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Modelling and Bayesian analysis of the Abakaliki smallpox data

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ABSTRACT

The celebrated Abakaliki smallpox data have appeared numerous times in the epidemic modelling literature, but in almost all cases only a specific subset of the data is considered. The only previous analysis of the full data set relied on approximation methods to derive a likelihood and did not assess model adequacy. The data themselves continue to be of interest due to concerns about the possible re-emergence of smallpox as a bioterrorism weapon. We present the first full Bayesian statistical analysis using data-augmentation Markov chain Monte Carlo methods which avoid the need for likelihood approximations and which yield a wider range of results than previous analyses. We also carry out model assessment using simulation-based methods. Our findings suggest that the outbreak was largely driven by the interaction structure of the population, and that the introduction of control measures was not the sole reason for the end of the epidemic. We also obtain quantitative estimates of key quantities including reproduction numbers.

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1. Introduction

In 1967, an outbreak of smallpox occurred in the Nigerian town of Abakaliki. The vast majority of cases were members of the Faith Tabernacle Church (FTC), a religious organisation whose members refused vaccination. A World Health Organization (WHO) report (Thompson and Foege, 1968) describes the outbreak, with information on not only the time series of case detections but also their place of dwelling (compound), vaccination status, and FTC membership. The outbreak has inherent historical interest as it occurred during the WHO smallpox eradication programme initiated in 1959. Although smallpox was declared eradicated in 1980, it regained attention as a potential bioterrorism weapon in the early 2000s (see e.g. Gani and Leach, 2001; Meltzer et al., 2001; Halloran et al., 2002) and continues to be of interest due to concerns about its re-emergence or synthesis (see e.g. Henderson and Arita, 2014; Eto et al., 2015; World Health Organisation, 2015 and references therein). Public health planning for potential future smallpox outbreaks requires estimates of the parameters governing disease transmission, and thus being able to accurately obtain such quantities from available data is of considerable importance.

Within mathematical infectious disease modelling, the Abakaliki smallpox data set has been frequently cited, the first appearance being (Bailey and Thomas, 1971). The data are almost always used to illustrate new data analysis methodology, but in virtually all cases most aspects of the data are ignored apart from the population of 120 FTC individuals and the case detection times, while the models used are not particularly appropriate for smallpox (see for example Becker, 1976; Yip, 1989; O'Neill and Roberts, 1999; O'Neill and Becker, 2001; Huggins et al., 2004; Boys and Giles, 2007; Lau and Yip, 2008; Clancy and O'Neill, 2008; Kypraios, 2009; Shanmugan, 2011; Xiang and Neal, 2014; McKinley et al., 2014; Golightly et al., 2014; Oh, 2014; Xu et al., 2016 and references therein). In Ray and Marzouk (2008) a more realistic smallpox model is used and account taken of the compounds where individuals lived, but again all non-FTC individuals are ignored.

The main objective of this paper is to present a Bayesian analysis of the full data set. To our knowledge, the only previous analysis of the full Abakaliki data is that of Eichner and Dietz (2003), where the authors define a stochastic individual-based transmission model that considers not only the case detection times but also the other aspects of the data. Their model takes account of the population structure, the disease progression for smallpox, the vaccination status of individuals and the introduction of control measures during the outbreak. The model parameters are then estimated by constructing and maximising a likelihood function which is itself constructed using various approximations. Specifically, the true likelihood of the observed data given the model parameters is practically intractable, since it involves integrating over all possible unobserved events, such as the times at which individuals become infected. Eichner and Dietz tackle this problem by first using a back-calculation method to approximate the distribution

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of unobserved event times for a given individual, and then by making various assumptions about independence between individuals in order to construct an approximate likelihood function.

An alternative solution to the intractable likelihood problem is to use data-augmentation methods to produce an analytically tractable (and correct) likelihood, which can then be incorporated in a Bayesian estimation framework by using Markov chain Monte Carlo (MCMC) methods along the lines described in O'Neill and Roberts (1999) and Gibson and Renshaw (1998). We adopt this approach to carry out a full Bayesian analysis of the Abakaliki smallpox data, whilst also assessing how well the Eichner and Dietz approximation method works in this setting. Our approach provides results which can be directly compared with those of Eichner and Dietz, specifically estimates of model parameters, estimates of associated quantities of interest such as reproduction numbers, and the sensitivity of the results to the disease progression assumptions. In addition, we also estimate quantities derived via data-augmentation, such as who-infected-whom and the time of infection for each individual, carry out various forms of model assessment to see how well the model fits the data, and explore particular aspects of the model via simulation. None of these additional elements feature in the Eichner and Dietz analysis.

The paper is structured as follows. In Section 2 we describe the data, stochastic transmission model and method of inference. Section 3 contains results and details of sensitivity analysis and model-checking procedures. We finish with discussion in Section 4. The supplementary material contains details of some likelihood calculations and the MCMC algorithm.

2. Data, model and inference methods

The outbreak is described in detail in Thompson and Foege (1968) and Eichner and Dietz (2003). There were 32 cases in total, 30 of which were FTC members. All of the infected individuals lived in compounds, which were typically one-storey dwellings built around a central courtyard, and capable of housing several families. The FTC members frequently visited one another and were somewhat isolated from the rest of the community, which is one reason why most previous data analyses only consider FTC members. Although FTC members refused vaccination, many of them had been vaccinated prior to joining FTC as described below.

2.1. Abakaliki smallpox data

Table 1 contains details of the 32 cases of smallpox recorded during the outbreak, specifying the date of onset of rash, compound identifier, FTC membership status and vaccination status. Note that we set a timescale by setting day zero of the outbreak to be the first onset of rash date. The composition of the affected compounds is provided in Table 2, where the total numbers of vaccinated and non-vaccinated FTC and non-FTC members within each compound are listed. Note that on day 25, four FTC individuals from compound 1 (three vaccinated and one non-vaccinated) moved to compound 2. In addition, quarantine measures were put in place in Abakaliki, but not until part way through the outbreak. The exact time these measures were introduced was not recorded.

Table 1
Smallpox cases in Abakaliki, Nigeria during 1967, taken from Thompson and Foege (1968). Compounds listed are those before the move of cases 7 and 8, and 2 other uninfected individuals, on day 25 from compound 1 to compound 2.

Case number	Day of onset of rash	Compound	Confession	Vaccination
0	0	1	FTC	No
1	13	1	FTC	No
2	20	1	FTC	No
3	22	1	FTC	No
4	25	1	FTC	No
5	25	1	FTC	No
6	25	1	FTC	No
7	26	2	FTC	Yes
8	30	2	FTC	Yes
9	35	1	FTC	No
10	38	4	FTC	No
11	40	5	FTC	No
12	40	1	FTC	No
13	42	1	FTC	No
14	42	1	FTC	No
15	47	1	FTC	No
16	50	5	FTC	No
17	51	2	FTC	No
18	55	1	FTC	No
19	55	2	FTC	No
20	56	6	Non	Yes
21	56	5	FTC	Yes
22	57	2	FTC	Yes
23	58	7	FTC	No
24	60	4	FTC	No
25	60	2	FTC	No
26	61	2	FTC	No
27	63	8	Non	Yes
28	66	3	FTC	No
29	66	9	FTC	No
30	71	5	FTC	No
31	76	2	FTC	Yes

2.2. Stochastic transmission model

We suppose that the residents of Abakaliki form a closed population with $N = 31{,}200$ individuals, labelled $0, 1, \ldots, N-1$. Individuals $0, 1, \ldots, n_{com} - 1$ are those inside the compounds, where $n_{com} = 251$ is the number of people within the compounds. Any individual $k = 0, \ldots, N-1$ may be categorised as type (c_k, f_k), where (i) $c_k = 1, \ldots, 9$ is the compound of k, with $c_k = 0$ if k is outside the compounds, and (ii) f_k is k's confession; FTC or non-FTC. These types may lead to differences in the mixing behaviour of individuals, but otherwise individuals are considered to be identical in their susceptibility to smallpox and their ability to infect others.

We now describe a stochastic disease-transmission model for the spread of smallpox throughout the population of Abakaliki. This model is essentially the same as that described in Eichner and Dietz (2003), and is a variant of a Susceptible-Exposed-Infective-Removed (SEIR) model. At any given time t each individual in the population will be in any one of six states, namely susceptible, exposed, with fever, with rash, quarantined or removed. For $j = 0, \ldots, N-1$, let e_j, i_j, r_j, q_j, τ_j denote, respectively, the times of exposure, fever, rash, quarantine and recovery for individual j. Any susceptible individual may become exposed, as described below, at which point they enter an incubation (or latent) period. They next enter the fever stage of the disease, at which point they become infectious and may hence infect others. During the rash stage which follows, the individual remains infectious but with a potentially different level of infectivity. We define the infectious period to be the combined time spent in the fever and rash stages. Infectious individuals will either become removed (namely recovery or death; we do not distinguish these) or isolated, in which the individual is quarantined and henceforth unable to infect others. Control measures, in which cases are placed into isolation soon after detection, are introduced part way through the outbreak at time t_q. We do not allow re-infection, so that individuals who have been infected cannot become susceptible again. The epidemic continues until there are no infectious or exposed individuals remaining in the population, at which point each person will either still be susceptible, or will have been quarantined/removed.

The lengths of time spent in each stage of the disease for different individuals are assumed to be mutually independent random variables with specified distributions, the parameter values of which are assumed known. We adopt the assumptions of the

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