkinsonism Due to Loss of Function Mutations in a Manganese Efflux Transporter

Manganese is an essential metal, but elevated cellular levels are toxic and may lead to the development of a parkinsonian syndrome. Historically, manganese-induced parkinsonism has been reported in individuals with elevated exposures from occupational or environmental sources, and in patients with impaired liver function who fail to excrete manganese through bile. Interestingly, in 2012, a new form of familial parkinsonism was reported to occur due to mutations in *SLC30A10*. The cellular function of SLC30A10 and the mechanisms by which mutations in this protein caused parkinsonism were unclear.

Using a combination of mechanistic and functional studies in cell culture, primary neurons and *C. elegans*, we discovered that SLC30A10 functioned as a cell surface-localized manganese efflux transporter that reduced cellular manganese levels and protected against manganese-induced toxicity. In contrast, mutations in SLC30A10 that caused familial parkinsonism blocked the ability of the transporter to traffic to the cell surface and to mediate manganese efflux. Although expression of disease-causing SLC30A10 mutants were not deleterious by themselves, neurons and worms expressing these mutants exhibited enhanced sensitivity to manganese toxicity. These results provide novel insights into the mechanisms involved in the onset of a familial form of parkinsonism and highlight the possibility of using enhanced manganese efflux as a therapeutic strategy for the potential management of manganese-induced parkinsonism, including that occurring as a result of mutations in SLC30A10.

Timothy Nurkiewicz
West Virginia University School of Medicine

Maternal Nanomaterial Inhalation During Gestation: Fetal Microvascular Consequences and Transcriptomic Dysregulations

The association between pulmonary xenobiotic particle exposure and cardiovascular dysfunction is established, yet the exact mechanisms linking exposure and effect remain unclear. A decade ago, we initially hypothesized that inflammatory mechanisms govern the systemic microvascular dysfunction that follows engineered nanomaterial (ENM) inhalation. To this end, we exposed rats and mice (male/female, young/adult, virgin/pregnant) to experimental atmospheres relevant to environmental, domestic, and occupational conditions. These atmospheres were generated with various ENM, including nano-titanium dioxide (nano-TiO_2). The primary particle size was ~21 nm; aerosol concentration ranged from 0 to 12 mg/m^3; and exposure duration was typically 4-6 hrs/d (sometimes repeated up to eight days, over a 2 wk period). Following inhalation exposure, intravital microscopy (with the spinotrapezius muscle or uterus) and isolated arteriole (from the heart, mesentery or muscle) experiments were performed. Endothelium dependent arteriolar dilation was significantly impaired after nano-TiO_2 exposure, and this was accompanied with widespread venular leukocyte trafficking (rolling, adhesion, extravasation). Cellular changes in the microvascular wall that supported these observations included: oxidative and nitrosative stress; myeloperoxidase deposition, and decreased nitric oxide bioavailability. Gender (female) and age (young) imparted partial protection against these extrapulmonary effects. Given these discoveries, and a dearth of understanding about the fetal transcriptome after maternal ENM exposure, we explored if maternal nano-TiO_2 inhalation produced gestational microvascular dysfunction, and if fetal outcomes also resulted. We have reported that nanomaterial inhalation during the second half of gestation significantly impairs uterine microvascular function, and this decreases litter size, pup mass and vascular function. In these fetal hearts, RNA-sequencing followed by Ingenuity Pathway Analysis identified a significant number of dysregulated processes including: decreased oxidative phosphorylation (56 markers, mks), and increased mitochondrial dysfunction (92 mks). Further, cardiovascular development and function was significantly dysregulated (1043 mks), and cardiotoxicity was represented largely by increases in markers of hypertrophy (227 mks), failure (149 mks), and necrosis (149 mks). Considered together, maternal ENM exposures may affect fetal cardiovascular health by impacting the transcriptome, possibly predisposing offspring to future adverse health outcomes. Ongoing studies are directly verifying these observations with functional cardiac and microvascular assessments.

Jill Poole
University of Nebraska Medical Center

Pattern Recognition Receptors Mediate the Lung Injury-Bone Loss Inflammatory Axis to Organic Dust Exposure(s)

Chronic inhalation of organic dusts, which are enriched in microbial cell wall components, cause significant airway inflammatory diseases including asthma, bronchitis, and chronic obstructive pulmonary disease, particularly in agriculture-exposed persons. We have utilized an animal model of inflammatory lung injury to delineate functional roles of specific components within complex organic dusts to provide mechanistic insights and inform future therapeutic strategies. We initiated a paradigm shift in this field by finding that gram-positive bacterial peptidoglycan (PGN), as opposed to gram-negative lipopolysaccharide (LPS), is a predominant driver of lung inflammatory consequences. Whereas a strong role was demonstrated for the Toll-like receptor 2 (TLR2) signaling pathway, the strongest phenotype found was for the common TLR/IL-1R adaptor protein, MyD88.

Using our newly created MyD88 bone marrow chimeric mice, our preliminary data show divergent roles for MyD88-dependent pathways in lung resident versus hematopoietic-derived immune cells to organic dust. Our studies also support new roles for MyD88 in regulating airway epithelial cell functions. We have also uncovered a novel role for MyD88 in regulating bone loss in response to inhalant organic dust exposure. Chronic airway inflammation is a risk factor for mediating systemic inflammation and debilitating bone disorders such as osteoporosis and fracture. Because agricultural workers have a high prevalence of musculoskeletal diseases, we sought to understand the systemic consequences of these inhalant exposures on bone homeostasis. Using state-of-the-art micro-CT imaging, we discovered significant bone loss following treatment with organic dust and its critical components, which established, for the first time, an animal model connecting inhalant lung injury to bone loss. Interestingly, we again observed discordance in component effects, highlighting the importance of specific pattern recognition signaling pathways. Inhalant PGN resulted in the greatest changes in lung histopathology, whereas inhalant LPS resulted in the greatest degree of bone deterioration. Furthermore, our studies have demonstrated that high vitamin D supplemental diets reduce organic dust-induced bone loss. Moreover, there is an age-effect in bone loss following organic dust mediated lung injury. Namely, young aged mice were more susceptible to dust-induced bone loss as compared to older aged mice.

Our recent studies are demonstrating that TLR4, but not TLR2, signaling pathway is critical in explaining the bone loss effects secondary to organic dust exposure, and that, the systemic IL-6 effector response is an important mediator. Collectively, these studies have helped identify mechanisms and potential therapeutic targets regulating lung injury and lung-injury induced systemic bone loss that could be translated to agriculture-exposed human cohorts to ultimately reduce disease burden.

Christina Porucznik
University of Utah School of Medicine

Periconceptional Biomonitoring

Mounting evidence supports that environmental exposures during gestation and the periconceptional period affect health. We designed the Home Observation of Periconceptional Exposures (HOPE) Study as a resource-efficient, low-technology means to collect biospecimens to analyze for environmental exposures timed to individual biological windows of sensitive development. Briefly, couples were taught to identify an estimated day of ovulation and fertile window based upon observation of changes in cervical mucus. Both members of the couple collected daily first-morning urine beginning at the onset of the fertile window with the woman continuing to collect through the end of the menstrual cycle. Once ovulation was identified, the man stopped collecting urine, and both members of the couple collected a saliva specimen. Couples collected a semen sample after the fertile window via intercourse using a semen collection device. Collection of biospecimens following this protocol continued for two menstrual cycles or identified pregnancy. 309 eligible couples were screened between 2011 and 2015, of which 183 enrolled. Eleven couples (6.0%) withdrew or were lost to follow up.

The most successful and cost effective recruiting strategies were word of mouth (40% of participating couples), posters and flyers (37%), and targeted Facebook advertising (13%). Pregnancy was achieved by 127 (69%) couples, and conception primarily occurred during the first three cycles of observation (n=95, 76%). Compliance with collection of saliva and semen samples was as follows: women's saliva 280/286=97.9%; men's saliva 278/286=97.2%; semen 257/286=89.9%. Compliance with collection of daily urine samples was also high. A urine specimen was collected on the estimated day of ovulation by the woman in 239 (86.6%) cycles and by men in 208 (75.4%) cycles, and women collected mean 16.7±5.2 and men collected mean 5.1±2.1 urine samples per cycle. Urine samples were analyzed for bisphenol-A (BPA) and disinfection-by-products using quantitative liquid chromatography-tandem-mass spectrometry.

This project demonstrated implementation of a cost-efficient approach which could be applied in larger studies for exposure monitoring timed to periconception or other developmental windows in a prospective cohort.

Jason Richardson
Northeast Ohio Medical University

Developmental Pesticide Exposure Causes Long-term Decreases in Sodium Channels and Glutamate Release in the Hippocampus

Recent work based on my ONES award found that developmental exposure to the pyrethroid insecticide deltamethrin caused long-term changes in neurochemistry and behaviors reminiscent of Attention-Deficit/Hyperactivity Disorder (ADHD). Epidemiological data revealed that increased urinary metabolites of pyrethroids in children was also associated with increased risk of ADHD and ADHD-like behaviors. Pyrethroids, such as deltamethrin, target voltage-gated sodium channels (Nav) and mutations that alter Nav expression have been shown to reduce action potential frequency and neurotransmitter release. Previous work investigating Nav regulation found that developmental exposure to Nav activators, including the pyrethroid insecticide deltamethrin can down-regulate Nav mRNA and protein in vitro, possibly as a mechanism to reduce hyperexcitability. However, it is not known whether or not this occurs following in vivo exposure. Repeated postnatal exposure (PND1-21) to deltamethrin (0.1-0.25 mg/kg/day) significantly reduced Nav protein levels at PND 30 and 75 in the hippocampus. Using isoform-specific antibodies, it was determined that Nav 1.1, and 1.6 were significantly downregulated, while Nav 1.2 levels were unchanged. Decreased levels of Nav were accompanied by a 50 percent reduction in 4-aminopyridine-evoked release of glutamate from hippocampal synaptosomes. This effect may be attributed to reductions in Nav protein as it was eliminated by pretreatment of synaptosomes with the Nav antagonist tetrodotoxin. Further, there was no reduction in vesicular glutamate transporter levels, suggesting there was no deficit in neurotransmitter loading into vesicles. Taken together, these new data demonstrate that postnatal exposure to deltamethrin has persistent effects on Nav protein expression and neurotransmitter release in the hippocampus. Given the importance of glutamate in learning and memory, these data provide a plausible mechanism by which developmental deltamethrin exposure may contribute to behavioral deficits.

Cheryl Rockwell
Michigan State University

The Role of Nrf2 in Immune Cell Polarization and Allergy

Nuclear erythroid 2 related factor 2 (Nrf2) is a transcription factor activated by cell stress, including oxidative and electrophilic stimuli, resulting in the upregulation of cytoprotective genes. Nrf2 can be activated experimentally by a number of different pharmacological agents, including tert-butylhydroquinone (tBHQ), which is also used commercially as a food additive. Our current studies demonstrate that activation of Nrf2 by tBHQ promotes Th2 differentiation and inhibits Th1 differentiation in isolated CD4+ T cells. Given that tBHQ is a food additive and promotes Th2 differentiation (which is associated with the development of allergy), we next sought to determine the effect of tBHQ in a mouse model of food allergy. Mice were administered a diet either with or without tBHQ (0.001%) for two weeks prior to sensitization with ovalbumin. Notably, this dose of tBHQ is lower than that normally present in standard rodent chow (0.0016%). The mice were sensitized weekly transdermally for 4 weeks. Sensitization to ovalbumin caused an increase in ovalbumin-specific IgE and IgG1 in plasma, which was greater in animals exposed to tBHQ. After sensitization, the mice were orally challenged with ovalbumin, causing mild/moderate immediate hypersensitivity, which was exacerbated by tBHQ. Overall, these studies suggest that low doses of the food additive, tBHQ, increase IgE response to food allergen and exacerbate clinical signs of immediate hypersensitivity.

Angela Slitt
University of Rhode Island

The Role of Nrf2 in Bisphenol-induced NAFLD

Hepatic biliary excretion is an essential process that exists to aid the in the elimination of foreign chemicals, and protects the body from accumulating chemicals and toxicants. Understanding the underlying processes by which specific transporters that aid in biliary excretion are regulated is integral for improving human health, predicting chemical exposure, and preventing disease associated with chemical exposure. Nutrition (i.e. dietary intake, fasting, and caloric restriction [CR]) is an important factor in the susceptibility/progression of a variety of diseases associated, especially those associated with aging and environmental exposure. Trans- resveratrol (RES) is an antioxidant in red wine with anti-aging effects that mimic CR, and is an agonist for the deacetylase Sirt1. Understanding how RES, fasting, and CR regulate the expression and function of liver transporters involved in hepatic excretion (i.e. Multidrug Resistance-Associated Proteins [MRPS]) is important for understanding mechanisms by which nutrition is beneficial against chemical exposure and disease.

Preliminary data demonstrates that RES increases the mRNA and protein expression of Mrp1-4, 6 in human hepatocytes and mouse liver along with induction of genes regulated by the transcription factor Nuclear Factor- E2-Related Factor 2 (NRF2), suggesting that RES activates human and mouse NRF2. Additionally, liver Mrp1, 2, and 3 expression, along with Ho-1 expression is increased during fasting, in which liver cAMP is increased and Protein Kinase A (PKA) is activated. Furthermore, constitutive activation of NRF2 in livers of KEAP1-null mice results in increased Mrp1-5 expression.

The hypothesis of this proposal is that RES, fasting, and CR induce expression of MRPs through Sirt1- and PKA- upstream regulation of NRF2-mediated transcription. Specific aims will determine whether 1) RES treatment induces MRP expression in human hepatocytes and mouse liver through NRF2, 2) fasting and CR induce MRP expression via uptream activation of PKA and downstream activation of Nrf2, 3) RES, fasting, and CR induce MRP via Sirt1, and 4) RES, fasting, and CR affect bisphenol A and PBDE disposition in mice.

Together, these studies will define mechanisms by which nutritional status alters expression of human and mouse MRPS, as well as demonstrate whether nutritional status enhances biliary excretion of environmental chemicals. Moreover, they will also provide novel insights into mechanisms that regulate NRF2-induction of human MRP genes. Project Narrative Nutritional status is an important factor for the development of many age-related diseases. Some environmental chemicals are thought to exacerbate or contribute to the development/progression of age-related diseases. The purpose of this project is to determine whether nutrition affects mechanisms involved in the liver's ability to uptake and clear environmental chemicals from the body.

Heather Stapleton
Duke University

A Flame Retardant Story: Bridging Science With Policy

In the latter half of the 20th century, several federal and state flammability standards were introduced in order to increase fire safety and reduce hazards associated with fires. Industry responded to several of these standards by manufacturing and using large volumes of additive chemicals that were applied to various textiles, resins and polymers to reduce their flammability. However, the use of these chemicals in consumer products has now led to increased exposure to flame retardant chemicals, both domestically and internationally. Several types of flame retardants are considered persistent and bioaccumulative, and research has demonstrated that exposure to these chemicals can elicit a variety of adverse health outcomes. While some flame retardants have been or are currently in the process of being phased-out, new flame retardants are being introduced and these new chemicals are often less studied. Several organizations and U.S. have proposed legislation that would ban several classes of flame retardant chemicals in specific types of consumer products. However, it is unclear how industry will respond to these chemical phase-outs, nor what alternatives exist that might be used as replacements. This presentation will provide an overview of the research conducted by Dr. Stapleton investigating children's exposure to flame retardant chemicals as part of her ONES award, and discuss its relation to scientific policy. This will include a discussion of exposure levels among the U.S. population, and summarize some of the more recent epidemiological studies published on flame retardant chemicals that have been used to support these new pieces of legislation.

Alexander Star
University of Pittsburgh

Role of Bio-coronas in the Enzymatic Oxidation of Single-walled Carbon Nanotubes

Carbon nanotubes (CNTs) have been proposed for application in multiple fields including composite materials, electrocatalysis, chemical sensors, and biomedicine. However, the large scale implementation of CNT technology has been hampered by reported biological side effects, specifically that of pulmonary toxicity upon inhalation of CNTs, similarly seen with asbestos. We have investigated the use of peroxidase enzymes for safe oxidation, and effective degradation, of CNTs both in vitro and in vivo. Our research has demonstrated the ability of peroxidase enzymes to oxidize single-walled carbon nanotubes (SWCNTs), multiwalled carbon nanotubes (MWCNTs), and graphene oxide (GO) ultimately showing that introducing structural defects consisting of oxygen functionalities enhances the enzymatic oxidation of CNTs. Here, we investigate the role of bio-coronas in oxidative degradation of SWCNTs catalyzed by neutrophil myeloperoxidase (MPO) using bandgap photoluminescence and Raman spectroscopy. Our results show diameter-dependence at the initial stages of the oxidative degradation of SWCNTs coated with surfactant, protein, DNA, and lipid molecules. In addition to studying semiconducting SWCNT bandgap luminesce (E_{11}) to extract chirality information of a sample, we have also investigated the formation of a new emission band (E_{11}^{-}), which is induced by the newly formed sp^3 defects on the surface of SWCNTs. Understanding the role of bio-coronas in enzymatic oxidation of SWCNTs is important for design of CNT-based nano-carriers with controlled circulation times and biological compatibility for medical diagnostics and therapeutics.

Biographies

Neelakanteswar Aluru
Woods Hole Oceanographic Institution

Neelakanteswar Aluru, Ph.D., is an assistant scientist in the Biology Department at the Woods Hole Oceanographic Institution in Massachusetts, where he studies the role of toxicants in altering DNA methylation. He received his doctoral degree in 2005 from the University of Waterloo, Ontario, Canada; shortly after, he completed his postdoctoral research at the University of Guelph (2006-2008) and Woods Hole Oceanographic Institution (2008-2012). He is an author/co-author of 30 publications including peer-reviewed articles and book chapters. Aluru served as an ad hoc member on the SIEE and XNDA study sections.

Aaron Bowman
Vanderbilt University

Aaron Bowman, Ph.D., is an associate professor in the Departments of Pediatrics, Neurology and Biochemistry at Vanderbilt University and Vanderbilt University Medical Center. He is also director of the Vanderbilt Training Program in Environmental Toxicology, an investigator of the Vanderbilt Brain Institute, and the Vanderbilt Kennedy Center for Research on Human Development. In addition, he holds appointments in the Vanderbilt Center in Molecular Toxicology and Vanderbilt Center for Stem Cell Biology. Bowman received his Ph.D. in biomedical sciences in 2000 from the University of California, San Diego. He completed postdoctoral fellowship training at Princeton University and Baylor College of Medicine. Bowman is a 2008 recipient of the Outstanding New Environmental Scientist (ONES) R01 award from the National Institute of Environmental Health Sciences (NIEHS). Bowman has a long record of service for the Society of Toxicology (SOT), including president of the Neurotoxicology Specialty Section (NTSS), senior council of the Stem Cells Specialty Section, member of the SOT Specialty Section Collaboration and Communication group, and chair of the SOT Education Committee Graduate Education sub-committees. Bowman serves on several national study sections, including standing membership in the Neurotoxicology and Alcohol (NAL) study section, NIEHS Specific Emphasis Panels, and the Veterans Affairs Merit Review Subcommittee (NUR-E). Bowman also serves on the editorial boards of Nature – Scientific Reports, NeuroToxicology, BMC Pharmacology and Toxicology, as well as performing other ad hoc editorial and peer review services.

The goal of Bowman's research is to define mechanisms of neuronal dysfunction and understand the basis of selective neuropathology, by characterizing the molecular function of disease genes and their interaction with environmental risk factors, especially metals. His work is supported by three R01 grants focused on the influence of manganese exposure in Huntington's disease (ES016931, PI) and parkinsonism (ES010563, MPI), and neurodevelopmental toxicity of methylmercury (ES07331, MPI, *pending*). Furthermore, he is a co-recipient of a NIEHS ViCTER R01 (ES010563-13S1, MPI) examining a role for manganese in restless legs syndrome. His lab utilizes patient-specific induced pluripotent stem cells, high throughput screening, mouse models, and biochemical approaches to examine gene-environment interactions in neurological disease. The long-term goals of the Bowman lab are to determine patient-specific and disease-specific toxicant vulnerabilities, understand the cellular pathways underlying these gene-environment interactions, and develop neuroprotective strategies to mitigate neurological diseases with environmental etiologies, especially manganese.

Ulrike Dydak
Purdue University

Ulrike Dydak received her Master of Science in physics from the University of Vienna, Austria, and completed her Ph.D. and postdoc in medical imaging at the Swiss Federal Institute of Technology (ETHZ), Zürich. She is associate professor of Health Sciences at Purdue and holds adjunct appointments with the Radiology and Imaging Science Department at Indiana University School of Medicine, the Biomedical Engineering Department at Indiana University – Purdue University Indianapolis (IUPUI), and a courtesy appointment with the Speech, Language and Hearing Science Department at Purdue University. Her research focuses on the development and application of novel magnetic resonance imaging (MRI) and spectroscopy (MRS) techniques to study brain metal accumulations and metabolic effects in welders exposed to a variety of metals through welding fumes. Over the past five years she built up a unique cohort of welders, who are being followed with neuroimaging, measures of biomarkers, cognitive and motor testing, and personal exposure measurements at their work place.

In 2015 Dydak received an NIH S10 instrumentation grant to buy a human MRI scanner and establish the Purdue Life Science MRI facility. Dydak will serve as director of this new facility, which starts operation in fall 2016. She also serves as charter member of the NIH MEDI study section and as executive committee member of the ISMRM Psychiatric MRI and MRS study group. In 2015, Dydak was named Purdue University Faculty Scholar.

Rebecca Fry
University of North Carolina at Chapel Hill

Rebecca Fry is an associate professor in the Department of Environmental Sciences and Engineering at the Gillings School of Global Public Health at University of North Carolina (UNC) at Chapel Hill. She also holds appointments in the Curriculum in Toxicology and the Lineberger Cancer Center. Fry is the director of UNC's Superfund Research Program funded by the National Institute of Environmental Health Sciences (NIEHS). Fry also serves as the director of graduate studies in the Curriculum of Toxicology at UNC. She is co-PI of the T32 training grant in biostatistics that trains more than 30 pre- and postdoctoral students at UNC. Fry's Ph.D. is in biology. Her postdoctoral training was in toxicogenomics and environmental health from MIT. Broadly speaking, her research focuses on elucidating biological mechanisms that relate prenatal toxicant exposure to detrimental health outcomes in children. Building off her expertise in the areas of DNA repair, toxicogenomics, and systems biology, her research at UNC has been ground breaking, identifying epigenetic mechanisms that potentially underlie children's health. A primary goal of Fry's research is to increase awareness of the deleterious impacts of exposures during the prenatal period and to improve public health initiatives to address this critical issue. She has served on the committee for the National Academies of Science (NAS) National Research Council for the IRIS review of inorganic arsenic, as a reviewer for the cancer and non-cancer risk assessment of arsenic in food by the Food and Drug Administration (FDA), and on the funding selection committee of the International Agency on Cancer Research (IARC).

Daniel Gorelick
University of Alabama at Birmingham

Daniel Gorelick received his Bachelor of Arts in music from the University of Pennsylvania in 1997. He received his Ph.D. in cellular and molecular medicine in 2005 working in the lab of Peter Agre at the Johns Hopkins University School of Medicine. Gorelick was an NIH-NRSA postdoctoral fellow at the Carnegie Institution for Science, Department of Embryology, in the lab of Marnie Halpern. From 2008-2009, he worked as a science adviser in the US Department of State as an AAAS science and technology policy fellow. Gorelick joined the Department of Pharmacology and Toxicology at the University of Alabama School of Medicine in 2012. He received the Outstanding New Environmental Scientist award from the National Institutes of Health in 2016.

Stacey Harper
Oregon State University

Stacey Harper is an assistant professor of nanotoxicology in a joint position between the Department of Environmental and Molecular Toxicology and the School of Chemical, Biological, and Environmental Engineering at Oregon State University (OSU). She received her doctorate in biological sciences from the University of Nevada, Las Vegas in 2003, and was a postdoctoral fellow at the U.S. Environmental Protection Agency from 2003-2005 and an NIEHS postdoctoral scholar at OSU from 2005-2009. In her current research, she uses rapid assays with whole organisms and communities of organisms to evaluate the biological activity and toxic potential of diverse nanomaterials. She has established a collaborative, open-source knowledge-base of nanomaterial-biological interactions (nbi.oregonstate.edu) to share her findings and data on the biological impacts of nanomaterials. Harper currently serves as the chair of ASTM International E56 Committee on Nanotechnologies and is a member of the ANSI-Accredited U.S. Technical Advisory Group for ISO/TC 229: Nanotechnologies. She is currently co-Leading the National Cancer Informatics Program Nanotechnology Working Group, a voluntary community that has coalesced around common interests in nanoinformatics (information and resources for the nanoscience community). She was awarded an Outstanding New Environmental Scientist award from the National Institute of Environmental Health Sciences in 2011, the L.L. Stewart Faculty Scholars award in 2012, and the Savery Outstanding Young Faculty award from OSU in 2014. From 2011-2012, Harper served as the president of the Pacific Northwest Association of Toxicologists, a regional chapter of the Society of Toxicology.

Yu-Ying He
University of Chicago

Yu-Ying He is an assistant professor of medicine at the University of Chicago. Recently, she was promoted to associate professor with tenure effective July 1, 2016. She received her Ph.D. in chemistry in 2000 from the Chinese Academy of Sciences in China. She received the Humboldt Research Fellowship working with Donat-P. Häder at the University of Erlangen-Nuerenburg in Germany. In 2001, she came to the NIEHS/NIH for her postdoctoral training with Colin Chignell. In 2007, she joined the faculty in the Department of Medicine at the University of Chicago. During her research career, He has received several awards, including the first NIEHS Science Day Early Career Award, the NIH Fellows Award for Research Excellence (FARE), the American Skin Association Research Scholar Award, the Outstanding New Environmental Scientist Award, and Leif B. Sorensen Faculty Research Award. Her research interests are in the genetic and environmental determinants of carcinogenesis.

Samir Kelada
University of North Carolina at Chapel Hill

Samir Kelada is an assistant professor in the Department of Genetics at the University of North Carolina at Chapel Hill. He is also a member of the Curriculum in Toxicology at UNC and a member of the Society of Toxicology. Kelada received a Master of Public Health degree in environmental health from the University of Michigan School of Public Health, followed by a Ph.D. in toxicology from the University of Washington, and finally completed postdoctoral training in genetics and genomics in the lab of Francis Collins, M.D., Ph.D., at NIH. Broadly speaking, his interests are in gene-environment interactions in obstructive airway diseases such as asthma. Along with colleagues at UNC and the Jackson Laboratory, Kelada utilized incipient lines of the Collaborative Cross (CC) mouse genetics resource to identify genetic loci that regulate allergen-induced airway inflammation. His current research program is focused on identifying genes and pathways that mediate the effect of air pollution on the subsequent development of allergic airway disease using the CC.

Michele La Merrill
University of California, Davis

Michele La Merrill conducts integrated toxicological and epidemiological studies to understand susceptibility to environmental diseases. These susceptibilities include environmental insults during the exquisitely sensitive developmental period, poor diet and ensuing metabolic diseases, and genetic and epigenetic predispositions. She earned her Ph.D. in toxicology from the University of North Carolina at Chapel Hill and her M.P.H. in epidemiology during her postdoctoral fellowship at the Mount Sinai School of Medicine. She is an assistant professor of environmental toxicology at the University of California, Davis where she teaches toxicology, genetics, and epidemiology courses with a focus on gene x environment interaction. She was a recent member of the IARC Monographs Working Group for volume 113, on some organochlorine insecticides and chlorophenoxy herbicides.

Kun Lu
University of Georgia

Kun Lu received his Ph.D. from University of North Carolina in 2009, studying formaldehyde-induced DNA adducts and its risk assessment. He did postdoctoral training at Massachusetts Institute of Technology to develop metabolomics methods for biomarker discovery. Since 2012, he has been appointed as an assistant professor at University of Georgia, with the overall research goal to better understand the health effects of environmental exposure and individual response by integrating the microbiome, exposome, -omics, and biomarker development. He was the winner of the Board of Publications Best Paper Award by the Society of Toxicology in 2011. His recent research focuses on understanding functional interactions between the microbiome and heavy metals, pesticides, and a number of other important environmental chemicals. In particular, his ONES award is to interrogate the functional interaction between the gut microbiome

and arsenic exposure.

William Mack
University of Southern California

William Mack is an associate professor of neurosurgery and neuroscience at the University of Southern California. He received his medical degree and completed neurosurgical residency at Columbia University. During training, he spent two years as a postdoctoral cerebrovascular research fellow in the laboratories of E. Sander Connolly Jr., M.D., and David J. Pinsky, M.D. Mack's postdoctoral training focused on the vascular biology of cerebral ischemia and reperfusion injury. His clinical practice involves caring for patients with cerebrovascular disorders. He directs a translational science laboratory that focuses on oxidative stress and inflammatory mechanisms of injury in cerebral ischemia and the design and implementation of experimental stroke models. Through interactions with mentors at the University of Southern California's Air Pollution and the Brain Network, he has become particularly interested in the effects of air pollution in cerebrovascular disease. Specifically, his group studies the impact of nanoparticulate matter on stroke, cerebral hypoperfusion, and vascular cognitive impairment. He is an active member of the Southern California Environmental Health Sciences Center (SCEHSC) and the Southern California Clinical Translational Science Institute (CTSI). Mack's overarching academic goal is to examine the effects of air pollution in experimental models of cerebrovascular disease and translate the findings from bench-to-bedside and beyond to the policy realm.

Maitreyi Mazumdar
Harvard Medical School

Maitreyi Mazumdar is a pediatric neurologist at Boston Children's Hospital and Harvard Medical School. Supported by a 2009 K23 award from the National Institutes of Environmental Health Sciences (NIEHS), Mazumdar became a key member of the Harvard Superfund Research Program (SRP) focused on understanding the effects of mixed metal exposures on neurodevelopment. While engaged in this work in Bangladesh, she saw many cases of children with myelomeningocele, a specific type of neural tube defect characterized by failure of the backbone and spinal canal to close prior to birth. Suspecting a connection between the number of children in Bangladesh with myelomeningocele and other neural tube defects and the concurrent arsenic epidemic, Mazumdar obtained pilot funding and established collaborations at Dhaka Community Hospital (DCH) to build a birth defect case-ascertainment program in rural areas of the country. Her initial studies suggest that folic acid supplementation, currently the primary strategy for neural tube defect prevention, may be less effective in areas of high environmental arsenic exposure due to interactions between arsenic and folate. With support from the NIEHS Outstanding New Environmental Scientist (ONES) R01 award, Mazumdar plans to investigate possible mechanisms, such as arsenic-folate interactions, that may link arsenic and neural tube defects.

Joel Meyer
Duke University

Joel Meyer studies the effects of genotoxic agents on human and wildlife health. He is interested in understanding the mechanisms by which environmental agents cause DNA damage, the molecular processes that organisms employ to protect prevent and repair DNA damage, and genetic differences that may lead to increased or decreased sensitivity to DNA damage. Mitochondrial DNA damage and repair are a particular focus. He studies DNA repair and other responses to DNA damage via PCR-based analysis of DNA damage and

repair, genomic and systems biology approaches, and organismal-level responses.

Dana Miller
University of Washington

Dana Miller is an assistant professor in the Department of Biochemistry at the University of Washington (UW) School of Medicine. She started her lab at UW in 2010. Before that, she completed postdoctoral research with Mark Roth, Ph.D., at the Fred Hutchinson Cancer Research Center. Miller received her Ph.D. from Johns Hopkins University in 2003, where she worked with Joel Schildbach, Ph.D. Miller's lab studies how changes in environmental conditions are translated into physiological effects in animals using biochemical, molecular, and cellular approaches. They are particularly interested in the effects of hypoxia and hydrogen sulfide, which is a common environmental toxin.

Somshuvra Mukhopadhyay
University of Texas at Austin

Somshuvra Mukhopadhyay is an assistant professor in the Division of Pharmacology and Toxicology at the University of Texas at Austin. His first degree is in clinical medicine [Bachelor of Medicine and Bachelor of Surgery (M.B.B.S.) from Topiwala National Medical College in Bombay, India in 2004; M.B.B.S. is equivalent to M.D. in the USA]. After completing his medical training, he switched to basic science research and obtained a Ph.D. in cell biology from New York Medical College in 2008. From 2008-2013, he trained as a postdoctoral fellow with Adam Linstedt, Ph.D., a world renowned expert on protein trafficking and Golgi biology, at Carnegie Mellon University in Pittsburgh, PA. Mukhopadhyay was recruited to the University of Texas at Austin as an assistant professor in January 2013.

Mukhopadhyay's research interest lies at the intersection of mechanistic cell biology and environmental toxicant-induced disease. A specific focus is on studying the subcellular regulation of metal ion homeostasis as a means to understand the pathobiology of, and develop therapies for, diseases that occur due to metal toxicity. His postdoctoral work and independent research has provided novel insights into the mechanisms of homeostatic control of manganese, an essential trace metal that induces parkinsonism at elevated exposures. Thus far, his work on manganese biology has been published in reputable journals including in PNAS (2011), Science (2012), and the Journal of Neuroscience (2014), and aspects of his findings have been covered by the scientific and mainstream media including by Nature, Nature Chemical Biology, The Washington Post, MSNBC, The Wall Street Journal, The Times of India, and National Public Radio. He has been supported by pre- and post-doctoral fellowships from the American Heart Association, a Pathway to Independence K99/R00 grant from the NIEHS (2011-2017; currently, in no-cost extension) and now the NIEHS ONES R01 (2016-2020). Support from the NIEHS has been instrumental in his career. The K99/R00 gave him the protected time necessary to develop his work on manganese-induced parkinsonism and successfully transition to an independent faculty position. The ONES grant is now poised to allow his group

to make important contributions in the area of familial parkinsonism due to inherited defects in metal metabolism.

Timothy Nurkiewicz

West Virginia University School of Medicine

Timothy Nurkiewicz was funded by the NIEHS Outstanding New Environmental Scientist mechanism in 2007. In the past nine years, he has ascended to the rank of professor; and for over five years has served as the associate chair for research in the Department of Physiology and Pharmacology at the West Virginia University School of Medicine in Morgantown, West Virginia. Nurkiewicz currently leads the Xenobiotic Toxicology Initiative in the West Virginia University Center for Cardiovascular and Respiratory Sciences. He is the vice-president of the Nanotoxicology Specialty Section of the Society of Toxicology (SOT), the past president of the Allegheny-Erie Regional SOT Chapter, as well as the Cardiovascular Toxicology Specialty Section (and a founding member). Nurkiewicz has been the director of West Virginia University's Inhalation Facility since its inception in 2007. His research program has been continuously funded by extramural agencies such as the National Institutes of Health, and Health Effects Institute since 2001; it was also a contributing laboratory in the NIEHS Nano Grand Opportunity Consortium. Nurkiewicz is a standing member of the Cardiac Biology/Regulation-Basic Sciences Study Section in the American Heart Association, and regularly serves as an ad hoc member for NIH study sections. He currently serves as the associate editor for Nanomedicine & Nanobiotechnology, Cardiovascular Toxicology, and Frontiers-Vascular Physiology, as well as on the editorial board of several other prominent toxicology journals. Nurkiewicz has been an adjunct scientist at the National Institute for Occupational Safety and Health since 2008.

Nurkiewicz's research program is active in the combined disciplines of microvascular physiology and toxicology, with a specific focus on pulmonary exposure to particulate matter air pollutants and engineered nanomaterials. His laboratories utilize multiple rodent models of inhalation exposure and subsequent in vivo (intravital microscopy) and in vitro (isolated microvessel) techniques. In 2004, the Nurkiewicz research program was the first to report that systemic endothelium-dependent arteriolar dilation is impaired after pulmonary particulate matter exposure. In the ensuing 12 years, they have been consistently revealing the mechanisms linking pulmonary toxicant exposure with systemic microvascular consequences. Most recently, his research program initiated novel investigations in the field of maternal nanomaterial exposures and fetal microvascular ramifications.

Jill Poole

University of Nebraska Medical Center

Jill Poole, M.D., is an associate tenured professor in the Division of Pulmonary, Critical Care, Sleep and Allergy Medicine and medical director of Allergy Services at the University of Nebraska Medical Center. She is a board-certified clinical allergist and immunologist with an active laboratory focused on understanding agriculture-related environmental organic dust-induced lung and systemic bone disease. This has been her focus for over ten years with grant funding from the National Institute of Environmental Health Sciences (recently renewed R01) and the National Institute for Occupational Safety and Health. She has had several important findings as part of her funded work. She developed an animal model of organic dust-induced airway disease that resembles the chronic inflammatory adaptation response observed in humans. Next, she initiated a paradigm shift in this field by demonstrating a strong role for gram-positive bacterial components as opposed to gram negative LPS with strooles for TLR2 and MyD88 signaling pathways. Finally, she recently reported the first description of animal model connecting lung injury with bone loss consequences, and she is investigating the mechanisms underlying the lung-bone inflammatory axis crosstalk.

Poole enjoys collaborating with agriculture research centers in Nebraska and Colorado, particularly in human cross-sectional and epidemiological studies, and mentors doctoral and postdoctoral students, and pulmonary fellows. To highlight some of her services, she serves as a grant reviewer for NHLBI R13 study section, ad hoc reviewer for R01/SIEE study section, special issue editor for the International Immunopharmacology Journal on Asthma Immunopathogenesis, serves on the editorial board for the Journal of Asthma, and will chair the Environmental and Respiratory Disease committee for the AAAAI. She has been invited to present her research discoveries at several allergy and lung focused meetings. She received the Basic Science Investigator Award Winner and Distinguished Scientist at UNMC. Poole has been the recipient of the American College of Allergy, Asthma, and Immunology Young Faculty Research Award and awarded an honorary plenary lecture award at the American Academy of Allergy, Asthma, and Immunology. She has been recognized as the Best Doctor in America Award since 2009, as well as several local teaching awards.

Christina Porucznik
University of Utah School of Medicine

Christy Porucznik, Ph.D., is an associate professor and the associate chief for education in the Division of Public Health of the Department of Family and Preventive Medicine at the University of Utah School of Medicine. Her area of research interest is in appropriately timing measurement of exposures. In the environmental domain, she studies endocrine disruptors and reproductive endpoints including fertility, pregnancy outcomes, and breastfeeding. Her other primary focus area is on prescription medications, primarily opioids, and the impact of policy changes on drug dispensing and adverse events.

Porucznik teaches in the Division of Public Health and serves on numerous graduate student committees (MPH, MSPH, and Ph.D.). She serves on the Board of Scientific Counselors for the National Center for Injury Prevention and Control of the Centers for Disease Control and Prevention (CDC). Before joining the faculty in 2005, Porucznik served as an epidemic intelligence service officer with the CDC at the Utah Department of Health and as a lieutenant commander in the Commissioned Corps of the United States Public Health Service. She worked for the U.S. Environmental Protection Agency, Human Studies Branch, while completing graduate study in epidemiology at the School of Public Health at the University of North Carolina at Chapel Hill, and was also a trainee in the NIEHS-UNC program (T32ES07018; Yrs. 22-23).

Jason Richardson
Northeast Ohio Medical University

Jason Richardson, Ph.D. DABT is a tenured professor in the Department of Pharmaceutical Sciences at Northeast Ohio Medical University, a board-certified toxicologist, and the founding director of a university-wide research focus area in neurodegenerative disease and aging. He received his bachelor's degree from Northeast Louisiana University (now University of Louisiana at Monroe) where he majored in toxicology. He received his Master of Science and Ph.D. degrees from Mississippi State University and then completed postdoctoral training at Emory University, where he was jointly appointed at the Rollins School of Public Health and the Center for Neurodegenerative Disease in the School of Medicine. Richardson spent ten years at Rutgers University as a faculty member at Robert Wood Johnson Medical School and the Environmental and Occupational Health Sciences Institute where he served as deputy director and then director of the joint graduate program in toxicology at Rutgers University. Richardson has authored or co-authored over 70 manuscripts and book chapters in the areas of developmental neurotoxicology, neurodegenerative disease, and pesticides. He has given over 50 invited lectures both nationally and internationally. Richardson is currently a member of the editorial boards of Toxicological Sciences, Current Molecular Pharmacology, Neurotoxicology, Neurotoxicology, and Teratology, and Toxics. He begins service as an associate editor for Neurotoxicology in 2016 and was an associate editor for BMC Neurology. He has served as a reviewer and chair for several NIH panels, the Michael J. Fox Foundation for Parkinson's Disease Research, Health Canada, and the United Kingdom Parkinson's Disease Society. Most recently, he was named to the Environmental Health Sciences Review Committee at NIEHS and the Committee on Emerging Science for Environmental Health Decisions at the National Academy of Science. Richardson received the Outstanding New Environmental Scientist Award from NIH/NIEHS and was the inaugural recipient of the Young Investigator Award from the Toxicology Division of the American Society for Pharmacology and Experimental Therapeutics. Richardson has been continually funded by NIH for his entire career. He has served as PI on grants with direct costs totaling ~$7 million. He is currently PI on NIH grants that aim to determine the role of genetic susceptibility to neurotoxicity in Parkinson's disease, identifying novel targets for reducing neuroinflammation in Parkinson's disease, and to develop drugs to mitigate acute neurotoxicant exposures as part of the HHS Counterterrorism Initiative (CounterAct). Additional grants pending at this time focus on the gene-environment interactions and health disparities in

Alzheimer's disease.

Cheryl Rockwell
Michigan State University

Cheryl Rockwell received her undergraduate degree from the University of Michigan. She worked in a contract lab prior to initiating her graduate studies in pharmacology and toxicology at Michigan State University. After receiving her doctorate in 2005, Rockwell began her postdoctoral training at the University of Missouri Kansas City before moving onto a second postdoctoral position at the University of Kansas Medical Center. Her work there in the lab of Curt Klaassen, Ph.D., resulted in a successful K99 transitional fellowship. She then accepted a tenure-track position at Michigan State University in the department of Pharmacology and Toxicology. She has published over 20 papers in a number of high-quality journals. She serves on the editorial boards for Molecular Pharmacology and Pharmacological Research. She has been very active in the Society of Toxicology, serving on a number of different committees and elected positions. She was the 2014 recipient of the Outstanding Young Investigator of the Year for the immunotoxicology specialty section. She has been invited to give a number of presentations, including a talk at the joint SOT-FDA Colloquium on immunotoxicology in April 2015.

Angela Slitt
University of Rhode Island

Angela Slitt's research interests focus on how 1) expression of drug transporters affects chemical (i.e. drug and environmental chemical) disposition and toxicity, 2) nutrition and intake of dietary antioxidants affects the expression of drug transporters, 3) liver disease (i.e. diabetes, cholestasis, and ethanol cirrohsis) affects transporter expression and chemical disposition, and 4) transporter expression affects cholesterol transport and susceptibility to gallstone formation.

Heather Stapleton
Duke University

Heather Stapleton is an associate professor of environmental chemistry and exposure science at the Nicholas School of the Environment at Duke University. Her research examines the fate and transformation of organic contaminants, with a particular emphasis on flame retardant chemicals. Her current research projects are focused on identifying flame retardant chemicals in consumer products and exploring the routes of human exposure to these chemicals, particularly in children. In 2008, she was awarded an Outstanding New Environmental Scientist Award (ONES) from the National Institute of Environmental Health Sciences for her research grant proposal entitled Children's Exposure to Brominated Flame Retardants: Effects on Thyroid Hormone Regulation. In 2012 she testified in front of the U.S. Senate Environment and Public Works committee on human exposure and toxicity of new-use flame retardants.

Alexander Star
University of Pittsburgh

Alexander Star is a professor of chemistry and bioengineering at the University of Pittsburgh. His current research is focused on synthesis and properties of carbon nanomaterials and their applications in sensors, energy conversion devices, and nanomedicine. Originally from Kazakhstan, Star received his Bachelor of Science and Ph.D. degrees in chemistry from Tel-Aviv University in 1994 and 2000, respectively. He then spent two years as a postdoctoral associate at California NanoSystems Institute at the University of California, Los Angeles, where he developed synthetic schemes to functionalize carbon nanotubes in a non-covalent fashion to improve their biocompatibility. Between 2002 and 2005, he served as senior scientist and manager of applications development at Nanomix, Inc. – a nanotechnology startup company – where he worked on development and commercialization of carbon nanotube-based sensors. He joined the Chemistry faculty at the University of Pittsburgh in 2005. During his academic career, Star co-authored over 100 peer-reviewed publications and was listed as a co-inventor on nine issued patents and over 20 patent applications. His research was recognized by Intel Award, three University of Pittsburgh Innovator Awards, Chancellor's Distinguished Research Award, NSF CAREER Award, and NIEHS Outstanding New Environmental Scientist (ONES) Award.

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