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# Estimation of the dynamics and rate of transmission of classical swine fever (hog cholera) in wild pigs

J. HONE[1], R. PECH[2] AND P. YIP[3,4]

[1] *Applied Ecology Research Group, University of Canberra, PO Box 1, Belconnen 2616 Australia*

[2] *CSIRO Division of Wildlife and Ecology, PO Box 84, Lyneham 2602 Australia*

[3] *Department of Statistics, La Trobe University, Bundoora 3083 Australia*

[4] *Department of Statistics, University of Hong Kong, Pokfulam Road, Hong Kong*

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## SUMMARY

Infectious diseases establish in a population of wildlife hosts when the number of secondary infections is greater than or equal to one. To estimate whether establishment will occur requires extensive experience or a mathematical model of disease dynamics and estimates of the parameters of the disease model. The latter approach is explored here. Methods for estimating key model parameters, the transmission coefficient ($\beta$) and the basic reproductive rate ($R_D R_S$), are described using classical swine fever (hog cholera) in wild pigs as an example. The tentative results indicate that an acute infection of classical swine fever will establish in a small population of wild pigs. Data required for estimation of disease transmission rates are reviewed and sources of bias and alternative methods discussed. A comprehensive evaluation of the biases and efficiencies of the methods is needed.

## INTRODUCTION

Modelling of the dynamics of directly-transmitted infectious diseases in animal hosts predicts a threshold abundance ($N_T$) of susceptible hosts above which the disease establishes and below which the disease disappears [1, 2]. Above $N_T$ an infected host, if introduced into a population of susceptible hosts, will transfer the infection to at least one susceptible host. The number of secondary infections is the basic reproductive rate ($R_D R_S$) [2] or initial relative infection rate [3]. If the disease is the subject of control or eradication efforts then an objective is to reduce host abundance to below the threshold. Alternatively if the disease is a biological control agent then host abundance must be above the threshold for the disease to establish and potentially regulate pest abundance.

This paper briefly reviews the modelling of disease dynamics in wildlife and the estimation of key model parameters. Classical swine fever (hog cholera) in wild pigs is used as an example. The relevance of the approach to eradication of classical swine fever (CSF) or other diseases and use of CSF for biological control of wild pigs are described.

Plans have been developed for eradication of exotic diseases of livestock, including those in wildlife should the diseases enter a country such as Australia

[4–6]. These diseases include directly-transmitted diseases such as foot-and-mouth disease, rabies and CSF. For example, if wild pigs (*Sus scrofa*) in Australia were infected with CSF then efforts would be made to eradicate the disease from their populations. An outbreak of CSF may be eradicated by culling of wild pigs. Other strategic options include vaccination, as investigated in the USSR [7]. In some cases control of wild pigs may not be needed because CSF may not become endemic in them. The relative value of these options has not been evaluated.

Classical swine fever is a virus disease of pigs [8, 9] spread primarily by close proximity between hosts. The disease is widespread in Europe, Asia and central and South America, though it does not currently occur in Australia. It was reported in Australia in 1903, 1927–8, 1942–3 and 1960–1 [10]. The disease was first reported in wild pigs in an enclosed park in Munich in 1906 [11]. Since then, CSF has been reported in wild boar or feral pigs in the USA [12–14], Australia [15], Germany and Austria [12], Pakistan [16], the USSR [7, 17], Yugoslavia [18] and Italy [19]. CSF may disappear naturally from wild pigs, as was reported in California [14], Pakistan [16], in western Europe [20] and apparently in Sardinia [19]. In contrast, it was reported that wild pigs could represent a reservoir of infection for CSF [21].

## MODELLING OF DISEASE DYNAMICS

There are two steps in the modelling of disease dynamics; firstly derive a model to show what determines $R_D$ or $R_S$ after entry of an infected host into a susceptible population, and secondly, obtain estimates of the parameters in the model. These will be described sequentially.

Models of disease establishment are of two basic types; deterministic and stochastic. The historical development of the models and their predictions have been described [1]. Of particular relevance here, is the threshold theorem which exists for both sorts of models. The theorem states that an infected host introduced into a population of susceptible hosts would not give rise to an epidemic outbreak unless the abundance of susceptibles exceeded a critical number – the threshold level ($N_T$). The derivation of $N_T$, and its application to disease control, has been subsequently described in many articles [22].

### *Deterministic modelling*

The number of secondary infections estimated by a deterministic model ($R_D$) is the ratio of the abundance of susceptibles ($X$) to $N_T$ [23, 24]:

$$R_D = \frac{X}{N_T}, \tag{1}$$

The equation for $N_T$ for a population with no births and natural deaths can be derived, for a directly-transmitted infectious disease as:

$$N_T = \frac{\alpha + v}{\beta}, \tag{2}$$

where $\beta$ is the transmission coefficient and $v$ is the recovery rate, the length of the infectious period is $1/v$, and the death rate from infection is $\alpha$.

$R_D$ for the disease is then:

$$R_D = \beta X/(\alpha + v). \tag{3}$$

By definition, a disease establishes when the $R_D$ is greater than or equal to one. A prediction of establishment of disease is only valid for the initial population and is not a prediction of persistence. For example, if survivors of infection develop permanent immunity the disease may disappear as the number of susceptibles declines.

*Stochastic modelling*

The stochastic model is based on describing the probability of a susceptible host becoming infected and then dying. Using this model the basic reproductive rate ($R_S$) of an infection similar to that described above is:

$$R_S = X\frac{\beta}{\gamma}, \tag{4}$$

where $X$ describes the initial number of susceptibles, and $\gamma$ is the rate of loss of infectives [3]. The rate of loss of infectives is the sum of the recovery rate ($v$) and the disease-induced mortality rate ($\alpha$).

## ESTIMATION OF PARAMETERS

An example of a suitable set of data for estimation of $\beta$ and other model parameters has been reported [16]. CSF was introduced into a population of wild boar in a forest plantation of 44·6 km$^2$ in Pakistan. The initial host population was estimated by drive and count census to be 465 and it was assumed that they were susceptible (i.e. $X = 465$). One wild boar was injected with live virus and released. Hence it was assumed that the initial number of infectives was one. Portions of a carcass of a previously inoculated wild boar were also placed in the forest. As the experiment occurred in July (summer in Pakistan) we assumed here that the carcass quickly decayed and the virus was inactivated as it is susceptible to putrification [10]. Hence the carcass was not a source of infection.

The number of wild boar found dead ultimately totalled 77 and was tabulated for days after introduction (Table 1). The number of wild boar remaining in the area 5·5 months later was estimated to be 87. The fate of the other 301 wild boar is not known. As the second census was over 5 months after the first, many wild boar could have died from causes other than CSF or moved out of the area.

The data for the first 69 days (Table 1) were used to estimate parameters in the deterministic model. For the stochastic model, calculation of $\beta$ and $R_S$ requires an estimate of the total mortality due to CSF at the end of the epizootic. There are two possible estimates of the total mortality. Firstly, if CSF did not persist after the 69th day, a total of 77 deaths can be attributed to the disease. Alternatively, assuming CSF died out after 69 days and before 5·5 months, the maximum number of possible deaths was 379 ($= 465 + 1 - 87$). In all cases, the models did not include the possibility of births or natural deaths in the wild boar population during the CSF epizootic.

The epidemiology of CSF in feral pigs has not been well described so data for domestic pigs were used where necessary. Domestic and wild pigs are equally

Table 1. *The cumulative number of deaths in a population of wild boar in Pakistan after introduction of classical swine fever. Details of the population and release of virus are described in the text and original reference* [16]

| Days since disease introduction | Cumulative number of deaths |
|---|---|
| 31 | 6 |
| 32 | 9 |
| 33 | 10 |
| 43 | 15 |
| 44 | 21 |
| 45 | 23 |
| 46 | 25 |
| 47 | 32 |
| 48 | 39 |
| 49 | 40 |
| 51 | 53 |
| 52 | 55 |
| 53 | 59 |
| 54 | 61 |
| 58 | 66 |
| 61 | 69 |
| 62 | 71 |
| 63 | 73 |
| 69 | 77 |

susceptible to CSF [12] and CSF in wild and domestic pigs does not differ in appearance [11]. CSF can develop as an acute, sub-acute, or chronic disease [9]. CSF in wild animals is always an acute disease [18] so that was assumed here. It was also assumed that vertical transmission of the pathogen did not occur and that there were no carriers of the infection.

*Incubation period*

The mean incubation period was assumed to be 4·75 days based on recent estimates of 2–8 days [9, 10, 25, 26]. An older estimate of 3–20 days [12] was inconsistent with that reported elsewhere so was excluded. The rate of change from incubating to infectious ($\sigma$) was 0·21/day.

*Recovery rate*

Pigs infected with a virulent virus, as in an acute infection, shed virus for 10–20 days [8]. A mean of 15 days was assumed so the rate of recovery ($v$) was 0·067/day.

*Mortality rate*

The mortality rate ($\alpha$) from an acute infection may reach 90% or even approach 100% [9, 10, 26, 27]. A mean mortality rate of 95% was assumed. This is a case mortality over an average of 15 days so $\alpha$ was 0·200/day.

*Transmission coefficient*

CSF is spread mostly by direct contact between infected and susceptible pigs [10]. Spread by fomites may also occur but is not considered here. The $\beta$ is difficult to measure during an epizootic in wildlife populations but can be estimated

Table 2. *Methods used to estimate the transmission coefficient in compartment models of the dynamics of directly-transmitted infectious pathogens and parasites. Methods 1–8 are for deterministic models and 9–11 for stochastic models. Method 4 estimates the transmission rate rather than the transmission coefficient though the two are very closely related*

| No. | Method | Ref. |
|---|---|---|
| 1 | Estimate by regression of prevalence and time since start of epidemic when population size constant and no incubation period | [2, 36] |
| 2 | Estimate from equation for basic reproductive rate when threshold density known | [23, 28] |
| 3 | Estimate from equilibrium prevalence | [37] |
| 4 | Estimate from age prevalence curves | [36] |
| 5 | Infer from behaviour data | [29] |
| 6 | Infer from data on 'contacts' between hosts | [38] |
| 7 | Iterative comparison of field prevalence data with model predictions | [39] |
| 8 | Iterative estimation by regression of trends in deaths over time since disease introduction | This study |
| 9 | Estimate from known initial population size and total deaths in an epidemic | [3] |
| 10 | Estimate from known initial population size and trends in prevalence of disease | [1, 40] |
| 11 | Estimates of susceptibles, infectives and host abundance each day | [41] |

indirectly, for example from $N_T$ using equation (2). This approach was used for rabies in foxes [23] and in raccoons [28]. Other methods are listed in Table 2. In this study two methods were used: one based on fitting a deterministic model to the observed trend in deaths and treating $\beta$ as an adjustable parameter, and a second where $\beta$ was calculated using equation (4) and estimates of $R_S$ obtained from a stochastic model.

The $\beta$ was estimated for the deterministic model by iterative least squares of the trend in cumulative mortality of wild pigs over time since introduction of the disease. The estimate used was that which minimized the mean square deviations between observed and expected deaths. The deterministic model was that derived for foot-and-mouth disease in feral pigs [29] modified by the assumptions that births and natural deaths were zero during the short term of the observations on CSF in Pakistan, and that immune pigs did not lose immunity to CSF. Pigs that survive infection have an immune response with peak antibody levels 3–4 weeks after infection [30]. Immunity is lifelong [27].

Two related Martingale-based techniques have been derived to obtain estimates and associated standard errors for $R_S$ and $\beta$ for a stochastic epidemic [3]. The first technique requires complete observation of the epidemic. It is essentially an estimate of maximum likelihood which requires daily information on the number of infectives and susceptibles and so was not used here. The second technique uses only the total number of individuals who were infected during the epidemic and the initial number of susceptibles in the population. It was shown that the

asymptomatic efficiency of the second method is at least 90% that of the first method for most cases [3]. The second method is very useful, especially in the case of incomplete data, and will be used here to obtain estimates of the $\beta$ and $R_S$.

An estimate of $R_S$ can be obtained [3], with the integrals replaced by summations:

$$R_S = X\left[\frac{1}{X}+\frac{1}{X-1}+\cdots+\frac{1}{X-Z+i+1}\right]\Big/Z, \tag{5}$$

where $Z$ is the number of removals at the end of the epidemic and $i$ is the initial number of infectives ($i = 1$). The standard error (S.E.) of $R_S$ is:

$$\text{S.E.}(R_S) = \left[X^2\left(\frac{1}{X^2}+\frac{1}{(X-1)^2}+\ldots+\frac{1}{(X-Z+i+1)^2}+R^2Z\right)\right]^{0\cdot5}\Big/Z. \tag{6}$$

When an estimate of $\gamma$ is available, then $\beta$ can be estimated by rearranging equation (4) to give:

$$\beta = \frac{R_S\gamma}{X}, \tag{7}$$

and the S.E. is given by:

$$\text{S.E.}(\beta) = \left(\frac{\gamma}{X}\right)\text{S.E.}(R_S). \tag{8}$$

*Vaccination*

Modelling can estimate the proportion of the population that needs to be vaccinated, shortly after birth, to ensure that the disease does not become endemic [22]. For example, the level of vaccination needed to control rabies has been estimated [23, 28, 31].

The aim of a vaccination programme is to make $R_S$ or $R_D$ less than one. If a proportion ($p$) of the population ($N$) is vaccinated then $R_S$ is reduced to $R_S(1-p)$. Since the reduced $R_S$ must be less than 1 then, $R_S(1-p) < 1$. By rearrangement, the proportion of the population to be vaccinated must be at least:

$$\left.\begin{aligned} p &= 1-(1/R_S) \\ &= 1-(N_T/N). \end{aligned}\right\} \tag{9}$$

## RESULTS

*Deterministic modelling*

The least squares estimate of $\beta$ was 0·000 99/day. The estimated $N_T$ for an acute infection of CSF is $N_T = 270$ pigs (equation (2)), or 6·1 pigs km$^{-2}$, and the estimate of $R_D$ is 1·7 ($= (465 \times 0{\cdot}000\,99)/(0{\cdot}200+0{\cdot}067)$). The initial abundance of the wild boar population in the forest in Pakistan was 465 pigs (10·4 km$^{-2}$) which is higher than the estimated threshold.

Using the values and the parameter estimates for $\sigma$, $v$, and $\alpha$ as above, numerical simulation showed that the epizootic could have lasted about 150 days with a total of 246 deaths.

*Stochastic modelling*

A lower estimate ($R_{S,L}$) of the initial relative infection rate is obtained by assuming that 77 pigs died ($Z = 77$). Using equation (5), $R_{S,L} = 1{\cdot}1$ ($\pm 0{\cdot}2$ S.E.). An upper estimate ($R_{S,U} = 2{\cdot}1 \pm 0{\cdot}2$ S.E.) is obtained by assuming that 379 pigs died ($Z = 379$).

Assuming $\gamma$ is 0·267 ($= 0{\cdot}200 + 0{\cdot}067$) then:

$$\beta_L = 0{\cdot}000\,63\,(\pm 0{\cdot}0001 \text{ S.E.})/\text{day},$$

from which $N_T$ is estimated to be 424 pigs, and:

$$\beta_U = 0{\cdot}001\,21\,(\pm 0{\cdot}000\,091 \text{ S.E.})/\text{day},$$

from which $N_T$ is estimated to be 221 pigs.

*Vaccination*

The estimated proportion of the pig population that needs to be vaccinated to control an acute CSF outbreak is estimated from the deterministic model as $p = 1-(1/R_D) = 0{\cdot}41$, or 41%. The estimates from the stochastic model are $p = 0{\cdot}09$ (9%) if $R_{S,L} = 1{\cdot}1$ and $p = 0{\cdot}52$ (52%) if $R_{S,U} = 2{\cdot}1$.

## DISCUSSION

This study indicates that six pieces of data need to be recorded during an epizootic of CSF in wildlife for later use in predictive modelling or for retrospective interpretation of epizootics. The data are the initial abundance of hosts, the number of infectives initially involved, the number of deaths during the epizootic, the incubation period, the recovery and disease-induced mortality rates.

The estimate for CSF, from the deterministic model in the present study, of the number of secondary infections ($R_D = 1{\cdot}7$) is intermediate between the estimates ($R_S = 1{\cdot}1$–$2{\cdot}1$) from the stochastic model. That is encouraging. The estimates of $R_S$ or $R_D$ are lower than those reported (4·3–18·0) for a variety of directly-transmitted human diseases such as measles, whooping cough and mumps [32]. Using the deterministic model, it is possible to extrapolate beyond the confirmed deaths in the first 10 weeks, to predict the likely extent of the CSF epizootic. The estimate of a total of 246 deaths is between the mortality estimates used to calculate $R_{S,L}$ (77 deaths) and $R_{S,U}$ (379 deaths). The conclusions in the present study must be preliminary, because the original data were incomplete. The precision of each estimate should be higher with more complete data. The value of the analyses reported here is that they show what data are needed, how such data can be analysed and how it can be used for management of disease in wildlife populations and management of possible vertebrate pests. Further modelling of the dynamics of CSF could examine the effects of vertical transmission and carrier pigs. This may be important if the virus is of low virulence.

The $\beta$ had the greatest effect on predictions of dynamics of foot-and-mouth disease in feral pigs [29] and that appears to occur here also. The $\beta$ is difficult to estimate and various methods used are listed in Table 2. Most require knowledge of population density or abundance at the start of the epidemic, and basic data on

disease mortality rate, incubation period and recovery rate after infection. Method 1 requires a constant size of host population and so was not used in this study. The relative biases and efficiencies of all the methods have not been comprehensively investigated. That is an area for future research.

The effects on parameter estimation of incomplete data can be examined. The estimate of initial host abundance ($X = 465$) is important. Estimating abundance of wildlife, especially of wild pigs, is difficult [33] and probably tends to yield underestimates. In the modelling here, such underestimates may lead to biased estimates of $R_S$. The values of $\alpha$ and $v$ have minor effects on estimates of the $R_D$ or $R_S$ because the rates are additive in equation (3). The stochastic modelling generated two estimates of $R_S$. It is considered that the estimate ($R_{S,L}$) using the value of 77 deaths is more accurate because the cumulative number of deaths appeared to be diminishing as the 69th day approached (Table 1). The estimate ($R_{S,U}$) may be biased by the assumptions of no births and natural deaths.

The estimates of $R_S$ may be biased by another source of missing data. Wild boar that survive infection with CSF become immune [27] but the number of immune boar was not tabulated in the original study [16]. As a result, the number of removals in the stochastic model will be underestimated. Hence the estimate of $R_S$ will be negatively biased and the estimate of the transmission coefficient will also be negatively biased.

Classical swine fever may not always be useful as a biological control agent. The disease may disappear in populations of less than about 430 wild pigs. The short duration of a CSF epizootic may be of strategic value if a short-term reduction in abundance of wild pigs is required. Alternatively, a knowledge that CSF will disappear is an advantage in attempting to dispel fears that the disease may spread to unvaccinated domestic pigs. The short duration of an epizootic may be a disadvantage as it produces extra costs for repeated introductions of a biological control agent. A threshold rate of pathogen introductions may occur for successful biological control [34]. The estimation of such a rate has not been attempted in this study.

The optimal characteristics of a pathogen for biological control were reported to be intermediate pathogenicity, high $\beta$ and ability to produce long-lived infective transmission stages [34]. In contrast, it was reported that biological control of persistent pests, which would include wild pigs, should use a parasite or pathogen that is persistent in a reservoir, carrier or the environment [35]. CSF does not appear to meet these requirements.

The disappearance, after introduction, of CSF in the Pakistan pig population is similar to independent reports of CSF in wild pigs in California [14], western Europe [20] and Sardinia [19]. The pig populations in each case may have been at a level before each outbreak lower than $N_T$ or alternatively the survivors were all immune and so insufficient susceptible pigs remained. Two studies [12, 14] concluded that wild pigs would not act as a reservoir of infection of CSF. The modelling results support those conclusions. One report [21] that wild pigs could act as a reservoir for CSF needs to be confirmed in the light of this analysis.

The estimates of the percentage of the wild boar population to be vaccinated ranged from 9 to 52%. Such a wide range is not particularly useful for control planning. Either less variable estimates are needed or the upper estimate (52%)

could be used as a conservative approach. The results suggest that use of vaccines for CSF control in wild pigs in the USSR [7] may not be necessary unless wild boar populations are very high. In particular it would be of interest to know if the average age at first infection with CSF was greater than the average age of vaccination of wild boar or less. In the latter case vaccination would not be as effective because animals previously exposed to the pathogen would be given vaccine. Ideally vaccine should be given only to susceptible animals.

The estimates of $N_T$, $R_D$ and $R_S$ for CSF suggest that only in good quality habitats such as wetlands and river systems, would CSF establish in Australia, as the carrying capacity in those habitats is thought to be well above the threshold. If true, this suggests that in such habitats eradication of CSF would require active culling of the pig population or vaccination. This conclusion assumes that the behaviour of pigs is similar in each country.

The modelling of disease dynamics and estimation of parameters of such models will be further advanced by closely linking data from epizootics with the models. There is a need for further study of parameter estimation and the application of unbiased estimates to understanding and control of infectious diseases in wildlife populations.

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