Model for Estimating Acute Health Impacts from Consumption of Contaminated Drinking Water

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Abstract: Disease transmission models predict the spread of disease over time through susceptible, infected, and recovered populations, and are commonly used to design public health intervention strategies. A modified disease model is linked to flow and transport models for water distribution systems in order to predict the health risks associated with use of contaminated water. The proposed framework provides information about the spatial and temporal distribution of health risks in distribution systems and is useful for understanding the vulnerability of drinking water systems to contamination events, as well as for designing public health and water utility strategies to reduce risks.

DOI: 10.1061/(ASCE)0733-9496(2006)132:4(293)

CE Database subject headings: Estimation; Public health; Potable water; Water quality.

Introduction

Contamination of drinking water distribution systems can result from cross-connections with nonpotable water, permeation and leaching of pipes, chemical reactions and microbial growth within pipes, or intentional acts of contamination. Hydraulic and water quality models can be used to model the fate and transport of contaminants within utility-specific distribution system networks (Rossman 2000; Uber et al. 2004a). Recently, new methods have been developed to allow for modeling of multiple interacting species in flow (Shang et al. 2004), for example, the full dynamics between chlorine and an organic contaminant. By linking flow and transport models to dynamic models for disease, a framework is presented for estimating the spatial distribution of health risks associated with ingestion of contaminated drinking water.

Contamination warning systems, sometimes referred to as early warning systems, have recently been proposed as a promising approach for reducing the risks associated with the intentional contamination of drinking water systems (USEPA 2005). Contamination warning systems use continuous online contaminant detectors or water quality sensors to detect potential contamination events and provide an early warning of potential health risks. The technology involved is early in its development, and costs are high; thus, there is a strong incentive to limit the number of sensors and locate them optimally. Most sensor location methods rely on spatial estimates of risk associated with potential contamination events [see, for example, Berry et al. (2005) and Uber et al. (2004b)]. The risk assessment approach described in this paper will provide a more comprehensive framework for estimating health effects associated with contamination events.

This framework is also useful for designing effective public health and water utility intervention strategies. By simulating the spatial and temporal health risks associated with consumption of contaminated drinking water, for instance, one can identify the locations of exposed populations in need of public health treatment. The same tools are useful for assessing the value of hydraulic control options for isolating or flushing contaminated water, as well as the potential for treating the water in situ. In addition, such tools should be useful for planning and preparing for contamination events and also for real-time planning of response actions.

Disease models have already been applied to waterborne disease outbreaks (defined as diseases that can be traced back to water by epidemiological evidence); see, for example, Eisenberg et al. (1998). The U.S. Centers for Disease Control and Prevention (CDC) along with the USEPA have collected data on waterborne disease outbreaks since 1971. In 2001–2002, 31 outbreaks were reported, causing illness among approximately 1,000 persons and resulting in seven deaths. Recent outbreaks in the United States were linked to *Cryptosporidium, Giardia, E. Coli, Salmonella, Campylobacter jejuni, Legionella*, and Noroviruses, among others (Blackburn et al. 2004).

The framework presented in this paper includes the capability of tracking waterborne disease outbreaks spatially. This is especially important because of the difficulty of linking disease outbreaks back to water sources. A CDC report found that reported water borne illnesses "probably represent only a small proportion of all illnesses associated with waterborne-disease agents" (CDC 1990). Most illnesses resulting from ingestion of chemicals are not collected by this system, nor are diseases resulting from long-term exposure to low levels of contaminants. Many small-scale outbreaks are probably not reported. Moreover, because of the difficulty in linking public health events to drinking water sources, the number of outbreaks reported is likely conservative.

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The modeling tools presented in this paper allow one to simulate both the contamination event in the water system and the resulting health impacts in a population, improving the capability of linking a public health event back to the water supply.

This paper links hydraulic models for water flow through distribution systems to models for estimating health impacts in order to predict the spread of disease over time in a population using contaminated water. The focus is on biological agents, such as bacteria and viruses, but some of the methodology would also be appropriate for chemical agents. In Section 2, the recommended approach for biological agents is examined, as well as the accepted risk assessment paradigm for chemicals, whereas Section 3 describes a disease transmission model in detail. In section 4 the infectivity rate that links the hydraulic models to the disease models is described in detail, and in Section 5 the common methods used to predict flow and transport in distribution systems are discussed. In Section 6 some additional assumptions and simplifications to the model are presented. Finally, the new framework is used to study a particular contamination example in Section 7. There are many potential applications for this framework; however, the focus of this paper is on developing the methodology.

Discussion of Methods for Quantifying Health Risks

Generally, attempts to quantify the risk associated with consuming contaminated water have used static models that determine the probability of individual illness based on a single exposure. This approach follows the accepted paradigm for chemical risk assessment. The dose and the dose-response curve—the amount of contaminant consumed and the probability of a given health response—are the main criteria used to determine health impacts. Such an approach has been used to estimate health risks associated with long-term exposure to low levels of toxic chemicals (Risk 1983).

The same information is important for assessing risks associated with exposure to viruses, bacteria, and protozoans. However, biological risk assessment requires the consideration of additional factors such as multiple infectivity paths (person to person, environment to person), possible secondary transmission paths (person to environment to person, environment to person to person), immunity to disease, microbial incubation periods, and the potential for asymptomatic carriers of disease. See Haas et al. (1999) for a complete treatment of this topic.

Recently, the use of dynamic disease transmission models has been introduced into the biological health risk assessment process (Eisenberg et al. 2002). Disease progression models predict the temporal spread of disease through subgroups of a population, such as susceptible, infected, and recovered subpopulations. While traditional risk assessment approaches use data-driven models, disease transmission models include physically based parameters that describe the disease process. Such models may also account for the population dynamics of pathogens within the host or the natural reservoir. Disease models have been used to assess health risks, to differentiate epidemics from endemic disease cycles, and to design treatment and control strategies for diseases, such as vaccination programs (Anderson and May 1991). Similar models have been used to estimate the spread of disease following a bioterrorism attack and to optimize public health intervention strategies (Barrett et al. 2005).

Dynamic Disease Model

The model used in this paper is derived from the general dynamic model proposed by Anderson and May (1991) and is similar to the model used by Chick et al. (2003). The model describes the spread of disease through a population of susceptible persons (S), infected but not symptomatic persons (I), diseased (infected and symptomatic) persons (D), recovered and immune persons (R), and those impacted fatally from disease (F). The disease can be transmitted person to person or from drinking water. The dynamic model is given by the following equations:

$$\frac{\partial S}{\partial t} = \gamma R - \lambda S \tag{1}$$

$$\frac{\partial I}{\partial t} = \lambda S - \sigma I \tag{2}$$

$$\frac{\partial D}{\partial t} = \sigma I - (\alpha + \nu) D \tag{3}$$

$$\frac{\partial R}{\partial t} = \nu D - \gamma R \tag{4}$$

$$\frac{\partial F}{\partial t} = \alpha D \tag{5}$$

where $S(x_i,t)$=number of susceptible persons at time t; $I(x_i,t)$=number of latently infected but not symptomatic persons at time t; $D(x_i,t)$=number of diseased (infected and symptomatic) at time t; $R(x_i,t)$=number of recovered and immune at time t; $F(x_i,t)$=number of fatalities due to disease at time t; and $\lambda(x_i,t)$=force of infection at time t and is discussed in more detail in Section 6.

The parameters in the model are:

- ν=per capita recovery rate (mean duration of illness is $1/\nu$);
- σ=rate at which hosts move from I to D (mean latency period is $1/\sigma$);
- α=per capita disease induced death rate; and
- γ=per capita rate of loss of immunity.

The disease model, Eqs. (1)–(5), is applied at each spatial node x_i in a distribution system model. Distribution system models are not continuous but rather discrete in space, thus, the subscript notation x_i. If the number of births in the population exactly balances the number of natural deaths (deaths not associated with exposure to the contaminant), the total population at each node is given by the constant N

$$N = S(x_i,t) + I(x_i,t) + D(x_i,t) + R(x_i,t) + F(x_i,t)$$

The populations can be summed in order to estimate the total number of infected, diseased, recovered, and fatally impacted in the total population of persons using water served by a specific drinking water system. Then, $\bar{N}=\bar{S}(t)+\bar{I}(t)+\bar{D}(t)+\bar{R}(t)+\bar{F}(t)$, where

$$\bar{N} = \sum_{i=1}^{i=N} N(x_i,t)$$

To solve the model given by Eqs. (1)–(5), appropriate initial conditions must be specified.

Infectivity Rate

In the disease model, Eqs. (1)–(5), λ is the force of infection or, more specifically, the per capita rate of acquisition of infection. In general, for any route of transmission, λ can be written as the product of the rate of exposure to the pathogen and the probability of infection given that exposure. In this paper, infection can be transmitted in two ways, from close person to person contact, or from ingestion of contaminated drinking water. Therefore, let $\lambda=\lambda_P+\lambda_W$ where the subscripts P and W refer to transmission from people or water. For an infectious disease transmitted person to person, λ_P is assumed to have the form

$$\lambda_P(x_i,t) = \beta(I(x_i,t) + D(x_i,t)) \tag{6}$$

where β=transmission parameter dependent on the disease-inducing organism and many environmental and societal factors. For instance, β may depend on personal hygiene, a person's age, and the seasonal variability in microbial behavior. In this form, the disease can be transmitted between persons near to each other spatially (i.e., located at Node x_i). Note that Eq. (6) could be modified to reflect the mobility of a population that interacts with people some distance away from Node x_i. Note also that λ_P may vary significantly from node to node because the population density of infected persons varies spatially as well as the likelihood of exposure.

The infectivity rate resulting from the consumption of contaminated drinking water is related to the amount of water consumed and the amount of contaminant in the water. Let $d(x_i,t)$ be the cumulative dose of a contaminant received by the population at Node x_i and let $r(x_i,t)$ be the corresponding probability of infection given dose d. The dose d can be calculated from flow and transport simulations, as described in the next section. The probability of infection r describes the percent of the population responding to a given dose, which is essentially a dose-response relationship as a function of space and time, $r(x_i,t)=r(d(x_i,t))$. The infectivity rate can be modeled as

$$\Lambda_W \equiv \frac{dr}{dt}\frac{S_0}{S} = \frac{\partial r}{\partial d}\frac{\partial d}{\partial t}\frac{S_0}{S} \text{ or } r(x_i,t) = \int_0^t \lambda_W(x_i,\tau)\frac{S(\tau)}{S_0}d\tau \tag{7}$$

where $\partial d/\partial t$=rate of exposure to the pathogen; and $\partial x/\partial d$=probability of infection given that exposure. $S_0=S(x_i,0)$=total number of customers at Node x_i that may be susceptible to infection. Therefore, λ_W at time t is the rate of new infections rS_0 at time t per total number of susceptibles S at time t. This formulation of λ_W is a generalization of that used by Chick et al. (2001).

For many contaminants, dose-response curves are available in the literature that were developed by matching experimental data with an understanding of disease kinetics in the human body. A dose-response curve generally has a sigmoidal shape, and an example of one is given by the curve shown in Fig. 1. Along the horizontal axis is the dose in number of organisms, and along the vertical axis is the percent of persons expected to become infected by the disease at a specific dose. Note that this dose-response curve corresponds to an ID_{50} dose (the dose at which 50% of the population would be infected) of 100,000 organisms.

Exposure to contaminated drinking water is possible from several routes, including ingestion, inhalation, and dermal contact. Ingestion is the focus of this paper; however, the model can be modified to incorporate other exposure routes as well. The cumulative dose of a contaminant ingested by the population at x_i at time t is calculated according to

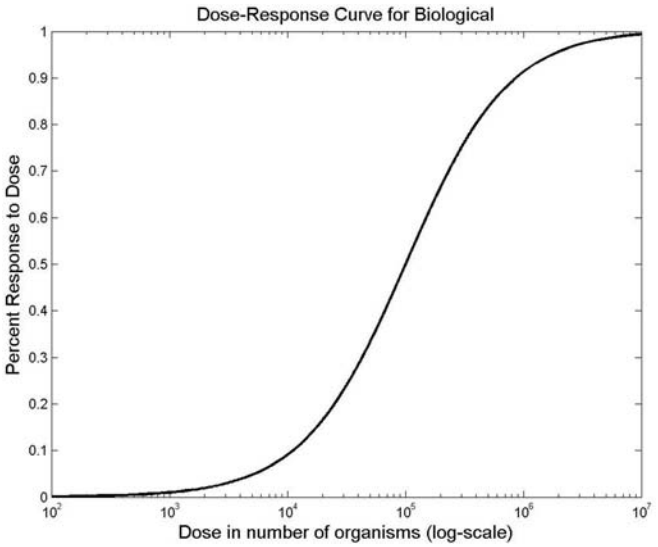

Fig. 1. Response curve as function of dose

$$d(x_i,t) = \int_0^t W(x_i,\tau)P_W(x_i,\tau)V_W d\tau \tag{8}$$

where d is measured in number or mass units; W=pathogen concentration in water; P_W=probability of consuming water at time t; and V_W=volumetric rate of water consumption, for example, 2 L/day.

Link to Hydraulic Models

Several disease transmission models have been developed to model transmission from drinking water sources. Eisenberg et al.(1998) studied a waterborne *Cryptosporidium* outbreak in 1993. In another example, Codeco (2000) studied endemic cholera and included in the model an equation for the population of *V. cholerae* in aquatic reservoirs. Cholera was transmitted through ingestion of untreated water, and infected persons shed pathogens back into the water reservoir. The *V. cholera* population, then, increased with growth and addition through shedding and decreased by a natural death rate. In another example, Colford et al. (2003) considered exposure to pathogens resulting from the application of biosolids to agricultural fields. Pathogens shed from infected persons accumulated in the biosolids, to which persons were exposed through inhalation or ingestion of surface water, groundwater, soil, or plants. This model included growth and death processes for the pathogens as well as increased concentrations due to human shedding of pathogens into sewage.

For all of the aforementioned applications of disease transmission models, the spatial distribution of the affected populations was not necessary to gain an understanding of an outbreak or to design public health interventions. However, to design a spatial network of sensors to detect contamination events, spatial information about the distribution of disease-causing agents within a pipe network and the resulting health impacts is required. In the rest of this paper, therefore, the disease model is applied at spatial locations to track the number of susceptible, infected, diseased, recovered, and fatally impacted at each location. The sum of these populations over all the spatial locations corresponds to the total number of affected persons served by the drinking water system.

The commonly used models for flow and transport in distribution systems will be combined with the disease model to predict the spatial distribution of health effects.

In drinking water distribution systems, the direction and magnitude of flow is determined by time-varying demands and the complex, looped geometry of the pipe network. Hydraulic models that include pipe lengths and diameters, pipe connectivity, and network operations are needed in order to predict time-dependent average water velocity in pipes. Most hydraulic models solve the flow equations in a pipe network by conserving mass at the pipe ends (nodes or junctions) and conserving energy in the pipes. EPANET (Rossman 2000) is a publicly available software package for simulating flow through pipes and forms the basis for many other commercially available software packages. For a complete treatment of pipe hydraulics, including the specific mass and energy balance equations in pipe networks, see Walski et al. (2003).

Using network hydraulic models, concentrations of chemicals or pathogens can be estimated spatially and temporally. Pathogens can be assumed to flow with the water in distribution system pipes, react with the chlorine residual, adhere to biofilms on pipe surfaces, and grow and decay according to natural processes. A general one-dimensional model for pathogen fate and transport in a distribution system is as follows:

$$\frac{\partial W}{\partial t} + \nu(x,t)\frac{\partial W}{\partial x} = f_1(W) + (b-a)W - g_1(W,C) \tag{9}$$

where $W = W(x,t)$ = concentration of the contaminant in the water. The concentration is advected with the average water velocity $v(x,t)$. Molecular diffusion is neglected, because mixing by turbulent flows is dominant in distribution system mains. The microbial concentration grows with rate b, dies with rate a, increases according to the source term f_1, and reacts with chlorine C, according to the reaction function g_1. Note that b is assumed to be a constant, but in reality may depend on such factors as nutrient and substrate concentrations, temperature, and pH. The constant a depends on natural death rates of the pathogen. The source term f_1 represents the addition of the pathogen to the water from human sources, such as shedding or contamination. If the reaction dynamics with chlorine or other disinfectants are known, the chlorine concentration can also be modeled by

$$\frac{\partial C}{\partial t} + \nu(x,t)\frac{\partial C}{\partial x} = f_2(C) - g_2(W,C) \tag{10}$$

In Eq. (10), the chlorine concentration increases with the source term f_2 and decreases as it reacts with the microbial agent and other organic and inorganic compounds according to the reaction term g_2. Eqs. (9) and (10) describe conceptually how pathogen-chlorine interactions could be modeled within a pipe network to support the spatio-temporal estimation of acute health impacts. For an example, see Propato and Uber (2004).

Assumptions and Model Simplifications

In this section, several assumptions are made in order to simplify the disease model and examine its basic dynamics in more detail. First, if the pathogen is not communicable but is transmitted only via the drinking water, then $\lambda = \lambda_W$. Second, if a person who has recovered from disease cannot reenter the susceptible state, then $\gamma = 0$ (i.e., after infection a person either gains permanent immunity or dies). Third, it is assumed that the death rate from the disease is proportional to the recovery rate, $\nu\theta = \alpha$, meaning that those in the disease state move at the same rate to both categories. The final assumption is related to the transition time between disease states. In the disease model, Eqs. (1)–(5), the transition time is exponentially distributed with a mean time between infected and diseased states of $1/\sigma$ and a mean time between the diseased and fatal states of $1/\nu$. If both of these transition times are assumed to always be constant, the model can be rewritten as a time delay model. For a discussion on arbitrary transition times in disease models, see Van den Driessche (2002). The new model is

$$\frac{\partial S}{\partial t} = -S_0(x_i)\frac{\partial r}{\partial t}\bigg|_{x_i,t} \tag{11}$$

$$\frac{\partial I}{\partial t} = S_0(x_i)\frac{\partial r}{\partial t}\bigg|_{x_i,t} - S_0(x_i)\frac{\partial r}{\partial t}\bigg|_{x_i,t-1/\sigma} \tag{12}$$

$$\frac{\partial D}{\partial t} = S_0(x_i)\frac{\partial r}{\partial t}\bigg|_{x_i,t-1/\sigma} - S_0(x_i)\frac{\partial r}{\partial t}\bigg|_{x_i,t-1/\nu} \tag{13}$$

$$\frac{\partial F}{\partial t} = \theta S_0(x_i)\frac{\partial r}{\partial t}\bigg|_{x_i,t-1/\nu} \tag{14}$$

This model can be solved exactly to obtain the following solutions:

$$S(x_i,t) = S_0(x)(1 - r(x_i,t))$$

$$I(x_i,t) = \begin{cases} S_0(x)r(x_i,t) & \text{if } 0 < t < t_\sigma \\ S_0(x)(r(x_i,t) - r(x_i,t-t_\sigma)) & \text{if } t > t_\sigma \end{cases}$$

$$D(x_i,t) = \begin{cases} 0 & \text{if } 0 < t < t_\sigma \\ S_0(x_i)r(x_i,t-t_\sigma) & \text{if } t_\sigma < t < t_\nu \\ S_0(x)(r(x_i,t-t_\sigma) - r(x_i,t-t_\nu)) & \text{if } t > t_\nu \end{cases}$$

$$F(x_i,t) = \begin{cases} 0 & \text{if } 0 < t < t_\nu \\ \theta S_0(x_i)(r(x_i,t-t_\nu)) & \text{if } t > t_\nu \end{cases}$$

where $t_\sigma = 1/\sigma$ = latency period; and $t_\nu = 1/\nu$ = disease duration. In this model, the number of susceptible persons decays linearly with the response function r. (Note, however, that r is not a constant but varies in space and time.) In turn, the number of infected persons increases linearly with rate r until after the latency period t_σ, when infected persons start showing symptoms and move into the diseased stage. After the latency period, the number of diseased persons grows linearly until after the duration of the illness, at which time a fraction of the diseased persons moves into the fatality stage. This simple model is solved for a specific example in the next section.

Case Study: Biological Contamination Event

An example case study is presented in which the health impacts of a contamination event are calculated according to the disease model, and the times for effective intervention are considered as well as the benefit of intervention. In this example, a large quantity of a pathogen is introduced at one particular location in a specific drinking water distribution system. The operations and hydraulics of this system are known, and the water velocity, $v(x_i,t)$, pressure head, $h(x_i,t)$, and pathogen concentration in the

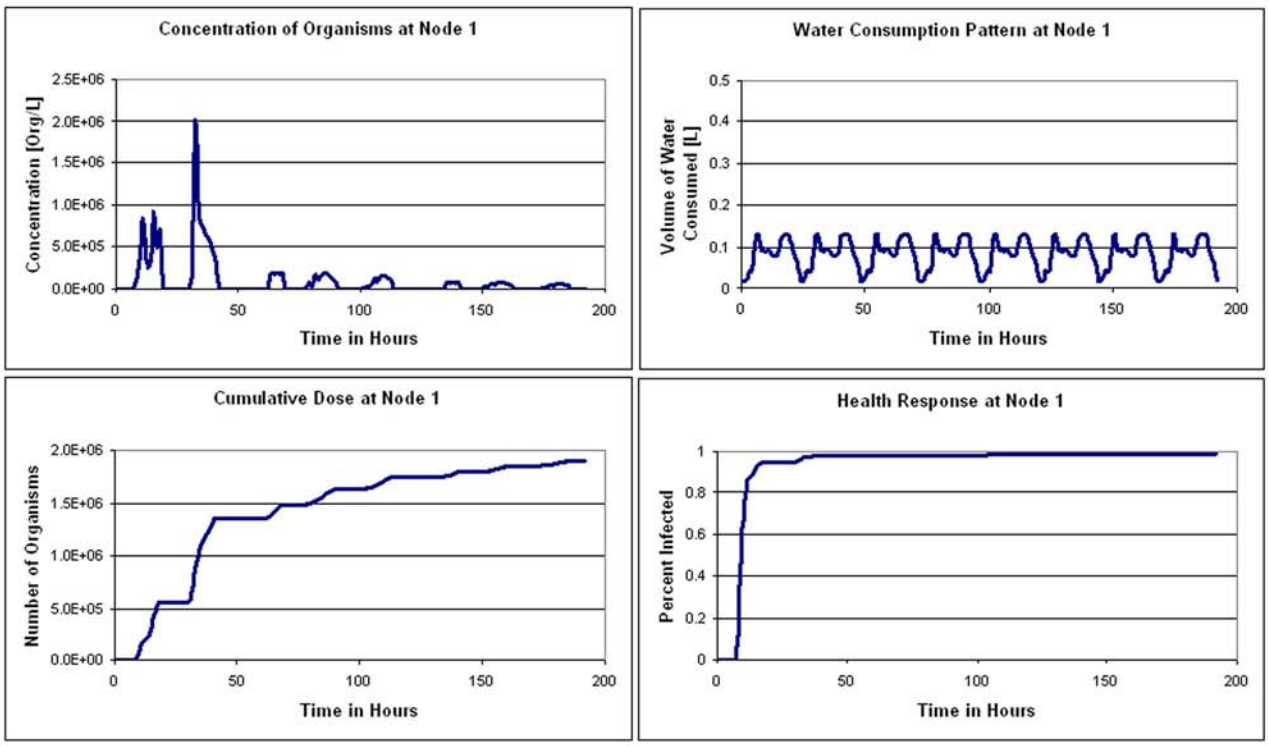

Fig. 2. At one node, concentration of contaminant, volume of water consumed, cumulative dose ingested, and response curve over time

water, $W(x_i,t)$, are predicted by EPANET. The population served by the drinking water system is approximately 200,000. The drinking water system covers approximately 100 mi^2, and draws water equally from surface water and groundwater sources. The hydraulic model includes approximately 3,000 nodes, 40 groundwater wells, and 30 storage tanks. At each node in the model, $x_n, n=1\ldots N$, a population of tens to hundreds is served water. The disease model, Eqs. (1)–(5), is applied at each node to predict the spread of disease among the population consuming water at that node.

The pathogen is a hypothetical biological agent with a latency period of 1 week ($\sigma=1/168$), a total disease duration of 2 weeks ($\nu=1/336$), and an untreated fatality rate of 30%. In this example, exposure to the contaminant occurs only through the ingestion route. Each person consumes 2 L/day of water. The probability that an individual at Node x_i consumes water at time t is assumed to be proportional to the ratio of the demand q at time t to the average demand over the time period T

$$P_W(x_n,t)=\frac{q(x_i,t)}{\frac{1}{T}\int_0^T q(x_i,\tau)d\tau} \tag{15}$$

This assumption reflects the average usage patterns of all the persons being served at a particular node. The dose-response curve predicts the probability of infection based on a given dose and is given by Fig. 1. Note that the dose-response curve corresponds to an ID_{50} dose of 100,000 organisms.

The initial conditions of the model are $S(x_i,0)=S_0$, $I(x_i,0)=0$, $D(x_i,0)=0$, $R(x_i,0)=0$, and $F(x_i,0)=0$, for all $i=1\ldots N$, where S_0=total population of persons served by the water system through node x_i. These conditions correspond to the case in which all persons are susceptible to the disease but no one is currently infected or has immunity.

Fig. 2 shows how one particular node in the system (downstream of the introduction location) would experience this contamination event. The figure shows four plots: (1) the concentration of contaminant that passes by the consumers at one node; (2) the water consumption patterns of consumers; (3) the cumulative dose received by consumers; and (4) the response function (cumulative percent of population experiencing a health response) over time. Note that the concentration profile is very complicated, because the spatial location is under the influence of a nearby tank. The contaminant is drawn inside the tank as the tank fills and is transported out as the tank drains.

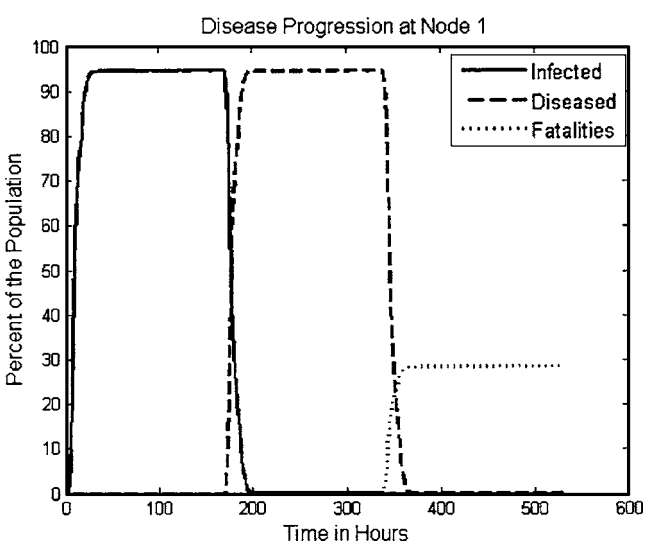

Fig. 3. Infected, diseased, and fatally impacted populations over time at one location in distribution system.

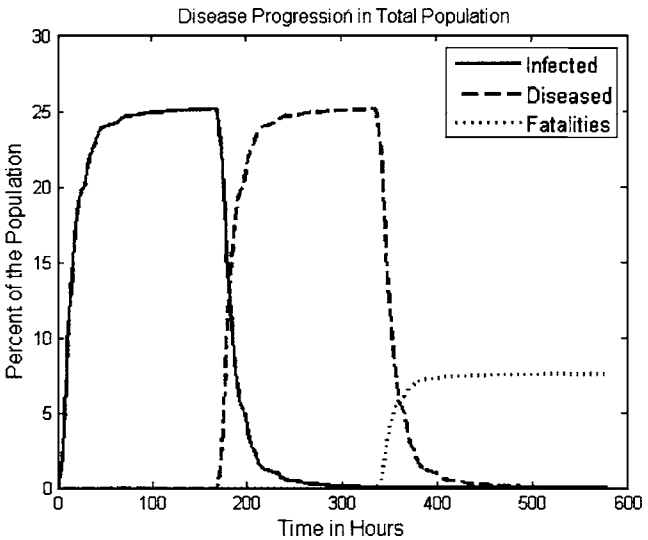

Fig. 4. Disease progression over time in total population

Fig. 3 shows the disease progression through the population at the same node shown in Fig. 2. The four curves show the number of susceptible persons, the number of infected persons, the number of diseased persons, and the number fatally impacted. The slope of the infections curve is directly related to the infectivity rate or the response function r, which encapsulates all the information about the hydraulics of the contamination event. The susceptible population quickly becomes infected and drops off to a very small asymptotic number. The infected population grows rapidly, sustains itself as the disease is latent (for one week), and then drops quickly as the infected persons transition into the diseased stage. Similarly, the diseased (symptomatic) population grows rapidly, sustains itself for the duration of the illness (one week), then a proportion of the diseased population recovers, while the remaining die. A similar set of curves could be drawn for each of the approximately 2,000 nodes in the network; however, each set of curves would be unique depending on the proximity to the location of contaminant introduction and the flow and transport dynamics near the node.

Fig. 4 shows the disease progression throughout the entire population (summed over all the nodes). Over the entire population served by the water system, a total of 25% of the population became infected after consuming contaminated water. It is interesting to note that the shape of these curves can change dramatically with different parameters. For some diseases, the latency period and disease duration are not equal; therefore, the I and D curves have different shapes. Figs. 5–7 show how the cumulative infections vary with three parameters: (1) the time of day of contaminant introduction; (2) the volume of contaminant introduced; and (3) the slope of the dose-response curve. In this example, the number of infections changes with the time of day of contaminant introduction, through the change is less than 1% of the total population. The number of infections increases with the volume of contaminant introduced, though it is not a linear relationship. Finally, as the slope of the dose-response curve increases and the curve steepens, the number of infections increases. Given that the data used to generate dose-response curves is often sparse and sometimes conflicting, this represents a source of great uncertainty in the model.

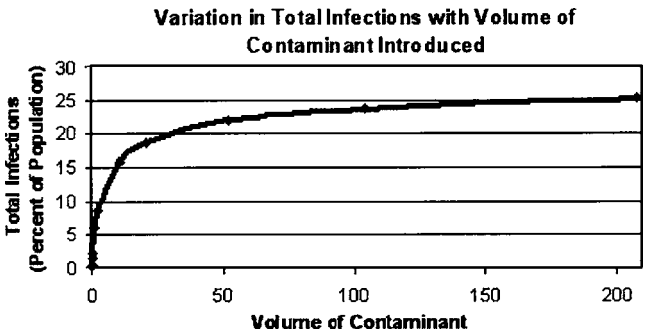

Fig. 6. Sensitivity of infections to volume of contaminant introduced

Information from Fig. 4 could be used by decision makers to plan public health and utility intervention strategies. In this case, the biological agent has a one-week latency period during which people would not yet be symptomatic or aware of the illness. This is a long period, during which a drinking water contamination warning system could provide the first detection of the incident. Following detection of a contamination incident, the utility could identify the contaminant through laboratory analysis and provide information to the public health sector about which neighborhoods were likely exposed to the specific contaminant, thereby informing the public health process. The data in Fig. 4 also shows, however, that the utility would need to detect and respond very rapidly in order to *prevent* exposure. Indeed, within 20 h of the contamination event, more than 50% of the exposures have already occurred. The modeling framework can also be used to compare the costs and benefits of various intervention strategies.

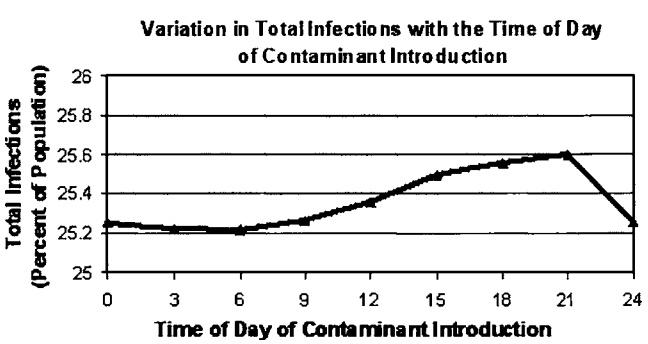

Fig. 5. Sensitivity of infections to time of day of contaminant introduction

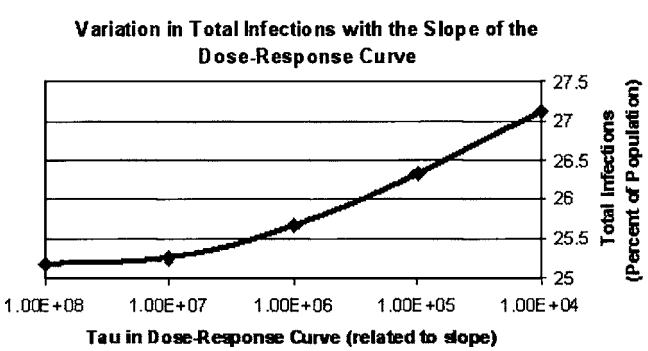

Fig. 7. Sensitivity of infections to slope of dose-response curve

Conclusions

This paper presents a new framework for estimating the spatial and temporal distribution of health impacts resulting from ingestion of contaminated drinking water. The example in this paper shows that the method is not restricted to small problems but can be applied even to large drinking water systems. Moreover, the method is flexible enough to accommodate most types of diseases that could be transmitted through water. The model could be extended to incorporate exposure to contaminated water through dermal and inhalation routes.

Though the focus of this paper is on describing the models and methods in detail, there are many important and useful applications that can be studied in future papers. This framework can be applied to both accidental and intentional contamination scenarios. Given the necessary parameter values for the health impacts of contaminants, the framework could be used to estimate the potential health risks of accidental backflows and intrusion events. Combined with flow information calculated in EPANET, the economic impacts of contamination events could be estimated (including public health costs and water utility cleanup and recovery costs). In addition, the public health costs and benefits of control options such as flushing and superchlorination could be examined.

In understanding the threat of intentional contamination of drinking water, this framework provides several useful tools. First, the number of infections or fatalities could be used as a metric in determining the optimal number and location of contamination warning system sensors (Berry et al. 2005; Uber et al. 2004b). Many methods for locating sensors attempt to minimize quantities such as the volume of contaminated water; however, the number of infections may be a more accurate reflection of public risk. In addition, the disease model could allow decision makers to determine the contaminant-specific time available for effective public health intervention strategies, such as vaccination, treatment, and "Do Not Drink" or "Do Not Use" orders. Finally, given the spatial estimates of health risk, decision makers could identify and prioritize populations and regions in the most urgent need of public health intervention.

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