**Association of Carbon Monoxide Exposure with Blood Pressure among Pregnant Women in Rural Ghana: Evidence from GRAPHS**

**Background and Objective:** The Ghana Randomized Air Pollution and Health Study (GRAPHS) is a community-level randomized-controlled trial of cookstove interventions for pregnant women and their newborns in rural Ghana. There have been many recent studies worldwide focusing on household air pollution (HAP) produced by the burning of biomass fuels in open fires used for cooking and heating and adverse cardiovascular outcomes. In Ghana, HAP is the most important risk factor contributing to disease burden, particularly cardiovascular disease (CVD). HAP from biomass combustion contains a mixture of volatile and particulate pollutants, including PM2.5 and carbon monoxide (CO). Many previous cookstove intervention studies have used CO monitoring as an indicator of woodsmoke exposure. Blood pressure (BP) is a known risk factor for CVD and appears to fluctuate with changes in air pollution, especially with PM2.5. The present study focused on whether CO exposure from woodsmoke was associated with BP measurements among 817 adult women in Ghana.

**Methods:** Participants were women in their first or second trimester of pregnancy living within one of the 35 communities in the Kintampo Health Research Center catchment area who were the primary cooks in their household, and were non-smokers. These women were previously enrolled in the GRAPHS cookstove intervention study in 2013. Women who had both a baseline BP (systolic and diastolic measurements) and an initial 72-hour personal CO measurement taken at the time of initial enrollment were eligible for this analysis. Multivariate linear regression models were used to evaluate the association between CO exposure and BP in 817 women who were in their first or second trimester of pregnancy. Both sets of measurements were taken at initial study enrollment. Covariates such as gestational age, maternal weight and height, marital status, educational level, and other indicators of socioeconomic status, such as ownership of property and occupation, were included in the regression model.
Results: Higher CO exposure levels were significantly associated with higher diastolic blood pressure (DBP), while a non-significant positive trend was observed for systolic blood pressure (SBP). A 1ppm increase in mean CO exposure over 72 hours was associated with a 0.43mmHg higher DBP [0.01, 0.86]. The table shows the crude and adjusted effects of personal CO exposure on BP.

<table>
<thead>
<tr>
<th></th>
<th>Crude Association</th>
<th>Adjusted Association</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Estimate (mmHg)</td>
<td>95% CI</td>
</tr>
<tr>
<td>SBP</td>
<td>0.31</td>
<td>[-0.28, 0.91]</td>
</tr>
<tr>
<td>DBP</td>
<td>0.59</td>
<td>[0.14, 1.03]</td>
</tr>
<tr>
<td></td>
<td>Estimate (mmHg)</td>
<td>95% CI</td>
</tr>
<tr>
<td></td>
<td>0.39</td>
<td>[-0.12, 0.90]</td>
</tr>
<tr>
<td></td>
<td>0.43</td>
<td>[0.01, 0.86]</td>
</tr>
</tbody>
</table>

a Standard Errors are cluster-robust. In each model, results are reported after removal highly influential variables with Cook's Distance > 0.04; maximum number removed observations = 2.

b Adjusted for age, BMI, gestational age, occupation, second-hand smoke exposure, and day of week of BP measurement.

Conclusions: The results from this cross-sectional study suggest that household air pollution from wood-burning fires is associated with higher blood pressure levels, especially DBP, in pregnant women in their first and second trimesters. Unfortunately, BP measurements were not available in the third trimester or close to delivery time, so CO exposure could not be assessed in women who developed hypertensive disorders later in their pregnancy. The authors caution that the clinical implications of the observed association leading to chronic hypertension and/or hypertensive complications of pregnancy remain unclear.

Longitudinal effects of prenatal exposure to air pollutants on self-regulatory capacities and social competence

In order to evaluate the influence of prenatal exposure to widespread urban air pollutants on the development of self-regulation and social competence, a longitudinal prospective cohort study of children born to nonsmoking minority women in New York City was conducted by researchers at the Columbia Center for Children’s Environmental Health.

The level of polycyclic aromatic hydrocarbon (PAH)-DNA adducts in maternal blood collected at delivery was used to estimate maternal air pollutant exposure. Generalized estimating equations were used to assess the influence of prenatal exposure to PAH on deficient emotional self-regulation (DESR) in children at 3–5, 7, 9, and 11 years of age, adjusted for gender and race/ethnicity. Second, the association of prenatal exposure to PAH with social competence, as measured by the social responsiveness scale (SRS), was assessed. In addition, the authors also examined whether impairment in self-regulation acted as a mediator in the association of prenatal exposure to PAH with social competence.

The results, published in the *Journal of Child Psychology and Psychiatry*, showed a significant interaction (p=.05) of exposure with time such that the developmental trajectory of self-regulatory capacity was delayed among exposed children. Multiple linear regression revealed a positive association between presence of PAH-DNA adducts and problems with social competence (p<.04), level of dysregulation and problems with social competence (p<.001), and evidence that self-regulation mediates the association of prenatal exposure to PAH with social competence (p<.007). These data suggest that prenatal exposure to PAH triggers long-term effects on self-regulatory capacities across early and middle childhood, and that these deficits may lead to emerging social problems with real-world consequences for high-risk adolescent behaviors in this minority urban cohort.


Seasonal Influenza Infections and Cardiovascular Disease Mortality

Influenza infection has long been thought to directly contribute to cardiovascular (CV) morbidity and mortality. In temperate regions, influenza epidemics recur annually in winter and coincide with excess cardiovascular deaths. A recent study was conducted by researchers in the Climate and Health Program to quantify the temporal association between population increases in seasonal influenza infections and mortality due to cardiovascular causes, and to test if influenza incidence can be a predictor of CV mortality during the influenza season. This study used time-series analysis of vital statistics records and emergency department visits in New York City among cardiovascular deaths that occurred during influenza seasons between January 1, 2006 and December 31, 2012.
Published in the journal *JAMA Cardiology*, this study showed that among adults 65 years and older, seasonal average influenza incidence was correlated year to year with excess CV mortality. In daily time-series analyses using four different influenza metrics, interquartile range increases in influenza incidence during the previous 21 days were associated with an increase between 2.3% and 6.3% for CV mortality and between 2.4% and 6.9% for ischemic heart disease mortality among adults 65 years and older. The strongest associations were observed for myocardial infarction mortality, with each interquartile range increase in influenza incidence during the previous 14 days associated with mortality increases between 5.8% and 13.1%. Out-of-sample prediction of cardiovascular mortality among adults 65 years and older during the 2009-2010 influenza season achieved an average 94.0% accuracy using four different influenza metrics. The results of retrospective estimates of influenza-related CV mortality, combined with accurate and reliable influenza forecasts can predict the timing and burden of seasonal increases in CV mortality and help inform proactive public health mitigation and response efforts.


Associations between Blood and Urine Arsenic Concentrations and Global Levels of Post-Translational Histone Modifications in Bangladeshi Men and Women

Chronic exposure to arsenic leads to bladder, lung, and skin cancers and is also associated with numerous non-cancer health outcomes, with susceptibility differing by sex. Although evidence from in vitro studies suggests that post-translational histone modifications (PTHMs) can be altered by arsenic, evidence in humans is limited. The study aimed to determine a) if arsenic exposure is associated with global levels of PTHMs: H3K36me2, H3K36me3, and H3K79me2 in a sex-dependent manner, and b) if %PTHMs are stable when arsenic exposure is reduced. Researchers examined associations between arsenic measured in blood and urine, and % PTHMs in peripheral blood mononuclear cells from 317 participants in the Bangladesh Folic Acid and Creatine Trial (FACT). The stability of %PTHMs after the use of arsenic-removal water filters was also examined.

The results published in *Environmental Health Perspectives*, revealed significant differences between men and women (p=0.01) in the associations between natural log-transformed (ln) urinary arsenic and PTHMs. Urinary arsenic adjusted for creatinine was positively associated with %H3K36me2 in men, while the association was in the opposite direction for women. Similar patterns of associations were also observed between histone modifications and blood arsenic, though not statistically significant. In addition, water filter use was associated with reductions in %H3K36me2, but this did not differ significantly by sex. Arsenic was not significantly associated with %H3K36me3 or %H3K79me2 in men or women.

Welcome Our New Center Members!

Jeanine Genkinger, PhD, is a cancer epidemiologist who has been driven to understand how modifiable factors, molecular pathways and related biomarkers may impact cancer risk and progression, particularly for rare but highly fatal cancers. Prevention through determining modifiable risk factors and improved early detection through identifying markers of risk and molecular pathways offer the most promising approaches to reducing morbidity and mortality of these diseases. Her area of methodological specialty is in nutritional epidemiology, longitudinal design and complex pooled and meta-analytic techniques. Dr. Genkinger has conducted her research in large scale international consortia, namely the Pooling Project of Prospective Studies of Diet and Cancer and the NCI Cohort Consortium, and has conducted research in numerous cohort studies, such as the Breast Cancer Family Registry. As a new Center member, Dr. Genkinger is looking forward to working with the Center to allow her to use facility cores and interact with leaders in the field of environmental health to conduct her research on early detection of pancreatic and ovarian cancer.

Liang Liu, PhD, has a long standing interest in studying how genes and environment interact to influence development and disease susceptibility. His research strategy relies on studying the basic biology and function of chromatin, which carries not only the genetic information but also the epigenetic information. Being the largest organ of the body, skin is an ideal in vivo model organ for mechanistic studies on gene and environment interactions because it interfaces directly with the outside environment and is easily accessible for biopsy and manipulation. Dr. Liu’s research centers on genetic and epigenetic crosstalk during skin development and UV-induced skin carcinogenesis. The overarching goal of his research is to elucidate the molecular network underlying UV-gene interactions and to identify actionable UV targets to develop mechanism-driven targeted approaches for prevention and treatment of skin cancer. He believes that becoming a CEHNM member will greatly enhance his research by providing access to important core facilities and services and also the opportunity to develop collaborations with other Center members.
**Andrea Baccarelli, PhD, MD**, serves as the Environmental Health Sciences Department Chair and the Director of the Laboratory of Environmental Precision Biosciences. As an epigeneticist and board-certified clinical endocrinologist, Dr. Baccarelli’s research explores epigenetic and molecular mechanisms as potential functional pathways linking exposures to environmental pollutants to human disease. His laboratory research activities are specifically focused on epigenetics, mitochondriomics, and computational epigenomics. Dr. Baccarelli is currently the PI of multiple NIH-funded projects and since 2010, his lab has produced publications at the intersection of epigenetics, molecular epidemiology and environmental health, with one of his recent papers named Paper of the Year by Environmental Health Perspectives, the leading journal in environmental health research. Recent and ongoing projects investigate health effects from environmental exposures, including particulate air pollution, metals, Bisphenol A, phthalates, and pesticides, and common risk factors, such as psychosocial violence, second-hand smoking, and maternal diet and metabolic alterations. Dr. Baccarelli’s Laboratory has been conducting studies on the U.S. population, as well as in highly-exposed groups or special conditions of exposure at several international locations in China, Canada, Mexico, Italy, Israel, Poland, Thailand, Oman, Bulgaria, Russia, and other countries.

**Ana Navas-Acien**, Professor of Environmental Health Sciences at Columbia University, is a physician-epidemiologist with a specialty in Preventive Medicine and Public Health (Hospital La Paz, Madrid ’01) and a PhD in Epidemiology (Johns Hopkins University’05). Her research investigates the long-term health effects of widespread environmental exposures (arsenic and other metals, tobacco smoke, e-cigarettes, air pollution), their interactions with genetic and epigenetic variants, and effective interventions for reducing involuntary environmental exposures. For more than 10 years she has been working on environment-related research in population-based cohort studies such as the Strong Heart Study, a study of cardiovascular disease and its risk factors in American Indian communities, and the Multi-Ethnic Study of Atherosclerosis (MESA), a study of cardiovascular, metabolic and lung disease in urban settings across the US. She is now starting a collaboration with the Trial to Assess Chelation Therapy 2 (TACT2), an NIH-funded multi-center clinical trial that will evaluate whether repeated chelation can reduce metal levels and whether this reduction can explain the beneficial cardiovascular effect of chelation therapy. Both in the US and internationally, she conducts research to evaluate exposure to tobacco smoke including emerging public health challenges such as water pipe smoking and e-cigarettes. By becoming a Center member, her goals are to enhance the impact of her research through new collaborations and to contribute to the reduction of environmental health disparities.
Dr. Markus Hilpert is a new Associate Professor in EHS, arriving from Johns Hopkins University, where he spent the past 14 years as a faculty member in the Department of Geography and Environmental Engineering. Dr. Hilpert has a doctorate degree in Civil Engineering from the University of Karlsruhe in Germany. He came to the U.S. in 1997 to do postdoctoral work at the University of North Carolina, Chapel Hill, in Environmental Science & Engineering and Public Health.

His research focuses on the interface between hydrology, environmental engineering, and environmental health. He applies his strong foundations in physics, mathematics, computational methods, and engineering towards solving problems pertaining to the movement of water, pollutants, and energy in the environment, and ultimately to human health. His research has found applications in many disciplines, including contaminant hydrology, environmental engineering, geophysics, soil microbiology, and environmental health sciences. Recent and current research projects include the following: (1) effects of bacterial chemotaxis on contaminant degradation, (2) colloid and pathogen transport in subsurface environments and water filters, (3) spreading of antibiotic resistance in poultry-waste impacted soil, (4) multi-fluid flow in porous media, (5) enhanced geothermal systems, and (6) chronic hydrocarbon releases at gas stations. He has been the PI on seven NSF-funded grants related to contaminant transport and remediation in subsurface environments.

Dr. Hilpert hopes that the Center, and in particular the Climate and Health Working Group and the Exposure Assessment Facility Core, will facilitate his research on the environmental health effects of VOC releases to the atmospheric environment during fuel transfer operations, e.g., at fuel dispensing facilities and gasoline distribution bulk terminals. Such VOC releases are particularly important in stressed urban environments such as the New York metropolitan area, because VOCs contribute to the formation of ground-level ozone.
On June 3rd, 2016, about 60 people gathered at Faculty House on the main campus for our annual NIEHS Center Retreat/External Advisory meeting. The attendees included about 22 Center members, six external advisors, pilot and career awardees, lab managers, the John Jay PriMER students, EHS students, postdocs, and WE ACT staff. The meeting began with Regina giving an overview of changes in Center membership and leadership in 2015-16, followed by updates on the pilot projects that were funded in Round 17 (Su) and 18 (Levy, Jack, Pon), and the newest two pilots reviewed at the end of the meeting and funded by the Center on July 1st (Greenlee and Re). The two newest Career Development awardees, Dr. Qixuan Chen in Biostatistics and Dr. Diana Hernández in SMS, were announced. The overview was followed by a fascinating seminar given by one of our newest EHS faculty members, Dr. Ana Navas Acien, who recently joined EHS from Johns Hopkins. The seminar titled, “Metals and Cardiovascular Disease: Opportunities for Prevention,” focused mainly on the health effects of exposure to lead, arsenic and cadmium, with an emphasis on prevention. Center members, Steve Chillrud and Darby Jack, gave an update on the new LDEO instrument awarded to Beizhan Yan and the Exposure Assessment Core called a “Sensitive Q Trap LC/MS/MS System for Biomarker Analyses.” The Core has also been developing real-time and integrative monitors for personal and residential exposure assessment. A Center funded pilot which led to R21 funding from NIEHS is focusing on inhaled doses of particulates for bicyclists and CV indicators. The Integrative Health Sciences Core will expand biostatistical services with the addition of Qixuan Chen, a Career awardee, who already collaborates with some Center members. Diane Levy, formerly of the Data Management Core, will teach a 3-hour basic data management workshop in the fall for research coordinators, students, postdocs, community members, and anyone else who is interested. The Trace Metals Core has been heavily involved in analyzing samples from Bangladesh, using the new Perkin-Elmer IC-MS instrument funded by the NIEHS.
The Community Outreach and Engagement Core (COEC) presented highlights of projects they were involved in during the past year including: 1) measuring community resilience after Superstorm Sandy; 2) the Johns Hopkins Center for Talented Youth Program at Columbia (a one-day program hosting 40 high school students and their parents introducing them to the field of environmental health sciences); 3) Environmental Health and Justice Leadership Training offered by WE ACT to community leaders from the Bronx, Washington Heights/Inwood and Harlem, as well as youths from the WHEELS Academy in Northern Manhattan; and 4) updating the Environmental Health Report Card which compares two sets of grades, a public grade and a community grade, for each of WE ACT’s 9 health indicators (e.g., air quality, pests and pesticides, quality of drinking water, etc.) in four areas of Northern Manhattan.

The afternoon featured an interesting seminar by Dr. Markus Hilpert, a new Associate Professor in EHS titled, “Chronic Hydrocarbon Releases at Gas Stations: Toward a Comprehensive Assessment of Environmental and Health Effects” and two exciting pilot talks by Dr. Amy Margolis in Psychiatry and Dr. Yalda Moayedi-Esfahani, a Post-doctoral Research Scientist working with Dr. Ellen Lumpkin in Dermatology.

There were discussion sessions related to defining the strategic vision for the Center over the next five years. Once the strategic vision is set, then we can modify our current working groups and fund pilots around these themes and goals to create an integrated Center. We discussed the current working groups and themes of the Center, with the suggestion of adding new “emerging working groups”, e.g., the microbiome, to existing working groups. The goals of the working groups must be clearly stated. There were also many ideas from the external advisors related to revamping the pilot projects program to increase the pool of applicants, shorten the review process to expedite funding of pilots, and fund good pilots to increase the likelihood of successful outcomes. The Executive Committee will be meeting throughout the summer and fall to focus on these issues, make decisions, and begin writing the first draft of the competitive renewal grant proposal.
**Abstract:** The hormonal milieu during adolescence may be important in determining breast development, morphology and breast cancer risk. The intestinal microbiome can have a profound impact on the bioavailability of estrogens and estrogen disrupting chemicals by contributing to the proportion of recirculated and excreted estrogens. The proposed pilot study maximizes the infrastructure and existing data of the LEGACY Girls Study. The LEGACY Girls Study is a multi-site NCI-funded cohort study of girls in the US and Canada. Half of the study participants are from high-risk breast cancer families, and half are from average breast cancer risk families. The proposed pilot study will add stool sample collection in girls and their mothers (n=10 evaluable pairs). All stool samples will be analyzed for fecal microbiota diversity and abundance using 16S rRNA sequencing at the Mailman School of Public Health’s Center for Infection and Immunity. A subset of the stool samples (n=2 pairs) will be assessed by shotgun metagenomic sequencing. Urinary estrogen will be measured via estrone-1-glucuronide (E1G), an estrogen metabolite that is an indicator of total circulating estrogens. Endocrine disruptors will be assessed by measuring urinary polycyclic aromatic hydrocarbon (PAH) metabolites. The primary aim is to assess the feasibility of collecting, processing and analyzing stool samples among mother-daughter pairs who consent for this ancillary study. Secondary aims are 1) To describe the fecal microbiota diversity and abundance in girls and their mothers, and to demonstrate our capacity for functional metagenomic analyses of the microbiome in this cohort, and 2) to measure i) total circulating endogenous estrogens (as assessed by E1G) and ii) endocrine disrupting chemicals (as assessed by PAHs) in girls and their mothers. Pilot study results will be used to inform the design of a planned R01 submission to NCI to investigate the association between endogenous estrogens, endocrine disrupting environmental chemicals, the fecal microbiome and breast development in girls participating in the LEGACY Girls Study. This pilot study represents a new collaboration among four faculty members of the Mailman School of Public Health.
Diane B. Re (aka Gourion-Arsiquaud), Ph.D., Ass’t Professor of Environmental Health Sciences; “Unraveling Gene-Environment Interactions and Window(s) of Exposure in TDP43-Linked Amyotrophic Lateral Sclerosis”; Award: $35K

Abstract: Amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig’s disease, is a rare adult-onset neurological disorder (incidence 1-2/100,000 persons) preferentially affecting the neurons which control muscles and voluntary movement, the “motor neurons”. It is characterized by progressive muscle weakness and muscle mass loss, and progresses aggressively to complete paralysis and death within 1-5 years after symptom onset. In ALS, the motor neurons are progressively dying, but the underlying pathogenic mechanisms remain elusive, and there is no treatment to slow or halt this process. Importantly, only 5-10% of ALS cases can clearly be explained by a genetic cause. Most patients have no familial history and are called “sporadic” cases. This indicates that patient exposure to unknown environmental factors may have a strong role in the development of the disease. Some studies have suggested that the number of ALS cases is constantly progressing, faster than the aging of industrialized population, while others have described strikingly high numbers of ALS cases in small geographical areas or among groups sharing the same occupation. Currently, 90% of the research effort against ALS is geared toward its rare genetic forms. More research on the potential implication of the environment in ALS is necessary today if we want to prevent and treat sporadic ALS tomorrow.

Several environmental/occupational neurotoxicants have been suggested to be associated with ALS (heavy metals, pesticides, cyanobacterial neurotoxins). However, none have been clearly demonstrated to cause ALS. This demonstration is indeed difficult to accomplish knowing that even sporadic ALS is rare and cannot be solely reduced to the exposure to an environmental toxin. The majority of persons in geographical areas or occupational groups where ALS is high still do not develop the disease, even though the probability to be affected is multiplied by 2-100. This means that only a combination of individual susceptibility factors (genetics) and exposure to a toxin can lead to the development of ALS. This phenomenon is called “gene-environment” interaction. Our long-term objective is to develop reliable and relevant cell and animal models to study these deleterious interactions and elucidate pathogenic mechanisms that could lead to a treatment for ALS. Although several genetic factors of susceptibility to ALS have been suggested, again, none has been confirmed and susceptibility models are lacking. In the present pilot study, we believe that we found an unprecedented opportunity to study gene-environment interaction in ALS. Recently, our collaborator at Columbia University, Dr. Neil Shneider, has developed a new generation of genetic animal models of ALS. In contrast with previous transgenic mice that were expressing mutant genes at non-physiological levels (~3-22 times higher), these new models express only one or two copies of the mutation, more similar to what is seen in ALS patients. Moreover, the mutant gene was introduced to replace the mouse gene at its exact original location, as-
suring it will be expressed where it should be in the body and at the right level. Dr. Shneider’s lab has generated such a mouse with a mutation in the protein TDP-43 which is thought to be implicated not only in some familial cases of ALS, but also in most of the sporadic cases of ALS and of frontotemporal dementia, where it has been shown to accumulate and aggregate in tissues. Unfortunately for geneticists such as our collaborator, this sophisticated near-perfectly designed genetic mouse model does not develop ALS over the short lifespan of the mouse (2 years vs. 50-60 years median age of the disease development in humans). However, this model of TDP-43 “silent ALS” mice represents a unique opportunity for environmental health scientists to unravel the potential origin and mechanisms of sporadic ALS.

Accordingly, our **first objective in this pilot study** is to use cells produced from these mice that are relevant to ALS pathology (motor neurons and supporting cells called astrocytes) to screen for environmental toxins suggested to be involved in ALS (lead, manganese, arsenic, organophosphorous pesticides, and cyanobacterial beta-methylamino-Lalanine) and identify which one(s) can spark a TDP-43-related ALS pathology in a dish. Once such a toxin is identified, our **second objective will be to expose TDP-43 silent ALS mice to this toxin following different temporal exposure paradigms (perinatal, adult, continuous ...) to study if they develop symptoms of ALS (motor weakness, TDP-43 accumulation and aggregates in tissues) and which window(s) of exposure is (are) critical in doing so. The data generated in this pilot study could represent an unprecedented breakthrough in the ALS field as well as in the neurotoxicity of neurodegenerative disease field. Indeed, this work could validate for the first time an ALS-inducing toxin and provide some insights into the timing of exposure necessary across the life course to trigger adult-onset neurodegeneration.
**Meet Our New Career Development Awardees**

Dr. Qixuan Chen was born in Fujian, China, and completed a B.A. in Economics at Nankai University in 2002. She then came to the U.S. to complete an M.S. in Applied Statistics at Bowling Green State University in Ohio in 2004, followed by a Ph.D. in Biostatistics from the University of Michigan in Ann Arbor in 2009. She became an Assistant Professor in the Department of Biostatistics at the MSPH soon after completing her doctorate. Her career has been motivated by her interest in statistics and her dedication to promote the health and wellbeing of under-served populations. Since she joined the Mailman School, she has been actively involved in a broad spectrum of population health research projects and in developing statistical methods motivated by population health research. She has authored or co-authored 37 peer-reviewed papers, 20 of which are on environmental health. She has also served as a Co-Investigator or lead statistician on multiple NIH research grants.

Dr. Chen’s current methodology research focuses on developing novel statistical methods to analyze complex survey data. Each year, the U.S., at great expense, conducts numerous national surveys to collect medical, health, and demographic information. These surveys provide rich data to examine the impact of environment on the health of the US general population. Although such datasets are usually large enough to study the entire population, the sample size can be small for some subpopulations, e.g., ethnic-minorities. Thus, direct statistical analysis for these subpopulations can be problematic. To enable more reliable inferences, she has developed a Bayesian multilevel model by borrowing information from other subpopulations. It can be applied to health surveys with various sampling designs and can be extended to study time trends using repeated surveys.

Recently she developed an interest in asthma and allergy research in collaboration with Drs. Perzanowski and Miller. Asthma and allergic diseases are common in all age groups and are associated with lower quality of life and high cost of health care. Although the literature suggested racial disparities in allergic sensitization, little is known about the prevalence rates by race and ethnicity. Moreover, understanding the environmental factors associated with the disparities could lead to interventions that would improve the health of individuals with asthma or allergic diseases. In a new R01 application to NIEHS, Dr. Chen plans to address these questions using the data from two important repeated national surveys – the National Health Interview Survey and the National Health and Nutrition Examination Survey. She will apply her Bayesian model for small subpopulations to estimate prevalence rates of allergic diseases and their trends over time by race/ethnicity and age groups. She will also extend her Bayesian model to allow multiple correlated exposures in examining risk factors associated with allergic diseases and the racial disparities in allergic sensitization.
Dr. Diana Hernández was born in the Bronx, NY. She attended Hunter College where she received a B.A. in Sociology in 2002, followed by an M.A. (2005) and Ph.D. (2010) also in Sociology from Cornell University in Ithaca, NY. She became an Associate Research Scientist in the Department of Sociomedical Sciences at the MSPH in 2009, and was promoted to Assistant Professor in SMS in 2011. Her scholarly interests center on housing and energy as social and environmental determinants of health. Drawing largely on qualitative and mixed-methods, her research examines intersections among the built environment (housing and neighborhoods), poverty and health with a particular emphasis on energy insecurity, a concept that she has spearheaded in the field of public health. Being a sociologist by training, she currently leads or collaborates on several research projects related to policy and structural-level interventions in low income housing (i.e., energy efficiency upgrades, cleaner burning fuel source conversions, capital improvements and financial restructuring in public housing, post-Sandy resilience among public housing residents and smoke-free housing policy (SFHP) compliance and enforcement in low-income multiple unit housing settings). These ongoing projects involve interdisciplinary collaborations that incorporate her expertise in qualitative and community-based research with quantitative methods that range from toxicological exposure assessments and large administrative datasets to longitudinal survey data. As these projects unfold, Dr. Hernández and her collaborators will accumulate the necessary empirical evidence to assess how poor housing quality and inefficient energy infrastructure affects the health and economic wellbeing of vulnerable groups while also evaluating the impacts of interventions set at the household, building and neighborhood levels.

Previous support from the CEHNM has enhanced both her program of research and career trajectory. A CEHNM pilot grant awarded in 2013 facilitated the incorporation of environmental exposure assessments in her research and enabled fruitful collaborations with Center members. The pilot project examined indoor/outdoor air quality in ten Northern Manhattan buildings prior to and one-year after cleaner-burning fuel conversion as mandated by NYC Clean Heat policies. In 2014, she expanded the study to include 30 buildings through an NIEHS-funded R21 grant with Drs. Chillrud, Liu and Perzanowski and WE ACT, our community partner. Preliminary results suggest that PM2.5 emissions decreased significantly over the study period and thermal comfort improved.

The pilot study paved the way for Dr. Hernández to become an inaugural recipient of the JPB Environmental Health fellowship at the Harvard T.H. Chan School of Public Health. This fellowship promotes multidisciplinary research on how the social and physical environments interact to influence health particularly in underserved communities. It provides training and collaborative research opportunities. As part of this fellowship, she has initiated a pilot project on SFHP compliance/enforcement in existing smoke-free low-income apartment buildings. Dr. Hernández and her colleagues are assessing environmental exposures to airborne nicotine and fine particulate matter in common areas of the buildings and placing nicotine badges and PM2.5 monitors in 60 apartments. In addition, they are testing an intervention model of SFHP implementation and enforcement that is resident-centered, informed by harm reduction principles and engages empowerment approaches that are responsive to the challenges of serving vulnerable populations.
**Fall 2016 NIEHS Center Seminar Schedule**

**September 22:** Rachel Morello-Frosch, PhD, MPH, Professor, Environmental Science, Policy and Management, UC Berkeley, CA; “The Haves, the Have Nots, and the Health of Everyone: Exploring connections between environmental equity and sustainability”; 722 W. 168th Street, Allan Rosenfield Bldg., 11th floor, Rm. 1101 (EHS Conference Room), **12:00-1:00 pm.**

**September 30:** Sam M. Hanash, MD, PhD*, Director, Red and Charline McCombs Institute for the Early Detection and Treatment of Cancer, The University of Texas MD Anderson Cancer Center, Houston, TX; “Blood biomarkers to increase the impact of lung cancer screening”; 722 W. 168th Street, Allan Rosenfield Bldg., 11th floor, Rm. 1101 (EHS Conference Room), **1:00-2:00 pm.** *Co-sponsored with the Columbia Center for Children’s Environmental Health (CCCEH) and the Herbert Irving Comprehensive Cancer Center.

**October 14:** Joseph Braun, PhD, Assistant Professor of Epidemiology, Brown University, RI; “Early Life Environmental Chemical Exposures and Children’s Health: The HOME Study”; 722 W. 168th Street, Allan Rosenfield Bldg., 11th floor, Rm. 1101 (EHS Conference Room), **12:00-1:00 pm.**

**November 17:** Martha (Mara) Téllez-Rojo, PhD, Senior Researcher at the National Institute of Public Health (NIPH) in Mexico; Topic: Exposure to Lead in Mexico; 722 W. 168th Street, Allan Rosenfield Bldg., 11th floor, Rm. 1101 (EHS Conference Room), **12:00-1:00 pm.**

**December 1:** Carmen Marsit, PhD, Professor of Environmental Health, Rollins SPH, Emory University, Atlanta, GA; Topic: Environmental Carcinogenesis and Epigenetics; 722 W. 168th Street, Allan Rosenfield Bldg., 11th floor, Rm. 1101 (EHS Conference Room), **12:00-1:00 pm.**

**Save the Dates**

**October 20th:** Center Meeting, 722 W. 168th Street, Allan Rosenfield Bldg., 11th floor, Rm. 1101 (EHS Conference Room), **12-2:00pm.** Lunch will be served.

**November 12th:** Conference jointly sponsored by WE ACT, the CEHNM, and the CCCEH, “NYC Communities Organizing for Climate Resilience and Social Cohesion: Advocating for Public Health, Energy Security & Emergency Preparedness”; 50 Haven Avenue, Bard Hall Lounge, 1st floor, **9:00am - 4:00 pm.** A light continental breakfast and lunch will be served.