Final Progress Report

Principal Investigator: Roy J. Rando, ScD, CIH
Professor and Vice Chair
Department of Global Environmental Health Science
Tulane School of Public Health & Tropical Medicine
1440 Canal St., Suite 2100
New Orleans, LA 70112-2704
(504) 988 3870
rando@tulane.edu

Institution: Tulane University Health Science Center
1430 Tulane Ave.
New Orleans, LA 70112

Co-Investigators: Henry W. Glindmeyer, DEng, Professor of Medicine (deceased, 2009)
John J. Lefante, PhD, Professor of Biostatistics and Bioinformatics
Robert N. Jones, MD, Emeritus Professor of Medicine

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List of Terms and Abbreviations

ANOVA:	analysis of variance
CI:	confidence interval
COPD:	chronic obstructive pulmonary disease
FEV1:	forced expiratory volume – 1 second
FVC:	forced vital capacity
FEF25-75:	forced expiratory flow between 25% and 75% of VC
GM:	geometric mean
GSD:	geometric standard deviation
HP:	hypersensitivity pneumonitis
HVAC:	heating, ventilation, air conditioning
LLN:	lower limit of normal
MVUE:	minimum variance unbiased estimator of the mean
NIOSH:	National Institute for Occupational Safety and Health
ODTS:	organic dust toxic syndrome
OSHA:	Occupational Safety and Health Administration
PEL:	permissible exposure limit
PM:	particulate matter
PNOR, PNOC:	particulates not otherwise regulated, classified
POR:	prevalence odds ratio
PRR:	prevalence rate ratio
SD:	standard deviation
SE:	standard error
TLV:	threshold limit value
TP:	toxic pneumonitis
TPM:	thoracic particulate matter

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Abstract

Hurricane Katrina devastated New Orleans in August 2005 with high winds and a tidal surge that flooded about 80% of the city for several weeks. In the aftermath of the hurricane flood event, restoration workers risked respiratory illness from inhaling airborne dust, mold, and bacteria. In order to investigate and quantitate this risk, we performed a 5-year epidemiologic study of a group of 898 workers from the New Orleans area, who mostly worked in the building, construction, and maintenance trades or custodial services, and most of whom had performed various restoration work activities, such as demolition, trash and debris management, sewer/waterline repair, landscape restoration, and mold remediation. We assessed exposure to dust, bacterial endotoxin, and microbial glucan for the study subjects for the period 2005 to 2012. For 2005 and 2006, we obtained and analyzed an OSHA database of dust measurements made during restoration work in Southeast Louisiana. We then collected additional samples for airborne dust between 2007 and 2012, and assayed some of these samples for bacterial endotoxin and microbial glucan. Average total and respirable dust exposures in 2005 were as high as 5.2 and 2.8 mg/m^3 respectively, and frequently exceeded the OSHA Permissible Exposure Limits (15 and 5 mg/m^3, respectively). Thereafter, measured dust exposures decreased by about an

order of magnitude within the first year after Katrina and then more gradually declined through 2012. Average exposures to endotoxin and glucan in 2005 were imputed to be as high as 256 EU/m^3 and 118 µg/m^3, respectively, and likewise decreased dramatically after 2005. Between 2007 and 2012, we administered annual lung function tests and respiratory health and occupational questionnaires to the study subjects. The associations between restoration work, exposure, and lung function and respiratory symptoms were examined by statistical techniques. Significant respiratory symptoms reported by the study cohort included episodes of transient fever with cough (29%), sinusitis (48%), pneumonia (3.7%), dyspnea (34%) and new-onset asthma (4.5%). Prevalence rates for post-Katrina sinus symptoms, transient fever with cough, dyspnea, and new onset asthma were significantly elevated for those who did restoration work compared to those who did not and the prevalence rates increased with increasing restoration work dust exposure dose. Lung function in the study population was slightly depressed overall but was not significantly different between those with and without restoration work exposure. While we did not find any exposure-related excess declines in lung function, there was some evidence of a healthy worker effect that could have obscured lung function changes in the exposed group. Overall, our results demonstrate that post-Katrina restoration work was associated with certain adverse effects on respiratory health, especially sinusitis, toxic pneumonitis, and asthma. These results support the need for continuing surveillance of the respiratory health of workers engaged in restoration work activities in the aftermath of major flood events. Management of flood restoration workers' exposures to dust and microbial contaminants, through engineering controls, such as wet suppression of dust emissions, and the proper use of respiratory protective equipment, is recommended to mitigate the risk of acquired respiratory disease.

NIOSH Grant No. 5R01OH008938
Respiratory Effects in Workers from Post-Katrina Related Airborne Exposures

Final Progress Report – Section 1

Significant Findings

- 73% of the study population of 898 workers reported performing some restoration work activity in the aftermath of Hurricane Katrina. Restoration work activities included demolition, trash and debris management, sewer and waterline repair, landscape restoration, and mold remediation. Demolition and trash and debris management accounted for the majority of the time spent in restoration work activity by the study population.
- Many restoration workers reported working extraordinarily long hours in the first year after Hurricane Katrina, with 16-hour work days and 7-day work weeks being common.
- Dust and bacterial endotoxin levels during restoration work in 2005 frequently exceeded recommended exposure guidelines, especially for demolition and trash and debris management, but rapidly decreased by more than an order of magnitude thereafter. Microbial glucan exposures were likewise elevated in 2005 and then decreased significantly.
- Important respiratory symptoms and conditions observed in the study population included: sinusitis (37%), dyspnea (34%), COPD (6.4%), asthma (14%), new-onset asthma post-Katrina (4.3%), pneumonia post-Katrina (4.1%), fever with cough (28%), abnormal lung function – flow (10%), and abnormal lung function – volume (11%).
- Sinusitis, new-onset asthma post-Katrina, dyspnea, and fever with cough were statistically more prevalent among those who did restoration work compared to those who did not. The prevalence of each of these respiratory conditions increased and statistically correlated with both the number of hours of restoration work performed and the cumulative dust exposure dose during the restoration work.
- Lung function was lower than expected in the study population overall but was not statistically related to any exposure measures. Change in lung function over time did not correlate with current or cumulative exposures but there was evidence of a possible healthy worker effect that might have made any such correlation difficult to detect.

Translation of Findings

The results of this study provide further evidence that exposure to microbial agents in dust and debris in the aftermath of flood events puts workers at risk of developing various respiratory illnesses. Inhalation exposures were found to be highly elevated during restoration work in the immediate time period after the hurricane flood event but decreased in magnitude rapidly thereafter. Adverse respiratory effects were found to be positively correlated with the elevated exposures. In order to protect the respiratory health of flood restoration workers, effective exposure control techniques are necessary such as applying dust suppression technology, limiting workers' duration of work/exposure, and implementing appropriate personal respiratory protective equipment programs, particularly in the early response period after a flood event. The results of this study also point to the need to develop comprehensive quantitative exposure and health surveillance guidelines for workers exposed to flood-contaminated dust and debris and the microbial agents, endotoxin and glucan, that are based on sound epidemiologic research and toxicity data.

Outcomes/Impact

Since the destruction of the World Trade Center on September 11, 2001, there has been an increasing focus on exposure assessment and protection of response and restoration workers' health during man-made and natural disasters. Restoration work in the aftermath of flood events in particular has garnered enhanced interest because of their relative frequency of occurrence, the generally widespread destruction common in such events, their impact on general environmental health of both the communities involved and the workforce, and continuing concerns over the adverse effects of exposure to microbial agents associated with water intrusion and dampness. Hurricane Katrina and the associated flooding of New Orleans can be considered a benchmark for such events in light of the massive amount of destruction and the enormous restoration and remediation effort that has taken place in its aftermath. There is a need to develop comprehensive quantitative exposure guidelines for flood restoration work. The results of this study provide unique quantitative exposure response information for flood restoration work that will be essential to regulators and others in developing effective exposure guidelines and standards.

Final Progress Report – Section 2

Scientific Report

BACKGROUND

In August of 2005, Hurricane Katrina devastated the New Orleans area with high wind, heavy rainfall, and a storm surge of about 7 m which caused the collapse of the levee system surrounding the city. Approximately 80% of the city was flooded to varying depths for many weeks before the U.S. Army Corps of Engineers was able to implement temporary levee repairs and install emergency pumping capacity.

In the aftermath of the flood event, the infrastructures of the city along with residences and commercial buildings were grossly contaminated with sediments deposited by the floodwaters and subsequently by microbial overgrowth supported by the residual moisture, high humidity, and elevated temperatures in the area. After floodwaters had receded, various surveys were conducted for measurement of indicators of microbial contamination in air, dust, and damaged building materials, including total and culturable mold spores, fungal fragments, mycotoxins, 1→3-β-D-glucan, and bacterial endotoxin. Generally, observed levels of microbial contaminants in these surveys were elevated, often extremely so, were relatable to the depth and duration of flooding, and indoor levels were typically higher than those in the surrounding outdoor environment.

Subsequent to the post-hurricane flooding event, there has been extensive rebuilding in the New Orleans area. Residents who personally performed repairs of their property, as well as various skilled and unskilled laborers working in the construction and building maintenance trades were at risk for inhalation exposures to dust containing microbial and other agents during demolition, removal, and repair of flood-damaged and contaminated infrastructure and building materials. Exposures to microbial contaminants in agriculture, waste management, and in water-damaged and moldy buildings have been linked to various upper and lower respiratory illnesses and adverse effects including rhinitis, hayfever, toxic pneumonitis (TP), hypersensitivity pneumonitis (HP), respiratory infections including pneumonia, and exacerbation or initiation of asthma. The potential for respiratory illness arising from inhalation exposure to bioaerosols and

microbial contaminants during restoration activities in the post-Hurricane Katrina environment was of particular concern. The overall objective of this project was to investigate the risk of respiratory illness associated with inhalant exposures while working in and around flood-damaged structures in post-Hurricane Katrina New Orleans. This was accomplished through a cross-sectional and 5-year longitudinal study of a cohort of workers from the New Orleans area, most of whom were involved in restoration activities.

SPECIFIC AIMS

- Year 1

1. Utilize federal, state and local governmental agencies and private agencies and employers to identify and recruit approximately 1,000 workers representing the specific types of occupations to be studied. Administer standardized spirometric tests to recruited workers, and compute baseline percent of predicted values for FEV_1, FVC, FEV_1/FVC and FEF_{25-75}.
2. Administer the respiratory, smoking and occupational (historical) questionnaires to the recruited workers.
3. Administer Post-Katrina specific questionnaires to document associated job tasks and durations, use of personal protective equipment, work practices, and other pertinent exposure-related information.
4. Conduct initial personal monitoring of the 1,000 post-Katrina/flooding remediation workers to be used as exemplars in the initial development of job and task specific exposures to thoracic (tracheobronchial and respirable) particle and microbial agents (endotoxin and β-glucan).
5. Begin to construct a job/task/time/exposure matrix using the field sampling results.

- Year 2-5

6. Investigate prevalence of COPD and asthma, incidence of new onset asthma and asthma-like symptoms, and symptoms possibly related to hypersensitivity pneumonia in years 2 through 5.
7. Continue to administer annual follow-up standardized spirometric tests and respiratory, smoking, occupational, and post-Katrina (associated job tasks and durations, use of personal protective equipment, work practices, and other pertinent exposure-related information) questionnaires to the study population, for a maximum 5 spirometric tests and questionnaires per subject over the study.
8. Continue to conduct personal monitoring of approximately 1,000 post-Katrina/flooding remediation workers per year to be used as exemplars in the development of job and task specific exposures to thoracic particulates matter and microbial agents (endotoxin and β-glucan), for a maximum of 5,000 samples, over the study; and continue to construct a job/task/time/exposure matrix using the field sampling results.
9. Assign cumulative and average exposures for thoracic particle and biomarkers for each worker in the study by coupling individual work history and the job/exposure matrix.
10. Identify individuals with an excessive decline in lung function and evaluate their decline together with their other respiratory health outcomes.
11. Investigate the development of new-onset asthma**,** respiratory symptoms, respiratory conditions, and accelerated annual decline in lung function associated with exposure to post-Katrina/flooding particulates and bio-aerosol exposures, while taking into account age, gender, race, absence or presence of historical toxic dust exposures, other contemporaneous confounding exposures, use of personal protective equipment, cigarette smoking habit and pack-years of smoking, and other possible confounders.

METHODOLGY

The final study cohort consisted of 898 adults residing or working in the greater New Orleans metropolitan area. Study participants were recruited from several sources: 1) employees of three large institutions in the City of New Orleans, two of which are academic and the third is

a branch of local government. All three institutions experienced heavy flood damage to their buildings and facilities and utilized their regular staff as well as contract labor to perform restoration work. Recruiting from the academic institutions focused primarily on workers from departments normally engaged in maintenance, custodial, and facilities services. Support personnel (clerical, managerial, etc.) from the targeted departments were included in the recruitment. 2) Members of a local union hall for the skilled and unskilled building trades . 3) Private building contractors and self-employed tradesmen. 4) Other residents of the New Orleans area, many of whom performed restoration work on their own properties.

Overall, about half of the study cohort reported a skilled or unskilled trade as their primary occupation, including carpentry, electrical, plumbing, paint/drywall, HVAC, groundskeeping, general construction, general maintenance, operating/building engineer, and mechanic/machinist. An additional 15% of study participants worked in custodial or janitorial services.

Testing was conducted in a mobile laboratory van outfitted with spirometry and interview work stations and ancillary equipment. The mobile laboratory van was moved to the work locations or union hall of the study participants for the duration of their respective testing period, generally 2 to 3 weeks each year, and to the parking lots of several large building supply stores, in order to allow private contractors and self-employed construction tradesmen to participate.

Spirometry testing procedures and equipment complied with the updated American Thoracic Society spirometric test criteria.[1] Spirograms were collected with a Sensormedics Model 1022 dry rolling seal spirometer interfaced to a laptop computer running OMI Spirometry software version 5.05.9 (Occupational Marketing, Inc., Houston, TX). All spirometric testing was conducted by the same research staff member who is a Certified Pulmonary Function Technician; in addition, all spirometric test results were quality assured and interpreted by senior study investigators (HWG and RNJ).

Predicted lung function parameters and lower limit of normal (LLN) lung function values for forced expiratory volume in one second (FEV1), forced vital capacity (FVC) and FEV1/FVC

[1] Glindmeyer HW, Jones RN, Barkman HW, Weill H. Spirometry: quantitative test criteria and test acceptability. *Am Rev Respir Dis.* 1987; 136(2):449-452.

ratio, were computed from predictive equations developed by Hankinson.[2] Separate predictive equations were used for Caucasians, African-Americans, and Latinos. Predicted values for study participants of Asian heritage were calculated using the equations for Caucasians. In addition to race, the predicted values were based on age, gender, and height. The LLN values were calculated by subtracting 1.645 SEE from the predicted values, where SEE is the standard error of the estimate and 1.645 is the 95th percentile of a standard normal distribution.

Those participants with chronic obstructive pulmonary disease (COPD) were identified according to the GOLD criteria, i.e., FEV1/FVC less than 70% and FEV1 % predicted less than 80% ;[3] however, only pre-bronchodilator lung function values were available, and thus may not have adequately differentiated asthma (with reversible obstruction) from COPD. Those reporting "ever asthma" on questionnaire were therefore excluded from the analyses of COPD prevalence as a function of exposure.

A demographic, medical, smoking and occupational questionnaire was administered to the study participants during the interview. The questionnaire accounted for a variety of risk factors and potential confounders for the development of airways disease including asthma, allergic disease, historical confounding exposures, serious childhood respiratory illness, cigarette smoking history, environmental tobacco smoke, and age, gender and race. Additional questions were designed to capture development of specific symptoms after Hurricane Katrina that might be associated with living and working in the post Katrina environment. These included post-Hurricane Katrina onset of asthma, sinus symptoms, pneumonia, and transient fever with cough absent infection, with the latter used as an indicator of possible hypersensitive (HP) or toxic (TP) reaction.

Asthma was defined dichotomously and required a positive response to both of the following questions: "Have you ever had asthma or attacks of shortness of breath with wheezing in the chest when not having a cold?" followed by "Do you still have asthma/ASOB?". The response to the question "How old were you when your asthma started?" was used in conjunction

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[2]Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. *Am J Respir Crit Care Med.* 1999; 159(1):179-187.

[3]Pauwels RA, Buist AS, Calverley PMA et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. NHLBI/WHO Global Initiative for Chronic Obstructive Lung Disease (GOLD) Workshop Summary. *Am J Respir Crit Care Med.* 2001; 163(5):1256-1276.

with the participant's date of birth to determine whether asthma onset was after September 30, 2005 (post-Katrina new onset asthma). Dyspnea was also defined dichotomously and required a positive response to the question "Do you have shortness of breath when hurrying on level ground?".

The interview also included queries on pre- and post-Katrina work and occupation, and detailed information was gathered on time spent post-Hurricane Katrina performing five specific types of hurricane/flood remediation work: demolition and rip-out, trash removal, landscape restoration, sewer line repair and mold remediation. Participation in any of these restoration work activities was assumed to result in occupational or vocational exposure to flood-related contaminants. Participants were asked to report the number of hours spent in each of the five restoration work activities, for each year since the hurricane up to the point of interview, and the type and relative frequency of any respiratory protective equipment that may have been used during the work. Restoration of personal property was included in the total time spent in restoration work along with any from the subject's regular employment.

Work/time/exposure matrices were constructed for the period of 2005 through 2012. For the years 2005 and 2006, exposure estimates for particulate matter were derived from data obtained from the public domain. These data were converted into the equivalent thoracic particle fraction as outlined below. For the years 2007 through 2012, project-specific thoracic particulate exposure monitoring was conducted on the cohort of workers participating in our longitudinal study.

Information on restoration workers' exposure to particulate matter during the year after Hurricane Katrina was derived from an Occupational Safety and Health Administration (OSHA) on-line database.[4] Shortly after Hurricanes Katrina and Rita in the fall of 2005, OSHA mobilized and began exposure monitoring of response and restoration workers in Louisiana, Mississippi, Alabama, and Florida. Exposure monitoring continued through the summer of 2006. Personal and area sampling for total and respirable particulate matter using both continuous direct-reading and integrated filter samples was performed. The database reports 1,085 measurements of total dust and 416 measurements of respirable dust. Most of the samples

[4] Occupational Safety and Health Administration: "2005 Gulf Coast Hurricane Response – Particulate Sampling Data". available at: http://www.osha.gov/katrina/lisareports/part_combined.html

were collected in Louisiana (n = 730) and Mississippi (n = 726) with far fewer in Florida (n = 25) and Alabama (n = 20).

Much of southeast Louisiana, and the metropolitan area of New Orleans in particular, is at or below sea level and protected by ring levee systems, so that the hurricane flood events were fundamentally different in Louisiana as compared to the other states. Because of this, only the Louisiana data were used in construction of the exposure matrix.

The OSHA data included information on the general location of the sampling, the type of work being done during sampling, and the sampled worker's job title or profession. This information was reviewed and, based on the professional judgment of the research team, samples were identified that represented one of the five following specific restoration work activities: demolition, trash & debris management, landscape restoration & tree-trimming, sewer/waterline repair, and mold remediation. Demolition included specific activities such as tear-out, interior gutting, removal of structural elements, and operation of heavy demolition equipment. Trash & debris management included collection, removal, haulage, debris reduction, and in some cases burning of demolition trash and debris. Landscape restoration included such tasks as landscape clearing, chainsaw and chipper operation, tree trimming, and brush removal. Mold remediation included removal of surface mold growth by wiping/scrubbing, power washing, stripping, and treatment with antimicrobials; however, no samples in the OSHA database could be identified as specifically fitting into the mold remediation restoration work activity.

Direct measurements of exposure to thoracic particulate matter in the 2005 – 2006 period were unavailable and had to be estimated from the available OSHA data on respirable and total particulate matter. For this purpose, estimation models relating thoracic to respirable and total particulate measures were developed from three data sets that employed side-by-side sampling for respirable, thoracic, and total dust: Model I from a previously published study of size fractionated fugitive dust levels around coal and limestone mine haulage roads,[5] Model II from a previously published investigation of size-fractionated sampler performance for industrial wood

[5] Organiscak, J.A., W.M. Randolph-Reed: Characteristics of fugitive dust generated from unpaved mine haulage roads. *Int. J. Surface Min. Reclam. Environ.* 18(4):236-252 (2004).

processing dust,[6] and Model III from a set of stationary site samples collected at a post-Katrina demolition site specifically for this study.

For the post-Katrina demolition site samples used in Model III, the respirable and thoracic fractions were collected with GK 2.69 cyclones (BGI Inc.) operated at a nominal flow of 4.2 L/min for the respirable fraction and 1.6 L/min for the thoracic fraction. The cyclones were fitted with 37-mm diameter, 0.8 µm pore size polycarbonate filters contained in polystyrene cassettes (SKC Inc.). The total dust fraction was collected at a nominal flow rate of 2 L/min onto the same type polycarbonate filters contained in 37-mm cassettes operated in the closed face mode. Filters were humidity conditioned for at least 48 hours over saturated potassium carbonate (RH ~ 52%) before and after sampling. Humidity conditioned filter samples were pre- and post-weighed on a Fisher analytical balance (± 10 µg) after electrostatic discharge with an alpha source. Sets of the three sampling devices were mounted side-by-side on a vertical 30-cm diameter round fiberboard cutout to simulate bluff-body (personal) sampling. Sampling was conducted at fixed site locations around and in close proximity to an operation in which an abandoned, severely flood-damaged school complex was being demolished manually and with heavy equipment. Dust emissions during the demolition were controlled with water spray.

In order to assess the similarity of the three models, a multivariable linear regression analysis was performed with all of the data from the three models combined, with thoracic PM as the dependent variable and respirable and total PM as the independent variables along with dummy variables for the models interacted with the respective total and respirable dust levels. In this analysis, the dummy variable/PM level interaction terms were all highly non-significant with p values ranging from 0.77 to 0.96, indicating that there were no statistical differences between the three models. The final form of each of the models was then derived from separate multivariable linear regressions through the origin with thoracic PM as the dependent variable and respirable and total PM as the independent variables. The regressions were performed using the data analysis pack within Microsoft Excel 2010. Separate models were created and used rather than a single overall model because the much larger amount of industrial wood processing data would have overweighted a combined model in comparison to the mine haulage and demolition data.

[6] Rando, R.J., H. Poovey, D. Mokadam, J. Brisolara, and H. Glindmeyer: Field performance of the RespiCon for size-selective sampling of industrial wood processing dust. *J Occup. Environ. Hyg.*, 2:219-226 (2005).

From 2007 to 2012, thoracic PM exposure monitoring was performed on the study cohort. Sampling for thoracic PM was conducted as described above for Model III data using GK 2.69 cyclones with polycarbonate filters. The majority of monitoring was conducted by personal sampling but some fixed area sampling was performed when personal sampling was not feasible. Monitoring was performed annually from 2007 to 2012 during 2 to 3 weeks at each of the primary worksites for the cohort members. For personal monitoring, participants wore the pump/cyclone/filter sampling train with the sampler placed in the breathing zone during the work day as they went about their normal work activities. Monitoring usually began shortly after the start of the work shift and concluded shortly before its end with target sample duration of approximately 6 hours. Also at the time of monitoring, information was collected from the participants on job title or profession and activities during the monitoring period including whether any of the previously described restoration work activities were performed: demolition and rip-out, trash and debris management, landscape restoration, sewer and water line repair, and mold remediation.

A subset of the collected thoracic PM samples that contained around 100 µg or more of dust was sent after gravimetric analysis to University of Iowa for assay of endotoxin and microbial glucan content of the dust. Filter samples were extracted in pyrogen-free water and three-fold serial dilutions were assayed for endotoxin using the kinetic chromogenic *Limulus* Amebocyte Lysate (LAL) assay as previously described.[7] (1→3, 1→6)-β-D-glucan was determined by sandwich enzyme-linked immunosorbent assay (ELISA) as previously described,[8] using custom mouse anti-glucan monoclonal antibody as the capture antibody and rabbit anti-scleroglucan polyclonal antibody as the second antibody. This assay provides specificity for microbial glucan in comparison to that derived from plants.

The exposure data were initially grouped by restoration activity and year for analysis. The data were examined for statistical differences across activities and years to determine appropriate groupings. In addition to being right skewed, most of the data groups contained some censored (below detection limit) measures. Statistical testing for differences across groups was therefore performed by non-parametric Mann Whitney Rank Sum test or Kruskal-Wallis

[7] Thorne, P.S.: Inhalation toxicology models of endotoxin- and bioaerosol-induced inflammation. *Toxicology* 152:13-23 (2000).
[8] Thorne, P.S., K. Kulhankova, M. Yin, R. Cohn, et al.: Endotoxin exposure is a risk factor for asthma in a U.S. national survey of housing. *Am. J. Resp. Crit. Care Med.* 172:1371-1377 (2005).

ANOVA with post-hoc multiple pair-wise comparisons by Dunn's Method, using SigmaPlot version 11.2 software (Systat Software, Inc.). Data across years for each activity group were combined when no statistical difference across years was observed with Dunn's multiple pair-wise comparisons method. For each final grouping of data, both the geometric mean and arithmetic mean and their respective standard deviations were determined. However the arithmetic mean was taken as the primary measure of exposure rather than the geometric mean, in accordance with recommendations for exposure – response studies.[9] For any group containing at least 5 measures, the mean was estimated from the minimum variance unbiased estimator (MVUE) as calculated from log-probit regression analysis performed with IHDataAnalyst version1.27 software (Exposure Assessment Solutions, Inc.); otherwise, the simple average of the data was calculated using Excel 2010.

The unadjusted prevalence rate ratios for each symptom or condition for those doing any restoration work vs. those not doing any restoration work, were calculated within smoking categories based on 2 X 2 contingency tables. The prevalence rate ratio was defined as $PRR = \frac{p_1}{p_2}$, where $p_1 = \frac{a}{n_1}$ and $p_2 = \frac{c}{n_2}$ represent the sample proportion of exposed (n_1) and unexposed (n_2) individuals with disease. If a and b represent the number of exposed subjects who do and do not have disease, respectively, and c and d represent the number of unexposed subjects who do and do not have disease, respectively, the asymptotic 95% confidence interval for prevalence rate ratio is calculated using the following standard logarithmic transformation: $\ln(PRR) \pm 1.96\sqrt{\frac{b}{an_1} + \frac{d}{cn_2}}$. Exponential transformations on the confidence limits of this log transformed interval provided the asymptotic 95% confidence intervals for prevalence rate ratio.

Multiple logistic regression analyses were used to compute adjusted prevalence odds ratios for each symptom or condition per 1,000 mg/m^3-hrs of cumulative exposure to thoracic particulate matter from restoration work, as well as to compute asymptotic 95% confidence intervals for prevalence odds ratios. Due to significant interactions between gender and restoration work, logistic regression analyses were adjusted for gender and run separately by

[9] Crump, K.S.: On summarizing group exposures in risk assessment: is an arithmetic or a geometric mean more appropriate? *Risk Anal.* 18(3):293-297 (1998).

smoking categories as well as in combination. All interactions between age, use of respiratory protective equipment (ever vs. never), and restoration work were initially considered, and were not significant.

Multi-level models for longitudinal data[10,11] were used to relate exposure measures to both level and annual change in spirometric measures of lung function including FEV1, FVC, FEF 25-75, and FEV1/FVC ratio. At the first level of modeling, lung function was regressed on time. At the second level of modeling, inter-individual differences in lung function level and annual change were regressed on exposure measures, demographic determinants, and other potential confounders. Both time-invariant (cumulative) and time-variant (contemporaneous) exposure measures were considered. SAS PROC MIXED[12] was used to fit these models.

RESULTS AND DISCUSSION

The majority of the study cohort was African American and male (Table 1). Hispanics accounted for 10% of the cohort. Current smokers comprised 27% of the cohort, whereas 18% were ex-smokers and 55% had never smoked. Upper and lower respiratory symptoms were prevalent in the cohort. Four percent of the study cohort reported having pneumonia after Hurricane Katrina and 37% reported newly developing sinus symptoms. Among those reporting never having had asthma prior to Hurricane Katrina (n = 784), 4.3% reported new onset asthma. Episodes of transient fever with cough occurring post-Hurricane Katrina were reported by about 28% of the study cohort. Multiple episodes of fever with cough were common in this reporting group.

Overall, lung function parameters were somewhat depressed in the cohort and correlated with cigarette smoking (Table 2). Average percent predicted FEV1 and FVC were statistically lower than 100% for both smokers and non-smokers at first measurement. Also the proportions of the cohort falling below LLN for FEV1 and FVC were somewhat elevated (5% being the

[10] Laird N, Ware JH. Random-effects models for longitudinal data. *Biometrics.* 1982;38:963–974.
[11] Singer JD, Willett JB. Applied longitudinal data analysis: Modeling change and event occurrence. New York, NY: Oxford University Press; 2003.
[12] Singer JD. Using SAS PROC MIXED to fit multilevel models, hierarchical models, and individual growth models. *Journal of Educational and Behavioral Statistics.* 1998;24:323–355.

expected proportion based on the definition of LLN), particularly for the current and ex-smokers (Table 1), as expected.

Almost 75% of the study participants reported having performed some restoration work activity post Hurricane Katrina. Demolition/rip out was the most commonly reported restoration work activity, followed by landscape restoration and trash/debris removal. Few study participants reported time spent in sewer line repair. Mold remediation was performed by about one-fifth of the study cohort. The distributions of time spent in these activities were highly skewed because many study subjects worked as much as 16 hours per day, seven days per week, for extended durations after Hurricane Katrina. The majority of the study subjects also reported time spent in more than one type of restoration work activity. Among those who reported performing restoration work, 80% reported some use of respiratory protective equipment (i.e., filtering facepiece or air-purifying cartridge respirator).

Out of 730 Louisiana samples from the OSHA exposure database, 663 were identified as representing one of the five specific restoration work activities. Of these, 482 were total dust measurements and 181 were respirable dust measurements. The majority of the samples (n = 631) were taken during demolition or trash & debris management activities. Sewer & waterline repair and landscape & tree trimming accounted for a total of 32 samples. No samples were identified that were clearly representative of the mold remediation activity. Other activities accounted for 67 samples and included occupations and actions such as office work, environmental inspections, hazardous materials response, and telecommunications repair.

Table 3 summarizes the results of the analysis of the OSHA data. For demolition, trash & debris management, and "all other activities", there were statistical differences between 2005 and 2006 and the data for each year were maintained as separate groupings; whereas for landscape & tree trimming and sewer & waterline repair, the differences between 2005 and 2006 were not statistically significant and the data for the two years were combined. None of the mean exposure values were above the applicable OSHA PELs, 15 mg/m^3 total dust and 5 mg/m^3 respirable dust for particles not otherwise classifiable (PNOC). Only one exposure mean, respirable PM for demolition in 2005, was above the action level. However exceedance fractions, modeled from the log-probit regression analysis, ranged from less than 0.1% to as high as 17.6%, the latter also for respirable PM for demolition in 2005.

A significant drop in levels of total and respirable dust from 2005 to 2006 for demolition, trash & debris management, and "all other activities" was observed and furthermore, the percentage of respirable dust in total dust decreased significantly as well. It is theorized that these observations resulted in part from the gradual introduction of water spray suppression of dust emissions for these activities. After Hurricane Katrina the municipal water supply system was completely disrupted and inoperable so that water spray dust suppression could not be used initially but became increasingly common as the water supply system was gradually returned to service.

Table 4 presents the results of multivariable regression modeling for estimation of thoracic PM from respirable and total PM. All three models showed good correlation of thoracic PM with the other PM measures in combination, with R^2 values ranging from 0.73 to 0.99. Respirable PM was the primary predictor of thoracic PM for all three models and the amount of total PM had much less influence on the final prediction of thoracic PM. Model I in particular showed a very strong correlation and predictive ability because the data used in that model are mean values for several sets of data. This is in contrast to Models II and III which are based on individual data and therefore show more variability with correspondingly lower correlation.

The respective mean values for respirable and total PM for each of the restoration work activity/year groupings from the OSHA data (Table 3) were input into each of the regression models of Table 4 to estimate the corresponding thoracic PM. The results of this process are presented in Table 5. The estimates of thoracic PM for each of the groupings were very similar across the three models and there were no statistical differences ($p = 0.12$) among the models by 2-way analysis of variance with activity/year and model as the treatment variables. As was observed for respirable and total PM, there is a decrease of about an order of magnitude in estimated thoracic PM from 2005 to 2006 for demolition and trash & debris management.

The estimated average exposure levels of thoracic PM in 2005 for demolition and trash & debris management, 5.8 and 4.2 mg/m^3, respectively, are in the range of exposure guidelines for nuisance dust or particles not otherwise specified or classified (PNOS or PNOC). Current OSHA permissible exposure limits (PEL) for PNOC are 5 mg/m^3 respirable fraction and 15 mg/m^3 total dust. While current ACGIH threshold limit values (TLV) for PNOS are based on respirable and inhalable fractions, the preceding TLV (1995) was 3 mg/m^3 respirable fraction and 10 mg/m^3 total dust. While there are no specific guidelines available for the thoracic

fraction of PNOS/PNOC, for the purposes of comparison, the PEL and TLV values for respirable and total PNOS/PNOC can be applied to the estimation models of Table 4 to generate an approximation of an equivalent guideline for the thoracic fraction. This results in values of 12 mg/m^3 from the PEL and 8 mg/m^3 from the TLV. Estimated average exposures to thoracic PM in 2005 for demolition and trash & debris management range from about 35% to 73% of these values. However, because the exposure data are log-normal, it is likely there were a significant number of individual exposure measures that exceeded these equivalent guidelines, as was observed for the total and respirable PM measures on which the thoracic PM estimates are based.

A total of 774 thoracic PM samples were collected from 2007 to 2012 in support of this study. Twelve percent of the samples were censored for gravimetric analysis based on an analytical limit of 10 µg dust per filter. Log-probit regression analysis of the data yielded an overall thoracic PM geometric mean of 0.13 mg/m^3 (GSD = 3.2) and MVUE of 0.27 mg/m^3 (SD = 0.45).

Table 6 shows that out of the 774 samples, a total of 267 were collected during the performance of any of the specific restoration work activities: demolition (n = 122), trash & debris management (n = 24), sewer & waterline repair (n = 49), landscape restoration & tree-trimming (n = 67), and mold remediation (n = 5). The remaining 507 samples were collected during various other activities including skilled and unskilled construction work, building maintenance and repair, custodial and janitorial services, general institutional labor, and clerical work.

Kruskal-Wallis ANOVA showed that there were statistically significant differences in thoracic PM across years for the demolition work activity ($p = 0.01$) and Dunn's method indicated that the data should be grouped by the years 2008, 2009, and 2010-2012 (Table 6); no data were collected in 2007 for the demolition activity. Thoracic PM exposures measured during the demolition activity decreased by about a factor of eleven from 2008 to 2011. For the other restoration work activities, there were no statistically significant differences across years in which the activity was sampled and the data were combined across all years for each of the activities. For the non-restoration work activity samples, the periods 2007 – 2010 and 2011 – 2012 were statistically different and decreased by almost a factor of three.

Table 7 reports the results of the assays of the collected thoracic PM samples for endotoxin and glucan content. Observed levels of the microbial agents in collected thoracic dust

were not significantly different across years for the various restoration work activities except for endotoxin in dust from demolition, for which the level in 2008 was about twice as high as for 2009 – 2012. Non-restoration work activities showed a similarly significant drop in endotoxin levels in thoracic PM, decreasing by about half from 2007-2008 to 2009-2011. Likewise, glucan content of thoracic PM decreased by about a factor of two from 2007-2008 to 2009-2010 and by more than an order of magnitude by 2011-2012.

In light of the extensive microbial contamination in the post-Katrina environment, the levels of endotoxin and microbial glucan in the thoracic fraction of dust and the resulting exposures may be of more concern than the exposures to thoracic PM. Unfortunately, the OSHA surveys in 2005 and 2006 did not include any measurements for the microbial agents. Our own data from 2007 to 2012 generally show gradual decreases in the content of endotoxin and microbial glucan in the thoracic dust samples over time. This is expected as the degree of microbial contamination would have lessened after the initial flood event and as more of the environment was remediated and contaminated materials were removed. The observed levels of endotoxin and microbial glucan in the dust for the early years of our own surveys (2007, 2008) therefore likely represent a conservative estimate of what the levels would have been in 2005 and 2006. Our data show average endotoxin levels in thoracic dust of 41.2 EU/mg for demolition and 61.0 EU/mg for trash & debris management. Integrating these values with estimated thoracic PM exposures for 2005 and 2006 for the demolition and trash management activities suggests minimum average endotoxin inhalation exposure levels in the range of 239 to 256 EU/m^3 for 2005 and 18 to 39 EU/m^3 for 2006. Likewise for microbial glucan, our observed average levels of 20.3 µg/mg and 18.7 µg/mg in the thoracic dusts from demolition and trash management, respectively, suggest minimum average microbial glucan inhalation exposure levels in the range of 79 to 118 $\mu g/m^3$ for 2005 and 9 to 12 $\mu g/m^3$ for 2006. In a similar fashion for landscape restoration and sewer & waterline repair, estimated minimum average endotoxin and microbial glucan exposure levels for 2005 and 2006 would be 28 EU/m^3 and 0.8 $\mu g/m^3$, and 5 EU/m^3 and 3 $\mu g/m^3$, respectively.

The 2005 values for demolition and trash & debris management in particular are much higher than those seen during residence remediation activities following the 2008 Cedar River flood in Iowa in which endotoxin levels ranging from 0.18 to 15 EU/m^3 and total glucan levels

ranging up to about 0.4 μg/m^3 were reported.[13] In contrast, a survey of the air quality inside 20 flood damaged residences in New Orleans from October 2005 yielded geometric means (geometric standard deviation) of 23.3 EU/m^3 (5.6) for endotoxin and 1.6 μg/m^3 (4.4) for microbial glucan.[14,15] These values equate to estimated means of 90 EU/m^3 and 4.5 μg/m^3, respectively. While these levels are also much higher than the Cedar River flood remediation data, they represent background indoor air levels of the microbial agents for flood-damaged residences, many of which were still unoccupied during the sampling, and so are not truly representative of airborne levels associated with restoration work. A better comparison with our data is provided from a smaller study of three flood-damaged homes in post Katrina New Orleans that were being actively remediated during the sampling.[16] Measured endotoxin levels in the homes during remediation work ranged from about 150 to 1500 EU/m^3 with an average of about 680 EU/m^3. Background levels in these same homes when remediation work was not being performed averaged 60 EU/m^3, much lower and in line with the results of the 20-home survey.

An occupational exposure limit for endotoxin of 90 EU/m^3 has been suggested based on adverse respiratory effects including irritation and inflammation.[17] Our analysis suggests that average exposure levels during demolition and trash & debris management restoration work activities in 2005 were well above this limit and restoration workers were therefore at risk for adverse respiratory effects. The contamination of airborne dusts with endotoxin and microbial glucan during early restoration work would have contributed to their irritancy and likely played a role in the etiology of Katrina Cough.

The contemporaneous exposure data (2007 – 2012) were also grouped according to job and analyzed statistically for differences across years. All jobs that were sampled were collapsed into 21 job groups. As was done with the exposure data by restoration work activity, job

[13] Hoppe, K.A., N. Metwali, S.S. Perry, T. Hart, et al.: Assessment of airborne exposures and health in flooded homes undergoing renovation. *Indoor Air* 22:446–456 (2012).

[14] Rao, C.Y., M.A. Riggs, G.L. Chew, et al.: Characterization of molds, endotoxins, and glucans in homes in New Orleans after Hurricanes Katrina and Rita. *Appl. Environ. Microbiol.* 73(5):1630-1634 (2007).

[15] Riggs, M.A., C.Y Rao, C.M. Brown, et al.: Resident cleanup activities, characteristics of flood-damaged homes, and airborne microbial concentrations in New Orleans, Louisiana, October 2005. *Environ. Res.*106(3):401-409 (2008).

[16] Chew, G.L., J. Wilson, F. Rabito, et al.: Mold and endotoxin levels in the aftermath of hurricane Katrina: a pilot project of homes in New Orleans undergoing renovation. *Environ. Health Perspect.* 114(12):1883-1889 (2006).

[17] Health Council of the Netherlands. *Endotoxins. Health-based recommended occupational exposure limit.* The Hague: Health Council of the Netherlands, 2010; publication no. 2010/04OSH.

exposure data were combined across years for any job group in which there were no statistical differences by year. The assigned exposure for a given job and year was taken as the arithmetic mean estimated from the MVUE determined by log-probit regression analysis. This process produced the job exposure matrices for thoracic PM, endotoxin, and glucan shown in Tables 8-10.

For each study subject in the cohort, contemporaneous exposure estimates for thoracic PM, endotoxin, and glucan were then developed for each year between 2007 and 2012 in which the subject was tested by coupling the subject's reported work history with the job exposure matrices. Likewise, cumulative exposure estimates for thoracic PM were calculated using the subject's self-reported number of hours spent in each restoration work activity for each year between 2005 and 2012. Cumulative exposure estimates for endotoxin and glucan were not developed because exposures to those agents during restoration work in 2005 and 2006 could not be estimated with sufficient certainty since the OSHA samples were only assayed gravimetrically for dust content. Table 11 summarizes the results of the exposure estimation for the study cohort. Those subjects for whom job histories were available and whose jobs were included in the job exposure matrices totaled 640, whereas 807 subjects provided information on restoration work activity and duration so that their cumulative exposure to thoracic particulate matter could be estimated.

Analysis of respiratory symptoms in the population showed that the prevalence rates for post-Hurricane Katrina episodes of transient fever and cough, new onset sinusitis, dyspnea, and new onset asthma were significantly elevated for those reporting any restoration work versus those who did no such work (PRR > 1.0, $p < 0.05$, Table 12). For sinusitis, fever with cough, and new onset asthma, the prevalence rate ratios were statistically significant for never-smokers but not for current and ex-smokers. The dyspnea PRR was also elevated for ever-smokers but was not statistically significant, whereas the elevated PRR for the overall study population was significant. Statistically significant elevations in prevalence rate ratios for those having done any restoration work were not observed for pneumonia, COPD, and being below LLN for any of the lung function parameters.

The prevalences of those post-Hurricane Katrina symptoms and conditions that showed statistically elevated prevalence rates for subjects who did any restoration work also increased with cumulative exposure to thoracic particulate matter. Table 13 shows that when analyzed by

logistic regression, the prevalences of dyspnea, fever with cough, and new onset asthma were statistically significantly associated with cumulative exposure to thoracic particulate matter during restoration work in never smokers, after adjustment for gender, with prevalence odds ratios of 1.15, 1.13, and 1.24 per 1,000 mg/m^3-hrs, respectively; however, the respective PORs for current and ex-smokers did not rise to the level of statistical significance. For post-Katrina sinus symptoms, the adjusted prevalence odds ratio was 1.22 per 1,000 mg/m^3-hrs for current and ex-smokers and was statistically significant. The POR for sinusitis in never smokers was also elevated but was not significant. The adjustments for gender in these logistic regressions were necessitated by observed restoration work exposure dose - gender interactions and may be confounded with job type since women in the study tended to be in the custodial/janitorial occupations, whereas men were more likely to be in the building, construction, or maintenance trades. The exposures associated with restoration work done within these two broad categories of occupation are qualitatively different.

The prevalence odds ratios for post-Katrina pneumonia, COPD, and having lung function below lower limit of normal with cumulative TPM exposure dose from restoration work showed no trends with increasing exposure dose and were not statistically significant by logistic regression analysis.

Table 14 presents the distributions of number of annual spirometric tests per study subject in the subgroups of the study population for whom we were able to estimate exposure. There were 807 participants with estimates of cumulative exposure to thoracic particulate matter arising from self-reported specific restoration work activities and durations from 2005 to 2012, whereas we were able to estimate contemporaneous exposures to thoracic particulate matter, endotoxin, and microbial glucan for 640 participants from 2007 to 2012 based on their concurrent jobs. The largest percentages of participants in each group had only one test and were lost to follow-up. Some of these individuals declined to participate in subsequent testing after their initial survey and some were lost to follow-up because they were non-local transient laborers who had responded to the immediate need for restoration workers after Katrina.

For the subgroups with estimates of exposure and multiple lung function tests, Tables 15-30 present the results of the multi-level regression analyses of lung function with exposure and demographic determinants. Results of model runs for study participants with at least 2 and with at least 3 lung function measurements are shown for each analysis. In these analyses, level and

annual change in the various lung function parameters were highly correlated with the expected demographic determinants including gender, race, height, weight, age, and smoking status, illustrating the robustness of the lung function data.

Overall there were no statistically significant adverse effects of either cumulative or contemporaneous exposures on any of the lung function parameters, either for level or annual change. FEV1 and FVC level were positively correlated with cumulative exposure (Tables 15 and 16) which may be indicative of a healthy worker effect. However this effect was statistically significant only for those with at least 2 lung function tests and not for those with at least 3 measures.

In summary, the results of this study indicate no adverse effect of restoration work exposures on lung function generally in the study population but there were exposure related increases in certain respiratory symptoms and in the prevalence of new onset asthma. These results tend to corroborate other reports of respiratory illnesses in various populations shortly after Katrina.

For example, in a survey of 525 New Orleans firefighters,[18] of whom 79% had contact with floodwaters following Hurricane Katrina, 38% reported new onset respiratory symptoms including sinus congestion, throat irritation, and cough. The prevalence rate ratio for those who had contact with floodwater vs. those who did not, was 1.9 and was statistically significant. Such first responders would have had significant exposures to flood sediments and associated contaminants, in addition to microbial agents. It is noteworthy that inhalation exposure to aerosolized sediment collected in the aftermath of Hurricane Katrina was shown to elicit significant pulmonary inflammation, increased airways resistance, and airway hyper-reactivity in a mouse model.[19]

There were widespread anecdotal reports of persistent non-productive cough, often with sore throat and rhinorrhea, in the population residing in New Orleans in the Fall of 2005. This symptom complex became known as the Katrina Cough. While an investigation of this

[18] Tak SW, Bernard BP, Driscoll RJ, Dowell CH. Floodwater exposure and the related health symptoms among firefighters in New Orleans, Louisiana 2005. *Am J Ind Med*. 2007; 50(5):377-382.
[19] Wang K, You D, Balakrishna S et al. Sediment from Hurricane Katrina: potential to produce pulmonary dysfunction in mice. *Int J Clin Exp Med.* 2008; 1(2):130-144.

phenomenon by the Louisiana Department of Health and Hospitals[20] concluded that visits to medical facilities for respiratory complaints in the population of New Orleans were not related to exposure to dust or molds at the residence or at work, there was evidence of a healthy worker effect confounding the analysis. It is likely that Katrina Cough was an irritant phenomenon resulting from a dry fall season with high levels of airborne particulate matter, coinciding with the start of the regular allergy and flu seasons.

The prevalence of episodes of fever with cough in the present study population is clearly elevated for those who have done restoration work and increased with increasing cumulative exposure dose for thoracic particulate matter. Some of these cases may be relatable to Katrina Cough. However, given the overall strong correlation with restoration work exposures, the common reports of multiple and distinct episodes of fever with cough, and the inclusion of the febrile component in the symptom complex, TP is likely to be underlying many of these reports and appears to be a common adverse effect of restoration work exposures in the post Hurricane Katrina environment.

Unlike hypersensitivity pneumonitis, it is uncertain whether toxic pneumonitis and inhalation fevers result in significant lasting decrements in lung function, and functional parameters are expected to return to baseline upon recovery from an episode. This study did not identify any restoration work related decrements in lung function parameters, nor in the prevalence of being below LLN. The World Health Organization, in its report on guidelines on indoor air quality related to damp indoor spaces and mold,[21] concluded that the evidence is inadequate to identify an association between damp indoor environments or the presence of mold with risk of alterations in lung function. However, in a recent study of 6,443 individuals in the European Community Respiratory Health Survey, lung function measurements across 9 years showed statistically significant excess declines in FEV_1 of -2.25 ml/year for women who reported dampness in the home and an additional -7.43 ml/year for those reporting visible damp spots in the bedroom.[22] Annual excess declines of such small magnitude are difficult to detect

[20] Louisiana Department of Health and Hospitals, Office of Public Health. *Katrina Cough.* April 18, 2006. http://www.dhh.louisiana.gov/offices/miscdocs/docs-249/KatrinaCoughReport.pdf .

[21] World Health Organization. *WHO Guidelines for Indoor Air Quality: Dampness and Mold.* Copenhagen, Denmark; 2009

[22] Norback D, Zock JP, Plana E et al. Lung function decline in relation to mould and dampness in the home: the longitudinal European Community Respiratory Health Survey ECRHS II. *Thorax.* 2011; 66(5):396-401.

over short time periods and are unlikely to result in a detectable group difference in function, measured after only a few years in the post Hurricane Katrina environment. Additionally, a healthy worker effect may be confounding the results of our longitudinal analysis of lung function in the overall study population and it is possible that adverse effects on lung function from exposures during restoration work in the more sensitive subpopulations are being masked by this effect.

It is generally accepted that exposure to flood related microbial contaminants can exacerbate existing asthma and there is mounting evidence that such exposures increase the risk of development of new asthma. In a recent extensive review and meta-analysis of the literature from 1980 to 2010, [23] overall odds ratios of 1.49 (C.I.: 1.28 – 1.72) and 1.68 (C.I.: 1.48 – 1.90) were found for the associations of asthma and wheezing, respectively, in children living in homes with visible mold. In our study, there was a significant elevation in prevalence of new onset asthma post-Hurricane Katrina which statistically increased with increasing restoration work exposures to thoracic particulate matter (POR for non-smokers = 1.24 per 1,000 mg/m^3-hrs cumulative TPM exposure, see Table 13).

CONCLUSIONS

Of the study population of 898 workers, 73% reported performing some restoration work activity in the aftermath of Hurricane Katrina. Restoration work activities included demolition, trash and debris management, sewer and waterline repair, landscape restoration, and mold remediation. Demolition and trash and debris management accounted for the majority of the time spent in restoration work activity by the study population. Many restoration workers reported working extraordinarily long hours in the first year after Hurricane Katrina, with 16-hour work days and 7-day work weeks being common.

Dust and bacterial endotoxin levels during restoration work in 2005 frequently exceeded recommended exposure guidelines, especially for demolition and trash and debris management, but rapidly decreased by more than an order of magnitude thereafter. Microbial glucan exposures were likewise elevated in 2005 and then decreased significantly.

[23] Tischer C, Chen CM, Heinrich J. Association between domestic mould and mould components, and asthma and allergy in children: a systematic review. *Eur Respir J.* 2011; 38:812-824.

Important respiratory symptoms and conditions observed in the study population included: sinusitis (37%), dyspnea (34%), COPD (6.4%), asthma (14%), new-onset asthma post-Katrina (4.3%), pneumonia post-Katrina (4.1%), fever with cough (28%), abnormal lung function – flow (10%), and abnormal lung function – volume (11%). Sinusitis, new-onset asthma post-Katrina, dyspnea, and fever with cough were statistically more prevalent among those who did restoration work compared to those who did not. The prevalence of each of these respiratory conditions increased and statistically correlated with both the number of hours of restoration work performed and the cumulative dust exposure dose during the restoration work.

Lung function was lower than expected in the study population overall but was not statistically related to any exposure measures. Level and change in lung function over time did not correlate with current or cumulative exposures but there was evidence of a possible healthy worker effect that could have made any such correlation difficult to detect.

Overall, the results of this study demonstrate that post-Katrina restoration work was associated with certain adverse effects on respiratory health, especially sinusitis, toxic pneumonitis, and asthma. These results support the need for continuing surveillance of the respiratory health of workers engaged in restoration work activities in the aftermath of major flood events. Management of flood restoration workers' exposures to dust and microbial contaminants is recommended to mitigate the risk of acquired respiratory disease.

Table 1: Demographics and respiratory symptoms for the study cohort (n = 898)

Age: mean (S.D.)	46.2 (13.5)
% male	70.2
% non-white	73.5
% never smoker	54.6
% current/ex- smoker	45.4
% with any restoration work	73.0
% dyspnea	34.2
% new onset sinus symptoms	37.2
% fever & cough	27.9
% ever asthma	13.5
% new onset asthma (*n* without asthma pre-Katrina)	4.3 (784)
% new onset pneumonia	4.1
% COPD	5.7
% < LLN, FEV1	9.8
% < LLN, FVC	10.6
% < LLN, FEV1/FVC	5.2

Table 2: Percent predicted (Hankinson) lung function of the study cohort at first test.

Lung Function Parameter	Percent Predicted Lung Function Mean (SD)	
	Never Smokers N = 468	**Ever Smokers N = 406**
FEV1	97.9 (14.2)	93.2 (16.8)
FVC	96.5 (13.4)	93.9 (15.0)
FEV1/FVC ratio	101.2 (7.0)	98.7 (9.2)
FEF25-75	103.8 (31.0)	92.8 (32.9)

Table 3: Post-Katrina restoration work activity groupings and summary statistics for Louisiana samples in the OSHA database

Restoration work activity	Year	Exposure Measure	N	GM (mg/m^3)	GSD	Mean (mg/m^3)	S.E.	PEL[A] Exceedance (%)
Demolition	2005	Total PM	32	0.87	6.3	4.1	2.4	6.1
		Respirable PM	5	0.95	5.9	2.8	2.0	17.6
	2006	Total PM	85	0.61	3.8	1.5	0.3	0.9
		Respirable PM	54	0.05	4.0	0.13	0.04	< 0.1
Trash/debris management	2005	Total PM	135	0.94	6.4	5.2	2.1	6.9
		Respirable PM	29	0.51	5.0	1.7	0.8	7.8
	2006	Total PM	207	0.73	4.7	2.4	0.5	2.5
		Respirable PM	84	0.07	3.6	0.16	0.03	< 0.1
Sewer & waterline repair	2005-2006	Total PM	4	0.62	7.3	1.5	1.2	---
		Respirable PM	1	---	---	0.19	---	---
Landscape & tree trimming	2005-2006	Total PM	19	0.35	3.9	0.80	0.32	0.3
		Respirable PM	8	0.06	2.7	0.09	0.03	< 0.1
All other activities	2005	Total PM	54	0.42	3.7	0.97	0.25	0.3
		Respirable PM	6	0.17	6.8	0.62	0.47	3.8
	2006	Total PM	4	0.69	1.39	0.72	0.11	---
		Respirable PM	3	---	---	< 0.03[B]	---	---

[A]OSHA PEL for Particles Not Otherwise Classified (PNOC): 15 mg/m^3 total dust; 5 mg/m^3 respirable dust

[B]2 of 3 samples were censored (< 0.015 mg/m^3)

Table 4: Regression models for estimation of thoracic particulate matter from respirable and total particulate matter in the OSHA database

Model --- Data Source	N	Thoracic PM = β_1(Respirable PM) + β_2(Total PM)		
		β_1 (S.E.)	β_2 (S.E.)	R^2
I Limestone quarry and coal preparation	8	1.57 (2.25)	0.21(0.25)	0.99
II Industrial wood processing	46	2.83 (0.33)	0.04 (0.03)	0.73
III This study, post- Katrina demolition	8	1.97 (0.40)	0.12 (0.12)	0.91

Table 5: Thoracic particulate matter exposures for restoration work activities estimated from OSHA respirable and total particulate matter exposure data, 2005 and 2006

Activity	Year	*Estimated Thoracic PM (mg/m^3)			
		Model I	Model II	Model III	Mean (S.D.)
Demolition	2005 2006	4.8 0.51	7.3 0.43	5.4 0.43	5.8 (1.3) 0.46 (0.05)
Trash & debris management	2005 2006	3.7 0.75	5.0 0.56	3.9 0.60	4.2 (0.7) 0.64 (0.10)
Sewer & waterline repair	2005-2006	0.62	0.61	0.56	0.59 (0.03)
Landscape & tree-trimming	2005-2006	0.36	0.35	0.32	0.34 (0.02)
All other activities	2005 2006	1.2 0.19	1.8 0.10	1.3 0.13	1.4 (0.3) 0.14 (0.04)

* 2-way ANOVA: activity/year, $p < 0.001$; Model, $p = 0.12$

Table 6: Post-Katrina restoration work activity and measured exposure to thoracic particulate matter, 2007 -2012

Restoration work activity	Years	N	GM (mg/m^3)	GSD	Mean (mg/m^3)	S.E.
Demolition[A]	2008	19	0.42	4.7	1.24	0.6
	2009	52	0.19	2.8	0.31	0.06
	2010 - 2012	51	0.08	2.2	0.11	0.01
Trash & debris management	2007 - 2012	24	0.12	4.3	0.31	0.13
Sewer & waterline repair	2007 - 2012	49	0.15	4.2	0.40	0.13
Landscape & tree trimming	2007 -2012	67	0.11	3.0	0.19	0.03
Mold remediation	2007-2011	5	0.23	1.7	0.26	0.06
All other activities	2007-2010	388	0.15	3.2	0.50	0.03
	2011-2012	119	0.09	2.6	0.17	0.02

[A]no data available for demolition in 2007

Table 7: Content of endotoxin (EU/mg) and (1→3, 1→6) β-D-glucan (µg/mg) in collected thoracic PM , 2007 -2012

Restoration work activity	Agent	Years[A]	N	GM (EU/mg) or (µg/mg)	GSD	Mean (EU/mg) or (µg/mg)	S.E.
Demolition	endotoxin	2008	10	13.4	5.2	41.2	25.4
		2009-2012	19	1.30	19	23.3	12.2
	glucan	2008 - 2012	29	3.21	7.5	20.3	14.9
Trash & debris management	endotoxin	2008 - 2012	16	12.7	6.7	61.0	43.5
	glucan	2008 - 2012	16	1.76	6.3	18.7	4.7
Sewer & waterline repair	endotoxin	2007 - 2012	13	2.98	5.2	8.72	4.22
	glucan	2007 - 2012	13	1.27	5.6	4.34	2.55
Landscape & tree trimming	endotoxin	2007 - 2011	21	5.80	12	82.3	99
	glucan	2007 - 2011	21	1.22	3.4	2.45	0.82
Mold remediation	endotoxin	2007- 2011	3	7.37	3.4	11.4	6.82
	glucan	2007 - 2011	3	0.71	2.3	0.89	0.42
All other activities	endotoxin	2007-2008	64	7.98	6.3	41.2	20.7
		2009-2011	50	3.22	8.3	24.3	15.4
	glucan	2007 - 2008	63	2.11	5.5	8.40	3.46
		2009 - 2010	31	1.41	3.9	3.33	1.12
		2011 - 2012	26	0.29	3.8	0.64	0.21

[A]no data available for demolition and trash & debris management in 2007; landscape & tree trimming and mold remediation in 2012

Table 8: Job exposure matrix for thoracic particulate matter (mg/m^3), 2007 – 2012

Code	Job	2007	2008	2009	2010	2011	2012
1	Painting	0.27	0.27	0.27	0.27	0.27	0.27
4	Masonry	0.45	0.45	0.45	0.45	0.45	0.45
5	Carpentry	0.30	0.49	0.49	0.23	0.23	0.23
6	Electrical	0.17	0.17	0.17	0.17	0.17	0.17
10	Heat Air-Conditioning	0.32	0.32	0.11	0.11	0.11	0.11
12	Plumbing	0.29	0.29	0.29	0.29	0.29	0.29
13	Janitorial	0.26	0.26	0.11	0.11	0.11	0.11
14	Grounds Landscaping	0.21	0.21	0.21	0.21	0.21	0.21
16	Heavy Equip Operator	0.16	0.16	0.16	0.16	0.16	0.16
19	Demolition-Ripout	0.85	1.24	0.31	0.11	0.11	0.11
22	Foreman/Supervisor	0.49	0.49	0.49	0.49	0.49	0.49
24	Machinist	0.18	0.18	0.18	0.18	0.18	0.18
27	Welder	0.93	0.93	0.93	0.93	0.93	0.93
35	Deputy	0.18	0.18	0.18	0.18	0.18	0.18
43	General Maintenance	0.18	0.43	0.43	0.43	0.17	0.17
58	Supervisor	0.18	0.18	0.18	0.18	0.18	0.18
69	Clerk	0.20	0.20	0.20	0.20	0.20	0.20
81	Mover	0.48	0.48	0.48	0.48	0.48	0.48
83	Medical Asst	0.10	0.10	0.10	0.10	0.10	0.10
88	Plant Control	0.61	0.61	0.61	0.61	0.61	0.61
136	Life Safety	0.10	0.10	0.10	0.10	0.10	0.10

Table 9: Job exposure matrix for endotoxin (EU/m^3), 2007 – 2012

Code	Job	2007	2008	2009	2010	2011	2012
1	Painting	1.94	1.94	1.94	1.94	1.94	1.94
4	Masonry	39.85	39.85	39.85	39.85	39.85	39.85
5	Carpentry	11.28	11.28	11.28	11.28	11.28	11.28
6	Electrical	1.03	1.03	1.03	1.03	1.03	1.03
10	Heat Air-Conditioning	7.50	7.50	7.50	7.50	7.50	7.50
12	Plumbing	0.78	0.78	0.78	0.78	0.78	0.78
13	Janitorial	2.49	2.49	2.49	2.49	2.49	2.49
14	Grounds Landscaping	19.70	19.70	19.70	19.70	19.70	19.70
16	Heavy Equip Operator	3.26	3.26	3.26	3.26	3.26	3.26
19	Demolition-Ripout	2.52	2.52	2.52	2.52	2.52	2.52
22	Foreman/Supervisor	2.53	2.53	2.53	2.53	2.53	2.53
24	Machinist	12.25	12.25	12.25	12.25	12.25	12.25
27	Welder	0.79	0.79	0.79	0.79	0.79	0.79
35	Deputy	0.86	0.86	0.86	0.86	0.86	0.86
43	General Maintenance	1.82	1.82	1.82	1.82	1.82	1.82
58	Supervisor	4.28	4.28	4.28	4.28	4.28	4.28
69	Clerk	0.64	0.64	0.64	0.64	0.64	0.64
81	Mover	7.30	7.30	7.30	7.30	7.30	7.30
88	Plant Control	1.32	1.32	1.32	1.32	1.32	1.32
136	Life Safety	5.56	5.56	5.56	5.56	5.56	5.56

Table 10: Job exposure matrix for (1→3, 1→6)-β-D-glucan (µg/m^3), 2007-2012

Code	Job	2007	2008	2009	2010	2011	2012
1	Painting	1.06	1.06	1.06	1.06	1.06	1.06
4	Masonry	0.53	0.53	0.53	0.53	0.53	0.53
5	Carpentry	0.75	0.75	0.15	0.63	0.06	0.06
6	Electrical	0.68	0.68	0.68	0.68	0.68	0.68
10	Heat Air-Conditioning	0.54	0.54	0.54	0.54	0.54	0.54
12	Plumbing	0.65	0.65	0.65	0.65	0.65	0.65
13	Janitorial	1.76	1.76	1.76	1.76	1.76	1.76
14	Grounds Landscaping	0.44	0.44	0.44	0.44	0.44	0.44
16	Heavy Equip Operator	0.32	0.32	0.32	0.32	0.32	0.32
19	Demolition-Ripout	0.66	0.66	0.66	0.66	0.66	0.66
22	Foreman/Supervisor	1.18	1.18	1.18	1.18	1.18	1.18
24	Machinist	0.39	0.39	0.39	0.39	0.39	0.39
27	Welder	0.11	0.11	0.11	0.11	0.11	0.11
35	Deputy	0.24	0.24	0.24	0.24	0.24	0.24
43	General Maintenance	0.29	0.29	0.29	0.29	0.29	0.29
58	Supervisor	0.32	0.32	0.32	0.32	0.32	0.32
69	Clerk	0.03	0.03	0.03	0.03	0.03	0.03
81	Mover	2.09	2.09	2.09	2.09	2.09	2.09
88	Plant Control	0.77	0.77	0.77	0.77	0.77	0.77

Table 11: Summary Statistics for exposures for the longitudinal study population. Contemporaneous exposures based on Job, 2007 - 2012 (n = 640). Cumulative exposure based on restoration work activities, 2005 – 2012 (n = 807).

Exposure	Minimum	Maximum	Mean	Standard Deviation	Geometric Mean	Geometric Standard Deviation
Thoracic particulate matter (mg/m^3)	0.11	1.24	0.29	0.16	0.25	1.6
Endotoxin (EU/m^3)	0.64	19.7	4.86	5.02	2.93	2.8
Glucan ($\mu g/m^3$)	0.03	1.76	0.70	0.58	0.44	3.0
Cumulative Thoracic Particulate Matter (mg/m^3-hrs)	0[#]	16,129	1,419	2,192	583*	8.4*

[#]zero cumulative exposure for no restoration work performed.

*For 581 study subjects who performed any restoration work.

Table 12: Respiratory symptoms and conditions in the study cohort with restoration work by smoking status post-Hurricane Katrina New Orleans, 2005 - 2012

Smoking Status	n	Univariate Prevalence Rate Ratio (PRR): any restoration work vs. none (95% Confidence Interval)								
		fever & cough	**new onset sinus symptoms**	**pneumonia**	**new onset asthma**	**dyspnea**	**COPD**	**FEV1 below LLN**	**FVC below LLN**	**FEV1/FVC below LLN**
Current & Ex-smokers	418	1.2 (0.8, 1.7)	1.2 (0.9, 1.6)	2.1 (0.6, 6.9)	0.8 (0.2, 3.0)	1.1 (0.8, 1.5)	1.0 (0.5, 2.1)	0.8 (0.5, 1.4)	0.8 (0.5, 1.4)	1.4 (0.6, 3.7)
Never smokers	479	**1.6*** (1.1, 2.4)	**1.4*** (1.0, 1.9)	0.8 (0.2, 2.7)	**9.2*** (1.3, 67.7)	1.3 (1.0, 1.9)	0.6 (0.2, 1.7)	0.5 (0.2, 1.1)	0.6 (0.3, 1.2)	0.4 (0.1, 1.0)
Overall	897	**1.4*** (1.1, 1.9)	**1.3*** (1.1, 1.6)	1.5 (0.7, 3.4)	**2.7*** (1.0, 7.5)	**1.3*** (1.0, 1.6)	0.9 (0.5, 1.7)	0.8 (0.5, 1.2)	0.8 (0.5, 1.2)	0.9 (0.5, 1.7)

*** $p < 0.05$**

Table 13: Results of logistic regression for respiratory symptoms versus cumulative exposure to thoracic particulate matter adjusted for gender. Values are Prevalence Odds Ratio per 1,000 mg/m^3-hrs (95% Confidence Interval).

	Sinusitis	New onset asthma	Pneumonia	Dyspnea	Fever with cough	COPD	<LLN FEV1	<LLN FVC	<LLN FEV/FVC
Never smoker	1.10 (0.99, 1.22)	**1.24* (1.06, 1.45)**	0.92 (0.58, 1.47)	**1.15* (1.04, 1.28)**	**1.13* (1.02, 1.25)**	1.06 (0.84, 1.34)	0.89 (0.69, 1.15)	0.88 (0.70, 1.11)	1.06 (0.84, 1.33)
Current/ex smoker	**1.22* (1.10, 1.35)**	0.89 (0.62, 1.27)	1.05 (0.89, 1.24)	1.01 (0.92, 1.11)	1.06 (0.97, 1.17)	0.99 (0.84, 1.17)	0.91 (0.78, 1.06)	0.86 (0.74, 1.10)	0.98 (0.81, 1.19)

*** $P < 0.05$**

Table 14: Distributions of number of annual spirometric tests per participant in study subgroups having cumulative and contemporaneous exposure estimates

Number of spirometric tests	Frequency (percent of total)	
	Subgroup with cumulative exposure estimates 2005-2012	**Subgroup with contemporaneous exposure estimates 2007-2012**
1	486 (60.2%)	376 (58.8%)
2	183 (22.7%)	143 (22.3%)
3	70 (8.7%)	56 (8.8%)
4	41 (5.1%)	39 (6.1%)
5	27 (3.4%)	26 (4.1%)
total	807	640

Table 15: Analysis of FEV1 level with cumulative exposure to thoracic particulate matter, 2005 – 2012

Parameter	Coefficient (L) SE p-value	
	≥ 2 tests N = 319	**≥ 3 tests N = 138**
Intercept	-1.4471 0.5684 0.011	-1.4036 0.8751 0.111
Age (per yr)	-0.03003 0.001773 <0.0001	-0.02957 0.002827 <0.0001
Weight (per lb)	-0.00194 0.000464 <0.0001	-0.00367 0.000817 <0.0001
Height (per cm)	0.03652 0.003569 <0.0001	0.03876 0.005502 <0.0001
Black	-0.3745 0.05528 <0.0001	-0.4120 0.08978 <0.0001
Sex	0.3739 0.07682 <0.0001	0.2685 0.1463 0.067
Ever Smoker	-0.04490 0.03666 0.22	-0.00670 0.05366 0.90
Cumulative TPM Exposure (per mg/m^3-hr)	0.000030 0.000012 0.018	0.000015 0.000020 0.44

Table 16: Analysis of FVC level with cumulative exposure to thoracic particulate matter, 2005 – 2012

Parameter	Coefficient (L) SE p-value	
	≥ 2 tests N = 319	**≥ 3 tests N = 138**
Intercept	-2.8523 0.6419 <0.0001	-2.5390 0.9539 0.0087
Age (per yr)	-0.02945 0.002007 <0.0001	-0.02886 0.003105 <0.0001
Weight (per lb)	-0.00281 0.000529 <0.0001	-0.00486 0.000901 <0.0001
Height (per cm)	0.04989 0.004033 <0.0001	0.05056 0.006002 <0.0001
Black	-0.5046 0.06277 <0.0001	-0.5252 0.09715 <0.0001
Sex	0.4445 0.08661 <0.0001	0.3728 0.1586 0.0193
Ever Smoker	-0.00129 0.04181 0.97	0.03633 0.05959 0.54
Cumulative TPM Exposure (per mg/m^3-hr)	0.000032 0.000014 0.024	0.000021 0.000021 0.99

Table 17: Analysis of FEF25-75 level with cumulative exposure to thoracic particulate matter, 2005 – 2012

Parameter	Coefficient (L) SE p-value	
	≥ 2 tests N = 319	**≥ 3 tests N = 138**
Intercept	1.2045 1.2163 0.32	-0.2868 1.8062 0.87
Age (per yr)	-0.04275 0.003833 <0.0001	-0.04343 0.005985 <0.0001
Weight (per lb)	-0.00176 0.001031 0.088	-0.00309 0.001756 0.080
Height (per cm)	0.02493 0.007656 0.0012	0.03737 0.01139 0.0011
Black	-0.3032 0.1171 0.0099	-0.3727 0.1809 0.040
Sex	0.4111 0.1633 0.0121	0.1496 0.2968 0.61
Ever Smoker	-0.1740 0.08175 0.034	-0.1658 0.1182 0.16
Cumulative TPM Exposure (per mg/m^3-hr)	0.000037 0.000026 0.16	0.0000022 0.000040 0.95

Table 18: Analysis of FEV/FVC ratio level with cumulative exposure to thoracic particulate matter, 2005 – 2012

Parameter	Coefficient (%) SE p-value	
	≥ 2 tests N = 319	**≥ 3 tests N = 138**
Intercept	100.60 7.7263 <0.0001	98.6310 11.1149 <0.0001
Age (per yr)	-0.1777 0.02374 <0.0001	-0.1737 0.03538 <0.0001
Weight (per lb)	0.007846 0.006007 0.19	-0.01516 0.01014 0.14
Height (per cm)	-0.07634 0.04838 0.12	-0.06175 0.06977 0.38
Black	0.5909 0.7627 0.44	-0.01441 1.1568 0.99
Sex	-0.09958 1.0541 0.92	-1.9329 1.8776 0.30
Ever Smoker	-1.1130 0.4725 0.019	-0.9081 0.6580 0.17
Cumulative TPM Exposure (per mg/m^3-hr)	0.000061 0.000172 0.72	-0.00002 0.000253 0.92

Table 19: Analysis of FEV1 annual change with cumulative exposure to thoracic particulate matter, 2005 – 2012

parameter	Coefficient (L per yr) SE p-value	
	≥ 2 tests N = 319	**≥ 3 tests N = 138**
Intercept	-0.2566 0.09350 0.0064	-0.2775 0.1176 0.020
Weight (per lb)	-0.00018 0.000095 0.065	-0.00031 0.000129 0.018
Height (per cm)	0.001489 0.000604 0.014	0.001884 0.000774 0.016
Black	-0.01488 0.009190 0.107	-0.00163 0.01187 0.89
Sex	0.01258 0.01313 0.34	-0.01612 0.02043 0.43
Ever Smoker	0.001162 0.007573 0.88	0.002203 0.09962 0.83
Cumulative TPM Exposure (per mg/m^3-hr)	-4.32E-7 2.01E-6 0.83	-1.36E-6 2.478E-6 0.58

Table 20: Analysis of FVC annual change with cumulative exposure to thoracic particulate matter, 2005 – 2012

parameter	Coefficient (L per yr) SE p-value	
	≥ 2 tests N = 319	**≥ 3 tests N = 138**
Intercept	-0.4761 0.1096 <0.0001	-0.4549 0.1373 0.001
Weight (per lb)	-0.00032 0.000110 0.004	-0.00047 0.000150 0.002
Height (per cm)	0.002920 0.000707 <0.0001	0.003090 0.000902 0.0007
Black	-0.03259 0.01069 0.003	-0.01345 0.01373 0.33
Sex	0.02298 0.01517 0.13	-0.00750 0.02353 0.75
Ever Smoker	0.004038 0.008765 0.65	0.003872 0.01143 0.74
Cumulative TPM Exposure (per mg/m^3-hr)	-47.28E-7 2.34E-6 0.76	-3.13E-6 2.89E-6 0.28

Table 21: Analysis of FEF25-75 annual change with cumulative exposure to thoracic particulate matter, 2005 – 2012

parameter	Coefficient (L per yr) SE p-value	
	≥ 2 tests N = 319	≥ 3 tests N = 138
Intercept	-0.3988 0.1998 0.047	-0.5849 0.2560 0.024
Weight (per lb)	-0.00023 0.000206 0.28	-0.00045 0.000282 0.11
Height (per cm)	0.002284 0.001294 0.079	0.003896 0.001687 0.022
Black	-0.01024 0.01968 0.60	-0.01489 0.02582 0.56
Sex	0.02309 0.02810 0.41	-0.03527 0.04429 0.43
Ever Smoker	0.02309 0.01649 0.89	0.004846 0.02190 0.83
Cumulative TPM Exposure (per mg/m^3-hr)	-1.415E-6 4.296E-6 0.74	2.945E-6 5.405E-6 0.59

Table 22: Analysis of FEV/FVC ratio annual change with cumulative exposure to thoracic particulate matter, 2005 – 2012

parameter	Coefficient (% per yr) SE p-value	
	≥ 2 tests N = 319	≥ 3 tests N = 138
Intercept	-0.7295 0.9706 0.45	-1.2193 1.1537 0.29
Weight (per lb)	0.000873 0.0001022 0.39	0.000869 0.001292 0.50
Height (per cm)	0.002814 0.006315 0.66	0.006266 0.007657 0.41
Black	0.1326 0.09848 0.18	0.09015 0.1204 0.45
Sex	-0.1492 0.1428 0.30	-0.2701 0.2090 0.20
Ever Smoker	-0.1035 0.08312 0.21	-0.07581 0.1037 0.47
Cumulative TPM Exposure (per mg/m^3-hr)	3.60E-5 2.10E-5 0.091	4.80E-5 2.508E-5 0.053

Table 23: Analysis of FEV1 level with contemporaneous exposure to endotoxin, glucan and thoracic particulate matter, 2007 – 2012

parameter	Coefficient (L) SE p-value	
	≥ 2 tests N = 263	≥ 3 tests N = 121
Intercept	-1.0685 0.6320 0.09	-1.5484 0.9047 0.09
Age	-0.02947 0.002086 <0.0001	-0.02619 0.003206 <0.0001
Weight (per lb)	-0.00188 0.000504 0.0002	-0.00402 0.00085 <0.0001
Height (per cm)	0.03388 0.003963 <0.0001	0.03875 0.005636 <0.0001
Black	-0.3851 0.05758 <0.0001	-0.3780 0.08628 <0.0001
Sex	0.4493 0.08898 <0.0001	0.2900 0.1519 0.057
Ever Smoker	-0.01652 0.04061 0.68	-0.01313 0.05648 0.82
Endotoxin Exposure (per EU/m^3)	0.002773 0.005242 0.60	0.009416 0.007341 0.20
Glucan Exposure (per µg/m^3)	-0.01188 0.03816 0.76	0.03247 0.05632 0.56
TPM Exposure (per mg/m^3)	0.04158 0.08301 0.62	-0.02605 0.1024 0.80

Table 24: Analysis of FVC level with contemporaneous exposure to endotoxin, glucan and thoracic particulate matter, 2007 – 2012

parameter	Coefficient (L) SE p-value	
	≥ 2 tests N = 263	≥ 3 tests N = 121
Intercept	-2.3485 0.7162 0.0012	-2.3616 1.0105 0.02
Age	-0.02909 0.002365 <0.0001	-0.02568 0.003584 <0.0001
Weight (per lb)	-0.00267 0.000574 <0.0001	-0.00504 0.000949 <0.0001
Height (per cm)	0.04670 0.004493 <0.0001	0.04853 0.006295 <0.0001
Black	-0.5226 0.06517 <0.0001	-0.5216 0.09627 <0.0001
Sex	0.5048 0.1008 <0.0001	0.4274 0.1695 0.01
Ever Smoker	0.04552 0.04621 0.33	0.04305 0.06324 0.50
Endotoxin Exposure (per EU/m^3)	-0.00278 0.005934 0.64	0.004769 0.008192 0.56
Glucan Exposure (per µg/m^3)	-0.05346 0.04338 0.22	0.03005 0.06308 0.63
TPM Exposure (per mg/m^3)	0.1434 0.09484 0.13	0.05327 0.1149 0.64

Table 25: Analysis of FEF25-75 level with contemporaneous exposure to endotoxin, glucan and thoracic particulate matter, 2007 – 2012

parameter	Coefficient (L) SE p-value	
	≥ 2 tests N = 263	**≥ 3 tests N = 121**
Intercept	2.0944 1.4150 0.14	-0.5649 1.9940 0.78
Age	-0.04189 0.004690 <0.0001	-0.03954 0.007126 <0.0001
Weight (per lb)	-0.00177 0.001152 0.13	-0.00437 0.001899 0.022
Height (per cm)	0.01895 0.008885 0.03	0.03935 0.01243 0.0017
Black	-0.3138 0.1280 0.015	-0.2330 0.1883 0.22
Sex	0.6107 0.1984 0.0022	0.1065 0.3325 0.75
Ever Smoker	-0.1823 0.09305 0.05	-0.1818 0.1278 0.16
Endotoxin Exposure (per EU/m^3)	0.01439 0.01166 0.22	0.01658 0.01605 0.30
Glucan Exposure (per µg/m^3)	0.04702 0.08691 0.59	-0.03792 0.1280 0.77
TPM Exposure (per mg/m^3)	-0.2391 0.1949 0.22	-0.2670 0.2371 0.26

Table 26: Analysis of FEV/FVC ratio level with contemporaneous exposure to endotoxin, glucan and thoracic particulate matter, 2007 – 2012

parameter	Coefficient (%) SE p-value	
	≥ 2 tests N = 263	**≥ 3 tests N = 121**
Intercept	102.75 8.4029 <0.0001	93.0156 11.2481 <0.0001
Age	-0.1614 0.02743 <0.0001	-0.1381 0.03921 0.0005
Weight (per lb)	0.005710 0.006387 0.37	0.002360 0.01025 0.82
Height (per cm)	-0.09863 0.05254 0.06	-0.03120 0.06999 0.66
Black	1.1556 0.7803 0.14	1.4132 1.0949 0.20
Sex	1.0029 1.1967 0.40	-1.5754 1.9146 0.41
Ever Smoker	-1.2336 0.5106 0.016	-1.1557 0.6690 0.085
Endotoxin Exposure (per EU/m^3)	0.1121 0.07095 0.11	0.1383 0.09283 0.13
Glucan Exposure (per µg/m^3)	0.8033 0.4867 0.100	0.3898 0.6625 0.56
TPM Exposure (per mg/m^3)	-2.1839 0.9946 0.029	-2.1288 1.1715 0.07

Table 27: Analysis of FEV1 annual change with contemporaneous exposure to endotoxin, glucan and thoracic particulate matter, 2007 - 2012

parameter	Coefficient (L/yr) SE p-value	
	≥ 2 tests N = 263	≥ 3 tests N = 121
Intercept	-0.2583 0.1064 0.016	-0.282 0.136 0.041
Weight (per lb)	-0.00017 0.00010 0.09	-0.00032 0.00014 0.025
Height (per cm)	0.001368 0.000677 0.045	0.00183 0.00088 0.038
Black	-0.01437 0.00984 0.15	-0.00412 0.01278 0.75
Sex	0.02113 0.01537 0.17	-0.00874 0.02315 0.71
Ever Smoker	0.00186 0.00841 0.83	0.00208 0.0110 0.85
Endotoxin Exposure (per EU/m^3)	0.000681 0.000854 0.43	0.00048 0.00104 0.65
Glucan Exposure (per µg/m^3)	0.01098 0.00809 0.18	0.01105 0.01169 0.35
TPM Exposure (per mg/m^3)	0.00941 0.03287 0.78	-0.00665 0.04310 0.88

Table 28: Analysis of FVC annual change with contemporaneous exposure to endotoxin, glucan and thoracic particulate matter, 2007 - 2012

parameter	Coefficient (L/yr) SE p-value	
	≥ 2 tests N = 263	≥ 3 tests N = 121
Intercept	-0.4546 0.1224 0.0002	-0.4874 0.1570 0.002
Weight (per lb)	-0.00029 0.00012 0.011	-0.00045 0.00016 0.006
Height (per cm)	0.002604 0.00078 0.001	0.003111 0.001010 0.002
Black	-0.03149 0.01126 0.006	-0.01694 0.01464 0.25
Sex	0.02440 0.01755 0.17	-0.0114 0.02650 0.67
Ever Smoker	0.00429 0.00960 0.66	0.000649 0.01250 0.96
Endotoxin Exposure (per EU/m^3)	0.001687 0.000982 0.09	0.002233 0.001206 0.07
Glucan Exposure (per µg/m^3)	0.01176 0.00919 0.20	0.01609 0.01331 0.22
TPM Exposure (per mg/m^3)	0.02531 0.03741 0.50	0.005168 0.04861 0.92

Table 29: Analysis of FEF 25-75 annual change with contemporaneous exposure to endotoxin, glucan and thoracic particulate matter, 2007 - 2012

parameter	Coefficient (L/yr) SE p-value	
	≥ 2 tests N = 263	≥ 3 tests N = 121
Intercept	-0.2469 0.2299 0.28	-0.3366 0.2906 0.25
Weight (per lb)	-0.00026 0.00022 0.25	-0.00055 0.00030 0.07
Height (per cm)	0.001257 0.001465 0.39	0.00256 0.00187 0.17
Black	-0.01214 0.02133 0.57	-0.01023 0.02738 0.71
Sex	0.06386 0.03331 0.06	0.003534 0.04956 0.94
Ever Smoker	0.01219 0.01846 0.51	0.02153 0.02375 0.37
Endotoxin Exposure (per EU/m^3)	-0.00192 0.001845 0.30	-0.00376 0.002236 0.09
Glucan Exposure (per µg/m^3)	0.01342 0.01759 0.47	-0.00977 0.02520 0.70
TPM Exposure (per mg/m^3)	-0.00899 0.07389 0.90	-0.02706 0.09545 0.77

Table 30: Analysis of FEV1/FVC ratio annual change with contemporaneous exposure to endotoxin, glucan and thoracic particulate matter, 2007 - 2012

parameter	Coefficient (%/yr) SE p-value	
	≥ 2 tests N = 263	**≥ 3 tests N = 121**
Intercept	-0.2094 1.0800 0.85	-0.1192 1.2631 0.93
Weight (per lb)	0.000393 0.001050 0.71	0.000143 0.00132 0.91
Height (per cm)	0.000344 0.006870 0.96	0.001129 0.008147 0.89
Black	0.1370 0.1023 0.18	0.1044 0.1223 0.39
Sex	0.1039 0.1612 0.52	0.01181 0.2226 0.96
Ever Smoker	-0.06922 0.08904 0.44	-0.01846 0.1074 0.86
Endotoxin Exposure (per EU/m^3)	-0.01293 0.008694 0.14	-0.0210 0.00976 0.03
Glucan Exposure (per µg/m^3)	0.03330 0.08619 0.70	-0.04081 0.1150 0.72
TPM Exposure (per mg/m^3)	-0.4782 0.3619 0.19	-0.3301 0.4502 0.46

Inclusion Enrollment Report

Study Title: Respiratory Effects in Workers from Post-Katrina Related Airborne Exposures

Total Enrollment: 898 **Protocol Number:** 09-00383

Grant Number: 5R01OH008938

PART A. TOTAL ENROLLMENT REPORT: Number of Subjects Enrolled to Date (Cumulative) by Ethnicity and Race					
Ethnic Category	**Females**	**Males**	**Sex/Gender Unknown or Not Reported**	**Total**	
Hispanic or Latino	25	67		92	**
Not Hispanic or Latino	231	563		794	
Unknown (individuals not reporting ethnicity)			12	12	
Ethnic Category: Total of All Subjects*	256	630	12	898	*
Racial Categories					
American Indian/Alaska Native					
Asian	2	4		6	
Native Hawaiian or Other Pacific Islander					
Black or African American	195	352		547	
White	33	205		238	
More Than One Race					
Unknown or Not Reported	26	69	**12**	107	
Racial Categories: Total of All Subjects*	256	630	12	898	*

PART B. HISPANIC ENROLLMENT REPORT: Number of Hispanics or Latinos Enrolled to Date (Cumulative)					
Racial Categories	**Females**	**Males**	**Sex/Gender Unknown or Not Reported**	**Total**	
American Indian or Alaska Native					
Asian					
Native Hawaiian or Other Pacific Islander					
Black or African American					
White					
More Than One Race					
Unknown or Not Reported	25	67		92	
Racial Categories: Total of Hispanics or Latinos**	25	67		92	**

* These totals must agree.

** These totals must agree.

Publications

1. Glindmeyer HW, Rando RJ, Freyder L, Lefante JJ, Jones R: [2010] Respiratory Health and Work in the Post-Katrina Environment. American Journal of Respiratory and Critical Care Medicine 181:A4701.
    - Peer reviewed abstract on preliminary cross-sectional description of lung function and respiratory symptoms in first 594 study participants

2. Rando RJ**,** Lefante JJ, Freyder LM, Jones RN: [2012] Respiratory Health and Flood Restoration Work in the Post Katrina Environment. In: Bioaerosols - 6th International Scientific Conference on Bioaerosols, Fungi, Bacteria, Mycotoxins in Indoor and Outdoor Environments and Human Health. (eds. E Johanning, PR Morey, P Auger), Fungal Research Group Foundation, Inc., pp. 61-67.
    - Peer reviewed manuscript reporting on initial cross-sectional analysis of lung function and symptoms in first 791 study participants. Prevalence of symptoms was evaluated statistically for those who did restoration work v those who did not.

3. Rando RJ**,** Lefante JJ, Freyder LM, Jones RN: [2012] Respiratory Health Associated with Restoration Work in Post Hurricane Katrina New Orleans. Journal of Environmental and Public Health. Vol. 2012. *8 pp.* doi:10.1155/2012/462478.
    - Peer reviewed manuscript describing detailed initial cross-sectional analysis of lung function and symptoms in first 791 study participants. Symptom prevalence was evaluated statistically for those who did restoration work v those who did not, and was further analyzed for correlation with the time spent performing restoration work.

4. Rando RJ, Kwon CW, Lefante JJ: [2013] Exposures to Thoracic Particulate Matter, Endotoxin, and Glucan During Post-Hurricane Katrina Restoration Work, New Orleans 2005 – 2012. Journal of Occupational and Environmental Hygiene. doi**:**10.1080/15459624.2013.839879. in press.
    - Peer reviewed manuscript detailing the analysis of exposures to thoracic particulate matter, endotoxin and glucan in the study cohort and the construction of the work/exposure matrix for 2005 to 2012.

Materials Available for Other Investigators

- Electronic datafile containing demographics, questionnaire responses, lung function measurements, and exposure estimates for 898 study subjects. Data is de-identified with study subject number only. SAS format.
- 572 37-mm polycarbonate filter samples containing thoracic particulate matter collected during the study, 2007 – 2012.