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Disease and disaster: Optimal deployment of epidemic control facilities in a spatially heterogeneous population with changing behaviour

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HIGHLIGHTS

- Epidemics of environmentally transmitted diseases may be minimised by the deployment of clinics and decontamination facilities.
- Where control facilities do not affect host behaviour, they should be placed where they may benefit most people.
- If infected people change their behaviour to seek treatment then the deployment of facilities offering treatment can lead to complex effects.
- Behavioural changes in response to control facilities can lead to critical facility numbers at which there is a radical change in the optimal configuration.

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ABSTRACT

Epidemics of water-borne infections often follow natural disasters and extreme weather events that disrupt water management processes. The impact of such epidemics may be reduced by deployment of transmission control facilities such as clinics or decontamination plants. Here we use a relatively simple mathematical model to examine how demographic and environmental heterogeneities, population behaviour, and behavioural change in response to the provision of facilities, combine to determine the optimal configurations of limited numbers of facilities to reduce epidemic size, and endemic prevalence. We show that, if the presence of control facilities does not affect behaviour, a good general rule for responsive deployment to minimise epidemic size is to place them in exactly the locations where they will directly benefit the most people. However, if infected people change their behaviour to seek out treatment then the deployment of facilities offering treatment can lead to complex effects that are difficult to foresee. So careful mathematical analysis is the only way to get a handle on the optimal deployment. Behavioural changes in response to control facilities can also lead to critical facility numbers at which there is a radical change in the optimal configuration. So sequential improvement of a control strategy by adding facilities to an existing optimal configuration does not always produce another optimal configuration. We also show that the pre-emptive deployment of control facilities has conflicting effects. The configurations that minimise endemic prevalence are very different to those that minimise epidemic size. So cost-benefit analysis of strategies to manage endemic prevalence must factor in the frequency of extreme weather events and natural disasters.

1. Introduction

Epidemics of water-borne infections, such as cholera and other diarrhoeal diseases, often follow flooding and other natural disasters when drinking water is contaminated and sewerage management is disrupted. It is mostly developing countries that are affected (Bouzid et al., 2013; Tatsuya Katsumata et al., 1998; Vollaard et al., 2004; Watson et al., 2007; WHO, 2006; Date et al., 2011; Mike Ahern et al., 2005; Noji, 2000; WHO, 2006). The World Health Organisation (WHO) estimates that *Vibrio cholerae* alone causes approximately five million cases each year, leading to 120,000 deaths (WHO, 2014). Here we use a mathematical model to examine how demographic and environmental heterogeneities can be exploited to manage such epidemics as effectively as possible given limited resources.

Cholera is an environmentally transmitted infection. Susceptible individuals can be infected by ingestion of water or food contaminated with *V. cholerae*. Infected individuals shed bacteria, further contaminating the environment and perpetuating the transmission cycle. Five main control measures have been shown to limit or break this cycle: treatment of infected individuals, vaccination of susceptible individuals, provision of clean water, provision of sanitation, and environmental decontamination.

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Treatment of infected individuals with oral rehydration salts or antibiotics reduces the duration of infection and intensity of shedding. Vaccination of susceptible individuals provides broad but waning immunity to infection. Provision of clean water reduces contact between susceptible individuals and the contaminated environment. Provision of effective sanitation reduces the proportion of bacteria shed by infectious individuals that enter the environment. Decontamination reduces the lifespan of bacteria in the environment (Andrews and Basu, 2011; Mwasa and Jean, 2011; Neilan et al., 2010; Eisenberg et al., 2013; Mukandavire et al., 2013; Tuite and Tien, 2011; Ochoche, 2013). Several mathematical modelling studies have examined the effects of these control measures on cholera epidemics. The epidemic risk in disease-free populations has been assessed using variants of the basic reproduction number. It has been shown that the risk is a product of social and economic factors with critical parameter values and population susceptibility dictating the possible epidemic behaviour. Sanitation affects the influence of the environmental reservoir on the risk (Codeço Claudia, 2001). The epidemic risk may be reduced most effectively by applying control strategies concurrently, rather than individually (Andrews and Basu, 2011; Mwasa and Jean, 2011), and targeting particular groups in heterogeneous populations (Eisenberg et al., 2013). Assessment of the endemic disease burden has shown that decontamination can be an effective control measure but may be insufficient to eradicate cholera if shed and contact rates are high (Ochoche, 2013). Assessment of the disease burden of a single epidemic has aimed to quantify the incidence reduction achievable through the provision of clean water, vaccination or antibiotics (Andrews and Basu, 2011). It has been shown that the ideal combination of control strategies depends on characteristics such as the ratio of asymptomatic to symptomatic cases, the average recovery rate and the duration of immunity (Neilan et al., 2010).

Mathematical modelling studies have also examined how cholera epidemics are affected by spatial structure and other heterogeneities in the population or environment. It has been shown that heterogeneities in clean water provision and sanitation affect outbreak severity at local and global scales (Lorenzo Mari et al., 2012; Njagarah and Nyabadza, 2014) and models with spatial heterogeneity predict the development of cholera epidemics better than homogeneous models (Lorenzo Mari et al., 2015). Models indicate that the hydrological network topology strongly affects the speed of the epidemic wave front as the disease propagates (Bertuzzo et al., 2007, 2010), and can result in transmission bottlenecks (Shuai and Driessche, 2014; Shuai et al., 2013). Human migration has been shown to be important for inter-catchment bacterial transport (Lorenzo Mari et al., 2012) and determining how best to allocate health care resources (Andrea Rinaldo et al., 2012). Here we contribute to understanding how human behaviour and population structures combine to influence the management of cholera by examining the effects of human movement, city structure and subpopulation heterogeneities on the optimal deployment of control facilities in endemic and epidemic settings. We model disease transmission in a large city with a basic spatial structure that accommodates heterogeneities in population density or clean water provision. We consider an epidemic associated with a natural disaster such as a flood and examine where, with respect to the heterogeneities, a limited number of control facilities should be deployed to minimise the number of infections. We show that the optimal distribution of control facilities depends on whether disease is absent or endemic before the perturbation, and what facilities are available. We also show that, when disease is endemic, the optimal pre-emptive deployment of control facilities to reduce the lifetime infection risk

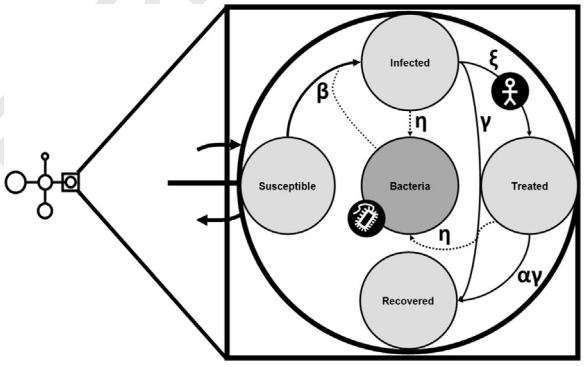

Fig. 1. Model structure and interactions. The city, shown on the left, is composed of patches in a star formation. Individuals are resident in a given patch. The peripheral patches are coupled to the central patch by the residents' habitual movement. Peripheral patches are not directly coupled to one another. Resident population density or contact rate with the environment may vary between patches, as denoted by the circle size. Peripheral patches are indexed in ascending order of contact rate or density. In each patch, disease dynamics occur as shown on the right; treatment and decontamination are shown in the black circles.

Table 1
Parameter values used throughout the analysis unless stated otherwise. (∗) indicates a parameter set option. There are homogeneous and heterogeneous population size N_j and contact rate β_j parametrisations. In addition there are contact rate parametrisations such that the disease-free equilibrium is stable, or the endemic equilibrium is stable. All rates are per day. The values of $\eta, \beta, \kappa, \gamma, \eta, \theta$ are based on those used in previous studies (Chao et al., 2011; Codeço Claudia, 2001; Gatto Marino et al., 2012; Lorenzo Mari et al., 2012; Mukandavire et al., 2011; Renaud Piarroux, 2011).

Parameter	Meaning	Value
N	Total population	7×10^5
N_j	Homogeneous resident population, all patches (∗)	1.4×10^5
N_j	Heterogeneous resident population, patch $j = [c, 1, 2, 3, 4]$ (∗)	$[1.4, 0.98, 1.26, 1.54, 1.82] \times 10^5$
μ	Birth/death rate, average lifespan 70 years	4×10^{-5}
σ	Residential interaction weight, non-infected	0.67
χ_1	Inertia due to treatment facility	0.2
χ_2	Animation due to treatment facility	2
β_j	Homogeneous contact rate with environment, all patches, endemic (∗)	1
β_j	Homogeneous contact rate with environment, all patches, disease-free (∗)	0.1
β_j	Heterogeneous contact rate with environment, patch $j = [c, 1, 2, 3, 4]$, endemic (∗)	$[1, 0.7, 0.9, 1.1, 1.3]$
β_j	Heterogeneous contact with environment, patch $j = [c, 1, 2, 3, 4]$, disease-free (∗)	$[0.1, 0.07, 0.09, 0.11, 0.13]$
κ	Half-saturation constant for transmission	2.5×10^7
ξ	Treatment rate	0.5
γ	Recovery rate, average infection duration 5 days	0.2
α	Recovery rate enhancement due to treatment	3
η	Unperturbed bacterial shedding rate, scaled by reservoir volume	17.86
ζ	Natural bacterial degradation rate	0.25
θ	Bacterial decontamination rate	0.5

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