Alexander Kolovos and George Christakos A BME approach to ozone exposure spatiotemporal health impacts

Among the challenges posed for environmental health scientists and integrated assessment modellers are exposure analysis and mapping of spatiotemporal pollutants in relation to their health effects. A general method for studying these effects of pollutants distributed in space and time was recently introduced in [1]. It is well known that human exposure to O_3 can have acute and chronic health effects, see e.g. [2]. The proposed method was implemented using ozone (O_3) concentration levels over the eastern US. The data used were provided by the Aerometric Information Retrieval System (AIRS) maintained by the US Environmental Protection Agency (USEPA).

Although exposure refers to the airborne pollutant concentration at a specific point in space and time, it does not stand for the the pollutant amount that will affect an organ in which important damage will occur. The variable that accounts for this quantity is burden, and for our case a knowledge of O_3 burden is necessary to an unambiguous evaluation of health effects ([3]). Toxicokinetic (or pollutokinetic) equations are used to calculate the burden from exposure. These equations take into consideration any spatiotemporal exposure variations in addition to variabilities due to biological and physiological characteristics of the individual.

The existence of the aforementioned variabilities and the uncertainties led to the use of spatiotemporal random fields (S/TRF, [4]) in order to represent the exposure distribution and the biological variables. According to this notion, the S/TRF domain is a space/time continuum. A S/TRF realization would be, e.g., a specification of the O_3 exposure values at all space and time points. In this scheme randomness manifests itself as an ensemble of possible realizations of the exposure and biological variables distributions. A combination of the above along with the available knowledge bases and a sound epistemological framework constitute the Bayesian maximum entropy (BME) approach (BME; [4]) that has been used for this study.

Using a sound epistemological framework means that the empirical investigation of space and time as a whole needs to be backed up by: (i) A geometry that best describes space/time. If a space/time continuum is considered that involves Euclidean spatial coordinates $s = (s_1, s_2)$ in \mathbb{R}^2 and a temporal coordinate t along the time axis T, a space/time vector $\mathbf{p} = (s, t)$ should follow a suitable spatiotemporal metric structure which need not necessarily be Euclidean. In fact, the appropriate metric should be dictated by the knowledge base available for the proposed exposure S/TRF $E(\mathbf{p})$ varying withing the above continuum. (ii) A logical framework for integrating and processing various knowledge bases. More specifically, the total knowledge \mathcal{K} may include all kinds of valid knowledge available at a given moment and can be obtained by the competent scientist using effectively a scientific procedure. Two prime knowledge bases can be further distinguished within \mathcal{K} : The first is the general knowledge base \mathcal{G} , which may include different types of physical laws, logical relations, statements of fact, scientific theories, as well as hard and soft data. With respect to our study, the general knowledge G available about the O₃ distribution is that the distribution is characterized by nonhomogeneous spatial patterns, nonstationary temporal trends and random local fluctuations. This information allowed for a choice of a proper S/TRF model for the O₃ exposure. The second knowledge base is called specificatory $S \,.\, S$ refers to our knowledge about the specific situation and includes two main groups of data available: Hard data and soft data. The former are measurements obtained from real-time observation devices, experiments on human subjects and other sources, whereas the latter may represent varying levels of understanding of uncertain observations leading to either a direct estimation of the probabilities or their indirect estimation from accumulated experience. For the present study the specificatory knowledge S consisted of the O₃ data set provided by the AIRS-USEPA.

The O_3 data set includes 1-hour O_3 concentrations (in parts per million) from 1228 monitoring stations for an area that covers the eastern USA east of 95°W longitude and north of 25°N latitude. Note that the concentration values at the space/time points also stand for the exposure values for receptors located at these points. As described in [1], BME mapping provides us with O_3 exposure estimates at any point in space and time for which observations are not available. The average O_3 exposure E(p) over a day period (t=24h) was calculated for a few days in July 1995 for a geographical region that includes New York City and Philadelphia (Figure 1). The outcome honors the data at points where measurements were taken, and the estimation error variances at unsampled points indicated a very good accuracy of the maps produced. The 24h averaging may smooth potential high peaks in the 1-hour O₃ concentration profile, thus not making the study proper for a purpose such as control of compliance with certain ambient air quality standards. Yet, it is appropriate for the present work's biological indicator and health effect analysis. Spatial exposure maps like the ones shown in Figure 1 enable detection of daily-averaged O₃ exposure variations, as well as spatiotemporal exposure patterns of interest (e.g. trends) in health studies.

As mentioned earlier, toxicokinetic (or pollutokinetic) models are used to predict burden distributions in the human organs and tissues as a result of exposure to pollutants. Compartment models [5] have been developed, that analyze the transfer and transformation processes occuring after the body has been exposed to a pollutant. The sub-category of physiological compartment models [6] has already been used [7] for the study of O₃ health effects. A stochastic one-compartment pollutokinetic model was used for this study. Using this model and the daily-averaged O₃ maps of Figure 1, maps of the burden B(p) on a receptor at p = (s,t) were plotted for a few days in July 1995 for our region of interest. These are shown in Figure 2 and demonstrate the spatiotemporal variability of burden resulting from environmental and biological factors acting either separately or interacting with each other. Such maps can assist the study of several health risk related issues, such as the amount of burden on target organs that could have been prevented for a certain reduction of O₃ exposures.

While the burden maps refer to 'representative' individual receptors (say, of a specific cohort) there is also a need for an assessment of the absolute or relative impact of

exposure on the population as a whole. To this end, one needs to relate the burden maps to the population health damage. There exist many factors, such as exposure duration, activities, receptors' pre-existing health conditions, age groups, that are taken into consideration for the development of burden-health response models. These models relate the population health effect H(p) with the O₃ burden B(p) in a manner that structual nonlinearity is accounted for, as well as biological uncertainties (inter- and intra-subject) (see [1]). Once H(p) has been quantified, the use of health damage indicators can provide specific answers about the health damage on a community-wide basis.

In the present study the population damage indicator $\Psi_{n}(p)$ has been used, which is the average local health damage to the population of the region v(s) at time t due to the health response H(p) for a specific health effect. $\Psi_{\nu}(p)$ is a also a function of the population density of receptors in the neighborhood v(s). Its units are in number of receptors affected per km². Figure 3a shows a map of $\Psi_{\mu}(p)$ that comes as a result of a certain health response H(p). A variety of cohorts can be considered, leading to a multiplicity of $\Psi_{\nu}(p)$. For example, using a different parameter in the model that quantifies H(p), with respect to some cohort characteristics other than the ones considered to create Figure 3a, leads to a different mapping of $\Psi_{\mu}(\mathbf{p})$, as shown in Figure 3b. Another useful indicator that was defined was the dimensionless normalized local damage indicator $\psi_{\nu}(\mathbf{p})$. This stands for the ratio of $\Psi_{\nu}(\mathbf{p})$ over the average damage $\Psi_{V}(p)$ occurred at the global region V, of whose v(s) is a subregion. The $\Psi_{V}(p)$ maps can help with the detection of areas where exposure has the highest probability to cause adverse health effects at the local population, whereas maps of the indicator $\psi_{v}(\mathbf{p})$ show how much larger the health damage at a local area v(s) is expected to be than the average damage of the entire region V.

The study presented here exhibits important improvements over previous ones. Firstly, the exposure mapping method presented uses the general and powerful BME analysis. Moreover, toxicokinetics modelling was incorporated into spatiotemporal health effect analysis and mapping. In addition to that, nonlinear burden-response curves with random coefficients were considered. And it is also important that exposure, burden and health effects were considered from a holistic perspective. This approach can be considered as an investigation tool that may offer a useful stochastic description of human exposure and an important basis for further analysis.

References

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Figures



Figure 1. Daily averaged O₃ exposure maps (ppm) at a region of eastern US.



Figure 2. Maps of daily accumulated burden (ppm) on receptors at a region of eastern US.



Figure 3. Maps of the health damage indicator $\Psi_V(\mathbf{p})$ (number of receptors affected/km2) for two different population groups on July 20.