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MINI GRANT PROJECT SUMMARY

Please complete the project summary and return the completed form to Alyssa Johnson, Administrative Assistant at the Institute on the Environment, at joh10074@umn.edu. Paper copies will not be accepted. Please also attach any photos, publications, brochures, event agendas or other materials that were a result of the mini grant summary.

Date of Report Submission:	04/06/2016
Project PI & Dept/School	Matteo Convertino (SPH)
Project Title:	*Attributing Cardiovascular and Respiratory Disease Mortality to PM2.5 Exposure: Is It to Blame? A Complex Systems Approach*
Grant Amount $:	$3000

Project Context & Purpose

Please include the original project purpose statement and revise for any changes that occurred in the project after the start date with a short explanation of the changes.

Cardiovascular and respiratory diseases (CVRD) are two of the most common causes of death and disability in the United States, causing a collective 1.02 million deaths in 2010 representing 41% of the total deaths from all causes (IHME 2010; Murphy 2010). Exposure to particulate matter less than 2.5 microns (PM2.5) has been identified as a significant causal factor in the incidence of these diseases (EPA 2010; California Air Resource Board 2010; Evans et al 2012). The primary sources of PM2.5 emissions in the U.S. are industry, motor vehicles, agriculture, biomass and fuel combustion, and personal household sources, such as tobacco smoke (EPA 2009). Industry and agriculture are particularly responsible for supply chain emissions at very different scales.
The project aimed to develop a complex system ''causal'' model that provides robust estimates of the incidence of cardiovascular and respiratory diseases (CVRD) and their attribution to various sources of PM2.5 exposure at different scales in the natural and built environment (Fig. 1). The model utilizes local, regional and global human mobility models of population in addition to spatially explicit facility level PM2.5 emissions from key industries, mobile emissions, global emissions and personal household tobacco smoke to provide better estimates of population exposure to PM2.5. A non- linear concentration-response function adjusted for social-demographic confounding factors is used to enable more robust predictions of the incidence of CVRD attributed to different sources of exposure.

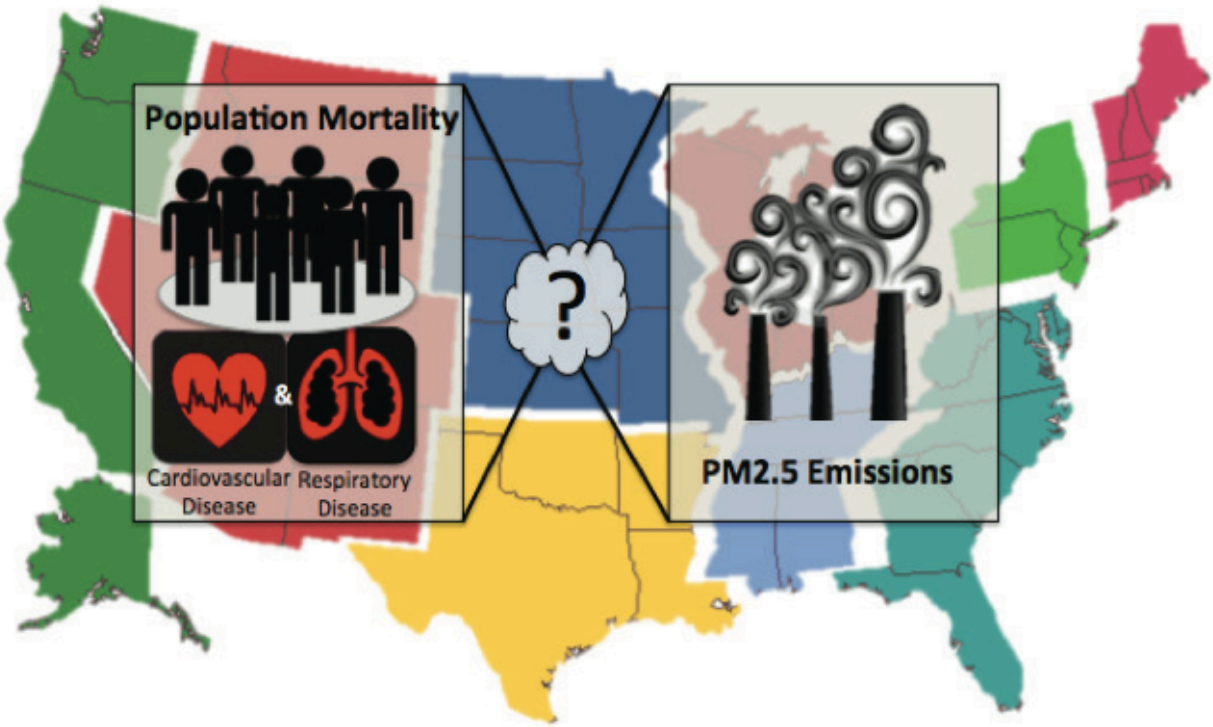

Figure 1. Framework of the study. The project aimed to develop a macroepidemiological model that provides robust estimates of the incidence of cardiovascular and respiratory diseases (CVRD) and their attribution to various sources of PM2.5 exposure at different scales in the natural and built environment

Work Completed

Please provide a summary of the work that was completed for the mini grant project.

The main outcome of the study was the creation of two models, a macro-epidemiological model and a mechanistic model, for the attribution of cardiovascular and respiratory diseases (CVRD) to different emission sources. The creation of the two models was also to compare the power of different models in analyzing and predicting population health outcomes that has relevance in epidemiology.

The following results have been found:

- CV/RD decreasing over time as well as PM2.5 for all macroregions considered in the USA.

- Scaling of CV/RD that is dependent on the macrogeographical area (both scaling validity and exponent); however PM2.5 is found as a robust predictor/driver of CV/RD as proven by the collapse test (Figs. 2, 4)

- Scaling of PM2.5 – dependent on different emission sources - with population density (Fig. 3)

For a given value in the incidence range, the likelihood (i.e., probability) of having that particular CVD incidence also varies greatly among the regions. For example, CVD incidence of 45000 has the highest probability (approximately 0.0003) in the Mountain region and relatively high probability (approximately 0.00007) in New England but extremely low probability (close to 0) in the other regions. The pdf of RD incidence indicates that number for RD incidence varies from one region to another as well. For instance, the approximate mean of RD incidence is 13000 in New England but greater than 45000 in South Atlantic. For a given value in the incidence range, the likelihood (i.e., probability) of having that particular RD incidence also varies among the regions. For example, RD incidence of 13000 has the highest probability (approximately 0.00058) in New England extremely

low probability (close to 0) in the rest regions. The pdf of PM2.5 concentration ($\mu g/m^3$) indicates that the Mountain region has the lowest PM2.5 concentration rate ($\mu g/m^3$), while the other regions have similar PM2.5 concentration rate.
In addition to the pdfs, the exceedance probabilities were also determined, which indicate the probability for which the incidence of the diseases and/or PM2.5 concentration for any given year will be exceeded.

Figure 2 indicates the existence of scaling relationship between PM2.5 actual exposures and incidence of CVD mortality in some regions, such as New England, Middle Atlantic, East North Central, West North Central, South Atlantic, and East South Central, while showing no scaling relationship in other regions, i.e., West South Central, Mountain, and Pacific. Figure 2 shows that scaling relationship between PM2.5 actual exposures and incidence of RD mortality only exists in regions of New England and Middle Atlantic, which is much fewer than that in the PM2.5-CVD case. All seven other regions are found not having scaling relationship between the two variables. The statistical relationship between PM2.5 actual exposures and incidence of CVD and RD mortality were re-plotted at the same horizontal and vertical magnitudes

A separate analysis was conducted another to investigate the relationship between population density and PM 2.5 emissions by sector for each county in the U.S. In order to determine whether a scaling relationship exists between the county-level population density and particular sources of PM 2.5 emissions, we used similar indicators (i.e., RMSE) as those conducted in the first part of the preliminary analysis, using population density as the independent variable and PM 2.5 emissions per sector as the dependent variables. The log-log relationship between population density and PM 2.5 emissions for the eight emission sectors are displayed in Figure 3. The approximate scaling relationship for each sector is notable, suggesting that the dependence of PM 2.5 emissions upon population density follows a power law scaling relationship. Not surprisingly, the selected emission sectors exhibit considerable differences in the fitting of the two variables. Obviously, population density shows strongest relationships with PM 2.5 emissions from dust and mobile sectors, which may be attributed to the close tie between human activities and the use of automobiles, and the potentially consequential increase in dust concentrations. All other six relationships show similar patterns between population density and PM2.5 emissions. Outliers toward the bottom of the six plots indicate that PM2.5 emissions from particular sectors do not exist in every county with population density. Some source sectors show a negative correlation between population density and PM 2.5 emissions. For instance, PM 2.5 emissions from agricultural and industry processing activities are observed to have a negative correlation with population density, which is not surprising because, agricultural activities in the U.S. are typically outside of heavily condensed population areas, and government regulations often mandate industry processing facilities to be zoned away from heavily populated areas due to city planning and health safety reasons. In the other four plots, population density shows a moderate relationship with PM2.5 emissions from commercial, electricity, fire, and combustion from fuel in industry sectors, respectively. This is somewhat expected because human activities are typically more directly involved in these sectors than in the agricultural and industry processing sectors. This analysis provides a more insightful understanding of potential human exposure to various sources of PM 2.5 emissions and could possibly serve as a reason to eliminate the emission sectors that are not strongly correlated with population density, as they may not be influential enough to be considered as causal factors of human exposure.

PM2.5 ~ Population^(1+H)
CV/RD ~ Population^(1+H)*

(alpha-2)=-1/(1+H)
H=Hurst's exponent

P(CV/RD) ~ cv/rd^(1-alpha)

h~1/(1+H)
h=Hack's exponent

Scaling

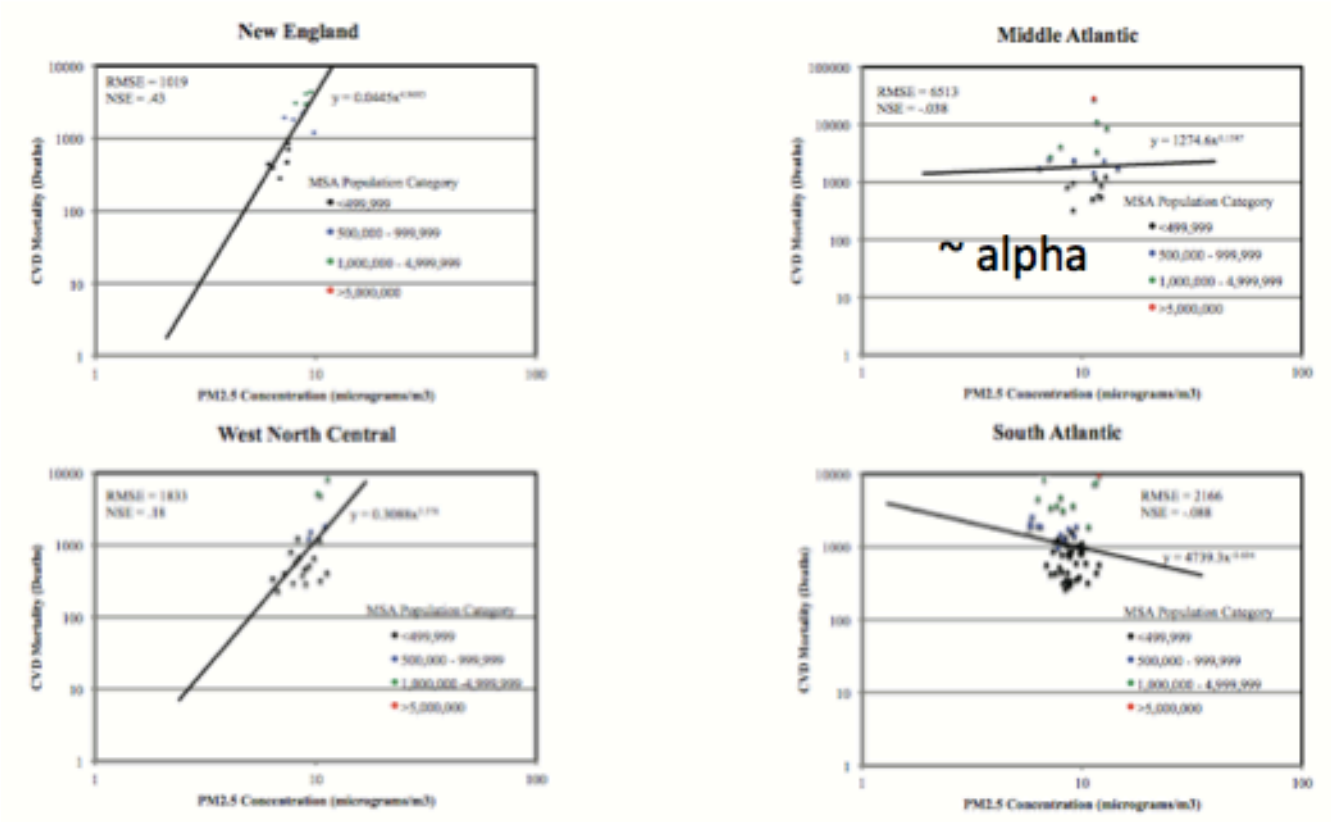

Figure 2. Scaling relationships between CVD and PM2.5 for all geographical regions as a function of MSA population classes. The scaling analytics is also reported. The Hurst exponent relates the exceedence probability distribution of CVD and RD with the population density that is a surrogate of PM2.5 via a power-law relationship.

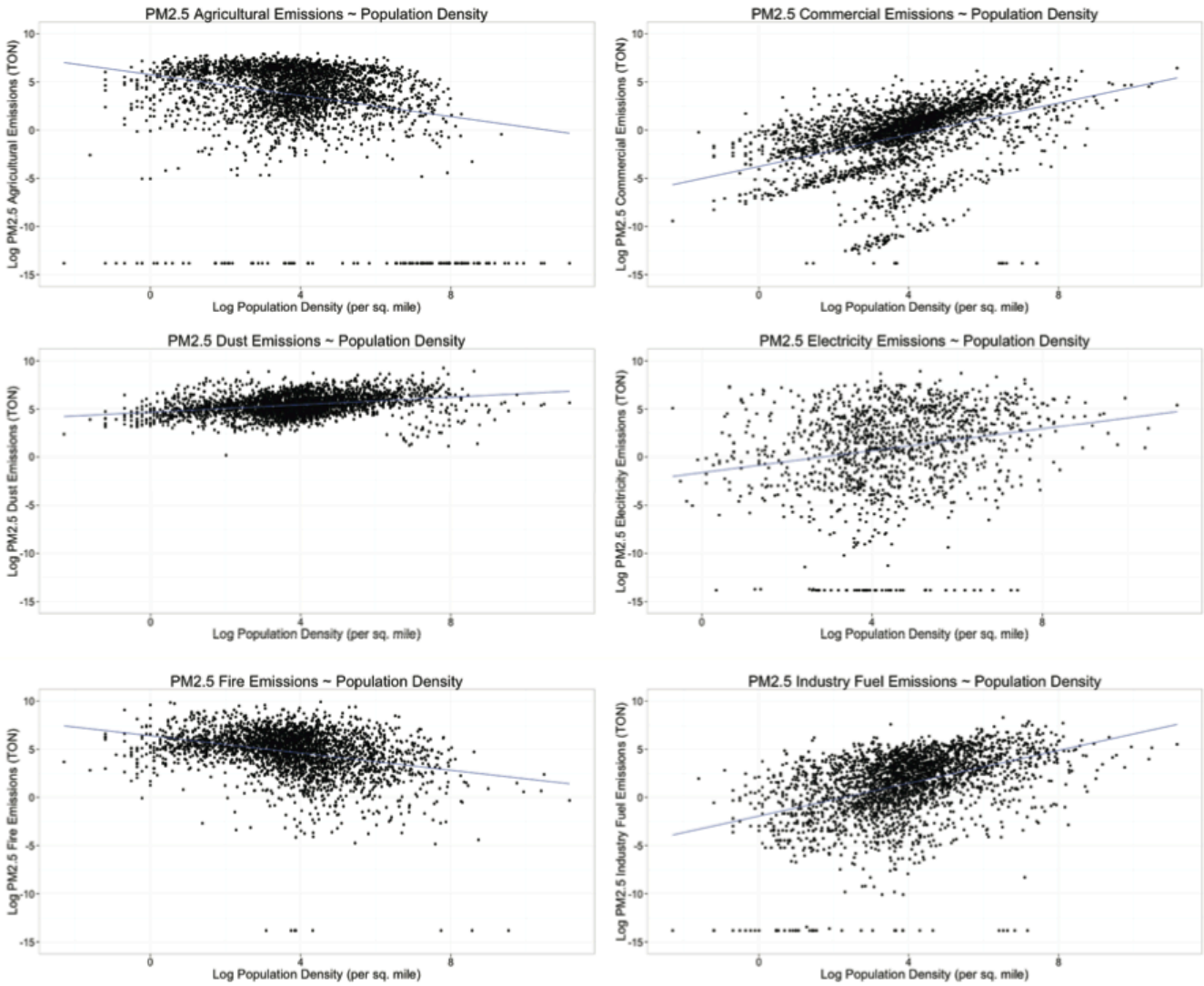

Figure 3. Attribution of PM2.5 to different emission sources as a function of population density.

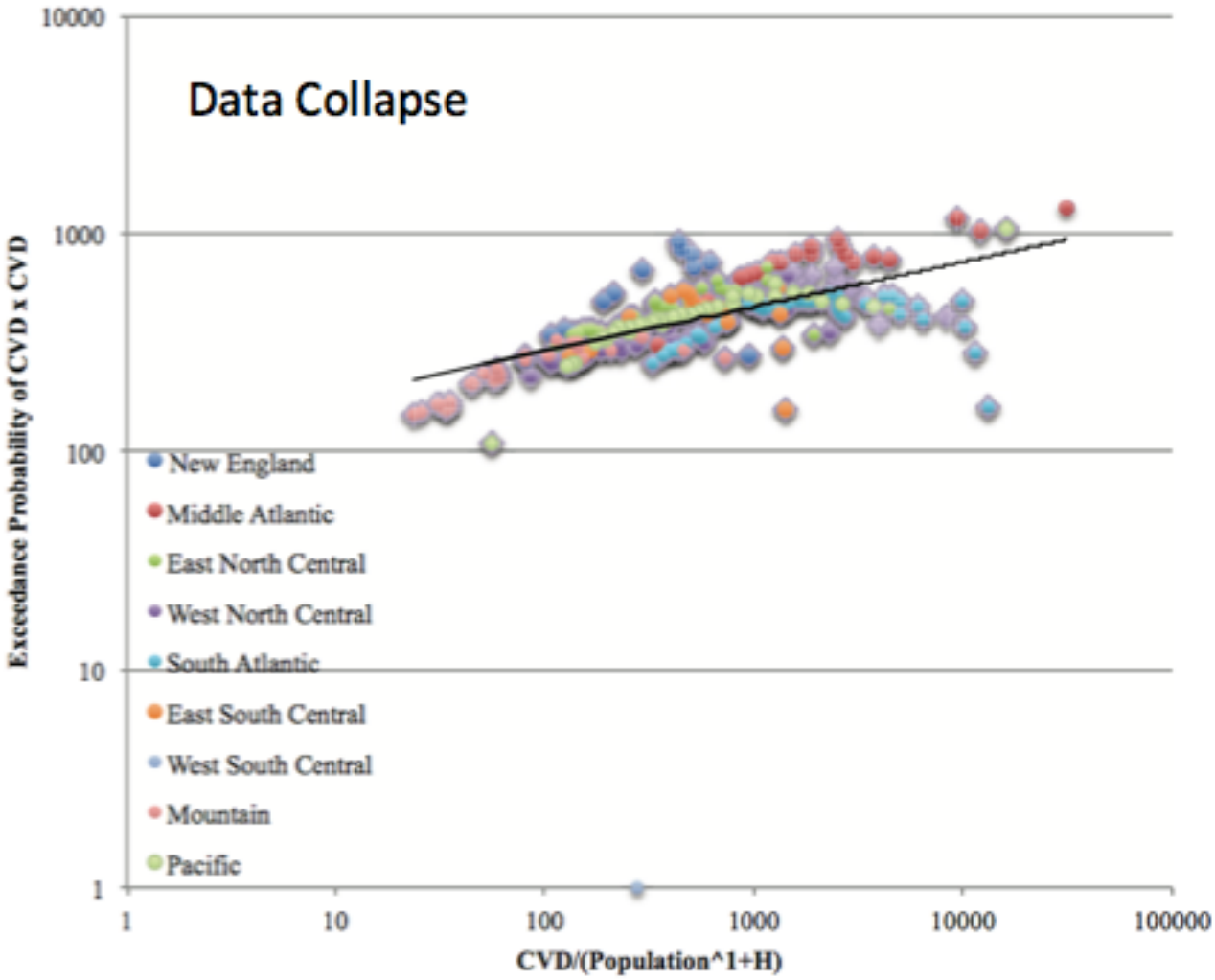

Figure 4. Collapse test verifying the scaling assumption. The exceedence probability of CV and RD for each macroregion is collapsed as a function of the population density and the Hurst exponent (see Fig. 2).

Partnerships & Collaborations

Please provide a summary of the project personnel, partnerships and collaborations that worked directly on the project or were started as a direct result of the mini grant project.

The project established a collaboration between the NISE and HumNat groups. Further effort will work on model integration where the NISE carbon emission models are going to be coupled to the epidemiological model of the HumNat group. This will lead to a great potential of collaboration with the industry and government entities.

Project Outcomes & Impacts

Please provide a summary of the outcomes and /or impacts of the mini grant project including future plans for the project.

Current publications

Convertino M, Timothy M. Smith, Rylie Pelton, Mo Li, Luyi Chen (2016), Macro-epidemiological Attribution of CVRD to PM2.5: A Scaling Approach, SERRA, submitted

Rylie Pelton, Mo Li, Luyi Chen, **Convertino** M (2015), Attributing Cardiovascular and Respiratory Disease Mortality to 10 Sources of PM2.5: What is to Blame? A Complex System Approach, Technical Report, HumNat Lab

Future studies

Further studies will consider more complex model than the macroepidemiological model from which we obtained the results. A key difference from the current study and other types of incidence and attribution studies is the characterization of the exposure estimate. While many studies use population- weighted concentration estimates and apply the estimates directly to the CRFs to determine incidences of health outcomes the envisioned future study will use more mechanistic methods, as the Stochastic Exposure and Dose Simulation model for Particulate Matter (SHEDS-PM), for estimating individual exposures at the county level, which will be further modified to consider inhalation rates to provide estimates of actual exposure. Because these actual inhaled exposure estimates concern a single individual in a population, they must be averaged across the population in the county and multiplied by the average body weight in the population in order to use these estimated inhaled exposures in the characterization of relative risks as the CRF is characterized for only population exposures. It is expected that using estimates of exposure that consider the concentrations that are actually inhaled, will provide better estimates of incidences than current estimates, which do not consider inhalation at the population level.