PROCEEDINGS OF THE EPA WORKSHOP ON INTERACTIONS BETWEEN SOCIAL STRESS AND ENVIRONMENTAL HAZARDS

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INTRODUCTION

The U.S. Environmental Protection Agency's Office of Environmental Justice and National Center for Environmental Research convened the Workshop on Interactions between Social Stressors and Environmental Hazards in May 2012 to examine the association between negative health outcomes and exposure to psychosocial and chemical stressors. This workshop sought to promote discussion among multiple stakeholders, including environmental health researchers, EPA regulatory scientists and community advocates.

The workshop represents a sustained effort by the EPA to advance the science of disproportionate environmental and health impacts experienced by minority and low income populations. It is a follow-up to discussions initiated on the issue of social stressors at the March 17-19, 2010 science symposium "Strengthening Environmental Justice Research and Decision Making: A Symposium on the Science of Disproportionate Environmental Health Impacts."

This report highlights the major issues presented and discussed during the May 2012 workshop.

PURPOSE AND BACKGROUND

Individuals with higher risk of exposure to environmental contaminants and an increased susceptibility to adverse health outcomes following exposure may also experience higher exposures to social stressors. Emerging evidence from epidemiological and toxicological studies suggests that social stressors may modify the effects of environmental exposures on health, potentially resulting in higher risks of adverse health effects in individuals exposed to both kinds of stressors. This workshop aimed to share emerging research from multiple disciplines related to the interactions between social stressors and environmental hazards.

Objectives for the workshop were as follows:

1. Review research on the physiological effects of stress and biological pathways through which psychosocial stress and social context may adversely influence health;
2. Share toxicological and epidemiological evidence of the interactions between stress, social context, and environmental hazards; and
3. Discuss how to focus future research efforts on these interactions to best inform policy decisions.

The workshop agenda divided presentations into three themes:

- Theme I: Science of the Impacts of Chronic Psychosocial Stress on Health
    - Robert-Paul Juster presented "The Life Cycle Model of Stress: Detecting how Chronic Stress and/or Trauma Harms Brains and Bodies" which gave an overview of the biological pathways involved in chronic, psychosocial stress and how stress may lead to adverse health outcomes.

- Theme II: Interactions between Chronic Psychosocial Stress, Social Context, Health and Environmental Hazards
    - Dr. Stacy Bilbo's talk, "Interactions between Social Context and Immune and Neuroendocrine Systems in Laboratory Animals," detailed the synergistic and gender-specific effects of early exposures to air pollution and social stressors on neural development and cognitive impairments in mice.
    - Dr. Rosalind Wright discussed how interactions between psychosocial stressors and environmental exposures in utero may lead to increased risk of wheezing and asthma in children during her presentation "Perinatal Stress and Physical Environmental Influences on Urban Childhood Asthma: Independent and Interactive Effects."
    - Dr. Joel Schwartz presented "Evidence Review from Epidemiological Data - Interactions between Air Pollution and Social Context and Implications for Risk Assessment," which reviewed the epidemiological evidence of the influence of social context on the effects of exposure to air pollution, lead, and temperature increases as well as the importance of incorporating population heterogeneity into risk assessments.
    - Dr. Deborah Cory-Slechta, in "Combined Effects of Lead and Stress on Impulsivity Measured and How These Effects are Mediated by Brain and HPA Axis," presented her research on the synergistic and gender-specific effects of exposure to lead and stress in animal models.
- Theme III: Measuring Chronic Psychosocial Stress and Social Context in Research
    - Robert-Paul Juster's second presentation, "The Allostatic Load Model: Indicators, Indices and Methods to Measure the Antecedents and Consequences of Chronic Stress throughout the Life Cycle," discussed the concept of the allostatic load model and the development of methods to measure chronic stress in a clinical setting.
    - Dr. Lynne Messer's presentation, "Measuring Neighborhood Context for Research Addressing Maternal and Child Health Disparities," shared her research on methods to measure the relationship between the built environment and maternal and child outcomes.
    - Dr. Jane Clougherty presented "Disentangling Spatial Patterns in Social and Environmental Exposures for Epidemiology" and detailed methods for using spatial data and geographic information systems to better characterize the relationship between stress, environmental hazards, and childhood asthma exacerbation.

These presentations were followed by a panel discussion entitled "communication/ translation of research on chronic psychosocial stress and environmental hazards to the public, policy makers, public health practitioners and clinicians." This discussion included environmental health researchers Dr. Cory-Slechta and Dr. Madeleine Scammel, community advocate Peggy

Shepard, executive director of WE ACT for Environmental Justice, and regulatory scientist Neal Fann of EPA's OAQPS.

This report presents a summary of each of the workshop themes and then describes several emerging areas of research that appeared across presentations, including the influence of timing of exposures on health outcomes, evidence for resilience to stress, and differences in susceptibility. It concludes with a summary of the panel discussion. Four appendices are included with this report: the workshop agenda, presentation abstracts, speaker biosketches, and a list of workshop attendees.

THEME I: SCIENCE OF THE IMPACTS OF CHRONIC PSYCHOSOCIAL STRESS ON HEALTH[1]

Over the course of evolution, humans have developed a "flight-or-fight" stress response to survive potentially life-threatening situations. This response involves subconscious physiological changes that allow individuals to quickly adapt to immediate threats to their well-being. Although these biological pathways evolved in response to urgent situations, these same physiological changes occur in response to chronic psychosocial stress. Activating the stress response to escape immediate threats to bodily harm offers a survival advantage. However, chronic activation of these pathways is thought to play a role in the development of numerous chronic diseases.

Mr. Juster and Dr. Wright gave broad overviews of the physiological mechanisms involved in the stress response. The body mediates stress through two physiological systems; the sympathetic-adrenomedullary (SAM) system and the hypothalamic-pituitary-adrenocortical (HPA) axis. Following exposure to external stressors, individuals automatically and subconsciously determine whether the situation presents a possible threat, in a process known as appraisal. If the individual perceives the situation to be stressful, signals from the hypothalamus stimulate the adrenal glands to produce hormones (epinephrine and norepinephrine) that stimulate the "fight or flight" response of the SAM system. This response increases heart rate, respiration, blood pressure, and blood flow to muscles, and decreases digestion, among other changes. The HPA axis regulates the body's stress response through the release of hormones in the brain (hypothalamus) and pituitary gland. These hormones tightly control the amount stress hormones, such as cortisol, released by the adrenal glands. Because elevated cortisol levels can be damaging, the body relies on negative feedback loops to lower cortisol levels when the stress response is no longer needed.

Chronic activation of the stress response affects multiple systems in the body and may lead to endocrine, metabolic, autoimmune, and mental health issues. Dr. Wright noted that stress may disrupt immune system function, increase oxidative stress, and alter gene expression. The immune system responds to feedback from the autonomic nervous system which in turn is influenced by hormones and cytokines (cell signaling molecules) released by the immune system. Dr. Cory-Slechta stated that abnormal HPA axis response is associated with numerous diseases including osteoporosis, heart disease, diabetes, autoimmune diseases, Alzheimer's disease, and psychiatric disorders.

[1] Mr. Juster gave the only full presentation on this theme. However, this section includes relevant information provided by other speakers during the workshop.

Chronic stress elevation brought on by long-term exposure to community violence or racism, as well as chronic effects from acute stressors such as job loss or emotional trauma may affect the development and size of certain brain regions. Different regions of the brain, including the amygdala, hippocampus, prefrontal cortex, and hypothalamus, are involved in the activation of the body's stress response. The amygdala regulates emotional response to events, especially fear, anger, and aggression. The prefrontal cortex controls cognitive processes such as moral behavior, planning, attention, and reasoning. The hippocampus is involved in learning and memory. Mr. Juster illustrated this concept using research observations on the effect of chronic stress on the development of mental health disorders such as Post Traumatic Stress Disorder (PTSD). Studies show that individuals with schizophrenia, depression, or PTSD are more likely to have a smaller hippocampus. Some researchers propose that stress has a neurotoxic effect that causes the hippocampus to shrink while other researchers argue that individuals with a smaller hippocampus are more vulnerable to developing certain mental health problems such as PTSD. One model that bridges these two hypotheses is the life cycle model of the effect of stress on development. Adversity early in life may affect brain development and lead to a smaller hippocampus. Animal studies show that maternal separation and poor maternal care accelerate the development of the amygdala and cause hippocampal atrophy. Children of depressed mothers have higher cortisol levels and larger amygdala and lower levels of cortisol produced in response to stress. Individuals with a smaller hippocampus may be predisposed to developing certain mental health issues following exposure to social stressors. In summary, the life cycle model suggests that chronic exposure to stress in early life has been associated with hippocampal atrophy, which in turn may impair the brain's response to social stressors later in life. A smaller hippocampus may predispose individuals to certain mental health disorders such as PTSD

Finally, presenters briefly highlighted the issues of stress perception and appraisal, as well as the concepts of harmful and helpful stress. Noting that the term *stress* has multiple colloquial meanings, Mr. Juster offered an explanation for psychosocial stress that is potentially harmful based on the exposed individual's appraisal, and described such potentially harmful stress as that which involves situations that are novel, unpredictable, threaten the ego or involve a sense of loss of control. Perception of stress was considered the "exposure agent" or trigger for physiological responses by some presenters who argued that external events must be perceived as stressful to lead to negative emotional and physiological responses. For example, loud noise is not bothersome if it is not heard. Dr. Messer argued that stressors may affect individuals negatively even without their direct acknowledgement of the stressor or of their response to the stressor.

THEME II: INTERACTIONS BETWEEN CHRONIC PSYCHOSOCIAL STRESS, SOCIAL CONTEXT, HEALTH AND ENVIRONMENTAL HAZARDS

Multiple human and animal studies illustrate how early exposure to pollutants in combination with severe or chronic stress can change the pattern of the developing brain, leading to lifelong social and psychological effects. The timing and duration of exposures to both chemical and psychosocial stressors play a critical role in the long term effect of exposure. Stress exposure may change the genetic programming of developing fetuses or children and can permanently alter physiology. The synergistic effect of exposures to pollution and social stress often leads to more severe health outcomes than either exposure alone.

TOXICOLOGICAL EVIDENCE OF THE INTERACTION BETWEEN CHRONIC PSYCHOSOCIAL STRESS AND ENVIRONMENTAL HAZARDS

Animal studies performed by Dr. Cory-Slechta and Dr. Bilbo involving exposure to pollutants at levels equivalent to higher inner city exposures demonstrate that prenatal and early postnatal exposures to social and chemical stressors have physiological effects that last into adulthood. Exposures during critical periods of development, such as *in utero*, may lead to permanent physiological or neurological changes. These studies in mice and rats focus on air pollution and lead exposures and suggest that maternal stress interacts with exposure to these contaminants and may harm developing fetuses.

Dr. Bilbo presented evidence that that concurrent exposure to air pollution and maternal stress increases the likelihood of cognitive impairment in adulthood. Mice exposed only to air pollution early in development (prenatally to diesel exhaust and postnatally to ozone) showed airway hyper reactivity as adults long after the exposure period ended. When these exposures were combined with maternal stress, markers of stress increased in both mothers and pups. Offspring weighed less and had higher stress hormone levels. Male offspring showed increased memory impairment and higher anxiety. The reason for these impairments may be that stress and diesel exhaust affect the immune response in the brain, leading to cognitive problems. These impairments may be mediated through impacts to microglial cells, which along with other immune factors, aid other neural cells in development and maturation. Both mothers and offspring showed evidence of neuroinflammation, including increases in the immune signaling molecules cytokines and chemokines. These early exposures to maternal stress and air pollution appeared to have lifelong health impacts in mice.

Dr. Cory-Slechta's research shows that prenatal exposure to lead in rats produced lifelong changes in the stress response which is amplified by concurrent prenatal exposure to social stress. Rats normally increase the stress hormone corticosterone over the first year of life, but animals exposed to both prenatal stress and lead did not show this developmental pattern. Male rats exposed to a plausible human exposure level of lead in utero displayed an elevated stress hormone response following a stress challenge. Interestingly, rats of either gender exposed at higher levels did not show this response. Rats exposed to both lead and stress prenatally had impaired cognitive functions, including learning impairments and greater impulsivity. Both lead and social stress appear to affect similar biological pathways, including the HPA axis and the mesocorticolimbic system, which is involved in learning and memory. Fetal exposure to either or both lead and stress appeared to cause lifelong

alterations to both of these pathways. Although the placenta protects the fetus against normal elevated maternal cortisol levels during pregnancy, it may not be able to protect against high levels due to chronic stress. A developing fetus will respond to the presence of excess fetal glucocorticoids or lack of nutrition by permanently altering its physiology. These physiological changes may be the mechanism for the cognitive impairments observed. Dr. Cory-Slechta noted that epidemiologic studies show an association between maternal stress during pregnancy and language development in human toddlers as well as attention deficits in children.

These researchers observed that the interaction effects of stress on the observed relationship between exposure to environmental toxins and stress and the development of cognitive deficits appears to be gender specific. Dr. Bilbo reported that in her research in addition to the observation that only males exposed to diesel exhaust *in utero* showed more anxiety-like behaviors and impaired memory, these effects were also aggravated by concurrent exposure to maternal stress *in utero*. Dr. Cory-Slechta noted that female rats exposed to both lead and stress *in utero* showed signs of learning impairments. Exposure prenatally to stress or lead alone did not produce these cognitive deficits. However, exposure to lead and stress prenatally increased impulsivity in both female and male rats. Further work by Dr. Bilbo suggests that a possible mechanism for observed gender-specific interactions between stress and cognitive effects is through neuroimmunomodulatory pathways in which the early immune system influences brain development during critical sensitive periods. She discussed experiments that showed that exposure to *E. coli* during the neonatal period alone did not lead to long-term effects. However, a second exposure during adulthood led to social and cognitive deficits in male mice only. The differences in the immune response observed in male and female mice may explain these findings. These animal studies suggest that exposures to social and chemical stressors during the prenatal and neonate periods lead to increased risk of adverse health and cognitive impacts later in life in a gender-dependent manner.

EPIDEMIOLOGICAL STUDIES ON THE INTERACTION BETWEEN CHRONIC PSYCHOSOCIAL STRESS AND ENVIRONMENTAL HAZARDS

Dr. Wright presented her research on the association between stress exposure and the risk of developing asthma. Her research measured the number of negative life events such as financial strain, racism, discrimination, interpersonal or community violence as well as exposure to allergens in children and mothers. Children whose mothers had traumatic life experiences had higher levels of certain immune factors indicative of asthma. These children took longer to recover following a stress challenge at six months of age, as measured behaviorally and via heart rate. Both prenatal and postnatal maternal stress, especially postnatal financial strain, was associated with repeated wheeze episodes before two years of age. Infants whose mothers reported the highest levels of social stress presented with wheeze patterns similar to children exposed to black carbon. This finding is important because repeated wheeze at a young age increases the risk of asthma. Children of atopic (allergic) mothers exposed to high levels of allergens at home showed a linear relationship between IgE (marker of reactivity to allergens) and stress. Non-sensitized mothers showed a large effect of prenatal stressors on repeated wheeze in their children. Both high community violence and

traffic-related air pollution were associated with repeated wheeze and may have had an interactive effect. Maternal cortisol levels may explain these findings. Mothers with children who wheezed had greater diurnal changes in cortisol levels. Prenatal maternal stress levels may lead to changes in fetal immune response and, therefore, increased risk for wheezing.

Dr. Schwartz presented multiple epidemiological studies that emphasized the importance of understanding how social and biological factors influence susceptibility to air pollution, temperature changes, and lead exposure. Dr. Schwartz pointed to evidence that lower socioeconomic status is associated with a higher prevalence of certain diseases, such as diabetes, that increase susceptibility to air pollution and high temperatures. All-cause mortality risk from air pollution also is inversely associated with educational attainment, a marker of socioeconomic status. Chronic exposure to variability in summer temperatures is associated with minority status. The odds of mortality, especially cardiovascular mortality, from exposure to extreme high temperatures are modified by race, diabetes status, and education. These results indicate that social factors influence the risk of mortality from air pollution and temperature changes.

Lead exposure has long been associated with cognitive impairment. Recent studies have shown that cognitive effects are seen below levels previously believed to be safe (10 ug/dL). Minority, lower socioeconomic status, and medically underserved children have a higher risk of exposure to lead and are more likely to be exposed to social stressors. Dr. Cory-Slechta noted that low socioeconomic status leads to increased risk of chronic elevation of stress hormones (such as glucocorticoids), which may increase the risk of certain diseases. Similar to the animal studies summarized previously, epidemiological studies presented by Dr. Schwartz suggest that concurrent exposure to lead and social stress increases the likelihood and magnitude of cognitive function deficits in both adults and children. Residence in neighborhoods with more psychosocial hazards was associated with cognitive deficits in older adults. Men who reported greater stress levels showed a stronger association between level of bone lead and cognitive impairments. The effects of lead exposure in children is also modified by socioeconomic status (as measured by parental education or paternal occupation), with lower levels of lead associated with cognitive deficits. Acute stress in these children has been associated with modified vascular response. Hypertension risk is greater for the combination of lead and social stress exposure than either exposure alone. Because the risk of negative cognitive effects from lead may be greater in children and adults concurrently exposed to high levels of social stress, policies that incorporate these findings may better protect against cognitive impairments due to lead exposure.

Moving toward advancing the integration of these findings with environmental policy, Dr. Schwartz outlined several problems with current risk assessment approaches, a key analytical and decision-making framework at EPA. Studies have established that age, health status, and genetics play a role in susceptibility to environmental exposures. However, most toxicological studies rely on healthy, same-age animals with a similar genetic make-up. This lack of diversity in animal models may lead to erroneous conclusions regarding the effects of certain exposures. Dr. Schwartz used examples from studies on particulate matter and heat to demonstrate that assuming average risk of adverse outcomes across a geographic area will fail to account for actual risk. Setting standards based on these averages may fail to protect

vulnerable individuals. Likewise, the mean response across multiple toxicological or epidemiological studies will appear normally distributed even if individual studies show a skewed response. Heterogeneity in human populations, whether due to social factors such as income, or innate factors such as genetics or disease status, is likely to lead to a range of thresholds for a given toxin. This range of thresholds may be best modeled assuming a linear concentration-response rather than a single threshold to find the standard that will protect vulnerable members of the population. Dr. Schwartz argued that heterogeneity has important implications for risk assessment and policy decisions. Incorporating factors that influence heterogeneity in exposure and susceptibility allows risk assessments to better reflect actual risks. Understanding factors that change susceptibility may allow risk assessments to better inform policy decisions and protect public health.

THEME III: MEASURING CHRONIC PSYCHOSOCIAL STRESS AND SOCIAL CONTEXT IN RESEARCH

Researchers have developed a number of techniques to quantify the level of exposure to chronic psychosocial stress. These measures include determining clinical biomarkers of chronic social stress, as discussed in Mr. Juster's presentation of the allostatic load model, as well as developing methods to measure the influence of the built environment on health outcomes, as detailed by Drs. Messer and Clougherty. These approaches to measuring chronic social stress allow researchers to better elucidate the relationship between stress, environmental exposures, and health impacts.

ALLOSTATIC LOAD MODEL

Mr. Juster presented the concept of allostatic load to understand how chronic stress can lead to health problems. This theory proposes that the body reaches stability by continuously changing physiological parameters to match environmental conditions in a process called allostasis. The body evaluates environmental stressors and reaches set points that both meet environmental demands and conserve resources. This framework of how social stress may lead to adverse health outcomes proposes that through the process of constant readjustment to match stressful conditions, the body may wear out physiological systems that allow adaptation to changing conditions. This "wear and tear," known as allostatic load, may be the mechanism for the development of adverse health outcomes from chronic stress exposure. The theory of allostatis allows a more complete understanding of how psychosocial stress may lead to certain health outcomes.

Traditional understanding of how the body maintains stability, known as homeostasis, posits that physiological systems rely on internal negative feedback loops to ensure that physiological parameters do not deviate from fixed set points. The model of allostasis was first proposed by Sterling and Eyer[2] in 1988 and offers a more dynamic understanding of the interaction of biological functions and describes how the body changes set points in response to external influences. For example, the allostatic model explains why chronically stressed rats will continue to have high blood pressure after the removal of experimental stressors.

2 Sterling, P. and Eyer, J., 1988, Allostasis: A new paradigm to explain arousal pathology. In: S. Fisher and J. Reason (Eds.), Handbook of Life Stress, Cognition and Health. John Wiley & Sons, New York.

Rather than returning to a baseline blood pressure, as expected with the homeostatic model, rats increased the set point of their blood pressure in response to external stressors. The allostatic model proposes that the body has mechanisms to anticipate physiological changes and that behavior can influence physiological response. Consider the experience of feeling dizzy after standing up too quickly. Once the individual realizes the cause (standing up too fast), he or she may choose to change behavior (stand up more slowly) to avoid feeling lightheaded. Rather than understand the dizziness response only as a physiological reaction to gravity, allostasis incorporates the change in behavior. Allostatic changes begin with primary mediators (e.g. stress hormones) that act as messengers to cause primary effects such as cellular changes. The combined effects of these subclinical changes may lead to secondary outcomes, such as changes in blood pressure, cholesterol, glucose levels, etc. These clinical markers can be a sign of the presence or risk of developing stress-related diseases (tertiary outcomes).

Because allostatic load may indicate the presence or risk for certain diseases, researchers such as Mr. Juster are investigating methods to allow clinicians to test levels of allostatic load. To measure allostatic load, researchers are developing an "allostatic index" consisting of four to ten biomarkers spanning the neuroendocrine, immune, cardiovascular, and respiratory systems and metabolic and anthropometric measures. Several major studies in the United States, Taiwan, and Europe have shown a correlation between higher allostatic load, as measured by an allostatic index, and lower socioeconomic status, lower self-rated health, less social mobility, higher job strain, career instability, minority status, and older age. Allostatic states that may be indicative of risk of poor health include both an inadequate stress response as well as a continued heightened response with failure to recover. Some of the ongoing research on allostatic load aims to find the parameters that best predict adverse health outcomes in order to give clinicians a tool to measure allostatic load prior to the development of disease. Generally speaking, the allostatic model suggests that a holistic approach to patient treatment in the clinical setting should aim to reduce arousal of the stress response in order to prevent or mitigate disease.

MEASURING BUILT ENVIRONMENT METRICS TO QUANTIFY IMPACTS OF CHRONIC SOCIAL STRESS

Drs. Messer and Clougherty presented their work developing methods to measure and analyze the impact of neighborhoods and the built environment on health outcomes. Their research focuses on designing and validating novel techniques to accurately assess how neighborhoods influence exposures to social stress and to environmental toxins as well as the risk of negative health effects from these exposures. Dr. Messer's work aims to quantify the effect of the built environment on pregnancy outcomes and Dr. Clougherty's research looks at factors that influence asthma exacerbation in urban children.

Dr. Messer noted that studies show neighborhoods have modest but lasting associations with health disparities, and that where people live can influence their level of exposure to both social stress and environmental toxins and their risk of adverse health outcomes. In order to better understand factors in the built environment that influence health outcomes, she assessed tax parcels in Durham, NC for descriptive variables, including the condition (peeling paint, broken windows, etc.), nuisances (such as litter, discarded furniture and graffiti), and

property descriptors (property type and vacancy status). She combined visual assessments of properties with data on crime, tenure, and amenities and analyzed data on a parcel-level, census block-level, and primary adjacency community level (parcels adjacent to index block). Her research found that certain housing and property metrics were associated with higher odds ratios for pre-term birth, small for gestational age, decreased birth weight, and decreased birth weight percentile for gestational age. In a follow-up study, the Healthy Pregnancy-Healthy Baby Cohort assessed the influence of genetic, environmental and social factors on birth outcomes. Unadjusted models showed relationships between certain built environment metrics and the mother's psychological state, while adjusted models demonstrated a significant relationship between living in a rental property and negative paternal support as well as "John Henryism" (working harder when faced with adversity). This research demonstrated a methodology to measure the built environment that assesses, rather than samples, an entire community and showed that neighborhood conditions may influence birth outcomes.

Dr. Clougherty presented her recent work designing a study in New York City that uses resident-defined neighborhood boundaries to elucidate the relationship between residential location, exposure to both traffic-related air pollution (TRAP) and social stressors, and childhood asthma exacerbation. This research will involve 25 focus groups where residents identify stressors of concern. Further surveys will determine if individuals' stress level is related to these stressors. The spatial patterns of social stressors and air pollution will be assessed using data from multiple sources, including census tracts, school districts, police precincts, etc. Because TRAP is a complex mixture of pollutants that varies across space and because social stressors and pollutants cluster spatially, this study will use statistical techniques that correct for spatial confounding and autocorrelation to understand the effects of air pollution, psychosocial stress, and socioeconomic status on asthma hospitalizations in children. Dr. Clougherty stated that stratifying geographic areas by effect modifiers of risk, such as markers of socioeconomic status (e.g., education) or other sources of variability, such as disease prevalence, will more accurately portray the distribution of risk. This study will examine the effects of social stressors and TRAP on hospitalization rates for asthma in children and explore whether social stress modifies the effects of air pollution on asthma exacerbation.

Drs. Clougherty and Messer discussed a number of issues related to data source availability for understanding spatial relationships of synergistic effects between social stressors and environmental contaminants. In order to accurately classify exposures and population characteristics by neighborhood, researchers need to integrate data from multiple sources. However, neighborhoods often do not map to administrative boundaries, such as census tracts or school zones and these administrative borders may not line up with each other. Dr. Clougherty's study will use statistical techniques to combine exposure and population variables from multiple, incongruous administrative areas. However, Dr. Messer compared several geographic units of analysis and found that census blocks and census tracts acted as good proxies for most neighborhood exposures, except for walkability. Dr. Clougherty discussed the potential for exposure misclassification when using metrics with differing scales of spatial variation. For example, the percentage unemployed varies block by block,

while differences in racial composition range across a larger geographic area. Additionally, social stressors identified by residents may be difficult to measure directly and researchers must consider related indicator variables as surrogates. Dr. Clougherty noted that the metrics chosen as markers for susceptibility may influence observed spatial differences in susceptibility. Dr. Messer noted that additional metrics not assessed in her work may influence birth outcomes and that further research will continue to refine metrics used to assess neighborhoods. Understanding these factors can lead to more accurate assessments of the interaction between social stressors, environmental hazards, and the risk of adverse health effects.

Understanding associations between neighborhood effects may lead to interventions that improve birth outcomes and reduce the rate of hospitalizations for asthma. Research that develops innovative methods for measuring the influence of neighborhoods on exposure to chronic social stress and environmental toxins, may give policy makers better tools to set standards that protect vulnerable populations.

CROSS CUTTING ISSUES/EMERGING THEORIES

IMPACTS OF THE TIMING OF SOCIAL STRESS EXPOSURE

Multiple presenters discussed the theory that timing of exposures to both social stress and chemical contaminants influences the nature of the interactions and the ultimate impacts of these exposures. Toxicological studies in animals and epidemiological evidence in humans support the idea that these exposures have the greatest impact during certain vulnerable periods during growth and development. Early exposures may cause genetic modifications that lead to lifelong cognitive and physical health problems. Susceptibility to social stressors and environmental contaminants may depend on the age and timing of exposure.

Evidence in both animal and human studies indicates that quality care during young childhood appears to prevent many health effects associated with exposure to social stress. Dr. Wright noted that the degree of vulnerability to maternal stress in children is highly regulated by social context. Exposure and response to maternal stress begins *in utero* and continues to develop after birth. Increased levels of stress hormones in children due to exposure to social stressors may impact immune function and lead to health effects in adulthood. Some individuals are more highly sensitive to stress exposure due to genetic make-up. However, one of the key factors in avoiding later health effects from exposure to social and chemical stressors is attentive and supportive care of young children. Responsive care seems to prevent increased levels of stress hormones in children and poor quality care seems to increase vulnerability to stress. Interventions during this period of development may improve lifelong health.

Exposures to social and chemical stressors early in life may influence health in adulthood due to genetic modifications known as epigenetics. This process involves molecular changes that regulate the level of expression of certain genes without altering the underlying DNA sequence. Epigenetic changes during fetal and early childhood development can have lifelong impacts. Dr. Cory-Slechta stated that exposure to elevated cortisol during fetal development may permanently alter the HPA axis in a similar manner to lead exposure. Mr.

Juster discussed how stress during prenatal development affects the programming of the HPA axis. From birth through two years, the hippocampus rapidly develops and may be most affected by stress early in life. As discussed previously, the hippocampus plays a key role in the stress response, signaling the adrenal gland to release stress hormones. Controlled experiments in animals support the idea that attentive care may mitigate the effect of social stress exposure on the hippocampus. Dr. Bilbo presented studies that indicate augmented maternal care in mice (attentive grooming and nursing behaviors) increases the expression of anti-inflammatory cytokines by altering the expression of the glucocorticoid receptor in the hippocampus through epigenetic changes. These levels of gene expression were not due to genetic inheritance alone, as mice fostered by more attentive mothers showed the same pattern of epigenetic changes and pups later fostered by less attentive mothers reversed these epigenetic changes. These molecular responses occurred during the first week of life and lasted into adulthood. As mentioned earlier, male mice exposed to *E. coli* as pups and again as adults showed signs of cognitive and social deficits. However, these results were seen only in pups lacking attentive care. Pups with augmented maternal care did not show deficits following the second immune challenge. Attentive maternal care appeared to give lifelong protection. These animal studies support the idea that intervention may mitigate the harm from social stress exposure, with long-term health benefits.

Concurrent early exposure to environmental contaminants and social stressors may affect physical and mental health in adults. Dr. Schwartz discussed how childhood socioeconomic status predicts adult socioeconomic status as well as lifetime lead exposure. Mr. Juster noted that the timing of stress exposure may influence which areas of the brain are affected. Epigenetic changes due to psychosocial stress may explain this association. For example, Dr. Schwartz noted that low optimism and higher anxiety scores were linked to increased gene expression (decreased methylation) in individuals exposed to air pollution. Evidence from both human and animal studies demonstrates that the timing of exposure to social stress and environmental contaminants may have lifelong effects.

RESILIENCE TO PSYCHOSOCIAL STRESS

Several presenters noted that despite clear associations between chronic social stress and negative health outcomes, certain types of stress may be beneficial by encouraging resilience. Appropriate stress response is important for normal social functioning. Mr. Juster discussed that among individuals who grew up in lower socioeconomic status households those who had the "shift and persist" personality trait had the lowest allostatic load. This personality trait tends to frame adversity more positively and continues to focus on the future despite hardships. This correlation was not seen in children from higher socioeconomic status households. Stress that motivates individuals may teach resilience rather than lead to negative health outcomes. However, most studies detailed in this workshop found chronic social stress detrimental to later health status.

DIFFERENCES IN SUSCEPTIBILITY

Multiple speakers presented findings showing that socioeconomic status, race, gender, genetics, disease status, and other factors influence the effect-modifying potential of social stress on the relationship between environmental exposures and adverse health outcomes.

Speakers emphasized that this heterogeneity should be taken into account when setting standards and that current policy decisions may not adequately protect vulnerable populations. Multiple toxicological and epidemiological studies demonstrate that social stress acts as an effect modifier for multiple environmental exposures, including air pollutants, lead, and temperature changes and that risk of exposure to social stress and to environmental toxins is correlated with socioeconomic and minority status. Studies in rodents show how gender influences the response to stress and pollutant exposures. Detailed analyses of geographic differences in susceptibility suggest that both exposure levels to social and chemical stressors as well as risk of adverse health outcomes from these exposures vary by neighborhood. By understanding characteristics that lead to heterogeneous responses to contaminants, policy makers can choose policy options that take into account distributional effects and design policies that reduce disparities in health.

PANEL DISCUSSION SUMMARY

The workshop ended with a panel discussion about ways to translate research on social stressors and environmental toxins to improve public policy and how best to communicate results to the public, policy makers, and clinicians. The panel was comprised of Peggy Shepard, a community advocate, EPA regulatory scientist Neal Fann, and environmental health scientist Dr. Madeleine Scammel. Dr. Cory-Slechta moderated the panel. The discussion was framed by a series of questions on how researchers, regulators, and communities can coordinate to improve public policy and public knowledge to reduce exposures to environmental and social stressors.

- *What are the traditional ways that EPA would incorporate information on sensitive populations in regulatory development decisions?*

Mr. Fann answered that EPA sets the standards for criteria pollutants, such as particulate matter (PM) and ozone, to be protective of sensitive subpopulations. The Transport Rule demonstrates an example of how environmental justice analyses are used to justify decisions, rather than used as an integral part of the rulemaking process. Through an environmental justice analysis EPA determined the baseline distribution of risk of mortality from PM by county and by race, income, and educational attainment. The analysis showed that PM-related mortality decreased across the population and the most substantial decrease was seen in these sensitive subgroups. Mr. Fann added that this analysis was completed later than the economic and other regulatory analyses and did not directly inform policy decisions made in developing the Transport Rule. These types of analyses are data- and time-intensive, and are often difficult to complete early in the regulatory process. However, incorporating this information earlier in the process may facilitate the inclusion of equity considerations throughout the policy development process.

Panelists debated whether EPA has a mandate to assess impacts on vulnerable subpopulations. Ms. Shepard stated that the 1994 Executive Order 12898 on environmental justice includes an unfulfilled mandate for such assessments and that guidance on cumulative impact from the 1990's has still not been incorporated into agency decision-making. Other panelists commented that EPA assesses impacts of rules on vulnerable subpopulations, even

when not mandated to perform these analyses. All panelists agreed that EPA needed to expand its ability to perform these assessments.

- *What types of data would be helpful to a regulatory analyst and decision maker in conducting environmental justice analyses?*

Better characterization of the distribution of exposures and health effects across subgroups would help inform analyses of environmental justice concerns. Reports detailing the monetized benefits for a given rule do not give information on risks for sensitive subgroups or on equity issues. The data needed to fully assess environmental justice concerns often comes from multiple sources, including state and county databases. For this reason, data on social factors and psychosocial stress may not be consistently formatted or straightforward to compile. EPA has had many discussions on ways to improve data interoperability, with the goal of providing spatial tools that combine data on exposure, health, ecosystem services, susceptibility factors, etc. These databases would allow researchers, community groups and community planners easier access these types of data.

Mr. Fann raised the importance of presenting data in a way that managers will feel confident in the conclusions of an assessment. Some methods used in environmental justice analyses are relatively new and EPA has not adopted standard approaches for these evaluations. For example, EPA has not yet established standard indicators for socioeconomic status. Current regulatory processes reflect careful review of existing analytical methods. For instance, the EPA has a well-vetted approach for analyzing the benefits and costs of rules. Therefore, a degree of "analytical inertia" exists that must be overcome in order to update these methods to include environmental justice concerns. Since each program office is autonomous, changes in approaches for incorporating environmental justice into regulatory analysis can take time. Nevertheless, EPA has made important strides in incorporating environmental justice issues into the regulatory process. As EPA continues to develop and refine approaches for characterizing vulnerable subpopulations and understanding the distribution of impacts, incorporating environmental justice concerns in the regulatory process will become easier. Including analyses that detail the impact on environmental justice in an executive summary of a regulatory impact analysis, rather than attached as an appendix, will help show that these considerations play an important role in the decision-making process.

- *Is there an opportunity to accommodate emerging data related to interactions between social stress and environmental hazards in your assessments?*

Two panelists spoke of pilot studies by EPA that improved knowledge of the exposure levels of vulnerable communities. Ms. Shepard mentioned a study where WE ACT and Columbia University worked with EPA to perform community-based monitoring of PM2.5 in Harlem. This study found PM2.5 levels twice as high as the standard. Ms. Shepard stated that these results were used to defend the new standards in court. Because of this study, EPA provided additional resources for community-based air monitoring in New York.

Mr. Fann reported that an EPA pilot in Detroit illustrated that states can develop innovative ways to implement NAAQS that incorporate environmental justice concerns. The study looked at two implementation approaches, the typically-used least cost approach and the

multiple risk based approach. The former method allows the state to just meet the standard. The latter aimed to find the maximum human health benefit by incorporating data on the distribution of exposure among the population and information on how controls on key industrial sectors would reduce exposure for highly exposed subgroups within the population. This approach both meets the air quality standard and improves health and equity across the population.

- *How can the research community best approach studies on social stress and environmental contaminants to inform EPA's regulatory process?*

Several researchers and EPA analysts spoke about the need for better communication between EPA and academic researchers. Dr. Scammel noted that often researchers and EPA are not aware of each other's data needs. She suggested regular conference calls between researchers and EPA to help encourage more policy-relevant research. Dialogue between academics and EPA would provide a forum for researchers to present results in a format that allows analysts to easily incorporate this information into regulatory analyses. For example, better understanding of dose-response information for vulnerable subgroups helps analysts better determine the distributional impacts of proposed rules. Dr. Cory-Slechta mentioned that the funding opportunities at other agencies, such as the National Institutes of Health do not always match EPA's research needs. Direct grants from EPA, such as the Cumulative Risk Grants, encourage researchers to improve understanding of distributional impacts on vulnerable and sensitive subpopulations.

Participants agreed on the need for cross-disciplinary and multidisciplinary research approaches. For example, the National Academy of Sciences' *Science and Decisions: Advancing Risk Assessment* offers a new paradigm for non-cancer risk assessment. The study of social stressors readily fits this updated risk assessment paradigm. Dr. Cory-Slechta noted that recently EPA recognized the importance of multidisciplinary approaches and added social and behavioral scientists to its Scientific Advisory Board. Audience members noted that environmental health risk assessors should work with researchers in the social and natural sciences to improve understanding of environmental justice concerns.

- *What are ways to help communities identify environmental hazards and learn how they can effect change in institutions to improve their lives?*

Panelists spoke of the importance of using community knowledge in understanding environmental justice issues. Ms. Shepard discussed how after WE ACT surveyed the community, the organization found that it was not working on any of the top concerns of residents (e.g. garbage, rats, litter, drugs, crime). Now the organization incorporates the community's interests into its agenda. She stated that workers are often aware of potentially harmful occupational exposures. Encouraging individuals to share this knowledge with their peers and neighbors can help initiate community action on these issues.

Community-based research that partners with EPA and academic researchers can inform studies on vulnerable subpopulations. These partnerships help shape research questions and allow data to be used to inform public policy. For example, Ms. Shepard discussed a partnership between the University of Pennsylvania and a small town in Pennsylvania that led

to improved water quality. Researchers heard from residents that the town had poor quality tap water. Upon further analysis, the researchers found that the town's water was contaminated and used that data to work with residents to ensure that they had access to clean water. Several community advocates who partner with Dr. Scammel on research efforts noted the importance of explaining to the community how results will be used to increase community participation and ensure that the concerns of the community are met. Encouraging community knowledge to guide scientific studies will improve understanding of environmental justice issues and lead to results that can be incorporated into regulatory decision making.

EPA Workshop on Interactions between Social Stressors and Environmental Hazards
May 14-15, 2012

Agenda

Day 1 - Monday May 14, 2012

Session Title	Speakers	Time
Welcome	Workshop Chairs: Onyemaechi Nweke, DrPH, U.S. EPA Devon Payne-Sturges, DrPH, U.S. EPA	1:00 p.m. - 1:15 p.m.
Theme I: Science of the Impacts of Chronic Psychosocial Stress on Health		
The Life Cycle Model of Stress: Detecting how Chronic Stress and/or Trauma Harms Brains and Bodies	Robert-Paul Juster, Center for Studies on Human Stress, McGill University	1:15 p.m. - 2:15 p.m.
Theme II: Interactions between Chronic Psychosocial Stress, Social Context, Health and Environmental Hazards - Evidence from Epidemiology and Toxicology		
Interactions Between Social Context and Immune and Neuroendocrine Systems in Laboratory Animals	Staci Bilbo, Ph.D., Assistant Professor, Duke Institute for Brain Sciences, Duke University	2:15 p.m. - 3:15 p.m.
BREAK		3:15 p.m. - 3:30 p.m.
Perinatal Stress and Physical Environmental Influences on Urban Childhood Asthma: Independent and Interactive Effects	Rosalind Wright, M.D., Associate Professor of Medicine, Channing Laboratory, Harvard Medical School and Associate Professor of Environmental Health, Harvard School of Public Health	3:30 p.m. - 4:30 p.m.
END OF DAY 1		

EPA Workshop on Interactions between Social Stressors and Environmental Hazards May 14-15, 2012

DAY 2: Tuesday May 15, 2012

Welcome Remarks/Re-cap	Workshop Chairs: Onyemaechi Nweke, DrPH, U.S. EPA Devon Payne-Sturges, DrPH, U.S. EPA	8:15 a.m. - 8:30 a.m.
Theme II: Interactions between Chronic Psychosocial Stress, Social Context, Health and Environmental Hazards - Evidence from Epidemiology and Toxicology *(continued)*		
Evidence Review from Epidemiological Data - Interactions between Air Pollution and Social Context and Implications for Risk Assessment	Joel Schwartz, Ph.D., Professor of Environmental Epidemiology, Harvard School of Public Health	8:30 a.m. - 9:30 a.m.
Combined Effects of Lead and Stress on Impulsivity Measured and How These Effects are Mediated by Brain and HPA Axis	Deborah Cory-Slechta, Ph.D., Professor of Environmental Medicine and Pediatrics, University of Rochester Medical School	9:30 a.m. - 10:30 a.m.
BREAK		10:30 a.m. - 10:45 a.m.
Theme III: Measuring Chronic Psychosocial Stress and Social Context in Research		
The Allostatic Load Model: Indicators, Indices and Methods to Measure the Antecedents and Consequences of Chronic Stress Throughout the Life Cycle	Robert-Paul Juster, Center for Studies on Human Stress, McGill University	10:45 a.m. - 11:45 a.m.
LUNCH		11:45 a.m. - 1:15 p.m.

EPA Workshop on Interactions between Social Stressors and Environmental Hazards May 14-15, 2012

DAY 2: Tuesday May 15, 2012 (continued)

Theme III: Measuring Chronic Psychosocial Stress and Social Context in Research *(continued)*		
Measuring Neighborhood Context for Research Addressing Maternal and Child Health Disparities	Lynne Messer, Ph.D., M.P.H., Assistant Research Professor, Center for Health Policy, Duke Global Health Institute	1:15 p.m. -2:15 p.m.
Disentangling Spatial Patterns in Social and Environmental Exposures for Epidemiology	Jane Clougherty, Sc.D., Assistant Professor and Director of Exposure Science, Graduate School of Public Health, University of Pittsburgh	2:15 p.m. - 3:15 p.m.
BREAK		3:15 p.m.- 3:30 p.m.
Panel Discussion: Communication/Translation of Research on Chronic Psychosocial Stress and Environmental Hazards to the Public, Policy makers, Public Health Practitioners and Clinicians	**Facilitator:** Deborah Cory-Slechta, Ph.D., Professor of Environmental Medicine and Pediatrics, University of Rochester Medical School **Panelists:** Peggy Shepard, WE ACT for Environmental Justice Madeleine Scammel, D.Sc., Boston University School of Public Health Neal Fann, U.S. EPA	3:30 p.m. - 4:30 p.m.
Adjourn		4:30 p.m.

EPA Workshop on Interactions between Social Stressors and Environmental Hazards May 14-15, 2012

Abstracts

Theme I: Science of the Impacts of Chronic Psychosocial Stress on Health

Title: The Life Cycle Model of Stress: Detecting How Chronic Stress and/or Trauma Harms Brains and Bodies (Robert Juster, Center for Studies on Human Stress, McGill University)

Chronic exposure to stress hormones, whether it occurs during the prenatal period, infancy, childhood, adolescence, adulthood or aging, has an impact on brain structures involved in cognition and mental health. Likewise, lifelong brain changes in turn shape our ability to adapt or mal-adapt to environmental stressors in measurable ways. Of critical importance is the timing and duration at which major stressors and/or traumas are experienced, as this will profoundly affect individuals' neurological development and their vulnerabilities or resistance. The consequences of chronic stress and/or trauma at different life stages depend on which brain regions are developing or declining at the time of the exposure. With regard to brain regions involved in the regulation of stress hormones, the (1) prefrontal cortex, (2) amygdala, and (3) hippocampus are each critically important and differentially sensitive to psychosocial stress. According to the life cycle model of stress, smaller regional volumes in these three substrates will lead to distinct biological signatures that can be used to predict differential risk-profiles for specific psychopathologies in adulthood, as well as inform when certain traumas might have occurred in early life. While direct measurements of central nervous system substrates are costly and invasive, indirect assessments using peripheral biomarkers routinely collected in blood draws and other vehicles (saliva, hair) can be used to determine physiological dysregulations among, for instance, stress hormone levels as well as in allostatic load biomarkers discussed under Theme III of this workshop.

Background reading:
Lupien, SJ. McEwen, BS. Gunnar, MR. Heim, C. 2009. Effects of Stress Throughout the Lifespan on the Brain, Behaviour and Cognition. Nature Rev. Neurosci. 10: 434-445. Doi: 10.1038/nrn2639
Available at: http://www.nature.com/nrn/journal/v10/n6/abs/nrn2639.html

Theme II: Interactions between Chronic Psychosocial Stress, Social Context, Health and Environmental Hazards - Evidence from Epidemiology and Toxicology

Title: Evidence from Toxicology - Interactions between Social Context and Immune and Neuroendocrine Systems in Laboratory Animals (Staci Bilbo, Ph.D., Assistant Professor, Duke Institute for Brain Sciences, Duke University)

Maternal and child health are influenced by multiple host, social, and environmental factors, but very little is known about the interactions of these forces at the neural level. Poverty is associated with combined environmental and psychological stressors, including toxin exposure, poor air quality, and sub-standard housing, which we believe can interact and possibly synergize to worsen physiological outcomes. Specifically, the presence of psychosocial stressors may increase vulnerabilities to chemical stressors such as air pollution during pregnancy, and result in adverse outcomes (such as asthma) in the

offspring that would not otherwise occur. Our work is designed to test the hypothesis that combined stressors early in life synergize to produce long-term changes in cognitive, affective, and physiological (e.g., asthma) outcomes. We are using a murine model that includes a significant social stressor (restricted bedding material) and an environmental exposure of known importance to health outcomes (air pollution). Recent data suggest that asthma and mental health problems, such as cognitive disability and depression, are strongly linked, independent of precipitating factors such as poverty. Thus we are interested in making the connection between neural and respiratory developmental outcomes associated with exposure to social and environmental stress. While we are using an animal model approach, we are working concurrently to understand the outcomes from this research within the context of human exposures and outcomes.

Background reading:

Bilbo, SD. Schwarz, JM. 2009. Early-life Programming of Later-life Brain and Behavior: A critical role for the immune system. Front. Behav. Neurosci. 3(14): 1-14. Doi: 10.3389/neuro.08.014.2009
Available at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2737431/

Title: Perinatal Stress and Physical Environmental Influences on Urban Childhood Asthma: Independent and Interactive Effects (Rosalind Wright, Associate Professor of Medicine, Channing Laboratory, Harvard Medical School and Associate Professor of Environmental Health, Harvard School of Public Health)

Children in lower socioeconomic status (SES) urban communities and ethnic minorities are at increased risk for wheezing respiratory illnesses and asthma compared to their higher SES counterparts. Such disparities may be explained by differential environmental exposures, including both psychosocial stressors and physical toxins, which cluster in more socially disadvantaged communities. Thus, transdisciplinary studies that consider both individual- and community-level psychosocial determinants and physical toxins together may help us better understand heterogeneities in urban asthma expression. Candidate factors identified in our ongoing urban cohort studies include community violence exposure, ambient pollution, and indoor aeroallergens. Both community- and individual-level stress have been linked to asthma expression, and urban low-SES families exposed to higher community violence are also more likely to experience chronic stress over other life domains (e.g., financial strain, discrimination, housing problems). Lower-SES populations, experiencing greater cumulative stress, may also be more likely to be exposed to other environmental toxins linked to asthma expression, such as household allergens related to poorer housing stock or traffic-related air pollution. Epidemiological data will be reviewed, demonstrating the independent effects of stress on childhood asthma when controlling for these co-varying physical determinants. These data also exemplify how co-occurring psychosocial and physical exposures may combine to influence or modify respiratory health risk in these urban children.

Background reading:

Peters, JL. Cohen, S. Staudenmayer, J. Hosen, J. Platts-Mills, TA. Wright, RJ. 2012. Prenatal Negative Life Events Increases Cord Blood IgE: Interactions with dust mite allergen and maternal atopy. Allergy. 67(4): 545-551. Doi: 10.1111/j.1398-9995.2012.02791.x
Available at: http://onlinelibrary.wiley.com/doi/10.1111/j.1398-9995.2012.02791.x/abstract;jsessionid=B4CAAD364E7ADB0F7F04523EBE174BEA.d02t01

Wright, RJ. 2009. Moving Towards Making Social Toxins Mainstream in Children's Environmental Health. Curr. Opin. Pediatr. 21(2): 222-229. Doi: 10.1097/MOP.0b013e3283292629
Available at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2752500

Title: The Interaction of Social Factors and Environmental Exposures in Risk: Issues and Results from Epidemiologic Studies (Joel Schwartz, Ph.D., Professor of Environmental Epidemiology, Harvard School of Public Health)

While the principle that social factors may modify response to environmental agents has long been understood, few quantitative risk assessments for major environmental rulemakings have taken this interaction into account. Conceptual issues, methodologic issues, and data limitations have contributed to this lack, but all of these are changing. Recent epidemiologic studies have provided the basis for differential responses to heat, air pollution, and lead. In addition, methodologic and data improvements are allowing examination of cumulative risk, critical windows for exposure, and transgenerational effects. As a result, it is becoming possible to examine not just the overall burden of environmental exposures, but the distribution of risk. This is illustrated with some examples from air pollution and climate change, as well as a simulation study.

Background reading:
Schwartz, J. Bellinger, D. Glass, T. 2011. Exploring Potential Sources of Differential Vulnerability and Susceptibility in Risk From Environmental Hazards to Expand the Scope of Risk Assessment. Am J Public Health. 101 Suppl 1: S94-101. Doi: 10.2105/AJPH.2011.300272
Available at: http://ajph.aphapublications.org/doi/abs/10.2105/AJPH.2011.300272?url_ver=Z39.88-2003&rfr_id=ori:rid:crossref.org&rfr_dat=cr_pub%3dpubmed

Title: Gender-Dependent Effects of Maternal Lead (Pb) Exposure, Prenatal Stress and the Combination on Impulsivity Assessed Using a Delay of Reward Paradigm (Deborah Cory-Slechta, Ph.D., Professor of Environmental Medicine and Pediatrics, University of Rochester Medical School)

Elevated Pb exposure and prenatal stress are co-occurring risk factors and have common adverse effects that include attention deficits. This may reflect the fact that both target the mesocorticolimbic system of the brain, critical to executive function, and the hypothalamic-pituitary adrenal (HPA) axis, the physiological system that mediates the body's stress response. Correspondingly, it might be predicted that when combined, Pb and prenatal stress would have enhanced effects relative to either alone. We examined the effects of Pb, prenatal stress and the combination of these factors on impulsivity, a component of attention deficit, in a rat model using a delay of reward (i.e., self-control) paradigm that provides a choice in each trial of the behavioral test session between a short delay followed by a small reward or a long delay followed by a large reward. The long delay value was increased over behavioral test sessions. Effects of Pb, prenatal stress and the combination were more pronounced in males, who exhibited a slower behavioral shift from the long delay to the short delay choice as long delay values increased, and, particularly Pb and prenatal stress-treated males, omitted more trials and responded prematurely more frequently. Females, particularly Pb and prenatal stress-treated, showed an almost exclusive long delay lever preference in initial training, but no differences in preference from controls as the long delay value increased. Pb and prenatal stress treated females, however, showed significant increases in inter-trial interval responses that were without functional value, as long delay value increased. These findings suggest difficulties, particularly in response to combined Pb and prenatal stress, in behavioral transition as reward value changes.

Background reading:
Cory-Slechta, DA. Virgolini, MB. Rossi-George, A. Thiruchelvam, M. Lisek, R. Weston, D. 2008. Lifetime Consequences of Combined Material Lead and Stress. Basic Clin. Pharmacol. Toxicol. 102(2): 218-227. Doi: 10.1111/j.1742-7843.2007.00189.x
Available at: http://www.ncbi.nlm.nih.gov/pubmed/18226077

Cory-Slechta, DA. Stern, S. Weston, D. Allen, JL. Liu, S. 2010. Enhanced Learning Deficits in Female Rats Following Lifetime Pb Exposure Combined with Prenatal Stress. Toxicol. Sci. 117(2): 427-438. Doi:10.1093/toxsci/kfq221
Available at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2940413/

Theme III: Measuring Chronic Psychosocial Stress and Social Context in Research

Title: The Allostatic Load Model: Indicators, Indices and Methods to Measure the Antecedents and Consequences of Chronic Stress Throughout the Life Cycle (Robert-Paul Juster, Center for Studies on Human Stress, McGill University)

The allostatic load model represents an interdisciplinary approach to comprehensively conceptualize and quantify chronic stress in relation to pathologies throughout the life cycle. Allostatic load represents the biological damage individuals experience when chronically stressed that is easily quantified using an index representing neuroendocrine, immune/inflammatory, cardiovascular, and metabolic system functioning. Our recent review of nearly 60 empirical studies suggests that allostatic load indices incorporating sub-clinical ranges for numerous biomarkers predict clinical outcomes better than traditional biomedical methods that address only clinical thresholds for single biomarkers. Importantly, allostatic load algorithmic inclusion of neuroendocrine and/or immune/inflammatory biomarkers is stronger than metabolic syndrome parameters or systemic clusters. In reviewing the literature, it was consistently found that increased allostatic load corresponds either cross-sectionally or longitudinally to a plethora of antecedents (e.g., socioeconomic disadvantage, poor social networks, workplace stress, maladaptive personality traits, genetic polymorphisms, etc.) with dire consequences (e.g., mortality, cardiovascular disease, psychiatric symptoms, cognitive decline, physical/mobility limitations, neurological atrophy, etc.). In addition to the cumulative physiological toll consistently found with increasing age in populations worldwide, the risk and protective factors of allostatic load differ according to sex at a given developmental period as well as a function of gender-specific factors that include social support, work/home balance, hostility, caregiving, and spirituality. Targeting these factors during key periods of development is therefore essential to improving public health. The allostatic load model offers a useful framework to foster refined prevention, detection, and treatment strategies vis-à-vis physical health and psychological well-being throughout the life cycle.

Background reading:
Juster, RP. McEwen, BS. Lupien, SJ. 2010. Allostatic Load Biomarkers of Chronic Stress and Impact on Health and Cognition. Neurosci. Biobehav. Rev. 35(1): 2-16. Doi:10.1016/j.neubiorev.2009.10.002
Available at: http://www.sciencedirect.com/science/article/pii/S0149763409001481

Title: Measuring the Neighborhood Context for Research Addressing Maternal and Child Health Disparities (Lynne Messer, Ph.D., M.P.H., Assistant Research Professor, Center for Health Policy, Duke Global Health Institute)

Background: The built environment, a key component of environmental health, may be an important contributor to health disparities, particularly for reproductive health outcomes.
Here I will report on research that investigates the relationship between seven indices of the quality of the residential built environment and adverse reproductive outcomes for Durham, NC.

Methods: We surveyed approximately 17,000 residential tax parcels in central Durham assessing over 50 individual variables on each. These directly-observed data were combined with tax assessor, public safety, and US Census data to construct seven indices (housing damage, property disorder, security measures, tenure, vacancy, crime count and nuisance count) representing important domains of the residential built environment. Fixed slope random intercept multilevel models estimated the association between the residential built environment and five adverse birth outcomes. Models were adjusted for maternal characteristics and clustered at the primary adjacency community unit.

Results: Five built environment indices (housing damage, property disorder, tenure status, vacancy status, and nuisances) were associated with each of our five outcomes (pulmonary tuberculosis, small- for-gestational-age [SGA], low birthweight, continuous birthweight and birthweight-percent for gestational age) in the unadjusted context, but some estimates were attenuated following adjustment. In models adjusted for individual-level covariates, housing damage remained statistically significantly associated with SGA, birthweight and birthweight-percent for gestational age.

Conclusion: This work suggests a real and meaningful relationship between the quality of the residential built environment and birth outcomes, which we argue are a good measure of general community health. It also suggests that measuring the built environmental context is feasible and yields important and unique information for use in health disparities work.

Background reading:
Miranda, ML. Messer, LC. Kroeger, GL. 2012. Associations between the Quality of the Residential Built Environment and Pregnancy Outcomes among Women in North Carolina. Environ. Health Perspect. 120(3): 471-477. Doi: 10.1289/ehp.1103578
Available at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3295337

Messer, LC. Vinikoor-Imler, LC. Laraia, BA. 2012. Conceptualizing Neighborhood Space: Consistency and variation of associations for neighborhood factors and pregnancy health across multiple neighborhood units. Health Place. In press. Doi: 10.1016/j.healthplace.2012.03.012
Available at: http://www.sciencedirect.com/science/article/pii/S1353829212000603?v=s5

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Title: Disentangling Spatial Patterns in Social and Environmental Exposures for Epidemiology (Jane Clougherty, Sc.D., Assistant Professor and Director of Exposure Science, Graduate School of Public Health, University of Pittsburgh)

Growing epidemiological and toxicological evidence suggests that chronic stress may modify individual susceptibility to environmental pollutants – possibly mediated through allostatic load pathways including immune, endocrine, and metabolic alterations. A persistent problem in the epidemiology of combined effects, however, is persistent spatial confounding between social and physical environmental exposures. A further complication is that publicly-available data on chronic community stressors is aggregated to different administrative data (e.g., crime rates by police precinct, poverty rates by census tract).

This presentation will explore GIS-based methods for validating the selection of community stressors and neighborhood scales, methods for re-appropriating stressor measures across administrative units, and for quantifying spatial autocorrelation among multiple social and pollution exposures (which indicates the potential for spatial confounding and effect modification). We will employ case examples and preliminary data from our New York City-focused EPA STAR grant.

Background reading:

Clougherty, JE. Kubzansky, LD. 2009. A Framework for Examining Social Stress and Susceptibility to Air Pollution in Respiratory Health. Environ Health Perspect 117(9): 1351-1358.
Doi:10.1289/ehp.0900612
Available at: http://ehp03.niehs.nih.gov/article/info:doi/10.1289/ehp.0900612

EPA Workshop on Interactions between Social Stressors and Environmental Hazards
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Speaker Biosketches

Staci Bilbo, Ph.D.
Assistant Professor, Duke Institute for Brain Sciences, Duke University

Dr. Staci Bilbo received a bachelor's degree from the University of Texas at Austin in 1998, her Ph.D. in Psychology and Brain Sciences from Johns Hopkins University in 2003, and continued her training with a postdoctoral fellowship in the Center for Neuroscience at the University of Colorado at Boulder. She is now an Assistant Professor of Psychology and Neuroscience at Duke University. Current research in her laboratory focuses on understanding the consequences of early life events, including infection, stress, environmental toxins, and maternal obesity on neural and immune system development, using rodent models.

Jane Clougherty, Sc.D.
Assistant Professor and Director of Exposure Science, Graduate School of Public Health, University of Pittsburgh

Jane Clougherty is an Assistant Professor and Director of Exposure Science at the University of Pittsburgh Graduate School of Public Health, Department of Environmental and Occupational Health. Her work focuses on differential susceptibility to air pollution by chronic stress, and she has developed several of the early epidemiological and toxicological models for exploring these synergistic effects. Dr. Clougherty completed her doctorate in Environmental Health at the Harvard School of Public Health in 2006. From 2008-2010, she managed the New York City Community Air Survey (NYCCAS), one of the largest GIS-based studies to date on intra-urban variability in multiple pollutants. She is currently leading an effort under an EPA STAR grant to extend the NYCCAS work to explore spatial patterning in chronic stress across NYC, and synergistic effects on childhood asthma hospitalizations.

Deborah Cory-Slechta, Ph.D.
Professor of Environmental Medicine and Pediatrics, University of Rochester Medical School

Deborah Cory-Slechta is a Professor of Environmental Medicine and Pediatrics at the University of Rochester Medical School. She previously served as Chair of its Department of Environmental Medicine and Director of the NIEHS Environmental Health Sciences Center, and as Dean for Research. She also was the Director of the Environmental and Occupational Health Sciences Institute (EOHSI) and Chair of the Department of Environmental and Community Medicine at the UMDNJ-Robert Wood Johnson Medical School. Dr. Cory-Slechta has served on multiple national review and advisory panels, including the National Institutes of Health, the National Institute of Environmental Health Sciences, the Food and Drug Administration, the National Center for Toxicological Research, the Environmental Protection

Agency, the National Academy of Sciences, the Institute of Medicine, and the Agency for Toxic Substances and Disease Registry. She currently serves on the Advisory Committee for Childhood Lead Poisoning Prevention of the CDC. Her research has focused largely on the relationships between brain neurotransmitter systems and behavior, and how such relationships are altered by exposures to environmental toxicants, particularly the role played by environmental neurotoxicant exposures in developmental disabilities and neurodegenerative diseases.

Neal Fann
Office of Air and Radiation, U.S. Environmental Protection Agency

Neal Fann serves in the Office of Air and Radiation of the U.S. Environmental Protection Agency, where he has developed extensive experience in quantifying and characterizing the human health impacts, and monetized benefits, of changes in criteria and toxic air pollution. In this role, he has performed technically complicated and policy-relevant benefits assessments in support of major EPA regulatory actions including the Cross-State Air Pollution Rule and the Mercury and Air Toxics Rule, among many others.

Mr. Fann manages the PC-based environmental Benefits Mapping and Analysis Program (BenMAP) software, a model relied upon by EPA, stakeholder groups, researchers and international analysts to quantify the impacts and monetized benefits of air quality improvements. To ensure that this model remains the state-of-the-science and is properly specified with the best available data, he continually and critically evaluates the newest air pollution epidemiology and economics literature.

Mr. Fann also regularly contributes to the academic literature, publishing air pollution, risk assessment, and environmental justice assessments in journals including Risk Analysis and Environmental Science & Technology. Most recently he wrote a commentary in Environmental Health Perspectives, articulating the benefits of tighter integration between air pollution epidemiology and risk assessment. His other recent publications include: a national assessment of the public health burden of recent levels of PM2.5 and ozone in the U.S. and a proof of concept approach for maximizing the public health benefits of air quality improvements while achieving a more equitable distribution of risk. Prior to joining EPA, Mr. Fann received a Master of Public Policy at the Sanford School of Public Policy at Duke. He holds a bachelor degree in Economics and Political Science from Trinity University.

Robert-Paul Juster
Center for Studies on Human Stress, McGill University

Robert-Paul Juster is currently a researcher and doctoral candidate in the Integrated Program in Neuroscience at McGill University and is affiliated with the Centre for Studies on Human Stress. His work applies the allostatic load model of chronic stress to detect health risks in vulnerable populations.

Lynne C. Messer, Ph.D.
M.P.H., Assistant Research Professor, Center for Health Policy, Duke Global Health Institute

Lynne C. Messer is a socio-environmental and reproductive / perinatal epidemiologist whose work focuses on the social and structural determinants of maternal and child health disparities. Her methodological work includes better-defining neighborhood environments, developing environmental exposure measures for infant mortality outcomes and addressing the limits of causal inference in observational studies. She is also interested in the psychosocial mechanisms through which socio-environmental exposures result in health disparities for women and children.

Madeleine Kangsen Scammell, D.Sc.
Boston University School of Public Health, Boston, MA

Madeleine Kangsen Scammell is an Assistant Professor of Environmental Health and a Core Director of the Partners in Health and Housing Prevention Research Center (CDC) and the BU Superfund Research Program (NIEHS). Her research is mostly community-driven and community-based and includes mixed-methods analysis that incorporate qualitative and quantitative data obtained using social science and epidemiological research methods. She applies these methods in mapping and monitoring community-identified environmental health hazards and analyzing cumulative exposures to chemical and non-chemical stressors. She is the PI of an EPA STAR grant to develop new analytic techniques for examining the cumulative risks of exposure to social and chemical stressors.

Joel Schwartz, Ph.D.
Professor of Environmental Epidemiology, Harvard School of Public Health

Dr. Joel Schwartz is a Professor of Environmental Epidemiology at the Departments of Environmental Health and Epidemiology, Harvard School of Public Health. He has extensive experience in air pollution. He examined both acute and chronic effects of air pollution exposure. His recent research has established that exposure to fine combustion particles in the air at concentrations well below current standards are associated with a range of adverse health effects from increased respiratory symptoms, to increased hospital admissions, to increased deaths. This work has led to a tightening of the U.S. air quality standards. In addition, he has also done considerable work on health effects of ozone exposure. He has several international collaborations underway in this area. Recent work has been focused on the cardiovascular effects of air pollution, and on factors that modify the response to air pollution. Recent work has suggested diabetics are more susceptible to such effects.

Peggy M. Shepard
Executive Director, Co-Founder, WE ACT For Environmental Justice

Peggy Shepard is co-founder and executive director of WE ACT For Environmental Justice (WE ACT), based in West Harlem, which has a 22-year history of engaging Northern Manhattan residents in community-based planning and campaigns to affect environmental protection and environmental health policy locally and nationally. WE ACT has successfully combined grassroots organizing, environmental advocacy and environmental health research to become a national leader in advancing the perspective of environmental justice in urban communities. A former journalist, Ms. Shepard has served as chair of the EPA's National Environmental Justice Advisory Council (NEJAC). She received the Jane Jacobs Medal from the Rockefeller Foundation in 2008 for Lifetime Achievement, the 10th Annual Heinz Award For the Environment, and an Honorary Doctorate from Smith College in 2010.

Ms. Shepard served as chair of the National Environmental Justice Advisory Council (NEJAC) to the U.S. Environmental Protection Agency, and currently serves as co-chair of its Research and Science workgroup. She is a member of the Regional Health Equity Council, Region 2, Office of Minority Health, HHS; the Environmental Justice Advisory Committee to the NYS Department of Environmental Conservation and the NYC Mayor's Sustainability Advisory Board. Ms. Shepard is a former member of the National Advisory Environmental Health Sciences Council of the National Institutes of Health and the National Children's Study Federal Advisory Committee, NIH. She has served as guest editor of an Environmental Health Perspectives monograph, Advancing Environmental Justice Through Community-Based Participatory Research, April 2002, and is co-author of Promoting Environmental Health Policy Through Community Based Participatory Research: A Case Study from Harlem, New York, Jan. 2006, Journal of Urban Health. A lecturer on issues of environmental justice and community-based health research, she graduated Howard University and Solebury and Newtown Friends Schools.

Rosalind Wright, M.D.
Associate Professor of Medicine, Channing Laboratory, Harvard Medical School and Associate Professor of Environmental Health, Harvard School of Public Health

Dr. Rosalind Wright is internationally recognized for work examining independent effects of stress as well as interactions between stress and other environmental toxins (e.g., air pollution, aeroallergens, diet) on asthma in urban, minority populations. Specifically, her research program focuses on the epidemiology of chronic respiratory disease in both adults (adult-onset asthma, COPD) and children (childhood asthma). Utilizing multiple longitudinal cohort designs, Dr. Wright is investigating the role of varied psychosocial stressors (e.g., adverse life events, violence, social deprivation) on asthma morbidity. Whereas earlier psychosomatic models have supported a role for psychological stress in contributing to variable asthma morbidity among those with existing disease, a growing appreciation of the interactions between behavioral, neural, endocrine, and immune processes suggest a role for these psychosocial factors in the genesis of asthma as well.

EPA Workshop on Interactions between Social Stressors and Environmental Hazards
May 14-15, 2012

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