Efficient Large-scale Network-based Simulation of Disease Outbreaks

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OBJECTIVE

This paper describes biologically-based mathematical models and efficient methods for early epoch simulation of disease outbreaks and bioterror attacks.

BACKGROUND

The effectiveness of public health interventions during a disease outbreak depends on rapid, accurate characterization of the initial outbreak and spread of the pathogen. Computer-based simulation using mathematical models provides a means to characterize both and enables practitioners to test intervention strategies. While compartmental differential equation models can be used to represent epidemics, they are unsuitable for early time simulations (first few days) when a small number of people are infected (and even fewer symptomatic), nor are they capable of representing spatial disease spread. Numerous models for disease propagation have been explored, including national scale network models for influenza [2] and social network-based [1] and probabilistic [3] models for smallpox. To be useful in a public health context, a model for disease propagation should be efficient (e.g., simulating several weeks of real time in an hour) and flexible enough to simultaneously represent multiple diseases and attack scenarios.

METHODS

The proposed model for disease transmission tracks individuals as they move about a transit-based network [5]. It generates detailed predictions of disease spread on a city scale (similar to EpiSims [1]), including tracking the first few symptomatic people, for use in a Bayesian inverse problem context to determine the initial location, time, and size of a bioterror attack [4]. A person may acquire a disease from a contaminated location (e.g., anthrax) or from contact with an infected person (e.g., smallpox). At each network location, a differential equation model quantifies transmission between collocated people and the site. Inter-individual variability for in-host pathogenesis is specified by probability distributions for infectious doses and length of disease stages.

Runtime is reduced through both parallelism and model-reduction techniques. Distributing the population across multiple processors greatly reduces runtime. Simulation on only a subset of the population also reduces runtime; the reduced model is determined by social network analysis such that disease spread remains accurate. One model-reduction method clusters based on population connectivity and runs the simulation on a population sampled from the clusters. A drawback of this method is that the original network's graph properties are not completely conserved. Another method efficiently identifies before each simulation all people who could possibly become infected; the simulation is then run on this population subset. This method has the advantage of preserving the graph properties in the sub-network.

RESULTS

The model has been used to simulate a smallpox attack affecting two locations in a model of Portland, Oregon (Figure 1). Red and orange indicate individuals in contagious stages of the disease, whereas blue denotes those in earlier exposed or prodromal stages. For short time simulation of contact transmitted diseases, the second model-reduction method above has been shown to effectively reduce the problem size by more than two orders of magnitude.

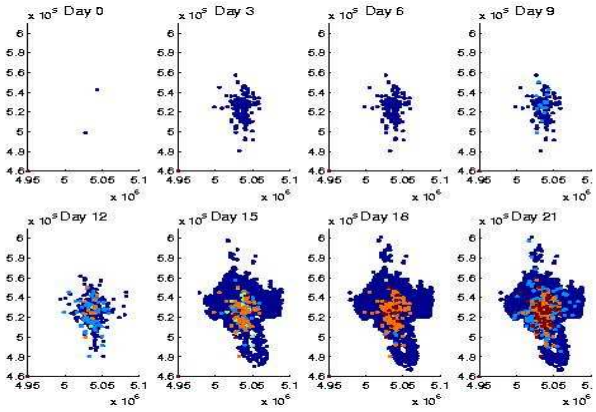

Figure 1: Geographic spread of smallpox in Portland, Oregon.

CONCLUSIONS

Through parallelism and model reduction, pathogen spread can be simulated in a timely manner. Fast simulations enable the use of the disease model in inverse problems to characterize the source of a disease outbreak. Once calibrated, it can predict disease spread on large time and spatial scales. The effect of varying disease characteristics and further improvements in computational efficiency are being explored.

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Sandia is a multiprogram laboratory operated by Sandia Corporation, a Lockheed Martin Company, for the United States Department of Energy's National Nuclear Security Administration under contract DE-AC04-94AL85000.